

MODULE

Food-borne Diseases

Degree Program

For Health Officers, Nurses, Environmental health Officers and Medical Laboratory Technologists



**Ethiopia Public Health
Training Initiative**

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UNIT ONE

INTRODUCTION

1.1. Purpose and Use of the Module

A big challenge in the training of well-versed health professionals in the different higher institutions in Ethiopia has emanated from the serious shortage of adequate number of contextual reference materials. To add to this problem, even the available reference materials sometimes fail to address the most important learning issues of the Ethiopian students. However, up to this day, efforts geared towards the preparation of reference materials by instructors in the different institutions in order to reduce this problem have remained meager.

This brings into focus the purpose of the preparation of this module, which is just one among many having been or being undertaken through the initiation made by the Carter Center, EPHTI. This module is prepared to help students develop knowledge, attitudes and skills required in their practice areas through active learning. To this end, it will enable the different categories of health professionals i.e., Health Officer (H.O), Nurse (N), Environmental Health Officer (E.H.O) and Medical Laboratory Technologist (M.L.Technologist) to be able to recognize and manage the important food-borne diseases as well as to prevent them from occurring from the outset.

Besides, it is believed that those already engaged in the service delivery working in different health facilities will benefit as well from reading this module. All individuals taking time to look at this document are reminded of the importance of consulting standard textbooks on the subject whenever possible, since this module is by no means meant to replace them.

1.2. Directions for Using the Module

Before starting to read this module, please follow the directions given below:

1. Go through all the contents of the core module by starting with the pretest.
2. Use a separate sheet of paper to write your answers and label it “pretest answers”.
3. The pretest has two portions: Part I, and Part II.

PART I: Contains common questions to be answered by all categories.

PART II: The questions are prepared for the specific categories; Health Officers, Nurses, Environmental Health Officers, and Medical Laboratory Technologists. Select and do the portion that corresponds to your professional category.

- When you are sure that you are through the core module proceed to read the satellite module corresponding to your profession or interest.
- Go through the task analysis for the team members in comparison with that of your own.
- Read the module for health extension workers under unit 3.5. in order to get an insight what roles they play in public health.

Note: You may refer to the list of abbreviations and glossary shown in Unit Five for terms that are not clear.

UNIT TWO

CORE MODULE

2.1 Pre-test

Write the answers of the following questions on a separate answer sheet and label it pre-test.

PART I: Questions Common for All Categories

Answer the following questions as appropriate.

1. Define food-borne diseases.
2. Describe the two major classifications of food-borne diseases and give one example for each.
3. Mention some factors contributory to the widespread occurrence of food-borne diseases in Ethiopia.
4. List the areas of emphasis in the general diagnostic approach of food-borne diseases.
5. What are the three most important basic principles in the prevention and control of food-borne diseases?
6. Which of the following food-borne diseases is different from the others?
 - a. Typhoid fever
 - b. Shigellosis
 - c. Cholera
 - d. Amebiasis
7. Write the most important clinical features for each of the following food-borne diseases,
 - a. Typhoid fever
 - b. Shigellosis
 - c. Cholera
 - d. Taeniasis
 - e. Amebiasis

8. The most important complication of cholera and its management is:
 - a. Dehydration and electrolyte disturbances-fluid and electrolyte replacement
 - b. Fever-antibiotic and antipyretic until fever subsides
 - c. Intestinal perforation-surgical repair of perforated intestinal loops
 - d. Blood loss-blood transfusion
9. Discuss the implications of each of the following practices in relation to food-borne diseases:
 - a. Inadequate cooking of food
 - b. Use of unclean water in food preparation and cleaning of dishware
 - c. Mixing raw with cooked ready-to-eat food
 - d. Personal hygiene of food handlers
10. Outline the steps in food-borne disease outbreak investigation.

PART II: Questions for Specific Categories

A. For Health Officers

Write the letter that indicates your best choice.

1. The most important symptom of typhoid fever is:
 - A. Severe headache
 - B. Abdominal pain
 - C. Diarrhea
 - D. Prolonged fever
 - E. Rash
2. Bloody diarrhea could be a manifestation in all of the following except:
 - A. Shigellosis
 - B. *E. coli* infection
 - C. *E. histolytica* infection
 - D. Inflammatory bowel disease
 - E. None of the above

3. Identify the true statement about cholera:
- A. Any patient suspected to have cholera should be immediately referred to a higher center for better care.
 - B. In the management of patients with cholera, fluid replacement is less important than antimicrobial therapy.
 - C. All patients with cholera should be managed with intravenous fluids.
 - D. One role of the use of antimicrobials is for prophylactic purpose in family members (contacts).
 - E. All are true statements
4. Which of the following is false about Widal test?
- A. It is most useful during the second and third weeks of infection.
 - B. It is the gold-standard for diagnosis of typhoid fever.
 - C. The presence of cross-reactive antibodies limits its use.
 - D. Serum and urine specimens give almost the same result.
 - E. B and D
5. Identify the food-borne disease not acquired through ingestion of raw or undercooked meat:
- A. Brucellosis
 - B. Anthrax
 - C. Taeniasis
 - D. Cysticercosis
 - E. None of the above
6. Which of the following diseases results from food poisoning?
- A. Ascariasis
 - B. Amebiasis
 - C. Brucellosis
 - D. Botulism
 - E. None of the above

B. For Nurses

Write the letter that indicates your best choice.

1. Which one of the following can be taken as an objective data when assessing a patient with food borne diseases?
 - A. Patient complaining of crampy abdominal pain
 - B. History of ingestion of contaminated foods
 - C. Very poor skin turgor
 - D. Patient telling you that the diseases has started about 24 hours back

2. During the nursing care for a patient with diarrhea secondary to food borne diseases, caffeine and carbonated beverage is limited because:
 - A. These is a potential source of food borne diseases
 - B. These extremely decrease the peristaltic action of the gastro intestinal tract.
 - C. These stimulate intestinal motility
 - D. All are answers

3. One of the following nursing interventions is not carried out for a patient with poisoning related to the ingestion of contaminated food with chemical poisons and poisonous plants.
 - A. Attaining control of the air way, ventilation, and oxygenation
 - B. Treatment of shock
 - C. Administering the specific chemical antagonist or physiologic antagonist
 - D. All are answers

4. Induction of vomiting is not recommended after ingestion of caustic substances or petroleum distillates
 - A. True
 - B. False

5. Identify an incorrect statement about the nutritional management of the patient with food borne diseases that has diarrhea.
- A. Give sips of weak tea, carbonated drinks or tap water for mild nausea.
 - B. Advise the patients to increase food products with a cellulose or hemi cellulose base.
 - C. Vary the diet to make eating more enjoyable, and allow some choice of foods.
 - D. Give clear liquids 12 to 14 hours after nausea and vomiting subsides.
6. Which one of the following nursing interventions is used to reduce anxiety of a patient with diarrhea secondary to food borne diseases?
- A. Providing an opportunity to express fears and worry about being embarrassed by lack of control over bowel elimination.
 - B. Assist to identify irritating foods and stressors that precipitate an episode of diarrhea.
 - C. Encourage to be sensitive to body clues that warn of impending urgency.
 - D. All are answers
7. Identify a measure that is helpful to prevent the spread of food borne infections to others.
- A. Hand washing
 - B. Blood and body fluid precautions whenever handling vomitus or stools
 - C. Provision of isolation according to the general rule of body substance isolation, or individual institution adaptation of isolation.
 - D. All are answers
8. One of the following drugs is not used in the treatment of Taeniasis (T-solium)
- A. Niclosamide
 - B. Mebendazole
 - C. Thiabendazole
 - D. Praziquantel

C. For Environmental Health Officers

Read the following questions carefully and give the appropriate answer.

1. One of the following is **NOT** the basic principle of food sanitation
 - A. Prevent contamination of food from microorganisms, toxins or chemicals.
 - B. Eliminate /destroy micro-organisms or their toxins
 - C. Prevent the growth of microorganism.
 - D. None of the above
2. Hazards at the stage of production of raw materials include the following except
 - A. Nutrinents
 - B. Environmental contaminants
 - C. Natural toxins
 - D. Microbial toxins
 - E. None of the above
3. The factors that are necessary for the transmission of a food borne disease are
 - A. Transmission of the causative agent from the environment to the food itself.
 - B. Growth support if the agent is biological.
 - C. Transmission of the agent from the source to a food.
 - D. A source and reservoir of transmission for the agent.
 - E. All of the above.
4. Which of the following factors leads to the occurrence of food borne disease outbreaks
 - A. Inadequate reheating
 - B. Insects and rodents
 - C. Storage at ambient temperature
 - D. Undercooking
 - E. All of the above
5. Elements of hazard analysis and critical control point (HACCP) include
 - A. Monitor critical control points
 - B. Identify critical control points
 - C. Determine hazards
 - D. B and C

- E. All of the above
6. One of the following statements is NOT true about blanching operations
- A. It is a mild pre-cooking process
 - B. It is a bactericidal process
 - C. It is used for vegetables
 - D. It used to reduce the bacterial load
 - E. None of the above
7. Which of the following techniques is (are) used to keep food safe
- A. Fermentation
 - B. Pickling
 - C. Irradiation
 - D. Chemical treatment
 - E. All of the above
8. List the different types of food samples collected for food-borne disease assessment.

D. For Medical Laboratory Technologists

Write the letter of your choice for the following questions on separate answer sheet .

1. Which of the following parasites is diagnosed by finding cyst and trophozoit stage?
 - A. E. histolytica
 - B. A. lumbricoid
 - C. Taenia saginata
 - D. All
2. Direct stool examination is used for diagnosis of
 - A. E. histolytica
 - B. G. lamblia
 - C. A. lumbricoid
 - E. All
3. Widal test is a serological test used for diagnosis of
 - A. Shigellosis
 - B. Typhoid fever
 - C. Cholera
 - D. Brucellosis
4. Stool culture is used for the identification of the following bacteria.
 - A. Shigella
 - B. Salmonella
 - C. V. cholera
 - D. All
5. Among the following tests typhoid fever is best diagnosed by:
 - A. Widal test
 - B. Culture
 - C. Direct microscopic examination
 - D. Gram stain
6. Duodenal aspirate can be used for diagnosis of
 - A. Amoebiasis
 - B. Ascariasis

- C. Taeniasis
 - D. Giardiasis
7. Which one of the following is spore forming bacterium?
- A. Salmonella
 - B. Shigella
 - C. Clostridium perfringens
 - D. Vibrio cholera



2.2 Significance and Brief Description of Food borne Diseases

As far back as the documentation of human history goes, consumption of food unsafe for health and its consequences have been one of man's major health problems. They still remain to be a major public health concern globally. Food-borne diseases are known to be responsible for a large proportion of adult illnesses and deaths; more importantly, as sources of acute diarrheal diseases, they are known to claim the lives of overwhelming numbers of children every day.

In developing countries like Ethiopia, the problem attains great proportions due to many reasons; basic among which are poverty and lack of public health awareness. Although well-documented information is lacking regarding the extent of food-borne diseases in the country, and many cases and outbreaks are unrecognized or unreported, they are unquestionably one of the major reasons or why people of all ages seek medical help. Most food-borne diseases manifest with gastrointestinal symptoms and signs, the latter being uniformly among the top diagnoses in health facilities at all levels. Besides, they commonly lead to epidemics that result in the losses of many lives, accompanied with severe economic repercussions.

In these modern days, in which food is usually not consumed immediately following and/or at the site of production, the risks of food-borne diseases are becoming increasingly important; the concern is obviously much more in areas where food storage and preparation safety measures are far below the optimum.

The role of well-trained health professionals not only in the prevention and control of food-borne diseases, but also in the recognition of individual cases as well as outbreaks and their timely and proper management in order to reduce mortalities and morbidities is very crucial.

2.3. Learning Objectives

▪ General

Upon completion of this module, the learner will be able to recognize, prevent and manage food-borne diseases.

▪ Specific

After completing this module the learner will be able to:

1. Describe the epidemiology of food-borne diseases.
2. Define and classify food borne diseases
3. Identify the causes of most common food-borne diseases in Ethiopia.
4. Describe the clinical features and complications of food born diseases.
5. Explain the general diagnostic and management approaches to some food born diseases.
6. Investigate and control outbreaks of food-borne diseases.
7. Develop preventive and control strategies for common food-borne diseases.

2.4. Case Study

Learning Activity 1

It was during the period of drought and famine that people were getting displaced to other parts of the country. Before the resettlement, they used to wait in groups in the nearby small town for few days or weeks. Among them, Fatuma, a 25 years old lady came to the nearby health center with one day history of nausea, vomiting and watery diarrhea. She was one of the cooks for the group. On examination, she looked weak with feeble pulse, tachycardia and her BP was 90/60 mmHg. Tongue and mucosa were dry.

After appropriate laboratory examination she was given appropriate management and advice. The next day morning 25 similar cases come to the Health Center from the group. They were also given appropriate management, and advice. Staffs from the Health Center supervised their temporary residence and come up with the following report: There were about 50 individuals living in four rooms within one compound. The houses were under construction with multiple openings and dusty floor. There was no toilet in the compound and it was observed that there were indiscriminate human excreta in the compound. The people were sitting in group. Cooking and eating utensils

were not clean and there was no appropriate storage for the food. Pipe water supply was available in the compound; but the people fetched the water using wide mouthed buckets for storage. Finally the staffs conducted appropriate intervention measures and no similar cases were seen subsequently.

Exercise:

Answer the following questions related to the case study

1. What is your suspicion about the case?
2. What are the possible sources for this problem?
3. What do you comment about the role of Fatuma in this disease process?
4. What are possible interventions to prevent such occurrences in the future?
5. Could food be considered as a possible cause to this outbreak? Why?

2.5. Definition of Food borne diseases

The term “food borne disease” is defined as a disease usually either infectious or toxic in nature, caused by agents that enter the body through the ingestion of food (1).

2.6. Epidemiology

Although food is a basic human need it can sometimes cause a number of illness arising from pathogenic and toxic substances, which find their way in to food through contamination or spoilage (2). Food borne illnesses have significant impact world wide including developed nation like the US. The Center for Disease Control and Prevention (CDC) estimates for the US are 76 million cases, more than 300,000 hospitalization and 5000 deaths in a year. In addition 400 – 500 out breaks are reported. The spectrum of food borne diseases is constantly changing. New and re-emerging food-borne illnesses have resulted from recent changes in human demographics, international travel and commerce, microbial adaptations, economic development, technology and industry, eating behavior and land use (5).

In the last couple of decades a number of diseases thought to be of unknown causes have been proven to result from food borne infections. For example, hemolytic uremic syndrome which is a very important cause of acute renal failure in children is caused by infection with E.coli 0157: H₇ (EHEC) and related bacteria (9).

Epidemiologic data related to food-borne diseases are meager in Ethiopia. But it can be evidenced that these are very common in Ethiopia because of many reasons including poverty, lack of awareness, poor water supply, poor personal hygiene and environmental sanitation, etc.

According to the 2002-2003 “Health and Health-related Indicators” published by the Planning and Programming Department of the Federal Ministry of Health of Ethiopia,

- Helminthic infections were the second leading cause of outpatient visits
- Dysentery and different parasitic infections were also among the ten top causes of outpatient visits
- Dysentery was among the leading causes of hospital admissions and deaths
- The national average access to safe water was 28.4% (75.7% for urban and 19.9% for rural)
- National figure for safe excreta disposal was 11.5% (49.7% for urban and 3.9% for rural)
- Typhoid fever, acute diarrheal diseases, bloody diarrhea and anthrax were reported as some of the major causes of outbreaks (27).

2.7. Classification and Etiology of Some Food Borne Diseases

Food borne diseases are classified into two major categories depending on the causative agent: food-borne poisonings/intoxications and food-borne infections. For further information, see Figure 2.1.

Food borne infections: are diseases whose etiologic agents are viable pathogenic organisms ingested with foods and that can establish infection. Table 2.1 shows some examples of food-borne infections including their category, and the foods commonly involved.

Food borne poisonings/ intoxications: diseases arising from the ingestion of toxins released by microorganisms, intoxications from poisonous plants or toxic animal tissues: or due to consumption of food contaminated by chemical poisons. (3, 4)

Table 2.2 shows the etiologies of some food-borne poisonings/intoxications including their category, and the foods commonly involved.



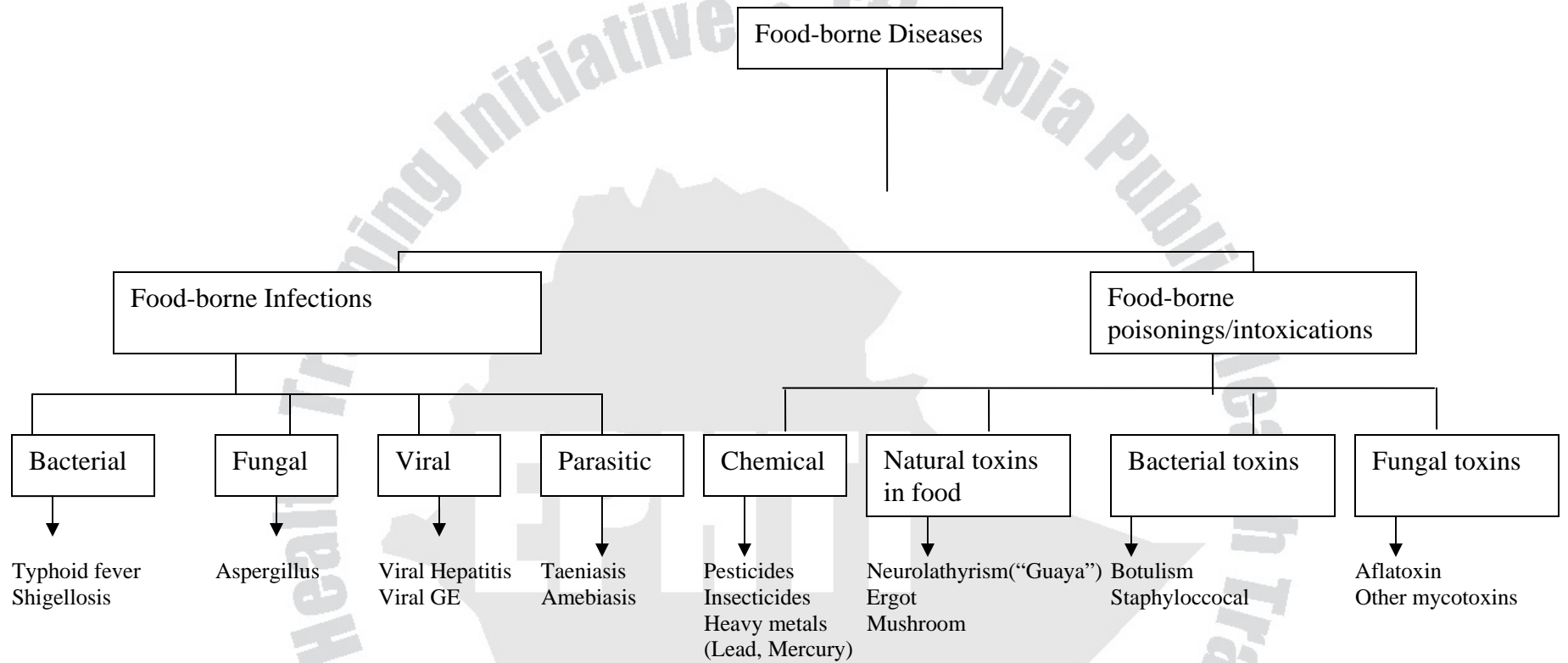


Figure 2.1: Classification of food borne diseases, with examples

Table 2.1: Etiologies of some food borne infections and foods commonly involved.

| Etiologic Category | Diseases | Causative organisms | Foods commonly involved |
|---------------------------|---------------------------|--|---|
| 1. Bacterial | Typhoid fever | Salmonella typhi and paratyphi | Raw vegetables and fruits, salads, pastries, un- pasteurized milk and milk products, meat |
| | Paratyphoid fever | Salmonella paratyphi | |
| | Shigellosis | Shigella species | All foods handled by unhygienic workers, potato or egg salad, lettuce, raw vegetables |
| | Cholera | Vibrio cholerae | Fruits and vegetables washed with contaminated water |
| | Non typhoid Salmonellosis | Salmonella species, e.g. Salmonella typhimurium | Eggs, poultry, undercooked meals, un-pasteurized dairy products, sea foods, sausages |
| | Brucellosis | Brucella species, mostly Brucella melitensis | Milk and dairy products from infected animals. |
| | Anthrax | Bacillus Anthracis | Contaminated raw and undercooked meat |
| | Bovine TB | M. Bovis | Un-pasteurized milk or dairy products from tuberculous cows, meat |
| | E.coli infections | E.coli | Beef, dairy products, fresh products, or raw produce (potatoes, lettuce, sprouts, fallen apples), salads. |
| | Listeriosis | Listeria monocytogenes | Milk, cheese, ice cream, poultry, red meat |
| 2. Viral | Viral GE | Rota virus, Norwalk virus, calici virus, astro virus | Any food of daily use with poor hygiene |
| | Viral hepatitis | Hepatitis A & E | Raw shellfish from polluted water, sandwich, salad, and desserts. |

| Etiologic Category | Diseases | Causative organisms | Foods commonly involved |
|---------------------------|--------------------|--------------------------------------|---|
| | Poliomyelitis | Polio virus | Any food of daily use with poor hygiene |
| | Rift valley fever | Rift valley fever virus | Any food contaminated with blood or aerosols from infected domestic animals or their abortuses |
| 3. Parasitic | Taeniasis | Taenia species | Raw beef, raw pork |
| 4. Fungal | Amoebiasis | Entameba histolytica | Any food soiled with feces |
| | Trichinosis | Trichnella spiralis | Insufficiently cooked pork and pork products |
| | Ascariasis | Ascaris lumbricoides | Foods contaminated with soil, specially foods that are eaten raw such as salads, vegetables |
| | Giardiasis | Giardia lamblia | Any contaminated food item |
| | Toxoplasmosis | Toxoplasma gondii | Raw or undercooked meat and any food contaminated with cat feces? |
| | Cryptosporidiosis | Cryptosporidium parvum | Apple juice, Any contaminated food item |
| | Hydatid disease | Echinococcus granulosus | Any food contaminated with dog feces |
| | Diphyllobothriasis | Diphyllobothrium latum | Raw or undercooked fish |
| | Trichuriasis | Trichuris trichuria | Any food contaminated with soil |
| | Fungal Infections | Aspergillus Penicillium Yeasts | Cereal, grains, flour, bread, corn meal, popcorn, peanut butter, apples and apple products, moldy supermarket foods, cheese, dried meats, refrigerated and frozen pasteries |

Table 2.2: Etiologies of some food borne poisonings/intoxications and foods commonly involved.

| Etiologic Category | Disease | Causative agent | Foods commonly involved |
|-----------------------------------|------------------------------------|---|---|
| A. Natural toxins in Foods | 1. Neurolathyrism | Beta oxalyl amino–alanine | “Guaya” (Lathyrus sativus) |
| | 2. Mushroom poisoning | Phalloidine and alkaloids found in some poisonous mushrooms. | Poisonous mushrooms such as species of Amanita phalloides and Amanita muscaria |
| B. Bacterial toxins | 1. Staphylococcal food poisoning | Enterotoxin from staphylococcus aureus | Milk and milk products, sliced meat, poultry, potato salad, cream pastries, egg salad |
| | 2. Perfringens food poisoning | Strains of Clostridium welchii/ C.perfringens | Inadequately heated or reheated meat, poultry, legumes |
| | 3. Botulism food poisoning | Toxin of Clostridium botulinum | Home-canned foods, low acid vegetables, corn and peas. |
| | 4. Escherichia coli food poisoning | Enterohemorrhagic Escherichia coli 0157:H7 | Ground beef, dairy products, raw beef. |
| | 5. Bacillus cereus food poisoning | Enterotoxin of Bacillus cereus | Cereals, milk and dairy products, vegetable, meats, cooked rice. |
| C. Fungal toxins | 1. Ergotism | A toxin (ergot) produced by a group of fungi called Cleviceps purpurea | Rye, wheat, sorghum, barley |
| | 2. Aflatoxin food poisoning | Aflatoxin produced by some groups of fungus (e.g Aspergillus flavus, Aspergillus parasiticus) | Cereal grains, ground nuts, peanuts, Cottonseed, sorghum. |

| Etiologic Category | Disease | Causative agent | Foods commonly involved |
|-----------------------------------|--------------------|--|---|
| D. Chemical food poisoning | Chemical poisoning | Heavy metals (e.g. Lead, mercury, cadmium) | <ul style="list-style-type: none"> - Fish, canned food - Foods contaminated by utensils made or coated with heavy metals |
| | | Pesticides and insecticides | <ul style="list-style-type: none"> - Residues on crops, vegetables, fruits. -Accidental poisoning where some chemicals may be mistaken with food ingredients. -When contaminated containers are used to hold or store foods. |
| | | Additives (unauthorized) | Various food items where unauthorized additives may be added as coloring agents, sweeteners, preservatives, flavoring agents etc. |

2.8. Pathogenesis and Clinical Features of Common Food-borne Diseases

2.8.1. Food-borne Infections

A. Bacterial Food Borne Infections:

i. Typhoid Fever (Enteric fever) and Paratyphoid Fever

Typhoid fever is a systemic disease caused by *S. typhi*.

S. paratyphi A and B cause paratyphoid fever.

Pathogenesis:

Following ingestion, the bacteria enter the epithelial layer of the small intestine, and are carried by macrophages throughout the body. They reach the reticuloendothelial system of the body. During bacteremic phase, they also seed in organs like the gall bladder (9).

Clinical features:

- After an incubation period of 3-21 days, patients present with prolonged fever, headache, anorexia, chills, malaise, abdominal pain, and diarrhea or constipation.
- On examination, one may find enlarged liver and spleen and abdominal tenderness.
 - Untreated patients may develop different complications, mainly gastrointestinal hemorrhage and/or perforation.

ii. Non-typhoidal Salmonellosis

Pathogenesis

Following ingestion, the non-typhoidal salmonella organisms reach the bowel where they cause damage to the intestinal mucosa causing inflammatory diarrhea.

Clinical Features

- The major clinical features are loose, non-bloody stools of moderate volume, nausea, vomiting, fever and abdominal cramps, which are seen following an incubation period of 6 – 48 hours.
- Large-volume watery diarrhea or dysentery can also occur.

iii. Shigellosis/Bacillary dysentery

It is an acute inflammatory colitis caused by a number of *Shigella* species.

Pathogenesis:

Orally ingested organisms invade colonic epithelial cells. Following this they multiply within the cells causing cell damage and death. This results in mucosal ulcerations.

Clinical Features:

After an incubation period of 1-7 days, patients present with non-bloody watery diarrhea or gross dysentery with tenesmus accompanied by fever which is particularly severe in children (40 – 41°C) and abdominal pain.

- Some of the complications of shigellosis are dehydration and bacteremia.

iv. Cholera

Cholera is an acute diarrheal disease caused by toxin released by *Vibrio cholerae*. It may result in death within hours if not treated.

Pathogenesis:

- Following colonization of small intestine, the organism releases a potent enterotoxin called cholera toxin.
- This toxin inhibits sodium absorption and activates chloride excretion resulting in the accumulation of sodium chloride in the intestinal lumen, which attracts water passively (9).

Clinical features:

- After an incubation period of 24 – 48 hours, patients experience sudden onset of profuse watery diarrhea accompanied by vomiting.
- Fever and abdominal pain are usually absent.
- Important complications include dehydration up to hypovolemic shock, electrolyte disturbances and acute renal failure.

v. *Escherichia coli* Infection

There are different strains of *Escherichia coli* which give rise to diarrhea by different mechanisms:

1. Enterotoxigenic *E.coli* (ETEC)

Pathogenesis:

Colonizes the small bowel and releases heat labile (LT) and heat stable (ST) toxins which stimulate fluid secretion.

Clinical Features:

The clinical features are watery diarrhea without mucus or blood and abdominal cramps.

2. Enterohemorrhagic *E.coli* (EHEC)

Pathogenesis:

Colonizes the colon and the ileum and produces Shiga-like toxin which results in inflammatory response in colonic mucosa.

Clinical Features:

The clinical features include initial watery diarrhea followed by grossly bloody diarrhea and significant abdominal pain. Hemolytic uremic syndrome may complicate EHEC infection.

3. Enteropathogenic *E-coli* (EPEC)

Pathogenesis:

Colonize the small bowel and adhere to the mucosa causing effacement of microvilli.

Clinical Features:

Manifests with diarrhea with mucus but no blood.

4. Enteroinvasive *E.coli* (EIEC)

Pathogenesis:

Invades and replicates within the colonic mucosa.

Clinical Features:

Produces a clinical disease that shares many features of Shigella infection (9).

vi. Brucellosis

Pathogenesis:

The organism invades the blood stream and localizes in the liver, spleen, bones, etc. In these tissues it induces inflammatory responses.

Clinical Features

The incubation period 1 week to several months and then patients manifest with many different symptoms and signs most common of which include fever, chills, sweating, myalgia, arthralgia, headaches, anorexia, weight loss, dry cough, etc.

Patients may appear well or may be very ill with any of the following manifestations: pallor, lymphadenopathy, enlarged liver and spleen, evidences of joint inflammation, rash, etc.

vii. Anthrax

Pathogenesis:

The organisms release anthrax toxin, which is responsible for the different manifestations of the disease.

There are three major clinical forms of anthrax:

1. Cutaneous anthrax (95%), which is the most common characterized by localized skin lesion with black central eschar of necrosis and non-pitting edema.
2. Inhalation anthrax (Wool sorter's diseases) characterized by hemorrhagic mediastinitis with high mortality rate.
3. Gastrointestinal anthrax, which is common in Ethiopia, and has high mortality rate.

Since gastrointestinal anthrax is the most important form of anthrax with respect to acquisition through contaminated food, the following discussion focuses on this form of anthrax.

Clinical features of gastrointestinal anthrax:

There are two major forms:

- Gastrointestinal anthrax manifesting with fever, nausea, vomiting, abdominal pain, massive and/or bloody diarrhea and occasional ascites.
- Oropharyngeal anthrax manifests with fever, sore throat and difficulty of swallowing, painful regional lymphadenopathy and respiratory distress.

B. Parasitic Food Borne Infections

Most common food-borne parasitic diseases to be considered are Amebiasis, ascariasis, taeniasis and giardiasis.

i. Amebiasis

Pathogenesis:

Motile trophozoites released from ingested cysts invade large bowel mucosa and cause mucosal ulcerations; they may also spread to other organs via the bloodstream to cause metastatic lesions (most commonly in the liver). Intestinal infections rarely cause mass lesion (ameboma).

Clinical features

There are various clinical syndromes

- Symptomatic intestinal amebiasis manifests with abdominal pain and mild diarrhea followed by diffuse abdominal pain, weight loss, malaise, and bloody-mucoid diarrhea. Fever occurs in less than 40% of patients.
- Amoebic liver abscess manifests with fever, abdominal and/or right lower chest pain, point tenderness over the liver and right-sided pleural effusion. Less than 30% have diarrhea.
- Complications include rupture of the abscess and formation of abnormal communications between the abscess cavity and the bronchi.

ii. Giardiasis

Pathogenesis:

- Ingested cysts release trophozoites in the small intestine.
- The trophozoites multiply by binary fission, adhere to the intestinal mucosa and lead to diarrhea and malabsorption; however, the exact mechanism by which *G. lamblia* produces diarrhea is not clear.

Clinical features

Most patients are asymptomatic. But in symptomatic individuals, the clinical features range widely and include diarrhea, abdominal pain, flatulence, anorexia, weight loss, nausea and vomiting.

iii. Taeniasis

a. Taeniasis Saginata

Pathogenesis:

- This is the common form of taeniasis in Ethiopia.
- Cysticerci deposited in the striated muscles of cattle infect humans when they are ingested together with raw or undercooked beef and they develop into adults in the small intestine.

Clinical features:

Patients notice passage of proglottids in the feces, perianal discomfort, abdominal discomfort or mild pain, nausea and anorexia.

b. Taeniasis solium

Pathogenesis:

T. solium is able to cause two different forms of infection in humans.

- Intestinal disease is infection with adult tape worms, acquired by ingestion of undercooked pork containing cysticerci.
- Cysticercosis is infection with larval forms in the tissues, most commonly the brain and skeletal muscles, and follows ingestion of *T. solium* eggs. Fecal-oral autoinfection is possible.

Clinical Features:

- Intestinal infection may be asymptomatic or may manifest with epigastric discomfort, nausea, hunger sensation, diarrhea, etc.
- Cysticercosis: the clinical features depend on the location and number of cysticerci and the degree of inflammatory response they induce in the tissue.

iv. Ascariasis

Pathogenesis:

- Eggs released with feces mature in the soil and become infective in weeks.
- When swallowed with contaminated food, they release larvae in the intestines, which enter blood, go to the lungs, enter the alveoli, ascend the bronchial tree and are swallowed into the bowel.

- In the small intestine, they develop into adult worms.

Clinical features:

Clinical manifestations result from:

- Larval migration in the lungs: cough, shortness of breath, blood-tinged sputum
- Effect of adult worms in the intestine: usually asymptomatic, but may produce intestinal obstruction, perforation; or worms may migrate to ectopic sites to produce other manifestations like biliary colic.

C. Viral Food Borne Infections

Different viruses may be transmitted via contaminated food; most produce mild self-limiting illness, but occasional severe illnesses and even deaths may also occur.

i. Viral gastroenteritis

Pathogenesis:

- Rota virus causes osmotic diarrhea due to nutrient malabsorption. Caliciviruses such as the Norwalk virus also produce diarrhea in a similar but slightly different mechanism that culminates in nutrient malabsorption.

Clinical Features:

- Rota virus infection causes sudden onset of vomiting followed by mild to very severe diarrhea mixed with mucus and fever.
- Norwalk illness results in abrupt onset of nausea and abdominal cramps followed by vomiting and /or diarrhea, low-grade fever, headache, myalgia after an incubation period of 18 to 72 hours.

ii. Viral hepatitis.

Pathogenesis:

- Almost exclusively the fecal-oral route transmits Hepatitis A and E viruses. None of the hepatitis viruses directly damages liver cells. Immunologic response of the host plays important role in the pathogenesis(9).

Clinical features:

- The incubation period varies according to the responsible agent.

- Prodromal symptoms include anorexia, nausea and vomiting, fatigue and malaise, arthralgia and myalgia, headache, photophobia, low-grade fever (38 – 39°C).
- These are followed by development of clinical jaundice; possibly accompanied by mild weight loss, tender enlarged liver, right upper quadrant pain.

2.8.2. Food poisonings / intoxications

A. Bacterial Food Poisoning

i. Clostridium perfringens

Pathogenesis

The spores are able to survive cooking, and if the cooked food (meat and poultry) is not cooled enough, they will germinate. The abrupt change in PH from stomach to intestine causes sporulation to occur, which releases the toxin. When massive dose of these organisms are ingested with food, toxins are elaborated in the intestinal tract which cause increased fluid and electrolyte secretion. (4)

Clinical Features:

- Incubation period: 6 to 24 hrs usually 8 to 24 hours after consumption of the food.
- The most common symptoms are diarrhea, abdominal cramp and little or no fever, nausea is common, but vomiting is usually absent. Illness is usually of short duration, usually 1 day or less. The disease is rarely fatal in healthy people. A cardinal symptom is explosive diarrhea. (4, 1, 9, 10, 6).

ii. Escherichia Coli 0157:H7

Pathogenesis:

Its somatic O and flagellar H antigens designate E-coli 0157:H7. Six classes of diarrhea-causing E-coli are recognized. They are enter hemorrhagic (EHEC), enterotoxigenic (ETEC), enteroinvasive (EIEC), enteroaggregative (EAEC), enteropathogenic (EPEC), and diffusely adherent (EDAEC). All enter hemorrhagic strains produce shiga toxin 1 and/ or shiga toxin 2, also referred to as Vera toxin 1 and Vera toxin 2. The ability to produce shiga toxin was acquired from a bacteriophage, presumably directly or indirectly from shigella (7). Enterotoxaemia E-

coli causes watery diarrhea by acting upon the upper small intestine (12). This bacterium attaches itself to the walls of intestine, producing a toxin that attacks the intestinal lining (7).

Clinical Features:

- Incubation period: The initial symptoms of hemorrhagic colitis generally occur 1 to 2 days after eating contaminated food, although periods of 3 to 5 days have been reported.
- Symptoms start with mild, non-bloody diarrhea that may be followed by a period of abdominal pain and short-lived fever.
- During the next 24 to 48 hours, the diarrhea increases in intensity followed by a 4 – 10 days phase of overtly bloody diarrhea, severe abdominal pain, and moderate dehydration.
- A life – threatening complication that may occur in hemorrhagic colitis patients is hemolytic-uremic syndrome, which may occur a week after the onset of gastrointestinal symptoms characterized by edema and acute renal failure. This occurs most frequently in children less than 10 years old.

iii. Bacillus Cereus

Pathogenesis:

The pathogenic agent of *Bacillus cereus* food poisoning appears to be an enterotoxin. This spore forming bacterium produces a cell-associated endotoxin that is released when cells die upon entering the digestive tract (4).

Clinical features

- Incubation period: From 1 to 16 hours in cases where vomiting is the predominant symptom; from 6 to 24 hours where diarrhea is predominant (10,7).
- The symptoms consist of nausea, vomiting, cramp like pains, tenismus, and watery stools. Fever is generally absent (4, 13, 7).

iv. Staphylococcal Food Poisoning

Staphylococcal food poisoning is the major type of food intoxication.

Pathogenesis

The disease is caused by enterotoxins produced by *Staphylococcus aureus*.

The toxins appear to act as neurotoxins that stimulate vomiting through the vagus nerve.

Clinical Features

Typical symptoms include severe abdominal pain, cramps, diarrhea, vomiting, and nausea. The onset of symptoms is rapid (usually 1 to 8 hours) and of short duration (usually less than 24 hours).

v. Botulism

Food-borne botulism is a form of food poisoning caused by *Clostridium botulinum*. It occurs in poorly canned foods, including home-canned foods and honey.

Pathogenesis

It is primarily caused by botulinum toxin, which is a neurotoxin that binds to the synapses of motor neurons preventing neurotransmission. As a consequence, muscles do not contract and flaccid paralysis results.

Clinical Features

Symptoms of botulism occur within 18 to 24 hours of toxin ingestion and include blurred vision, difficulty in swallowing and speaking, muscle weakness, nausea, and vomiting. Without adequate treatment, 1/3 of the patients may die within a few days of either respiratory or cardiac failure.

Infant botulism is the most common form. The infant becomes constipated, listless, generally weak, and eats poorly. Death may result from respiratory failure.

B. Chemical Food Poisoning

i. Heavy Metals

a. Lead poisoning

Possible sources of contamination include residues migrating into foods from soldered cans, leaching from utensils, contaminated water, glazed pottery, painted glassware and paints.

Metabolism

Lead is absorbed through ingestion or inhalation, and excreted in small amounts mainly in the urine and in the feces. Toxicity occurs due to its affinity for cell membranes and mitochondria, as a result of which it interferes with mitochondrial oxidative phosphorylation and sodium, potassium, and calcium transport.

Clinical Features

Lead poisoning is characterized by abdominal pain and irritability followed by lethargy, anorexia, pallor, ataxia, and slurred speech, joint pain, peripheral motor neuropathy and deficits in short-term memory and the ability to concentrate. Convulsions, coma and death due to generalized cerebral edema and renal failure occur in most severe cases. Sub clinical lead poisoning can cause mental retardation and chronic renal failure. The impact is greatest when the exposure is of long duration.

b. Mercury Poisoning

Possible sources include seafood, and mercury fumes in industries.

Pathogenesis

It is well absorbed by lungs and gastrointestinal tract, and excreted in small amounts in urine and/or feces. Toxicity manifestation occurs due to its local effect and its retention in kidneys.

Clinical features

Inhalation of mercury vapor manifests with cough, dyspnea, and tightness or burning pain in the chest.

Acute high dose ingestion of mercury can cause nausea, vomiting, hematemesis abdominal pain, diarrhea and tenesmus.

Major complications of mercury poisoning include:

- Respiratory distress, pulmonary edema, lobar pneumonia and fibrosis.
- Neurological toxicity.
- Acute renal failure and circulatory collapse

c. Arsenic

Sources for arsenic contamination of food include seafood, pesticides and herbicides.

Pathogenesis:

After absorption, inorganic arsenic accumulates in the liver, spleen, kidneys, lungs, and gastro intestinal tract. It is then rapidly cleared from these sites but leaves a residue in keratin-rich tissues. Arsenic, particularly in its trivalent form, inhibits critical sulfhydryl containing enzymes. In the heptavalent form, the competitive substitution of arsenic for phosphate can lead to rapid hydrolysis of the high – energy bonds in compounds such as ATP.

Clinical Features:

Major clinical features of arsenic poisoning include nausea, vomiting, diarrhea, abdominal pain, and delirium. In chronic arsenic poisoning, skin and nail changes may be seen.

2.9. Diagnosis of Food-borne Diseases

A variety of infectious and non-infectious agents should be considered in patients suspected of having a food borne illness. However, establishing a diagnosis can be difficult, particularly in patients with persistent or chronic diarrhea, those with severe abdominal pain and when there is an underlying disease process. The extent of diagnostic evolution of food borne diseases can be based on history, clinical features, environmental assessment and laboratory investigations. If applicable, radiological examinations may be implemented.

2.9.1. Clinical Assessment

The clinical diagnosis can be based on the clinical features discussed earlier in section 2.8.

A case history may be important clue in determining the sources and causes of the diseases and the type of foods involved. Therefore, obtain the history regarding the following points (15).

- Where, when, and what has been consumed?
- How soon after consuming the food did the symptoms occur?
- Duration of the resultant illness,
- Whether the consumed food had an unusual odor or taste?
- Inquiry on whether any other person or individuals have consumed the same food.

- Did any one else become ill from eating the same food?
- Any other evidence suggesting the cause of the illness.

Also, thorough physical examination should be done on any patient suspected to have food-borne disease.

2.9.2. Laboratory Investigations

The laboratory investigations will help to identify the causative agents. These investigations include macroscopic examination, microscopic examination, culture and biochemical tests, serology and toxicological tests. Different biological specimens such as stool, blood, liver aspirate, duodenal aspirate and muscle biopsy can be used for the investigation (16).

Macroscopic Examination

- Routinely examine fecal specimens and identify the physical characteristics of the stools (color, consistency, presence of blood, and mucus).
- Identify gravid segments of tape-worm from the stool specimen.
- Observe adult ascaris worm passing with stool, vomitus, or through the mouth or nose: (16)

Microscopic Examination

- The direct examination of stool specimen is essential to detect motile parasite, cyst and helminthes eggs. Because only a few eggs and cysts are usually produced even in moderate and severe infection, concentration technique should be performed.
- Gram stain to detect Gram–positive and Gram–negative bacteria (17)

Culture and biochemical tests

- General and enrichment culture media can be used to grow bacteria
- A selective medium is used to isolate the bacterial pathogen that causes food borne diseases.
- Stool cultures are indicated if the patient is, febrile, has bloody diarrhea, , or if the illness is clinically severe or persistent. It is also recommended if many fecal leukocytes are present.

- Blood cultures should be obtained when bacteremia or systemic infection is suspected (15).
- Following culture, biochemical tests are often required to identify pathogens by their enzymatic and fermentation reactions. (17)

Serology

Serological technique most frequently used in laboratories are those that can be performed simply and economically, uses stable reagents, do not require special equipment and enable specimen to be tested individually or in small number. Such techniques include agglutination test, flocculation technique and enzyme immunoassay (17).

- In agglutination test used to detect antibody in a patient serum, a known antigen suspension is used. The antigen particles are agglutinated if the serum contains the corresponding antibody.
- Flocculation tests for antibody detection are based on the interaction of soluble antigen with antibody, which results in the formation of a precipitate that can be observed microscopically or macroscopically.
- In ELISA, an enzyme is labeled or linked to a specific antibody or antigen. A substrate is added after the antigen-antibody reaction. This substrate is acted on (usually hydrolyzed) by the enzyme attached to the antigen-antibody complex, to give a color change. The intensity of the color gives an indication of the amount of bound antigen or antibody.

Toxicological Tests

Occasionally, the toxicology laboratory is asked to aid in the diagnosis of possible chemical intoxication by taking blood or urine sample from the affected individuals (22).

2.9.3. Environmental Assessment

It is important to conduct environmental assessment and collect environmental samples for suspected and potential causes of food borne illnesses especially of out breaks. The assessment may include survey of the source of the out-break with critical evaluation of:

- Source of the suspected food;
- How the food is prepared including cleanliness of table and kitchenware;
- Personal hygiene and health status of food handlers;
- Sanitation of the food preparation and service premises;
- Storage of the food before and after its preparation;
- Presence of potential or actual contaminants;
- Availability of safe and adequate water supply;
- Availability of safe and adequate sanitary facilities;
- Type and quality of food storage, and service equipments including food contact surfaces.
- Collection of samples from suspected foods and dishware as well from vomitus and stools of cases.
- Power failure before the outbreak and breakdown of refrigeration

Outbreaks and incidents of food poisoning and food borne infection require careful histories of the food vehicle, with environmental studies of the areas of food production and preparation as far back as possible. Sites of infection and areas of spread may include the farm of origin, dealers, markets, processing areas, wholesale or retail outlets to catering establishments, restaurants and domestic kitchens. Transport conditions for live animals and for food-stuffs may enhance spread also. (13).

2.10. General Management Approaches of Food-borne Diseases

The management approach to food-born diseases depends on the identification of specific causative agent, whether microbial, chemical or other. In addition determination of whether specific therapy is available and / or necessary or not is very important issue to consider. The management interventions for food born diseases may involve one or more of the following.

- Symptomatic and supportive therapy
- Specific antimicrobial, antitoxin, or antidotes, etc therapy.
- Surgical therapy (15)

Many episodes of acute gastroenteritis are self-limiting and require only fluid replacement and supportive care. If an antimicrobial is required the choice should be based on:

- Clinical symptoms and signs
- Organism identified from specimens
- Antimicrobial susceptibility test, and
- Availability of drugs for the identified organisms.

However, the limitation of facilities will have a negative impact on the choice of the specific management approach.

2.11. Prevention and Control of Food-borne Diseases

Prevention and control of food-borne diseases, regardless of the specific cause, are based on the same principles:

1. Avoidance of food contamination
2. Destruction or prevention of contaminants
3. Prevention of further spread or multiplication of contaminants.

Specific modes of intervention vary from area to area depending on environmental, economic, political, technology and socio cultural factors.

The preventive and control strategies may be approached based on the major site in the cycle of transmission or acquisition where they are implemented. These involve the following activities performed at the source of infection, environment and host.

1. Interventions at the source of infection include:

- Thorough cooking of raw food
- Thorough washing of raw vegetables with clean water
- Keeping uncooked animal products far separate from cooked and ready-to-eat foods.
- Avoiding raw milk or foods made from raw milk.
- Appropriate treatment of food items before consumption
- Active immunization of animals
- Inspection of food

- Sanitary disposal of human wastes
- Treatment of cases
- Washing hands, knives, cutting boards, etc. after handling uncooked foods.
- Avoiding contact with materials contaminated with pet excreta or soil.
- Decontamination of animal products, e.g., wool, goat hair
- Burying intact or cremating of infected animal carcasses.
- Isolation
- Recognizing, preventing, and controlling of infections in domestic animals, pets.
- Washing hands after contact with animals
- Training and supervision of food handlers and homemakers
- Treatment of carriers.
- Proper care for patients with food-borne illnesses.
- Avoidance of food from animals with obvious infection, e.g., mastitis in cows
- Treatment of infections in food handlers such as skin and throat infections

2. Environment interventions: involve stringent follow-up from production to consumption. Some of the interventions include:

- Freezing, salting, etc. of food items during storage
- Control of flies, rats, cockroaches
- Public education on environmental and personal cleanliness
- Surveillance of food establishments
- Avoiding contamination of food after cooking.
- Maintenance of sanitary food area.
- Proper handling and storage of leftover foods
- Kitchen cleanliness
- Safe canning at home
- Careful storage and use of chemicals (storage away from foods)

3. Host:

- Active or passive immunization of susceptible hosts
- Health education on the above areas.

“The Ten Golden Rules” of WHO for Safe Food Preparation (10)

1. Choose foods processed for safety
2. Cook food thoroughly
3. Eat cooked foods immediately
4. Store cooked foods carefully
5. Reheat cooked foods thoroughly
6. Avoid contact between raw and cooked food
7. Wash hands repeatedly
8. Keep all kitchen surfaces meticulously clean
9. Protect food from insects, rodents and other animals
10. Use safe water

Safe Food Handling:

- Require strict personal hygiene from all employees, and relieve infected employees of food handling duties. Instruct employees not to touch cooked food or food that will not be cooked.
- Identify all potentially hazardous foods on your menu and create a flowchart to follow these foods throughout the entire operation.
- Obtain foods from approved sources.
- Use extreme care in storing and handling food prepared and ready to eat foods via hands, equipment, and utensils, clean and sanitize food contact surfaces and equipment after every use to avoid cross contamination.
- Cook or heat processed food to at least the recommended temperatures.
- Store or hold foods at temperatures of below 4.4⁰C or above 60⁰C during preparation and holding for service.
- Cooked food should be chilled to 4.4⁰C or below within four hours in shallow pans in a refrigerator, in a quick chilling unit, or in an ice water bath and stirred or agitated frequently during the chilling.
- Reheat leftovers quickly to an internal temperature of at least 73.6⁰C within two hours.

2.12. Investigation of Outbreaks of Food-borne Diseases

Outbreaks of food-borne diseases can lead to deaths of many people within short periods, and hence their timely detection and proper management should not be undermined. When the Health Team receives information regarding an outbreak of a possible food-borne disease, action should start immediately. This action has to be integrated from the outset since the investigation and management of any outbreak requires the concerted effort of all health professionals concerned. In addition, being prepared beforehand for such outbreaks, by collecting the necessary information on food-borne diseases and previous outbreaks (in the area in particular) is important.

The objectives of investigating an outbreak of a food-borne disease are to:

- Identify the causative agent responsible for the outbreak
- Identify the food items, handlers, etc. responsible for the outbreak
- Identify and trace the location of the source of the outbreak
- Determine the conditions and mechanisms that led to the contamination of the food item identified, e.g. perform sanitary survey
- Limit the impacts and arrest the progression of the outbreak
- Be able to use information obtained from the current outbreak for the prevention of subsequent outbreaks

The health team should do the following in addressing a possible food-borne disease outbreak:

- Obtain as detailed information as possible from all available informers, cases, care-takers, clinicians, etc.; this involves interviewing of infected individuals, management, and food handlers; all the obtained information should be systematically registered using prepared questionnaires.
- In collecting this information, attempt should be made to determine the mean incubation period of the outbreak.
- The exact date and time at which the suspected food was consumed should be sought; and of those who ate and did not eat the food, the number and proportion of those who got sick should be calculated in order to know the attack rate. It will be helpful to have and keep a list of symptoms and signs during assessing these

individuals for the presence or absence of the suspected food-borne illness (nausea, vomiting, diarrhea, abdominal pain, fever, headaches, etc.).

- One has to keep in mind that the association between illness and exposure for the suspected food does not have to be “perfect”; in fact, this is rarely so because of different factors, one of which may be that the implicated food may not be contaminated throughout; in addition, host susceptibility varies as does dosage (the quantity consumed), and there may be errors in reporting food histories (faulty recall, uncertainty); there may also be errors in recording.
- If the outbreak is large and it is not possible to interview all participants, a random sample should be selected and questioned for symptoms and food exposure history.
- Develop a hypothesis based on the initial clinical features and other information obtained regarding the probable food-borne disease in order to devise case management plans to treat the sick individuals.
- Tell informers and cases to retain or recover all suspected food items, the original containers and packages.
- Collect specimens of suspected food, stool and vomitus from ill persons and send them to a reference laboratory immediately for identification of the agent.
- Obtain and use appropriate sampling equipments such as sterile containers and other apparatus.
- Visit the institution or place where the outbreak is suspected to have started. During this visit, all members of the team should go and analyze the environment and other situations in a systematic way; they have to keep records of all things observed.
- Analyze and interpret all the information collected using the different techniques outlined above and try to trace the exact source of the food implicated
- Finally, take remedial actions.
- Report the findings to the concerned authorities, and keep a document of it for future use from the experience gained.

Summary of steps in the investigation of food-borne disease outbreak investigation

1. Verify the existence of an outbreak

- Compare the current number of cases with the past
Note: Consider seasonal variations
2. Verify the diagnosis
 - Review clinical and laboratory findings
 3. Describe the outbreak with respect to time, place and person
 4. Construct an epidemic curve
 5. Calculate food-specific attack rates
 6. Formulate and test hypotheses
 7. Search for additional cases
 8. Analyze the data
 9. Make a decision on the hypotheses tested
 10. Intervene and follow-up
 11. Report the investigation
 12. Inform the public on the prevention and control of the disease.

N.B.

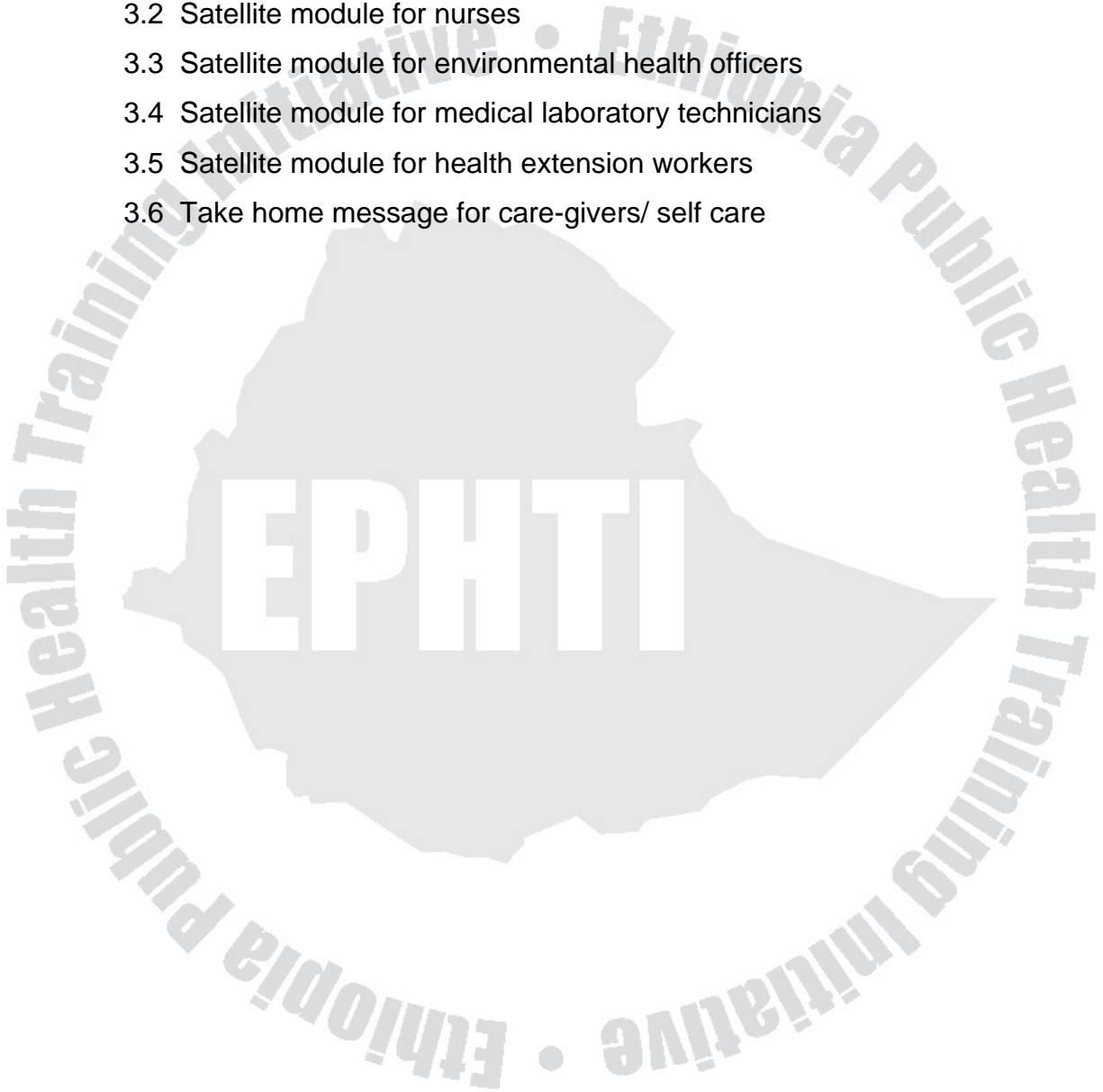
While investigating an outbreak the proper treatment and care for patients should not be ignored.

Now you are through with the Core Module, but the satellite module remains. Please go to the Satellite Module that applies to your specific professional category for continued thorough study.

UNIT THREE

SATELLITE MODULES

- 3.1 Satellite module for health officers
- 3.2 Satellite module for nurses
- 3.3 Satellite module for environmental health officers
- 3.4 Satellite module for medical laboratory technicians
- 3.5 Satellite module for health extension workers
- 3.6 Take home message for care-givers/ self care



3.1. SATELLITE MODULE FOR HEALTH OFFICERS

3.1.1 Directions for Using This Module

- Make sure you have thoroughly read the core module before you begin to read this satellite module.
- Read through this satellite module carefully.

3.1.2 Learning Activity

CASE I: Answer questions 1 and 2 based on the following case.

Ato Amsalu, a 28 year old patient, came from Asayta town to your health center complaining of fever and chills of a week's duration accompanied with headache, dry cough, abdominal pain, and reduced frequency of stooling. On examination, you find the following:

- The patient is acutely sick looking
- Axillary temperature is 38.5⁰ C
- Pulse rate is 74 beats per minute
- Blood pressure is 100/80 mm Hg right arm supine
- He has pale conjunctiva and mildly icteric sclera
- He has dry tongue and buccal mucosa
- On abdominal examination, you find enlarged liver and spleen together with diffuse mild abdominal tenderness.

1. Which of the following investigations is not as useful as the others in this patient to clarify his problem?

- a) Blood film
- b) Chest X-ray
- c) Widal test
- d) Weil-felix test
- e) All are equally useful

2. Your health center has recently run out of reagents needed for carrying out blood film, Widal test as well as Weil-felix test. Among the following, which one do you think will be the most appropriate action to take?

- a) Urgent referral to a higher health facility for better investigation and treatment.
- b) Empiric treatment for malaria and typhoid fever with oral medications.
- c) Empiric treatment for malaria and close follow-up of the patient.
- d) Admission of the patient for treatment with broad-spectrum parenteral antibiotics.
- e) Treatment for 2 weeks with oral antimicrobial effective against typhoid fever.

CASE II: Answer questions 3 and 4 based on the following case.

Yesterday evening, W/o Tsehay arrived to Harar town after a long day's journey from Addis. Starting early this morning, she noticed passage of watery stools and vomiting of ingested matter. She came to your health center and told you that she has passed only 2 loose stools up to the time of presentation and that she has no fever but mild crampy abdominal pain. On examination, you find that her vital signs are all within normal limits and she has no signs of dehydration. In addition, there is no abnormal finding on abdominal examination. Your request for stool examination returns with the following report: the gross appearance showed only loose stool with no mucus or blood and the microscopy revealed no fecal leukocytes or parasite.

3. Which of the following organisms is the least possible responsible agent in this patient's problem?
 - a) *Staphylococcus aureus*
 - b) *Clostridium perfringens*
 - c) *Bacillus cereus*
 - d) *Shigella dysenteriae*
 - e) *Enterotoxigenic Escherichia coli*
4. The most important measure in the management of this patient is:
 - a) Oral fluid support
 - b) Use of antidiarrheal agents such as Lomotil
 - c) Prescription of ciprofloxacin for 5-7 days
 - d) Investigation for possible typhoid fever
 - e) A and D.

3.1.3 Learning Objectives

By the end of this satellite module, you are expected to be able to:

- ❖ Explain the pathogenesis of common food-borne diseases
- ❖ Describe the clinical features and complications of common food-borne diseases
- ❖ Diagnose common food-borne diseases
- ❖ Outline a management plan for some food-borne diseases
- ❖ Identify some control and prevention measures for some food-borne diseases

3.1.4. Pathogenesis, Clinical Features, Diagnosis and Treatment of Common Food-borne Diseases

3.1.4.1. Food-borne Infections

A. Bacterial Food-borne Infections

I. TYPHOID FEVER (ENTERIC FEVER)

Pathogenesis:

- Ingested salmonella organisms traverse the stomach, resisting the acidic environment, to reach the small intestine, which they colonize. Patients with reduced gastric acidity such as those with antacid ingestion have increased susceptibility to salmonella infection (9,23)
- Then they cross the intestinal barrier mainly through phagocytic cells overlying Peyer's patches.
- Infiltration of the mononuclear cells into the small-bowel mucosa accompanies replication of the organisms (9)
- After crossing the epithelial layer of the small intestine, they undergo phagocytosis by macrophages and are in this way disseminated throughout the reticuloendothelial system via lymphatics (protected by the macrophages from polymorphonuclear leukocytes, the complement system, and antibodies).
- They colonize the reticuloendothelial tissues (liver, spleen, lymph nodes, bone marrow)
- They circulate in the blood (*bacteremia*) and seed into the gall bladder, intestines, and other tissues (9,23)

Clinical Features:

- Patients usually have a prodrome of non-specific symptoms such as chills, headache (mild to very severe), anorexia, dry cough, weakness, sore throat, dizziness, and muscle pains before the onset of fever (9,23,25)
- Fever-prolonged, 38.8-40.5 °C, with or without a stepladder rise pattern
- Abdominal pain (20-40%) and tenderness (9)
- Variable gastrointestinal symptoms-diarrhea or constipation; diarrhea is more common among children and HIV+ patients
- Rash (“rose spots”)-faint erythematous maculopapular rash on the trunk and chest. This is difficult to appreciate in dark-skinned individuals. (9)
- Hepatosplenomegaly
- Epistaxis
- Relative bradycardia for the degree of fever (9)
- Neuropsychiatric manifestations

Complications:

- Gastrointestinal hemorrhage and/or perforation, usually occurring in the third and fourth weeks of infection, are mostly seen in adults who did not receive treatment. (9,25)
- Rare complications include: hepatitis, hepatic and/or splenic abscesses, pancreatitis, endocarditis, pericarditis, orchitis, meningitis, pneumonia, bone and/or joint infections.
- Relapse rates are around 10% even in patients with intact immunity who have received prompt and adequate antibiotic treatment.
- Chronic carriers account for up to 1-5% of patients with enteric fever. The figure is more in females and patients with hepatobiliary abnormalities. These individuals continue to shed organisms in their urine and/or stool for more than a year. (9)

DIAGNOSIS:

- Clinical features:

The most prominent symptom of typhoid fever is prolonged fever (38.8-40.5°C) with or without a stepladder rise pattern.

- Laboratory investigations (9,23,25)
 - ❖ The gold standard for the diagnosis of typhoid fever is culture from stool, blood, urine or other specimens.
 - ❖ Tube dilution agglutination test (Widal test) –serum agglutinins rise sharply during the second and third weeks of salmonella infection. At least two serum specimens, obtained at intervals of 7-10 days are needed to prove a rise in antibody titer. The interpretation is as follows:
 - High or rising titer of O ($\geq 1:160$) suggests active infection
 - High titer of H ($\geq 1:160$) suggest past infection.
 - ❖ Rapid (10-15 minutes) immunochromatographic test has recently been developed.

N.B.

Results of serologic tests for salmonella infections must be interpreted cautiously; the possible presence of cross-reactive antibodies limits the use of serology in the diagnosis of salmonella infections. However, in areas where facilities for culturing are not available, the Widal test if performed reliably and interpreted with care, in addition to clinical features, can be of value in diagnosing typhoid fever.

Supportive laboratory evidences include (9,25):

- Anemia, leucopenia (neutropenia), thrombocytopenia
- Elevated PT and PTT (evidences of DIC)
- Elevated liver enzymes
- Presence of mononuclear leukocytes in the stool
- Serial plain abdominal films may be taken in search for evidence of intestinal perforation.

DIFFERENTIAL DIAGNOSES:

- Malaria
- Typhus
- Relapsing fever
- Pneumonia
- Tuberculosis

- Infective endocarditis (especially in known cardiac patients)
- Brucellosis
- Hepatitis
- Acute pyelonephritis

TREATMENT:

It may be carried out as inpatient if the individual is acutely ill, or as an outpatient for mildly ill individuals.

1. General Measures (9,24,25) include:

- Fluid and electrolyte support
- Antipyretic-analgesics as required such as paracetamol
- Close monitoring of the clinical course of the patient
- If there is suspicion of gastrointestinal hemorrhage or perforation, the patient should be immediately referred to a better health facility for appropriate management (blood transfusion, surgery).

2. Chemotherapy:

See Annex V for drugs available for the treatment of typhoid fever. Drug resistance is becoming an increasingly important problem. Many strains are nowadays resistant to chloramphenicol, ampicillin, amoxicillin, and co-trimoxazole.

For severe cases:

- a) Chloramphenicol 1 gram bolus IV every 6 hours until the patient remains afebrile for at least 48 hours followed by 500 mg PO every 6 hours to complete a total of 14 days of treatment; for children, 25 mg/kg IV bolus every 6 hours followed by 25 mg/kg PO every 6 hours after the patient has remained afebrile for 48 hours to complete 14 days of treatment.
- b) Ceftriaxone as above is also suitable for severe typhoid fever.

For chronic carrier state:

Treat for 6 weeks with one of the following drugs (9):

- a) Ampicillin 1 gram po qid
- b) Trimethoprim-sulfamethoxazole 800/160 mg po bid
- c) Ciprofloxacin 500 mg po bid

CONTROL MEASURES (6,10,26)

- Sanitary disposal of human excrement
- Proper management of patients, e.g.
 - Proper disposal of the excrements of patients (stool and urine)
 - Advice to families on how to prevent transmission
 - Teaching about carrier state
- Control of flies, rats, roaches
- Pasteurization of milk
- Chlorination of water supplies
- Education of public concerning personal cleanliness
- Proper handling of food, water, and human waste
- Vaccination

iii. NON-TYPHOIDAL SALMONELLOSIS

PATHOGENESIS:

- The disease is mainly caused by *S. typhimurum* and *S. enteritidis*
- Similar to enteric fever, the organisms have to traverse the stomach and gain access to the small intestine.
- Then they cause a localized infection that induces the massive infiltration of neutrophils in both the small and large intestinal mucosa with self-limited gastroenteritis (9)
- The degranulation and release of toxic substances by neutrophils may lead to damage of the intestinal mucosa, causing inflammatory diarrhea (9,23)

CLINICAL FEATURES:

- Patients present with nausea, vomiting, diarrhea (loose, nonbloody, moderate volume), abdominal cramping, fever (38-39⁰C); the gastroenteritis is self-limiting, resolving in 3-7 days (9,25)
- There may be signs of dehydration

DIAGNOSIS:

- Clinical features

- Nontyphoidal salmonella gastroenteritis is diagnosed when salmonella are cultured from stool; the organisms may also be cultured from blood or other body fluids such as joint fluid and the CSF in cases with metastatic infections(9,23,25).

TREATMENT (9,24,25)

1. **General measures:** as for typhoid fever

2. **Chemotherapy:**

- The symptoms are usually self-limited and antibiotic treatment is generally not recommended for Salmonella gastroenteritis.
- However, for patients at increased risk of metastatic infection, antibiotic treatment should be considered (neonates, patients older than 50 years of age, HIV infected patients)-with oral or intravenous antibiotics for 2-3 days or until defervescence. The antibiotics of choice are the same as those of typhoid fever.

CONTROL MEASURES:-

As for typhoid fever.

III. SHIGELLOSIS

PATHOGENESIS:

- Orally ingested *Shigellae* (*Shigella dysenteriae*, *S. flexneri*, *S. boydii*, *S. sonnei*) have the genetic capability to withstand the low stomach pH.
- They attach to and invade the colonic epithelial cells and spread to adjacent cells (This cell-to-cell spread helps the organism to evade host defense).
- The intracytoplasmic multiplication causes cell damage and death, leading to the development of characteristic mucosal ulcers.
- Another virulence factor is the Shiga toxin, which interferes with protein synthesis and affects cell membrane elements (9, 23)

N.B.

- Enterohemorrhagic strains of *E. coli* (EHEC) produce Shiga-like toxins, which are partially responsible for the typical, grossly blood diarrhea produced by this organism.
- Enteroinvasive strains of *E. coli* (EIEC) cause shigellosis-like syndrome through invasion of and replication within the colonic mucosa (9).

CLINICAL FEATURES:

- Patients may present with nonbloody or bloody-mucoid diarrhea, crampy abdominal pain, tenesmus, and fever (which can become high grade, particularly in children) (9, 25)
- There may be signs of dehydration.

Complications:

- Toxic colonic dilatation (toxic megacolon)
- Colonic perforation
- Protein-losing enteropathy
- Bacteremia, sepsis
- Hemolytic-uremic syndrome (HUS) characterized by hemolytic anemia, thrombocytopenia, and evidences of renal failure (oliguria, etc.); in addition one may find leukemoid reactions, hyponatremia, severe hypoglycemia, CNS manifestations, etc. This usually develops towards the end of the first week (9)
- Seizures
- Reactive arthritis (Immunologically mediated joint inflammation seen in some patients following shigellosis; it may also follow some other bacterial infections, e.g. E. coli infection)
- Metastatic infections like pneumonia, meningitis (rare)

DIAGNOSIS:

- Clinical features: Shigellosis should be considered whenever a patient presents with bloody diarrhea.
- Laboratory diagnosis (9,23,25)
 - Gross appearance of the stool may show blood, pus, and mucus.
 - On microscopy, there may be red blood cells and leukocytes in the stool.
 - The specific diagnosis is based on culture of Shigella from stool.
 - Leukemoid reactions (white cell counts of more than 50,000/ μ l)

DIFFERENTIAL DIAGNOSES:

- Invasive intestinal amebiasis
- Other bacterial diarrheas, such as those caused by E. coli
- Acute exacerbation of inflammatory bowel disease, especially ulcerative colitis

TREATMENT (9, 24,25)

1. General measures:

- Fluid replacement
- Analgesic-antipyretic

2. Chemotherapy:

- See annex V

N.B.

Antidiarrheal agents such as diphenoxylate and loperamide are generally contraindicated.

CONTROL MEASURES:

As for typhoid fever.

IV. CHOLERA

PATHOGENESIS:

- Cholera is a disease caused by toxin released by *Vibrio cholerae*.
- Ingested organisms colonize the small intestine, using a pilus for adherence. A large inoculum size is required in order to traverse the acidic medium of the stomach.
- Then they elaborate cholera toxin, a potent protein enterotoxin.
- The toxin, by increasing the level of cyclic AMP inside the cells, leads to inhibition of sodium absorption from and activation of chloride excretion into the intestinal lumen.
- The resulting intraluminal accumulation of sodium chloride drags water from the tissues along osmotic gradient.
- Isotonic fluid accumulates in the intestinal lumen in this way, and when it exceeds the capacity of the lumen, results in diarrhea (9, 23)

CLINICAL FEATURES:

- Then the patient manifests with sudden onset of painless, voluminous, cloudy with flecks of mucus, watery diarrhea ("rice-water" stool). Stool volume can sometimes exceed 250 ml/kg in the first 24 hours. Vomiting accompanies the diarrhea.

- Fever is usually absent (9)
- Some patients have abdominal cramps, muscle cramps
- Symptoms of dehydration-thirst, weakness, postural dizziness, reduced urine output, etc.

DIAGNOSIS

- a) Based on clinical features---assessment of the degree of volume deficit is most important.
- b) Laboratory diagnosis (9, 23)
 - Dark field microscopy to see characteristic motile vibrio
 - Stool culture
 - Laboratory anomalies of severe dehydration (e.g., elevated hematocrit), acid-base disturbances (e.g., acidosis), prerenal azotemia (e.g., elevated levels of blood urea nitrogen and creatinine) and electrolyte disturbances (e.g., hyponatremia, hypokalemia, hypochloremia).

TREATMENT (9, 24)

1. General measures:

- It is most important to assess the degree of fluid loss and to replace it. This may be done orally for mild cases using ORS, increased intake of fluid diets, and cereal-based rehydration therapy; or intravenously for severe dehydration. For intravenous fluid replacement, the best fluid to give is Ringer's lactate as it also helps to correct acidosis, which is common in severely dehydrated patients. Whether by intravenous or oral route, the fluid replacement should be monitored against fluid loss. In addition, supplementation of potassium, preferably by the oral route, is recommended.
- Giving prophylactic antimicrobials such as tetracycline for contacts helps to prevent spread.

2. Antimicrobial therapy (see annex)

This is not necessary for cure, but will diminish the duration and volume of fluid loss and will hasten clearance of the organism from the stool.

- For adults and older children, tetracycline 500 mg PO qid for 3-5 days or 2 gram PO stat; or doxycycline 100 mg PO bid for 3 days or 300 mg PO stat-for children, it may be given as 6 mg/kg/d for 3 days (weigh the risk and benefit); or ciprofloxacin 30 mg/kg PO stat (maximum 1 gram), or 15 mg/kg bid for 3 days (maximum 500 mg PO bid); erythromycin 40 mg/kg/day in 3 divided doses for 3 days
- For younger children, trimethoprim-sulfamethoxazole 8/40 mg/kg/day in 2 divided doses for 3-5 days.
- For pregnant mothers, furazolidone 100 mg PO qid for 7-10 days.

CONTROL MEASURES (6,10,26)

- Careful food selection, e.g., avoidance of unpeeled raw fruits and vegetables, as well as raw or undercooked seafood;
- Safe disposal of human waste;
- Tetracycline or doxycycline for contacts with doses indicated above;
- Vaccine has been developed but is not readily available.

v. BRUCELLOSIS

PATHOGENESIS:

- The Brucella organism is acquired most commonly through the ingestion of raw milk, milk products like cheese, meat, blood or bone marrow.
- The organisms become ingested by polymorphonuclear leukocytes and activated macrophages, but they resist intracellular killing by the phagocytes.
- They multiply and reach the bloodstream via the lymphatics.
- Then they localize in the liver, spleen, bones, kidneys, lymph nodes, heart valves, nervous system, and testes. Again in these organs, they become ingested by macrophages
- In the tissues, they induce caseating and noncaseating granulomas as well as abscess formation (9, 23)

CLINICAL FEATURES:

- Brucellosis is a systemic disease with many varied manifestations.
- Symptoms may start either abruptly or gradually.
- Fever with or without diurnal variation, chills, sweating, headaches, myalgia, fatigue, anorexia, joint and low-back pain, weight loss, constipation, sore throat, dry cough
- Physical Examination: usually reveals no abnormalities; but some patients may have pallor, jaundice, lymphadenopathy, hepatosplenomegaly, arthritis, spinal tenderness, epididymo-orchitis, rash, meningitis, cardiac murmurs, evidence of pneumonia. (9)

DIAGNOSIS

1. Clinical features and history of exposure to animal products
2. Laboratory diagnosis (9, 23)
 - Isolation of the organism from blood, discharge, bone or other tissue using culture is necessary for diagnosis
 - Serology (tube agglutination test)

TREATMENT

All suspected cases should be referred to higher centers for confirmation of diagnosis and appropriate treatment using multiple antimicrobial drugs.

CONTROL MEASURES (6, 10,26)

- Avoid ingestion of all raw milk
- Avoid contact with any fluid from infected animals
- For occupational exposure: Caution in handling animals and animal products (use of protective goggles and gloves), vaccination of animals against the disease

○ GASTROINTESTINAL ANTHRAX

PATHOGENESIS:

- Gastrointestinal (GI) anthrax usually results from ingestion of raw or inadequately cooked meat from animals infected with the organism

- Organisms that survive gastric defense establish primary infection in the intestine, producing lesions accompanied by hemorrhagic lymphadenitis. Oropharyngeal anthrax can also occur, with the primary lesion found most often in the tonsils (9)
- The organisms can invade the bloodstream and multiply rapidly
- They release the anthrax toxin, which has three different constituent proteins: Protective Antigen (PA), Edema Factor (EF), and Lethal Factor (LF) (9, 23)
- PA mediates entry of EF and LF into target cells
- EF induces the formation of edema in lesions
- LF induces the production of reactive oxygen species by macrophages, release of cytokines, shock and death (9)

CLINICAL FEATURES (9):

Intestinal Anthrax:

Fever, nausea, vomiting, abdominal pain, bloody diarrhea (occasionally massive), rapidly developing ascites

Oropharyngeal Anthrax:

The major features are fever, sore throat, difficulty of swallowing, painful regional lymphadenopathy; and there also may be respiratory distress.

DIAGNOSIS

1. Clinical features and exposure to animals (consumption of raw animal tissues)
2. The organism is occasionally isolated from blood or other infected body tissues.
3. Tests for antibody to B. anthracis are useful in confirming the diagnosis of anthrax (9, 23, 25)

TREATMENT

- Supportive treatment such as provision of IV fluid support
- Crystalline penicillin G 2-3 million IU every 4 hours for 7-10 days for adults
- Since these patients may need very close monitoring and extensive supportive management, they are best referred to higher centers.

CONTROL MEASURES:

- Similar to Brucellosis
- Avoidance of consumption of raw or undercooked meat.

B. VIRAL FOOD-BORNE INFECTIONS

a. VIRAL GASTROENTERITIS

PATHOGENESIS:

- **Rotavirus** infects and kills the mature cells of the small intestinal villi. This leads to nutrient malabsorption resulting in osmotic diarrhea (9, 23)
- **Enteric caliciviruses** like Norwalk virus result in disturbance of the architecture of the small intestine with shortening of villi and infiltration of lamina propria by polymorphs. These changes are accompanied by steatorrhea and carbohydrate malabsorption (9, 23)

CLINICAL FEATURES:

- **Rotavirus infection** can cause mild diarrhea to very severe fatal illness due to dehydration. It usually causes abrupt onset of vomiting followed by watery-mucoid diarrhea. One-third of children with rotavirus diarrhea may have fever of more than 39°C (9)
- **Norwalk infection** causes abrupt onset of nausea and abdominal cramps after an incubation period of 18-72 hours followed by vomiting and/or diarrhea. Half of patients have low-grade fever (9)

DIAGNOSIS

- Clinical features
- Stool examination reveals white and red blood cells in less than 15% of cases
- Specific diagnosis of etiology is not feasible in all Ethiopian health center settings, since isolation of viruses requires extremely specialized laboratories; moreover etiologic diagnosis of viral diarrheas is not important for case management.
- The most important measure in the diagnosis is to assess the extent of dehydration.

TREATMENT

The disease is usually self-limited and thus treatment focuses on supportive care in the form of fluid replacement (9)

- Assess the degree of dehydration and manage accordingly.

CONTROL MEASURES

- Safe disposal of human waste
- Personal hygiene
- Proper food preparation and handling

b. VIRAL HEPATITIS

PATHOGENESIS:

- Food-borne viral hepatitis is caused by hepatitis A or/and E viruses (HAV or HEV).
- None of the hepatitis viruses cause direct cytopathic effect on the liver cells (9)
- Immunological response to the presence of the viruses in the liver has important role in the pathogenesis of hepatic damage
- In hepatitis E, cholestasis is a common histologic feature. This is also possible in hepatitis A.
- Neither HAV nor HEV causes chronic liver disease (9)

CLINICAL FEATURES:

- Incubation period varies depending on the agent (15-45 days for hepatitis A; 14-60 days for hepatitis E.
- Prodromal symptoms including anorexia, nausea and vomiting, altered smell and taste, fatigue, malaise, arthralgias, myalgias, headache, photophobia, pharyngitis, cough, coryza, and low-grade fever (38-39°C) may precede the icteric phase by 1-2 weeks; some patients have dark urine and clay-colored stools 1-5 days before the onset of the jaundice. Others may have steatorrhea.
- Icteric phase manifests with clinical jaundice, mild weight loss (2-5 kg), enlarged tender liver, right upper quadrant pain and discomfort
- 10-20% of patients have splenomegaly and cervical lymphadenopathy.
- Complete clinical and biochemical recovery is expected to occur in 1-2 months in all cases of hepatitis A and E.
- Significant percentages of patients never become jaundiced (anicteric hepatitis) (9, 25)

DIAGNOSIS

1. Clinical features-history of exposure should be sought; exclude drugs and alcohol as possible causes
2. Laboratory diagnosis (9)
 - Marked elevation of liver transaminases
 - Mild elevation of alkaline phosphatase
 - Bilirubin level may be normal to markedly elevated; if elevated, both the conjugated and un-conjugated fractions will be increased
 - Serologic markers to identify viruses
 - Anti-HAV IgM indicates recent infection.
 - Marker for HEV infection has recently been reported.
 - For severe hepatitis, measure PT and PTT, albumin, electrolytes, glucose to assess for complications

DIFFERENTIAL DIAGNOSES:

1. Drug-induced hepatitis
2. Alcoholic hepatitis
3. Hepatic malignancy , mostly secondaries
4. Pre- and post-hepatic causes of jaundice
5. Typhoid fever, malaria and other acute febrile illnesses
6. Tuberculosis involving the liver

TREATMENT (9, 25)

- Is mainly supportive; no specific pharmacologic therapy is required or available.
- Usually treatment as outpatient is adequate
- High-calorie diet with some restriction on protein and fat intake is recommended.
- Role of multivitamin supplementation is controversial but may be helpful; all other drugs should be avoided as they may aggravate the hepatic damage.
- The patient should rest until general condition improves
- Educate patient and family regarding proper hygiene

- Patients with suspected coagulation defects, disturbances of fluid and electrolyte and/or acid-base balances, impaired renal function or, in general, severe hepatitis should be referred for better management.

CONTROL MEASURES

- Proper sanitation and hygiene
- Passive (immune globulin) and active immunization have been developed for hepatitis A virus. It is recommended for close contacts of cases and workers in institutions with multiple cases.

C. PARASITIC FOOD-BORNE DISEASES

I. AMEBIASIS

PATHOGENESIS:

- Infection occurs following ingestion of cysts from water, food or hands contaminated with feces.
- Motile trophozoites are released from the cysts in the small intestinal lumen
- The trophozoites remain as commensals in most hosts
- In some, however, they cause invasion and ulceration of the large bowel mucosa or enter the bloodstream to cause distant infections in organs like the liver, lungs or the brain.
- Various products and their cytopathic effect contribute to the tissue damage, which they cause.
- Rarely, intestinal infection leads to the formation of a mass lesion called ameboma (9)

CLINICAL FEATURES:

a. Intestinal Amebiasis:

- Most patients with intestinal Amebiasis are just asymptomatic cyst passers. They do not have symptoms but are sources of infection for others.
- Symptomatic intestinal Amebiasis:
 - The incubation period is 2-6 weeks

- Clinical features include gradual onset of lower abdominal pain, mild diarrhea followed by malaise, weight loss, diffuse lower abdominal or back pain and more severe bloody diarrhea (9, 25)
- Severe infection manifests with passage of many bloody stools per day (10-12 times), severe abdominal pain, high fever
- Patients with ameboma present with an asymptomatic or tender abdominal mass.
- Some patients develop a chronic form of the disease (9)

b. Amebic Liver Abscess:

- The liver is the most common site of extra-intestinal Amebiasis
- Most patients have fever, right-upper quadrant pain (dull or pleuritic), point tenderness over the liver and right-sided pleural effusion (common), jaundice (rare) (9)
- Fewer than 30 % of patients have active diarrhea (9)
- Patients from endemic areas may present with prolonged fever, weight loss and hepatomegaly
- 10-15 % of patients present only with fever.

DIAGNOSIS

1. Clinical features

2. Laboratory diagnosis:

- **Intestinal disease**

- Stool microscopy to see hematophagous trophozoites of *E. histolytica* (it is important to examine at least 2 more fresh stool specimens if the first specimen is negative) is the gold-standard diagnostic test. In addition, evidence of gastrointestinal bleeding and few or absent neutrophils may be found.

- **Amebomas** are usually first identified using barium studies. Sigmoidoscopy and biopsy may be done in higher centers for confirmation.

- **Amebic liver disease:**

- Patients may have leukocytosis, anemia, normal or minimally elevated liver enzyme levels, and elevated alkaline phosphatase level.
- Stool examination may or may not yield trophozoites and/or cysts
- Serology is a very useful tool but mostly unavailable
- Imaging studies, particularly ultrasonography, are helpful to visualize the hepatic lesions.

TREATMENT (9, 24)

- **General measures:**

- Supportive care and symptomatic therapy including analgesic-antipyretics, replacement of fluid loss, and proper nutrition.

- **Drug therapy:**

- See annex V
- Treatment of asymptomatic cyst carriage is of questionable role in Ethiopia because of the high prevalence of this infection; however, if there is decision to treat, it has to be remembered that metronidazole or tinidazole are not effective.

- **Other therapeutic interventions:**

- Patients with amebic liver abscess may need percutaneous aspiration. Thus, all patients suspected of having an amebic liver abscess should be referred to a hospital.

CONTROL MEASURES

- Avoidance of eating raw fruits and vegetables
- Proper disposal of human waste
- Personal hygiene (hand washing)

II. GIARDIASIS

PATHOGENESIS:

- Following ingestion, the cysts excyst in the small intestine.
- The released trophozoites multiply and remain localized to the small intestine

- They adhere to the intestinal epithelium, but do not cause any invasion.
- The exact mechanism by which *G. lamblia* causes diarrhea is not clear. (9)

CLINICAL FEATURES:

- Giardiasis may be asymptomatic or a serious disease with severe complications.
- Most infected persons are asymptomatic
- The incubation period is 1-3 weeks
- Symptoms of acute giardiasis may start suddenly or gradually and include diarrhea, abdominal pain, bloating, belching, flatus, nausea and vomiting.
- Patients with chronic giardiasis manifest with less severe diarrhea, but increased flatus, loose stools, weight loss, etc. These symptoms may be episodic or continuous and may last for years.
- The disease can be severe, leading to malabsorption, weight loss, growth restriction in children, and dehydration. (9, 25)

DIAGNOSIS:

1. Clinical features

2. Laboratory diagnosis:

- Depends on the identification of cysts in the feces or of trophozoites in the feces. Direct and concentration examination techniques should be used. Repeated examination of the stool may be necessary since cyst excretion is variable and may be undetectable at times.
- Tests for parasite antigens have been developed but generally are unavailable in Ethiopia.

TREATMENT:

1. **General measures:**

- Supportive and symptomatic therapy such as fluid replacement

2. **Drug therapy:**

- **See annex V**

CONTROL MEASURES

- Similar to those of Amebiasis

III. ASCARIASIS

PATHOGENESIS (LIFE CYCLE):

- Ascarid eggs released in the feces become infective after several weeks of maturation in the soil and can remain infective for years
- Following swallowing in contaminated food, etc. the eggs release, in the intestine, larvae which invade the mucosa
- The larvae migrate in the blood to the lungs where they enter the alveoli, go up the bronchial tree, and are swallowed back into the gastrointestinal tract to reach the small intestine. This larval migration occurs about 9-12 days after ingestion of eggs.
- In the small intestine, they develop into adult worms, which live in the host for 1-2 years.
- The mature female in the small intestine produces hundreds of thousands of eggs, which are released into the environment with feces (9)

CLINICAL FEATURES:

Larval migration phase:

- Patients may have irritating nonproductive cough, substernal burning discomfort worsened during coughing or sighing, dyspnea, blood-tinged sputum, and they also commonly have fever.

Adult worms in the small intestine:

- Adult worms in the small intestine are usually asymptomatic.
- Heavy infections may lead to pain and small intestinal obstruction due to formation of large bolus of worms. This may sometimes be complicated by perforation, intussusception, or volvulus.
- If single worms migrate to ectopic sites, they may produce different manifestations, such as biliary colic, cholecystitis, cholangitis, intrahepatic abscess (migration into the biliary tract), coughing and oral expulsion of worms (migration up the esophagus).
- Intestinal and biliary ascariasis can mimic acute abdomen (9)

DIAGNOSIS:

- Most cases are readily diagnosed through the detection of the characteristic eggs in the stool microscopically.
- Some patients present following the passage of adult worms in the stool.
- During the early phase of larval migration in the lungs:
 - Larvae may be found in sputum or gastric aspirates
 - There is usually prominent eosinophilia
 - Chest X-ray may reveal evidence of eosinophilic pneumonitis, with oval or round infiltrates a few millimeters to several centimeters in size.
- Imaging studies such as plain abdominal films or contrast studies of the gastrointestinal tract sometimes accidentally detect the adult worms (9)

TREATMENT (9, 24)

- Drug treatment
 - See annex V

N.B.

* Mebendazole and albendazole are contraindicated during pregnancy, especially in the first trimester; however, pyrantel pamoate and piperazine are safe.

- Patients with partial intestinal obstruction secondary to bolus of ascaris should be managed with:
 - Nasogastric suction
 - Intravenous fluid administration
 - Instillation of piperazine through nasogastric tube
- Patients with complete intestinal obstruction may need immediate surgical intervention, and thus need an urgent referral.

CONTROL MEASURES:

- Proper disposal of human excrement
- Treatment of cases
- Mass deworming

- **TAPEWORM INFECTION**

a. TAENIASIS:

PATHOGENESIS:

- Adult worms of both *T. saginata* and *T. solium* reside in the upper jejunum.
- They have thousands of proglottids, each of which produces many eggs.
- The eggs and proglottids are released into the environment with stool
- The eggs of *T. saginata* live on vegetation for months to years until cattle ingest them. Those of *T. solium* are infective for both humans and animals.
- After ingestion by intermediate hosts, the eggs embryonate, penetrate the intestinal wall, and are carried to many tissues with a predilection for striated muscles. The embryos in the striated muscle become transformed into cysticerci.
- Ingestion of the cysticerci in raw or undercooked beef (*T. saginata*) or pork (*T. solium*) leads to human intestinal infection.
- Infections that cause human cysticercosis follow the ingestion of *T. solium* eggs, mostly from food contaminated with feces, or auto infection from eggs produced in the intestine.

CLINICAL FEATURES:

- The infection may be asymptomatic
- Passage of proglottids in the feces which may be accompanied by the passage of perianal discomfort
- Mild abdominal pain, nausea, anorexia, weakness, weight loss
- Infection with *T. solium* may manifest with epigastric discomfort, nausea, hunger sensation, diarrhea, and weight loss.

DIAGNOSIS:

1. Clinical features
2. Laboratory diagnosis-
 - Identification of eggs or proglottids in the stool; use of scotch-tape may be helpful as in pinworm infection as the eggs are sometimes present in the perianal area. Distinguishing *T. saginata* from *T. solium* requires examination of mature proglottids or the scolex.
 - Patients may have eosinophilia.

TREATMENT:

- See annex V

CONTROL MEASURES:

- Avoidance of eating raw or undercooked meat
- Good personal hygiene to prevent the auto-infection seen in *T. solium* infection
- Sanitary disposal of human excreta

b. CYSTICERCOSIS:**CLINICAL FEATURES:**

- Since cysticerci may be found anywhere in the body, the clinical features vary depending on their localization and their number.
- The most common sites are the brain and skeletal muscle.
- Cysticerci in the brain produce manifestations of intracranial space-occupying lesions, including seizures and increased intracranial pressure, or they may also cause chronic meningitis or stroke.

DIAGNOSIS AND TREATMENT:

- The diagnosis can be very difficult, and may need expensive imaging techniques. All patients suspected of having cysticercosis should be referred to higher centers for better diagnosis and management.

3.1.4.2. FOOD POISONINGS/INTOXICATIONS

A. BACTERIAL FOOD POISONING:

a. PREDOMINANT GASTROINTESTINAL MANIFESTATIONS

A very useful approach to evaluating diarrhea is distinguishing inflammatory from non-inflammatory diarrhea (see ANNEX IV).

PATHOGENESIS:

- ***Staphylococcus aureus*** enterotoxins act on receptors in gut that transmit impulse to medullary centers through the vagus to induce vomiting; they may also act as superantigens.
- ***Bacillus cereus*** enterotoxins formed in food or in gut from the growth of the organism induce vomiting and diarrhea.
- ***Clostridium perfringens*** enterotoxins produced during sporulation in gut cause hypersecretion. The spores are able to survive cooking, and if the cooked food (meat and poultry) is not cooled enough, they will germinate. The pH change from the stomach to the intestine is believed to induce sporulation.
- **Enterotoxigenic *Escherichia coli*** strains produce heat-labile (LT) and heat-stable (ST) enterotoxins, which cause hypersecretion in the small intestine.
- **Enteropathogenic *Escherichia coli*** strains cause mucoid diarrhea through an effect on the intestine referred to as the “effacing lesion” (effacement of microvilli) (9, 23)
- **Enterohemorrhagic *Escherichia coli* O157:H7** produces shiga-like toxin.

CLINICAL FEATURES (See Annex III):

- **Staphylococcal food poisoning** produces, after an incubation period of 1-8 hours (rarely up to 18 hours), abrupt onset of intense vomiting accompanied by watery diarrhea and crampy abdominal pain for up to 24 hours followed by recovery in 24-48 hours
- ***B. cereus* poisoning** mainly manifests with vomiting following incubation period of 2-8 hours (emetic form); or mainly diarrhea following incubation period of 8-16 hours (diarrheal form)

- **Cl. perfringens poisoning** manifests with abrupt onset of profuse diarrhea after an incubation period of 8-16 hours; vomiting is occasional; without treatment, recovery usually takes 1-4 days
- **Enterotoxigenic strains of E. coli** produce abrupt onset of diarrhea after an incubation period of 24-72 hours; vomiting is rare; in adults the infection is usually self-limited, lasting only for 1-3 days
- **Enteropathogenic strains** produce watery-mucoid diarrhea following an incubation period of 1-2 days; the disease is usually self-limiting, but may sometimes persist for weeks (9)
- **Enterohemorrhagic strains** produce bloody diarrhea which resembles shigellosis.

DIAGNOSIS:

- Many cases of non-inflammatory diarrhea are self-limited and can be treated empirically; the determination of a specific etiology is of very little significance in the clinical management; thus one can proceed to treatment using the information obtained from history, assessment of level of dehydration, and stool examination.
- In cases of common-source outbreaks, attempt should be made to isolate the responsible organism from the suspected food item(s).

TREATMENT: can be based upon the clinical features (9)

- If the patient has watery diarrhea (no blood in stool, no fever, no distressing abdominal pain, no fecal leukocytes), and
 - If he/she has only 1-2 loose stools per day with minimal discomfort, consider only oral fluids.
 - If he/she has several loose stools per day with distressing symptoms, consider an antibacterial drug such as:
 - o Trimethoprim-sulfamethoxazole 160/800 mg PO bid for 3 days; for children, 4/20 mg po bid for 3 days
 - o Ciprofloxacin 500 mg PO bid for same duration
 - o Norfloxacin 400 mg PO bid for same duration

- If the patient has dysentery or inflammatory diarrhea or fever, investigate and manage accordingly (the patient may have shigellosis, intestinal amebiasis, or typhoid fever, etc.).

b. BOTULISM

PATHOGENESIS:

- Botulism is a paralytic disease produced by the potent neurotoxin of *Clostridium botulinum*.
- There are four recognized clinical forms:
 - Food-borne botulism results from ingestion of preformed toxin in contaminated food.
 - Wound botulism results from toxin produced in contaminated wound.
 - Infant botulism and adult infectious botulism follow ingestion of spores and production of toxin in the intestine
- The activity of the neurotoxin involves several steps culminating in the proteolysis of the components of the neuroexocytosis apparatus curtailing the release of acetylcholine at the myoneural junction. The end result of this is paralysis (9).

CLINICAL FEATURES (9):

Infant botulism:

- This is the most common form of botulism
- The severity ranges from a mild transient illness to a fatal paralytic one.
- Constipation is usually the early sign
- The infant may show loss of head control, loss of sucking ability and of facial expression and verbalization
- Symmetric descending paralysis, with initial cranial nerve involvement, is then noted
- Deep tendon reflexes may be diminished or absent
- The infant is typically afebrile

Food-borne botulism:

- Here also, the illness can vary from a mild illness not needing medical attention to a rapidly fatal severe disease
- The incubation period is usually 18-36 hours

- Cranial nerve involvement is the usual initial manifestation, manifesting with diplopia, dysarthria, and/or dysphagia, depressed gag reflex
- Symmetric descending weakness/paralysis progresses usually rapidly to involve the neck, arms, thorax, and legs; the weakness is occasionally asymmetric
- Non-specific symptoms of nausea, vomiting, abdominal pain, dizziness, blurred vision, dry mouth and dry-sore throat may precede or follow the onset of paralysis
- Paralytic ileus, severe constipation and urinary retention are common
- Patients are typically alert, with no fever; they may sometimes be drowsy or anxious
- Most have ptosis; depressed papillary reflexes may be present
- Half of patient have fixed or dilated pupils
- The deep tendon reflexes may be normal or suppressed

Adult infectious botulism:

- The mechanism of acquisition of the toxin is similar to that of infant botulism
- Clinical features are similar

DIAGNOSIS (9, 23)

- Clinical features-if patient presents with symmetric descending paralysis without sensory findings and without fever, the diagnosis of botulism should be suspected.
- The demonstration of the organism or its toxin in intestinal secretions (vomit, stool) strongly suggests the diagnosis. However, due to limitation of facilities, one has to base presumptive diagnosis on clinical features.

DIFFERENTIAL DIAGNOSES:

- Poliomyelitis
- Diphtheria
- Chemical intoxications
- Myasthenia gravis
- Guillain-Barre syndrome

TREATMENT:

- Any patient suspected of having botulism should be immediately referred to a higher center for proper management, since treatment should be given as inpatient in a facility with the maximal monitoring capabilities, especially for respiratory support.

CONTROL MEASURES:

Educate the public:

- To cook food well (boiling for 10 minutes or cooking at 80°C for 30 minutes can destroy the toxin)
- To avoid honey in the first year of life, as this has been identified to be one of the food items commonly associated with infant botulism.
- Not to eat or taste food from bulging cans

B. CHEMICAL POISONINGS:

a. HEAVY METAL POISONING (LEAD, MERCURY, ARSENIC)

DIAGNOSIS:

- History of exposure
- Consistent clinical features
- Laboratory diagnosis:-determination of serum level (this is available only in few centers to which patients suspected of having these poisonings should be referred)

MANAGEMENT:

- Termination of exposure
- GI decontamination using induction of emesis, gastric lavage, activated charcoal administration
- Use of chelating agents, e.g. , edetate calcium disodium, dimercaprol (9)
- Referral for further management

b. INSECTICIDES:

The most important of these are organophosphates.

DIAGNOSIS:

- History of exposure
- Clinical features
- Laboratory investigations for specific diagnosis are not available in most Ethiopian settings

MANAGEMENT:

- Termination of exposure and decontamination-remove all contaminated clothing, and wash the skin with soap and water
- Use charcoal to decontaminate the GIT

- Supportive measures:
 - Oxygen administration
 - Ventilatory support
 - Treatment of symptoms, such as seizures with benzodiazepines
- Antidote therapy: atropine 0.5-2 mg IV every 5-15 minutes until bronchial and other secretions have dried (9)

N.B. Use of atropine is not effective in reversing the CNS effects.

C. POISONOUS PLANTS:

a. NEUROLATHYRISM (LATHYRISM)/ “Guaya”:

DIAGNOSIS:

- History of exposure in the form of prolonged consumption of “guaya”
- Clinical features, mainly consisting of spastic paraplegia, sphincter dysfunctions, and sensory disturbances.

TREATMENT:

- The treatment is almost exclusively supportive and patients suspected of having this condition should be referred to health facilities with better supportive facilities (25)

b. MUSHROOM POISONING:

DIANGOSIS:

- History of ingestion of mushroom
- Clinical features:
 - The usual initial clinical features are nausea, vomiting, diarrhea and abdominal cramps
 - The subsequent manifestations vary depending on the mushroom group involved as well as the amount ingested
 - In addition, some mushrooms are known to produce gastritis and others produce hepatitis.
- For detailed discussion of the manifestations that may result from the ingestion of different types of mushroom, please refer to standard textbooks

TREATMENT:

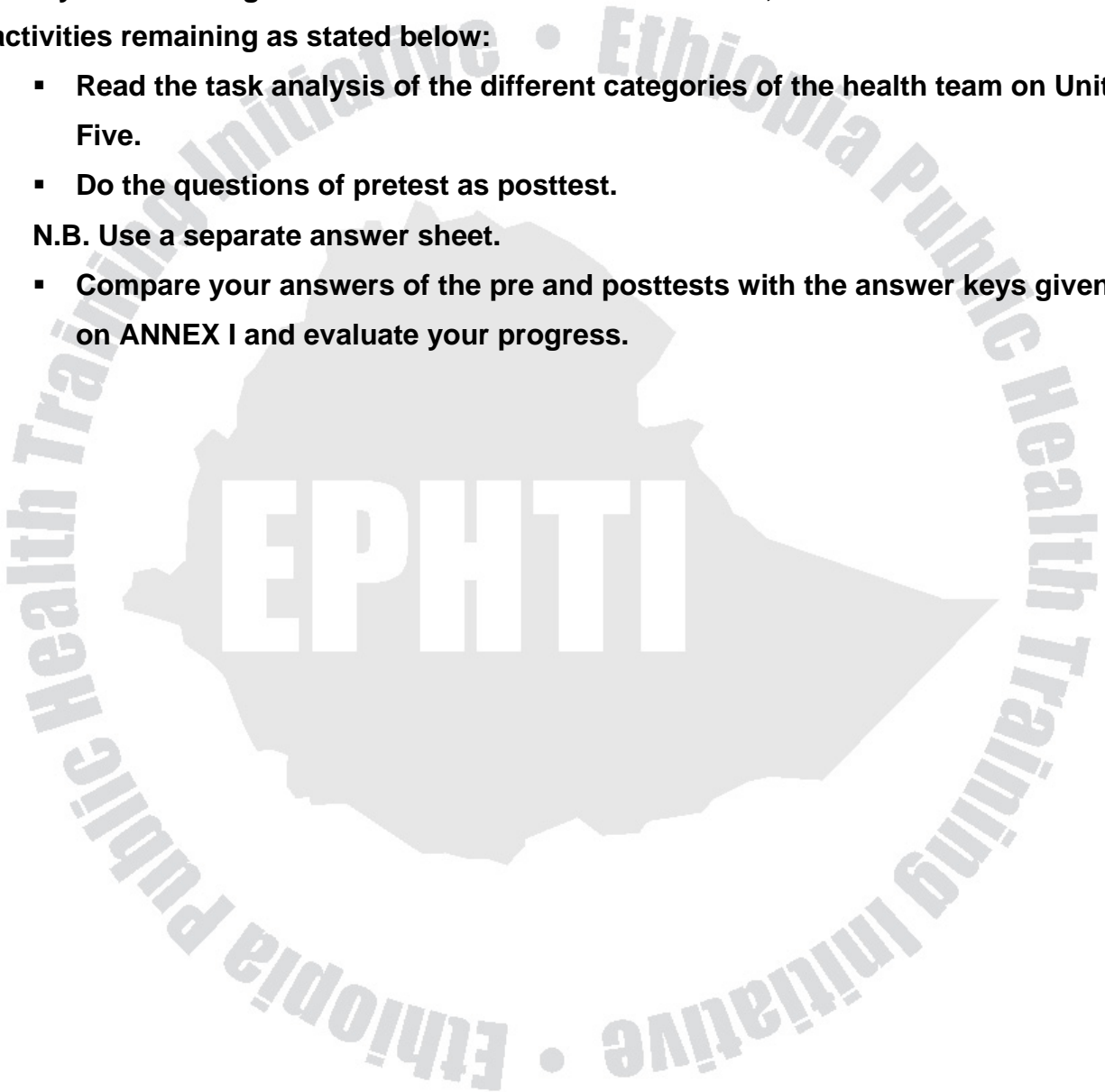
- Activated charcoal for gastrointestinal decontamination
 - Intensive supportive care
-

Now you are through with the core and satellite modules, but there are still some activities remaining as stated below:

- Read the task analysis of the different categories of the health team on Unit Five.
- Do the questions of pretest as posttest.

N.B. Use a separate answer sheet.

- Compare your answers of the pre and posttests with the answer keys given on ANNEX I and evaluate your progress.



3.2. SATELLITE MODULE FOR NURSES

3.2.1. DIRECTIONS FOR USING THE MODULE

- Before reading this satellite module be sure that you have completed the pre – test and studied the core module.
- Read this satellite module
- Refer to the core module if required

3.2.2. LEARNING OBJECTIVES

A. General

After completing this module the learner will be able to assess and manage cases of food borne disease.

B. Specific

After reading this module you will be able to:

- Assess the patient with food borne disease
- Make the Nursing diagnosis
- Plan the Nursing intervention
- Implement the planned intervention
- Evaluate the outcomes of the intervention

3.2.3. MANAGEMENT OF PATIENTS WITH FOOD BORNE DISEASE

Nursing assessment of the patient with food-borne disease makes use of subjective and objective data.

A. Nursing Assessment of the Patient with Food Borne Disease

i. Subjective Data

- Onset and duration of the disease (14)
- History of ingestion of contaminated food (food with unusual odor or taste, uncooked vegetables, raw meat etc.) (14,28)

- Did anyone else become ill from eating the same food?
- Ask about the health of other family members. (14)
- Sign and symptoms of the disease that the patient reports. (14,28)
- History of ingestion of foods known to have natural toxins or fungal toxins. (14)
- History of ingestion of food sources possibly contaminated by insecticides / pesticides and heavy metals. (11,9)

ii. Objective Data

- Patient's appearance
- Assess vital signs, sensorium, Central Venous Pressure (CVP) if indicated, and muscular activities.
- Weigh the patient.
- Slight abdominal distension (14)
- Signs of dehydration – dry mucus membrane, poor skin turgor.
- Inspection of the stool – consistency, color, odor.
- Results of the diagnostic tests - from samples of food, gastric contents, vomitus, serum and stool (28).

B. Nursing Diagnosis

Based on the classification of the food borne diseases and findings of the nursing assessment the following actual and potential nursing diagnosis can be made:

i. Actual Nursing Diagnosis

- A. Poisoning related to the ingestion of contaminated food with chemical poisons, poisonous plants and toxins. (4)
- B. Pain related to the diseases process
- C. Altered bowel elimination related to the disease process
- D. Altered nutrition: less than body requirements related to anorexia, vomiting and diarrhea (28).
- E. Anxiety related to frequent, uncontrolled elimination
- F. Knowledge deficit about possible causes of the disease and preventive measures related to lack of information. (14)

ii. Potential Nursing Diagnosis

- A. Risk for fluid volume deficit related to vomiting and increased loss of fluids and electrolytes from gastro-intestinal tract. (14,28)
- B. High risk for spreading of the infection to others. (14)

C. Planning the Nursing Intervention

To plan the nursing intervention:

1. Set a priority

- Consider urgency of the problem
- Give priority to physical needs of the patient

2. Establish goals for the nursing intervention

- To remove or inactivate the poison before it is absorbed.
- Relief pain
- Regain normal bowel elimination patterns
- Attain an optimal level of nutrition
- Reduce anxiety
- Increase patient understanding about possible causes of the disease & preventive measures
- Maintain fluid balance
- Prevent the spreading of the infection to others (14,28)

3. Establish expected outcomes

The patient:

- Reveals reduced/ no effects of the poisoning chemical, poisonous plant or toxins
- Reports less pain
- Reports a decrease in the frequency of diarrheal stools
- Tolerates small frequent feeding
- Verbalizes concerns and fears
- Reports the different causes and preventive measures of food borne disease

- Has no observable signs and symptoms of fluid balance
- Prevents spread of the infection to others

D. Nursing intervention /implementation

1. Reducing / eliminating the effects of the poisonous chemical, poisonous plant or toxins

- Attain control of the air way, ventilation, and oxygenation
 - Prepare for mechanical ventilation if respirations are depressed.
 - Administer oxygen for respiratory depression, unconsciousness, cyanosis, and shock.
 - Prevent aspiration of gastric contents by positioning
 - Insert an indwelling urinary catheter to monitor renal function.
 - Obtain blood specimen to test for concentration of the poison
 - Monitor neurologic status (including cognitive function)
 - Conduct a rapid physical examination.
 - Monitor vital signs
- Treat shock
- Remove the toxin or decrease its absorption. Use gastric emptying procedures as; the following may be used:
 - Syrup of ipecac to induce vomiting in the alert patient. (Do not induce vomiting after ingestion of caustic substances or petroleum distillates).
 - Gastric lavage. Save gastric aspirate for toxicology screens.
 - Activated charcoal administration if poison is one that is absorbed by charcoal.
 - Cathartic, when appropriate.
- Give specific therapy. Administer the specific chemical antagonist or physiologic antagonist as early as possible to reverse or diminish effects of the toxin.
- Support the patient having seizures. Poisons may excite the central nervous system or the patient may have seizures from oxygen deprivation.

- Carry out procedures, if indicated, to promote the removal of the ingested substance if the above are not effective:
 - Diuresis for agents excreted by the renal route
 - Dialysis
 - Hemoperfusion (process of passing blood through an extracorporeal circuit and a cartridge containing an adsorbent [such as charcoal or resins], after which the detoxified blood is returned to the patient).
 - Multiple doses of charcoal
- Monitor central venous pressure as indicated
- Monitor for fluid and electrolyte balance.
- Reduce elevated temperature (14)

2. Measures to Relief Pain

To ease anal irritation (pains) caused by diarrhea, clean the area carefully and apply a repellent cream, such as petroleum jelly, warm sitz baths and application of witch hazel compresses can also soothe irritation. (28)

3. Establishing a Regular Pattern of Bowel Elimination and Maintaining Nutritional Balance

- Administer medications, as ordered, correlate dosages and routes with the patient's meals and activities.
- Control nausea
 - Administer an anti-emetic medication (give 30 to 60 minutes before meals)
 - Give sips of weak tea, carbonated drinks, or tap water for mild nausea.
 - Give clear liquids 12 to 24 hours after nausea and vomiting subsides.
 - Gradually progresses to a low residue, bland diet i.e. advice the patient to avoid food products with a cellulose or hemi cellulose base (nuts, seeds).
- Vary the diet to make eating more enjoyable, and allow some choice of foods.
- During an episode of acute diarrhea encourage the patients to rest in bed and take liquids and foods that are low in bulk.

- Limit caffeine and carbonated beverage intake because these stimulate intestinal motility.
- Very hot and very cold foods should be avoided.
- Milk and milk products, fat, whole grain products, fresh fruits, and vegetables may be restricted for several days.
- Monitor fluid status carefully. Take vital signs at least every 4 hours, weigh the patient daily, and record intake and output.
- Watch for signs of dehydration, such as dry skin and mucous membranes, fever, and sunken eyes.
- If dehydration occurs, administer oral and I.V. fluids. If necessary, a potassium supplement may be added to the I.V. solution. If the patient is receiving a potassium supplement, be especially alert for the development of hyperkalemia (14,28,29).

4. Reducing Anxiety

- An opportunity is provided for the patient to express fears and worry about being embarrassed by lack of control over bowel elimination. This fear of embarrassment is often a major concern.
- The patient is assisted to identify irritating foods and stressors that precipitate an episode of diarrhea. Eliminating or reducing these factors helps control defecation. The patient is encouraged to be sensitive to body clues that warn of impending urgency (abdominal cramping, hyperactive bowel sounds). Special absorbent underwear, which will protect clothes if there is accidental fecal discharge, may be helpful.
- An understanding, tolerant, and relaxed demeanor on the part of the nurse is essential. The patient's efforts to use coping mechanisms are supported and encouraged.

5. Teaching about Possible Causes of the Disease and Preventive Measures

- Teach the patient about his or her specific disease and therapeutic regimens. She or he is instructed about personal hygiene and the maintenance of the home environment to prevent the disease. Teach also about proper storage of the food items, chemicals, insecticides/ pesticides, detergents and petroleum products brought to home for household purposes. Instruct the patient to thoroughly cook foods, to properly preserve perishable foods, to always wash his hands with water and soap before handling food, especially after using the bath room toilet, to clean utensils thoroughly, and to eliminate flies and roaches in the home.
- Inform the family about the disease problem and how they can seek additional health care. (14,28,29)

6. Avoiding Fluid Volume Deficit

To prevent fluid volume deficit maintaining fluid balance is important. But fluid balance is difficult to maintain during an acute episode of the disease because the feces are propelled through the intestines too quickly to allow for water absorption; and vomiting that leads to water loss; output exceeds intake. When a patient experiences such a condition the nurse assesses for dehydration (decreased skin turgor, tachycardia, weak pulse, decreased serum sodium, thirst) and keeps an accurate record of intake and output. Urine specific gravity can be monitored to assess hydration status. The patient is weighed daily. The nurse encourages oral fluid replacement in the form of water, juice, and commercial preparations. Parenteral fluids are administered as necessary. (14)

7. Preventing the Spread of the Disease to Others

- To prevent the spread of the infection wash your hands thoroughly after giving care (see figure 3.2.1), and use blood and body fluid precautions whenever handling vomitus or stools. In general all patients with such disease should be treated as potentially infectious until they are proven to be otherwise.

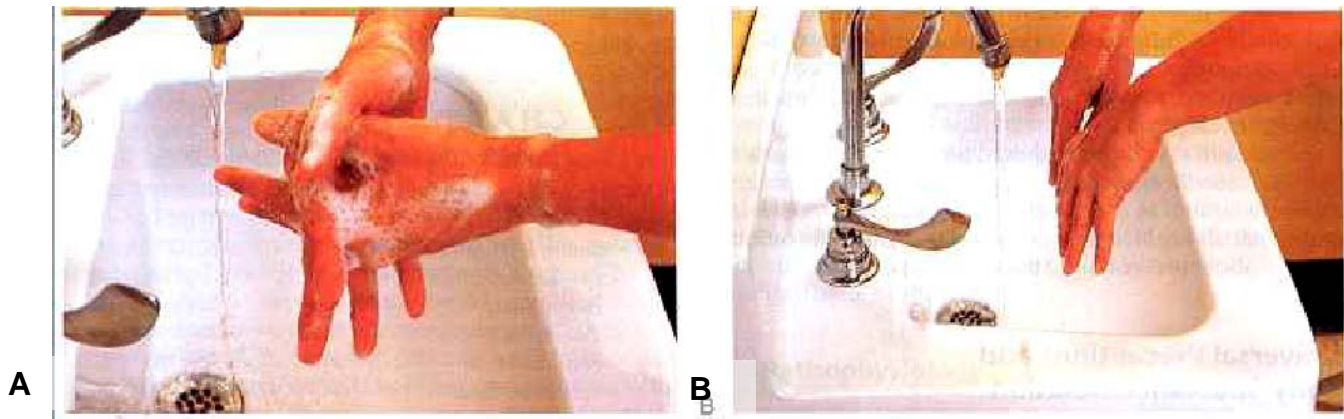


Figure 3.2.1 Effective hand washing. (A) Washing hands and forearms with firm rubbing and circular motions. (B) Rinsing thoroughly. (Adopted from Brunner and Suddarth's Text Book of Medical-Surgical Nursing).

- Gloves must be used when handling any body fluid from the patient. Gloves must be changed between patient care activities and hands must be washed after gloves are removed .
- To prevent patient-to-patient infection spread provide isolation according to the general rule of body substance isolation, or individual institution adaptation of isolation. Ensure that patients with highly transmissible organisms are physically separated from other patients if hygiene or institutional policy dictates.
- Teach the patient and family about means of preventing the spread within the home. (14,28)

E. Potential Complications

Based on the assessment data, a potential complication is cardiac dysrhythmia related to electrolyte depletion.

Monitoring and managing Potential Complications

Serum electrolyte levels are monitored daily. Vitals signs, including apical pulse and changes in tendon reflexes and muscle strength, are monitored frequently.

Electrolyte replacements can be made. Evidence of dysrhythmias or a change in the level of consciousness is reported immediately. (14)

F. Evaluation

Evaluate nursing intervention based on the outcome criteria setted in section 3.2.3 under planning the nursing intervention (under C).

G. Treatment of Specific Food-Borne Diseases

Food-borne diseases for which their specific chemotherapy is not indicated in this section please refer annex-v

i. Food-borne infections

Apart from the chemotherapy management of food-borne infections include fluid and electrolyte replacement. Supportive care and rest are particularly the corner stone of management for viral infections.

ii. Food poisonings/intoxications

➤ **Bacterial food poisoning:**

a. Staphylococcal food poisoning

- Fluid replacement and close observation
- Antibiotics are rarely used

b. Botulism

- Penicillin should be given to eradicate *Clostridium botulinum* from the site, even though the benefit of this therapy is unproven

➤ **Chemical poisoning:**

a. Heavy metals

- Terminate exposure
- Use chelating agents

b. Insecticide poisoning (organophosphates and carbamate ingestion)

- Use activated charcoal
- Supportive measures:
 - Oxygenation

- Ventilatory assistance
- Treat seizure
- Atropinization: 0.5-2 mg IV every 5-15 minutes until bronchial and other secretions have dried

c. Poisonous plants

1. Mushroom poisoning:

- Gastric emesis with ipecac
- Decontamination with activated charcoal with sorbitol for catharsis
- Atropine
- Withdraw ingestion of poisonous plants
- Supportive therapy

2. Neurolathyrism (lathyrism):

- Withdraw ingestion of the plant
- Supportive therapy (9,11,12)

3. Fungal toxins

▪ Aflatoxins:

- Treatment in Hepatocellular carcinoma includes drugs 5- fluorouracil and mitomycin, and surgery.

▪ Ergot toxin:

- Refer standard text books

3.3. SATELLITE MODULE FOR ENVIRONMENTAL HEALTH OFFICERS

3.3.1 Directions for Using the Module

- Make sure you have thoroughly read the core module before you begin to read this satellite module.
- Read through this satellite module carefully.

3.3.2 Learning Objectives

By the end of learning this satellite module the reader will be able to:

1. Explain the basic principles of food sanitation
2. Describe the transfer of contamination in food borne diseases
3. Discuss the different sources for food borne diseases.
4. Identify factors leading to food borne disease outbreaks.
5. Design prevention and control measures of food borne diseases.

3.3.3 Learning Activity

Case study

There is a busy cafeteria at a boarding school in the town of Bullhawo. The boarding school accommodates over 1200 students; and all are served in this cafeteria. The cafeteria is located in front of the students' dormitories in about a 50 meter distance. In most cases the direction of the wind blow is from the dormitories to the cafeteria. The dormitories harbor toilets with a water flush design but as water is scarce it is common to observe piles of human excreta with a buzzing population of flies feeding on the excreta. The campus compound, though has some trees, is dusty. The problem of water is alleviated by fetching water with trailer tankers from bore holes at a distance of about 20 KM. The water then is filled, for storage, to open barrels or narrow mouthed jerrycans with plastic hoses pulled over the floors in the kitchen of the cafeteria. The cafeteria lacks adequate dishes but this is compromised by rotating the utensils to serve more students. During this rotation the dishes are simply rinsed in a bowl of water before they are given to the next user in the queue. However, after a session of service the utensils are finally washed for the next session in a three-compartment manual dish washing system filled with cold water and at the first compartment having detergents.

The dishes are placed to drip and dry in perforated plastic racks placed on the floor for ease of sliding over the floor. The floor of the kitchen is rough and usually wet. However, it is frequently cleaned to drain but not usually mopped, as this is a tedious task.

The number of workers in the kitchen and cafeteria is enough to manage the required service. However, most of them are with low skills and they have been on the job for long period. Despite this fact the management of the boarding school is not prepared to train them on proper food handling assuming that they have the experience and the training requires additional cost.

The wastes including garbage from the kitchen and the cafeteria are given to pigs that scavenge around these facilities. The sewage drains to underground sewers but there is frequent blockage that leads at times to overflow. The overflows facilitate growth of green grasses surrounding the cafeteria. Moreover, this wastewater is used to water vegetables planted in the backyard. It is common to smell odors arising from the garbage and the wastewater. This is not given much attention by the school management as they consider it to be normal to kitchens and cafeterias.

The campus clinic record shows that most students come with complaints of diarrhea. The clinic head reports that mass diarrhea complaints are commonly observed but are usually not serious. As the clinic is so busy, the staffs have no time to visit the cafeteria. In addition, the head of the clinic believes that giving proper care to the sick is easier and better than wasting time assessing the cafeteria.

Questions related to the above case study

1. Based on the case study, make an assessment of the overall sanitation of the cafeteria?
2. What do you think are the potential sources of food contamination in this cafeteria?
3. How do you evaluate the dish handling and washing practice?
4. Do you think training of food handlers can address any problem related to food hygiene in the cafeteria? If so, discuss on some of them.

5. Are the toilets of the students' dormitories of any threat to the food hygiene in the cafeteria? How?
6. Discuss the presence of pigs in relation to the food hygiene of the cafeteria.
7. What do you feel about the attitude of the management about training of food handlers?
8. Do you have any recommendations with regards to growing of vegetables in the above case study?
9. Is the clinic addressing the diarrhea problem appropriately? If not, what other things should be done?
10. Do you believe medical certification of food handlers that will be renewed every 6 months plays an important role in reducing food borne diseases?

3.3.4 The Basic Principles of Food Sanitation

The word sanitation is derived from the Latin word “sanitas”, meaning “health”. Applied to the food industry, sanitation is “the creation and maintenance of hygienic and healthful conditions”. A sanitation program is “a planned way of practicing sanitation”. It results in a number of crucial benefits for the public and the businesses. Most owners or managers of food facilities want a clean operation. Sanitation is the application of a science: to provide wholesome food handled in a clean environment by healthy food handlers, to prevent contamination with microorganisms or toxic chemicals that cause food borne illness, and to minimize the proliferation of food spoilage microorganisms. Effective sanitation refers to the mechanisms that help accomplish these goals (7). However, unsanitary operations frequently result from a lack of understanding of the principles of sanitation and the benefits that effective sanitation will provide (7). Because of lack of awareness on issues of sanitation food borne diseases are among the major health problems in Ethiopia.

Food borne diseases are not limited to the activities of microbes or their products. Food borne diseases can also be caused by a variety of chemicals that may lead to illness and deaths of people who may have consume foods contaminated by these chemicals. The course of action of chemicals depends on the type, dose and their concentration.

An effective program of food sanitation includes the following benefits. To mention some:

- a. Reduced Public health risks
- b. Improved product shelf life
- c. Improved customer relations
- d. Improved product acceptability
- e. Increased trust of compliance agencies and inspectors.
- f. Decreased product salvaging, and
- g. Improved employee morale in food catering establishments.

Food sanitation is an applied sanitary science related to the production, harvesting, storage, distribution/ transport, processing, preparation, and handling of food. Sanitation applications refer to hygienic practices designed to maintain a clean and wholesome environment for food production, preparation, and storage. Sanitation is equated with more than cleanliness. This applied science relates to the physical, chemical, and biological factors that constitute the environment. (7).

The basic principles for food sanitation to control food borne illnesses and outbreaks can be summarized to three essential activities:

- Prevention of contamination of the food from microorganisms, their toxins or other chemicals of health hazard..
- Elimination / destruction of micro – organisms or their toxins.
- Prevention of the growth of microorganism or the inhibition of toxin production (4)

3.3.5 Stages and Processes at Which Food Contamination May Occur

Although food is a basic human need, it can sometimes cause a number of illnesses arising from pathogenic and toxic substances which may find their way into food through contamination or through spoilage (2). Contamination of food can be either from biological agents or chemicals (19). Biological agents in food that are of concern to public health include pathogenic strains of bacteria, viruses, parasites, helminthes, protozoa, algae, and certain toxic products they may produce. Similarly there are, many

chemical contaminants too with several pathways. Figure 3. 3.1 below illustrates some of the various biological or chemical contaminants of foods (2).

See figure 3.3.2 an example of pathways to food for selected chemical contaminants of food (19).

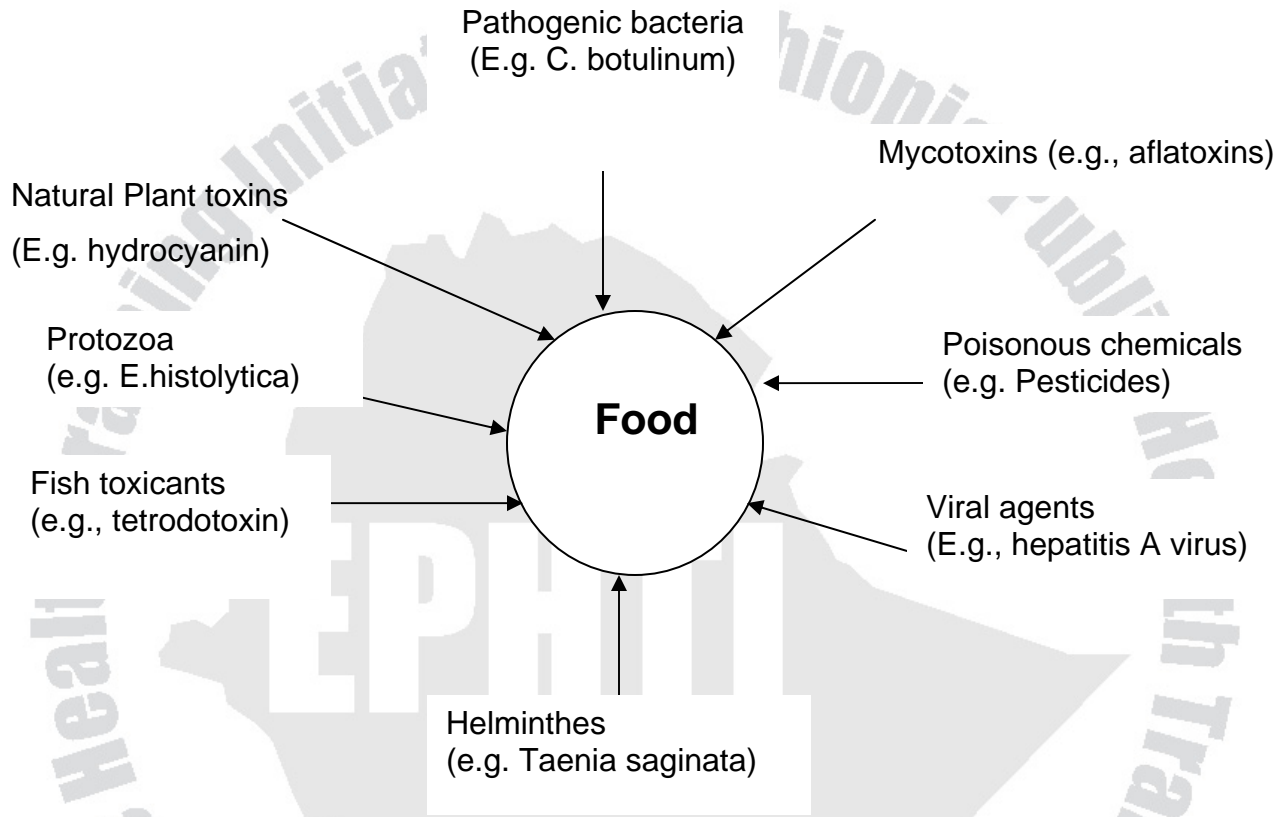


Figure 3.3.1. Contaminants of food

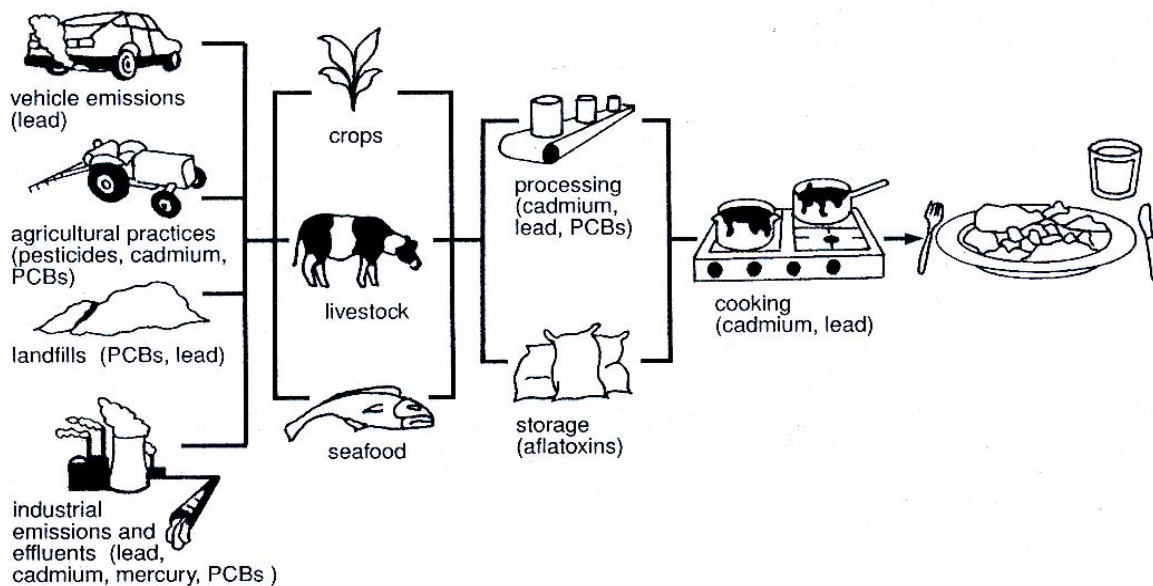


Figure 3.3.2. An example of path ways to food for selected chemical contaminants of food (19)

Food can be contaminated in the chain of its production and distribution, i.e.:

Farm → harvest → Transporting → Processing → Sorting → consumer (6).

1. **Farm:** Biological and chemical agents can contaminate food on the farm. Disposing of human waste in unsanitary manner may contaminate the food with pathogenic organisms, similarly different chemicals, such as pesticides, herbicides, and fungicides may be deposited on to and absorbed by various crops and vegetables (6,19).
2. **Harvest:** Harvesting food into contaminated receptacles can spread causative agents of disease, or may also lead to its contamination by poisonous chemicals if the receptacle was used to store such chemicals (6).
3. **Transporting:** During transportation, food can be contaminated by people, storage containers and so on (6).
4. **Processing and storage:** Food is liable for contamination during its processing and storage if stringent sanitation measures are not in place. Food may come in contact with chemicals that are toxic in storage areas. Rodents, insects (such as cockroaches) and other vermin are capable of contamination.

5. **Food preparation and consumption areas:** Restaurants cafeterias, mess halls, kitchens, bars, dining rooms, service tables, and utensils etc. can be conducive to growing and spreading of pathogens as well as chemical and physical agents of disease.

The flow of raw food materials to actual consumption is schematically presented in figure 3.4.3 including the accompanying hazards and risks at each stage. In principle the same flow scheme applies to both the food industry and to locally produced foods for private consumption (19)

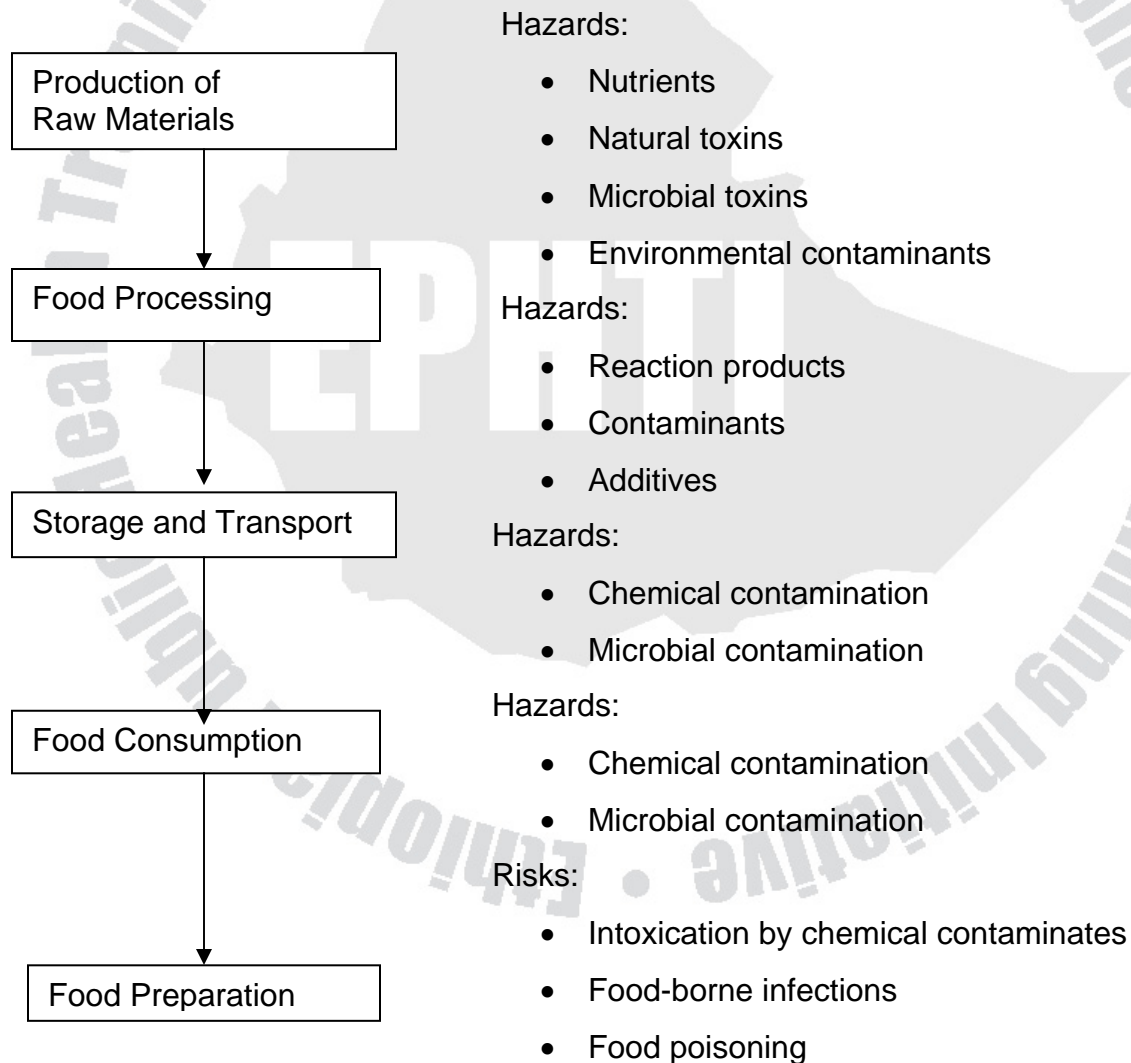


Fig. 3.3.3: Flow scheme of food production to food consumption

Sources of contamination of food:

Food products are rich in nutrients required by microorganisms and may become contaminated. Major contamination sources are **(7, 19,4)**:

- **Water:** water serves as a cleaning medium during sanitation operation and is an ingredient added in the formulation of various foods. If a safe water supply is not used it then becomes a source of contamination of the food (chemical or biological agents).
- **Sewage:** Raw, untreated sewage can contain pathogens that have been eliminated from the human body, as well as other materials including toxic chemicals from the environment. Examples are microorganisms causing typhoid and paratyphoid fevers, dysentery, and infectious hepatitis. If raw sewage drains or flows into potable water lines, wells, rivers, lakes, and ocean bays the water and living organisms such as seafood are contaminated.
- **Air:** Contamination can result from airborne microorganisms and chemicals in food processing, packaging, storage, and preparation areas. This contamination can result from unclean air surrounding the food or from contamination through improper sanitary practices.
- **Food Equipment:** contamination of equipments used for processing, preparing or serving food occurs during production (manufacture) and when the material is not properly cleaned.
- **Employees:** Of all the viable means of exposing microorganisms to food, employees are the largest contamination source. The hands, hair, nose, and mouth harbor microorganisms that can be transferred to food during processing, packaging, preparation, and service by touching, breathing, coughing, or sneezing. This is because the human body is warm; microorganisms proliferate rapidly, especially in the absence of good hygienic practices.
- **Adjuncts and additives:** Ingredients (especially spices, flavoring and coloring agents, preservatives) are potential vehicles of harmful or potentially harmful microorganisms and toxins. The amounts and types of these agents vary with place and method of harvesting, type of food ingredient, processing technique,

and handling. There could be hazards connected to these ingredients if there is lack of awareness of the incoming individual ingredients.

- **Insects and rodents:** Flies and cockroaches are associated with living quarters, eating establishments, and food processing facilities, as well as with toilets, garbage, and other filth. These pests transfer contaminants to food through their waste products; mouth feet, and other body parts; and during regurgitation onto clean food. Rats and mice as well transmit filth through their feet, fur, and intestinal tract. Like flies and cockroaches, they transfer filth from garbage dumps and sewers to food or food processing and food service areas.
- **Soil:** Soil may contain microorganisms as well as poisonous chemicals. These agents may get access to food either due to direct contamination or through dusts.
- **Plants and plant products:** Most of the organisms found in soil and water are also found on plants, since soil and water constitute the primary sources of microorganisms to plants. Chemicals sprayed to plants are other potential health risks.
- **Other animals' bodies:** From the intestinal tracts of animals, microorganisms find their way directly to the soil and water. From there, they may find their way into plants, dust, utensils and or food. Meat of animals can get contaminated during slaughtering, cutting, processing, storage, and distribution. Other contamination can occur by contact of the carcass with the hide, feet, manure, dirt, and visceral contents. Like wise drugs used to prevent disease and promote growth in animals may also become potential risk for human health due to persisting of these drugs in the meat or milk products.
- **Others:**
 - Vegetables and fruits treated with insecticides and which are not thoroughly cleaned before consumption.
 - Utensils from which toxic chemicals may leach out.
 - Mistaken use of a toxic chemical in the preparation seasoning or sweetening of food or by children believing it is a drink.

- o Deliberate and malicious contamination of food by a person for some irrational
- o Water polluted by chemicals from farm and or spraying food trees (4, 6).

3.3.6 Transfer of Contamination

Before a food-borne disease can occur, food-borne disease transmission requires that several conditions be met. There are two related models that illustrate the relationship among factors that cause food-borne diseases. These are (7):

a. Chain of infection:

This is a series of related events or factors that must exist or materialized and be linked together before an infection will occur. These links can be identified as Agent, Source, Mode of Transmission, and Host. The essential links in the infectious process must be contained in such a chain. The factors that are necessary for the transmission of a food borne diseases are (7):

1. Transmission of the causative agent from the environment in which the food is produced, processed, or prepared to the food itself.
2. A source and reservoir of transmission for each agent.
3. Transmission of the agent from the source to a food.
4. Growth support if the agent is biological.

These are conditions such as required nutrients, moisture, PH, Oxidation – reduction potential, lack of competitive microorganisms, and lack of inhibitors for contaminates to survive and grow. Moreover, the contaminated food must remain in a suitable temperature range for a sufficient time to permit growth to a level capable of causing infection or intoxication (7).

The infection chain emphasizes the multiple causations of food-borne diseases. The presence of the disease agent is indispensable, but all of the steps are essential in the designated sequence before food-bore diseases can result (see also figure 3.3.4 below)

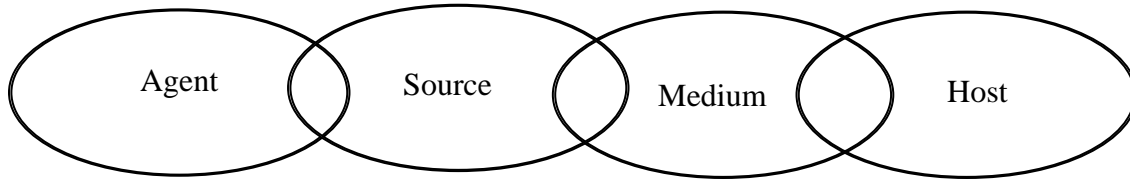


Fig. 3.3 4: The chain of infection

b. Web of causation:

This is a complex flow chart that indicates the factors that affect the transmission of food-borne diseases. This presentation of disease causation attempts to incorporate all of the factors and their complex interrelationships (7).

3.3.7 Factors Commonly Contributing to Food-borne Disease

Outbreaks (13, 4)

There are a number of factors that may lead to the occurrence of food-borne illness outbreaks. The major ones are (4,13):

- Preparation of food more than half a day in advance of needs
- Storage at ambient temperature
- Inadequate cooling
- Inadequate reheating
- Use of contaminated processed food (cooked meats and poultry, and the like)
- Undercooking
- Cross contamination from raw to cooked food from utensils, and contamination from other food contact surfaces in kitchen environment
- Infected food handlers or poor personal hygiene of food handlers
- Unsanitary dishware, utensils and equipment
- Improper food handling procedures such as unnecessary use of the hands during preparation and serving of food
- Improper food storage that may lead to cross contamination by agents of diseases (micro-organisms, poisonous chemicals), or exposure to moisture that may facilitate microbial growth
- Insects and rodents

3.3.8 Prevention and Control of Food-borne Diseases

The quality and safety of food is a topic of interest to the general public. Food quality from a more scientific point of view includes a number of safety aspects such as the presence of environmental contaminants, pesticide residues, use of food additives, microbial contamination, and nutritional quality. In practical terms, safe food can be defined as food that, after being consumed, causes no adverse health effects (19).

To ensure high quality of the food supply a number of parties must play specific roles. The main actors include the government, consumers, and the food industry. The government is responsible for the establishment of standards or codes of practice as well as the enforcement of laws and regulations. Furthermore, it should encourage the food industry to undertake voluntary measures to improve food safety. Consumers in turn should be well aware of the quality of the food they buy, prepare and consume and should adopt appropriate practices of food handling at home. At the industry level, all segments, including agriculture, should establish some system for safety assurance of their products and employ appropriate procedures and technologies (19).

Adequate monitoring of food quality is usually more difficult to achieve. But, it is critical that preventive measures for ensuring food safety should be given great attention to prevent and or reduce food borne diseases. The following are possible preventive measures for ensuring food safety at various stages:

1. Production of raw materials:

To ensure safe food production, it is important to look at the agricultural level, where foods are initially produced, and improve the hygienic quality of raw foods. By improving the conditions under which crops, fruits, vegetables and food animals are raised, the hygienic quality of raw food products can be significantly improved. Furthermore, use of both pesticides and fertilizers should be reduced, and residue levels of toxic chemicals used to improve crop production should be systematically monitored. Prohibition of use of untreated sewage water for irrigation of vegetable fields is also an area of attention. Food safety at this stage can also be improved through measures

aimed at reduction of industrial and vehicle emissions and disposal of hazardous waste materials that can enter the food chain.

2. Food Processing:

Greater demands are being made on the food-processing industry as a result of increasing urbanization. As consumers continue to move further a way from the sources of production, they will require an effective and safe food distribution system. This separation of the customer from the production sector means a loss of the traditional methods used by the consumer to ensure the safety of food. Substantial losses of food by contamination and spoilage can be prevented through the use of carefully controlled technology and well designed food-processing infrastructure (19).

The mainstay of microbiological food safety programs has been inspection. Inspection programs have serious limitations, however, as they sometimes overlook critical factors that are not part of the inspection protocol. Inspection services are usually inadequate or non-existent in many developing countries in which Ethiopia is inclusive. A different approach to food safety in modern industrial food production and in food establishments is the *Hazard Analysis and Critical Control Point (HACCP) system*. This is an attempt to make a significant impact on the prevention of food-borne diseases. The HACCP system consists of a series of interrelated actions that should be taken to ensure the safety of all processed and prepared foods at critical points during the stages of production, storage, transport, processing, preparation, and service. The elements of the HACCP system are summarized in Box 1 as follows (19).

Box 3.3.1. Hazard Analysis and Critical Control Point (HACCP) system elements (4,19)
(For further details, please see Annex vi)

Box 3.4. 1

- Determine hazards and assess their severity and risks.
- Identify critical control points.
- Institute control measures and establish criteria to ensure control.
- Monitor critical control points.
- Take action whenever monitoring results indicate criteria are not met.
- Verify that the system is functioning as planned.
- Establish a documentation system for procedures and records. Develop and maintain procedures and practices for record keeping.

Definitions:

- i. **Hazard:** Means the unacceptable contamination, growth or survival of microorganisms of concern to safety or persistence in foods or products of microbial metabolism (E.g. Toxins, enzymes, histamine) or the presence of chemicals of a harmful level of concentration or of a potential risk to health (4).
- ii. **Critical control Point:** Is a location, practice, procedure, or process at or by which control can be exercised over all or more factors that, if controlled, could minimize or prevent the hazard (4).

3. Food Preservation and Storage

The aim of food preservation is to eradicate or prevent the growth of pathogens during manufacturing, processing and preparation of food so that it will remain, safe to eat for longer periods of time. Bacterial growth is enabled by a number of conditions, the most important being the presence of a good substrate (in this case a food item); an infection with viable organisms; a temperature that allows growth of bacteria; proper pH; and sufficient water for growth. To guard against microbial growth, at least one of these conditions should be hindrance (19).

4. Food Preparation in the Home:

The household is perhaps the most relevant place for developing strategies to combat food borne illness, as it is the location where the consumers, can exert the most control over what they eat. Clearly, one of the most significant components of keeping food pathogen-free in the household is maintaining a clean and hygienic environment in the kitchen or other food preparation areas. Proper sanitation facilities, cleanliness of household members who prepare the food, and control of pests are all essential for the presentation of acceptable food.

Consumption cooked food, while still hot will not cause food borne infection. The chemical risks in food preparation at home are the same as those present during food processing. The general public should be made aware of these risks.

Keeping chemicals away from kitchens and areas of food preparation is important. If needed, use chemicals cautiously.

Many bacterial pathogens are able to multiply in food because of the temperature at which the food is stored. Figure 3.4.5 shows the temperatures at which bacteria can be killed or controlled (4.19).

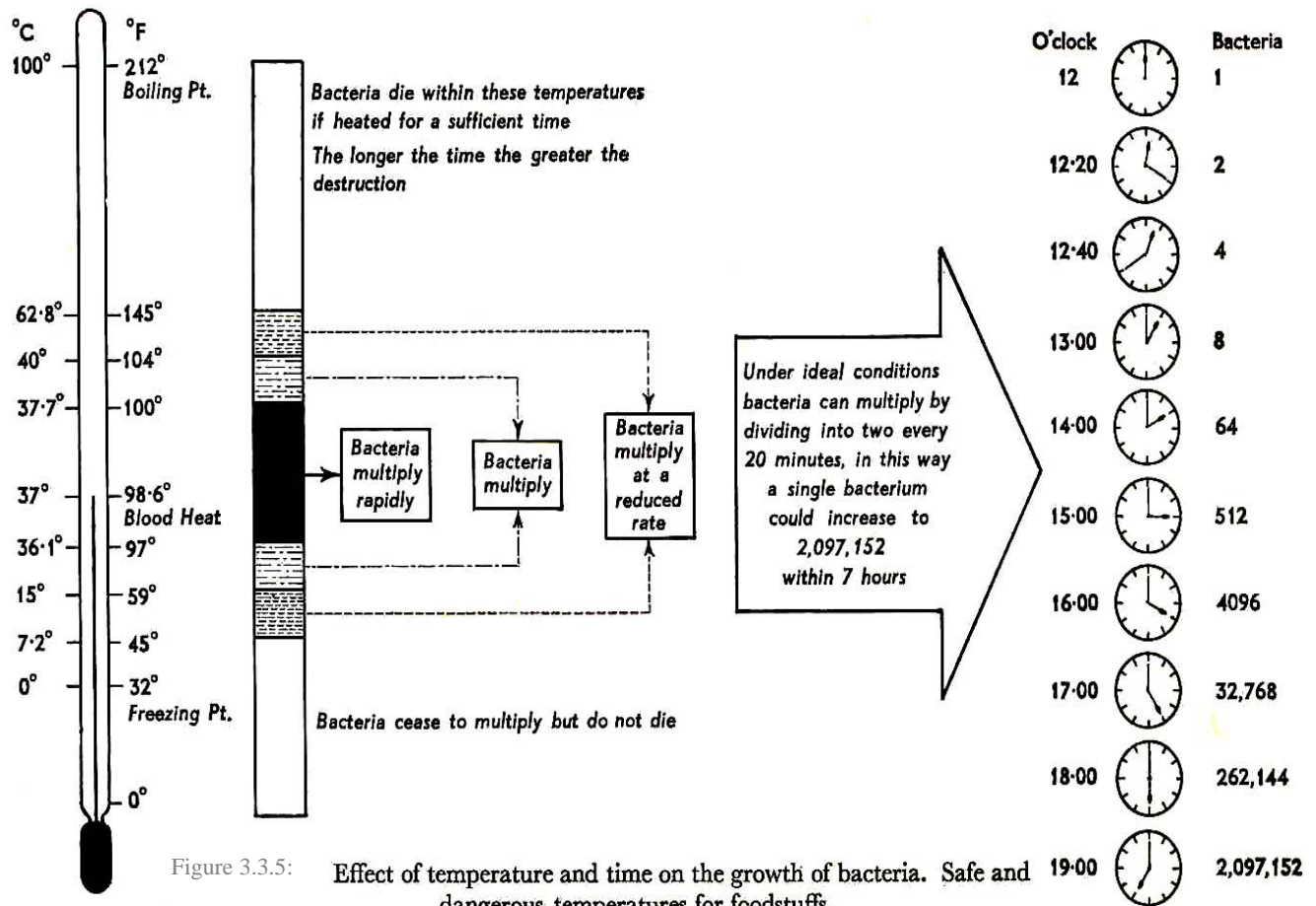


Figure 3.3.5: Effect of temperature and time on the growth of bacteria. Safe and dangerous temperatures for foodstuffs.

Taken from Food Poisoning & Food Hygiene. Third Edition. 1974. By Hobbs Betty C.

5. Food preparation in the food service industry:

The consequences of improper food preparation in food services such as canteens and restaurants can be much greater than that in the household, simply because a large number of individuals may be simultaneously exposed to unsafe food items. It is essential to have a quality control program (inspection) that will ensure the maintenance of food product standards during all stages of handling, processing and preparation; it must also be applied to all areas and equipment that come into contact with food and beverages. Street foods are particularly prone to lapses in safe food preparation, hence requiring stringent control measures (19).

The prevention and control strategies for food borne diseases emanate from the three basic principles (described in section 3.4.4): the prevention of contamination, destroying the pathogenic agent or retarding the growth and multiplication of the biologic agent as well as retarding the production of toxins (4). The different methods for applying the above principles are discussed below:

Methods to keep food safe

The art of keeping food safe and preservation requires knowledge of bacteria and the effect of the environment on microorganisms. Methods of keeping food safe and preservation include modern innovations such as vacuuming and filtration techniques, pressure canning and radiation processes. The primary objective of keeping food safe is to prevent food from acquiring hazardous properties during preparation, shipment, or storage. The principal methods and the techniques used to keep food safe include temperature control (including pasteurization, cooking, canning, refrigeration, freezing and drying), fermentation and pickling, chemical treatment and irradiation (2, 3, 4, 6, 7).

1) Temperature control:

a) The use of high temperature:

Heat is one of the oldest methods of destroying microorganisms in food. The greatest advance in food hygiene was inadvertently made when man discovered the advantages of boiling, roasting, cooking and other heat treatments of food. Heat renders the destruction of microorganisms / pathogens and in some forms also destroys the toxin produced, such as in the case of the toxin of clostridium botulinum. Heat treatment may involve the following techniques.

- Cooking / boiling / frying operations
- Blanching operations: Blanching is a mild pre-cooking operation involving brief scalding by hot water or steam, which is often used to reduce the bacterial load and insects on vegetable foods.
- Canning: This is the process of placing prepared (heat-treated) food in cans, exhausting the air from the cans, sealing the cans, sterilizing the sealed can and cooling it.

- Pasteurization: A process of heat treatment of food that kills pathogenic microorganisms without destroying taste, digestibility and nutritive value of food. It also destroys some food spoilage microorganisms.
- Drying (Desiccation): Bacteria cannot multiply in the absence of water (moisture). This can be achieved by application of heat or chemical treatment.

b). The use of low temperature

Unlike high temperature, low temperature (cold) is not an effective means of destroying microorganisms and toxins in foods except retarding their multiplication and metabolic activities there by reducing toxin production.

- Chilling (cold storage or refrigeration): is reducing food temperatures to below ambient temperatures. This is a suitable temperature to preserve perishable food items that may get spoiled at freezing temperature.
- Freezing: This is a dehydration method because the water in the food is transformed to ice, thus rendering it unavailable for microbial metabolic function. Freezing temperature depends upon the kind of food and the intended storage time.

2) Fermentation and pickling:

In fermentation the food is transformed into an acid state based on the pH control principle. Some fermented foods have high amount of alcohol, which is antimicrobial. Pickling on the other hand refers to the immersion of certain foods in concentrated natural acid solution such as vinegar.

3) Chemical treatment:

This involves osmotic balance disturbance or direct actions of the chemicals on the microorganisms. Chemicals that increase osmotic pressure with reduced water activity below the level that permits growth of most bacteria can be used as bacteriostatic. Liquids pass into or out of bacterial cells by the process of osmosis. Examples for osmotic actions are salting and sugaring. Some other chemicals may destroy or inhibit growth of microorganisms in food. Examples include application of nitrites and smoking.

4) Radiation: this is a process of exposure of the food to high- speed electrons to destroy microbial cells. Beta, gamma or x-rays irradiate microorganisms in foods. A cell inactivated by irradiation cannot divide and produce visible growth (7).

5) Other important methods /supportive procedures that facilitate the safety of food:

- Health education
- Good personal and environmental hygiene
- Availability of safe, ample and convenient water supply
- Training of food handlers and managers
- Stringent inspection and control actions
- Legislative support (ordinances and codes), licensing
- Good-house keeping practices including separate storage and care of toxic chemicals.
- Understanding about additives and restrictions of unauthorized use.
- Food equipment selection to avoid chemical poisoning arising from the material constituency and or coatings of some food utensils.
- Avoidance and care of insecticide use in food processing and preparation areas.

3.3.9 Collection of food samples

The need for sample collection:

The following factors may determine the essentiality of sample collection in food borne disease outbreaks:

- For diagnosis of outbreak
- For epidemiological reasons
- For legal issues
- For preventive aims
- For designing appropriate actions

Types of samples for assessment of food-borne diseases:

- Official samples: suitable for initiating prosecutions in court
- Informal samples: generally collected for the purpose of obtaining information, e.g., general survey

- Standard samples: for the purpose of establishing standards for foods
- Post-seizure samples: collected under court order from goods under seizure
- Documentary sampling: is done when samples are too heavy, too expensive, or too bulky

Sampling Plan:

Before instituting a food sampling plan, the following steps should be followed:

- Discuss the plan with laboratory personnel
- Determine the analytical capability of the laboratory
- Determine how sample is to be taken
- Decide how often and under what conditions sampling is to be done

Criteria for sample collection:

- Type of food
- Size of the lot to be sampled
- Representativeness of the sample
- Acceptance and rejection criteria: of pathogens, adulteration, tolerance limits, composition standards, net contents
- Degree of hazard to human health

Types of tests:

- Physical
- Bacteriological
- Chemical/toxicological

Now you are through the core and satellite modules, but there are still some activities remaining as stated below:

- 1. Read the task analysis of the different categories of the health team on Unit Five.**
- 2. Do the questions of pretest as posttest.
N.B. Use a separate answer sheet.**
- 3. Compare your answers of the pre and post tests with the answer keys given on ANNEX I and evaluate your progress.**

3.4. SATELLITE MODULE FOR MEDICAL LABORATORY TECHNOLOGISTS

3.4.1 Directions for Using the Module

- Before you start reading the Satellite Module, be you have completed the Core Module and performed the pre-test questions.
- Read through this satellite module carefully.

3.4.2 Learning Objectives

At the end of this satellite module, the reader is expected to be able to:

- Explain the safety procedures that should be taken in the processing of the specimens during diagnosis of food borne disease.
- Describe how to collect and handle different specimens in the investigation of food borne disease.
- List the laboratory investigations employed for diagnosis of food borne diseases.
- Describe the appearance of stool specimens during investigation processes in the diagnosis of food borne disease.
- Illustrate the morphological features of etiological agents of food borne disease.

3.4.3 Learning Activities

Refer to the case study under learning activity section 2.4 in the core module and discuss the following questions.

1. What type of specimen should be collected?
2. How should this specimen be collected?
3. What could be the etiology of the disease?
4. What type of investigations can be done?

3.4.4 Laboratory Diagnosis

A. Collection and handling specimen

Proper collection of specimen is essential since the final laboratory results are dependent on the initial proper quality of the sample. The cause of food borne disease may be identified in the laboratory by examining specimens such as stool, blood, vomit, rectal swab, liver and duodenal aspirate; macroscopically, microscopically, culture and immunologicly (16). If food poisoning is suspected because of a cluster of cases are

related to the eating of common foodstuff a sample of the suspected food should be collected (17).

i. Safety

Some organisms are more hazardous to handle and are more likely to infect laboratory workers than others, e.g. Hepatitis virus. Infection may be acquired through the skin, eye, mouth and respiratory tract so laboratory staff must practice the following safety precautions. (16)

- Washing of hands after handling specimens and infected materials, when leaving the laboratory and at the end of the day's work.
- Covering any cuts, insect bites, open sores or wounds on the hands or other exposed parts of the body with a water proof adhesive dressing.
- Wearing closed shoes and not walking bare foot.
- Not eating, drinking, chewing gum and smoking in the laboratory.
- Not licking gummed labels or placing pens, pencils or other articles near the mouth, eye or in hair.
- Protective clothing should be worn over normal clothing to protect the major parts of the body from splashes, droplet of liquids containing microorganisms and hazardous chemicals.
- Do not touch your eyes, nose or other exposed skin parts with your gloved hands.
- Make sure that specimen container is tightly closed and the cap is not leaking before transporting
- Decontaminate infectious materials and laboratory wastes before disposing
- Sterilize non-disposable items.

ii. Collection of stool specimen

For clinical purposes, a fresh faecal specimen is required. It should be uncontaminated with urine and collected in to a suitable size, clean, dry and leak-proof container. This container need not to be sterile but must be free of all traces of antiseptics and disinfectants. Several specimens collected on alternative days may be required for detecting parasites that are excreted intermittently e.g. G.lamblia Avoid using containers made from leaves, paper or cardboard because this will not be leak proof,

may not be clean and can result in the faecal contamination of hands and surfaces. Dysenteric and watery specimens must reach the laboratory as soon as possible after being passed (within 15 minutes), otherwise motile parasites; such as *E. histolytica* and *G. lamblia* trophozoites may not be detected. Other specimens should reach the laboratory within 1 hour of being collected. Specimen must be labeled correctly and accompanied by a correctly completed request form (16). Fecal specimens like other specimens received in the laboratory, must be handled with care to avoid acquiring infection, from infectious parasites, bacteria, or virus. Feces may contain infective forms of:

- Parasites such as *E. vermicularis*, *T. solium*, *G. lamblia*, *E. histolytica* or *C. parvum*.
- Bacteria such as *V. cholerae*, shigella or salmonella species.
- Viruses including hepatitis virus, HIV and rotavirus.

Whenever it is difficult to get feces, rectal swab should be obtained but rectal swab is unsatisfactory unless it is heavily charged and visibly stained with feces, which collected from the rectum, not anus. (17)

iii. Collection of Blood Specimens

The following precautions need to be followed during collection of blood sample.

- Syringes and needles used for collecting blood samples must also be chemically clean and dry
- Follow a safe technique and wear protective gloves.
- Specimen container must be leak proof, sterile and chemically clean, should be well washed with detergent, and rinsed in several changes of clean water.
- Blood should be collected before antimicrobial treatment has been started and at the time the patient's temperature is beginning to rise
- To increase the chance of isolating a pathogen it is usually recommended that at least two specimens (collected at different times) should be cultured
- Blood for culture must be collected as aseptically as possible.
- If anti-coagulated blood is required, add the correct proportion of blood to the anticoagulant in to the tube or bottle and mix it by gently inverting the container several times.

B. Laboratory tests for some food borne diseases

i. Parasitic food borne infections:

a. Amebiasis

Macroscopic Examination:

Amoebic dysentery contains blood and mucus

Microscopic stool examination:

The laboratory diagnosis of amoebic dysentery is by finding *E. histolytica* trophozoites in fresh dysenteric fecal specimen. Specimen must be examined without delay; otherwise identification of the trophozoites becomes impossible because the amoebae lose their motility.

Trophozoite of *E. histolytica* has the following general characteristics

- Average size about 15-30 μ m
- Shows active amoeboid movement in fresh warm specimen,
- Contain ingested red cells and
- Single nucleus is present which has a central karyosome.

Cyst of *E. histolytica* is morphologically identical with *E. dispar* but genetically distinct. Cysts formerly reported as *E. histolytica* should now be reported as *E. histolytica* / *E. dispar*. (16)

Cyst of *E. histolytica* / *E. dispar* has morphological characteristic of:

- Round, measuring 10 – 15 μ m
- Contain 1- 4 nuclei with a central karyosome
- Chromatoid bodies can be seen particularly in immature cyst.

Only one-third of infected patients are identified from a single stool specimen and it is recommended that at least three separate specimens be evaluated before excluding the diagnosis (18).

Culture:

Culture of amoeba is more sensitive but is not routinely available (9).

Serology:

Serology is an important addition to the methods used for the parasitological diagnosis of invasive amoebiasis. Kits for performance of agar gel diffusion

assay and ELISA are commercially available, and the result of tests are positive in more than 90% of patients with colitis, amebomas, or liver abscess (9).

b. Giardiasis:

Macroscopic Stool Examination

Fecal specimens containing *G. lamblia* may have an offensive odor and are pale colored, fatty and float in water.

Microscopic diagnosis of giardiasis is by:

- Finding *G. lamblia* trophozoites in fresh diarrheic specimens particularly in mucus. They are often difficult to detect because they attach themselves to the wall of the intestine.

- Finding *G. lamblia* cysts in more formed specimens:

The cysts are excreted irregularly. Often large number may be present for a few days followed by fewer numbers for a week or more.

Several specimens may need to be examined and a concentration technique used. *G. lamblia* cyst can be concentrated using the modified formal ether centrifuge technique (16).

Excretion of *G. lamblia* trophozoites and cysts often intermittent, so microscopical examination should be made on specimen collected at different time intervals (17).

Trophozoite of *G. lamblia*:

- Small pear-shaped flagellate with a rapid tumbling and spinning mobility often likened to a falling leaf.
- Measures 10-20µm in length and 5-9 µm in width
- Has a large concave sucking disc on the ventral surface.
- It has four pairs of flagella, two axonemes, and two nuclei.
- A single or two curved median bodies are present.

Cyst of *G. lamblia*

- Small and oval measuring 8 – 12 µm (double walled, elliptical shaped)
- Internal structures include two to four nuclei, grouped at one end, axonemes, median bodies, and remains of flagella.

Occasionally giardiasis can be diagnosed by detecting *G. lamblia* trophozoites in duodenal contents, but this should only be considered when giardiasis is clinically suspected and no parasites are detected after examining several faecal specimens (20).

Diagnosis of *G.lamblia* using an antigen test:

Several tests are commercially available for detecting *Giardia lamblia* specific antigen in faecal specimens using a monoclonal antibodies reagent. The antigen is stable and can be detected in fresh or preserved faeces. The presence of antigen indicates active infection. It is produced as *G. lamblia* multiplies in the intestine. Most *Giardia* antigen tests use an enzyme immuno assay (EIA) method in micro plate or membrane format. No equipment is required to perform or read the assay. Each test device contains a positive control. A circular blue – green spot in the test area indicates the presence of *Giardia* antigen in the specimen. The assay has 92.6% relative sensitivity and 98.1% relative specificity (16).

c. Taeniasis

The laboratory diagnosis of *T. saginata* infection is by:

Macroscopic Examination

- Identifying gravid segments recovered from clothing or passed in faeces macroscopically. The segment appears white and opaque and measures about 20mm long by 6mm wide when freshly passed.

Microscopic Examination

- Identifying the ova in the stool

A concentration technique and the examination of several specimens may be necessary to detect *Taenia* eggs in fces. The eggs can be concentrated by formal ether technique. It is round to oval measuring 33 – 40 µm. Embryo is surrounded by a thick brown wall, hook lets are present in the embryo. Eggs may also be present in the perianal area; thus, if proglottids or eggs are not found in the stool, the perianal region should be examined with use of a cellophane tap swab (9).

d. Ascariasis

The laboratory diagnosis of *Ascaris lumbricoides* is by:

Macroscopic Examination

- Identifying *A. lumbricoides* worms expelled through the anus or mouth. Freshly expelled ascaris worms are pinkish in color. They measure 12 – 35cm in length and taper at both ends.

Microscopic Examination

- Microscopically identifying *A. lumbricoides* egg in faeces:

Usually fertilized eggs are found in faeces but occasionally infertile eggs are produced. Fertile egg has yellow – brown oval or round shell is often covered by an uneven albuminous coat; contains a central granular mass, which is the unregimented fertilized ovum. Infertile egg is dark in color and has a thinner wall more granular albuminous covering, more elongated than a fertilized egg, and contains a central required mass of large granules.

ii. Bacterial food borne infection

a. Enteric Fever (Typhoid and paratyphoid fever)

Salmonella typhi and *salmonella paratyphi* causes enteric fever, which is endemic in many developing countries. (17)

General characteristics

- *Salmonella* are enterobacteria, gram negative rods
- They are motile, non capsulated and non sporing
- They survive freezing in water for long period
- They usually produce H₂S
- *Salmonella* grow readily on simple media but they almost never ferment lactose or sucrose.

Diagnostic laboratory Test

Specimen: Blood, urine, stool and bone marrow can be used to identify the organism.

A. Culture:

Culture is the diagnostic gold standard. The yield of blood culture is quite variable; it can be high as 90% during the first week of infection and decrease

to 50% by the third week. Organism usually from fecal specimen can be isolated from 40 – 50% of patients from the second week of infection. A diagnosis can also be based on cultures of urine and Bone marrow (21).

- For fecal specimen Eosin Methylene Blue and MacConkey are some different media and Salmonella shigella agar, Xylose Lysine Deoxycholate agar and deoxycholate citrate agar are selective media. For fecal specimen before inoculating on the plate agar, it is better to use selective broth such as selenite F to enhance the growth of salmonella which is usually found in small number.
- Blood sample primarily cultured in Thioglycolate broth and to the plate agar
- Biochemical reaction and slide agglutination tests with specific sera are used for the identification of suspected colonies from solid media. The differentiation of suspected salmonella colonies using motility indole urea (MIU) medium and Kligler iron agar (KIA) are described on annex II.

B. Serology

For serological examinations, paired acute and convalescent samples of serum should be collected at an interval of about 10 days in suspected enteric fever (17). Several serological tests including the classic Widal test for febrile agglutinins are available; however, it gives high rate of false positivity. The Widal test is a serological test for the presence of salmonella antibodies in patient's serum when facilities for culturing or antigen testing are not available. Widal test if performed reliably and interpreted with care (with clinical finding) can be of value in diagnosing typhoid and paratyphoid fever. When investigating typhoid the patient serum is tested for O and H antibody (21).

Most widal tests used as slide or tube serial dilution with manufacturers providing details for both slide and tube test. Before use the antigen suspension must be allowed to warm at room temperature and well-mixed, sufficient serum for Widal test can be obtained from 3 – 5 ml of patient

venous blood collected in to a clean dry tube and allowed to clot. The serum should be free from red cell and must not be heated.

The Widal test is reported by giving the titer from both O and H antibody (antibody titer is the highest dilution of serum in which agglutination occur). If no agglutination occur report as:

S. typhi O titer less than 1:20

S. typhi H titer less than 1:20.

In typhoid endemic areas in developing countries active typhoid is suggested if the titers of H or O or both, agglutinins are significantly raised (i.e. titer greater than 1 in 180 or 1 in 200)

Raised O or H titers other than active typhoid associated with vaccination with typhoid vaccine, infection with other salmonella species, chronic liver disease and immunological disorder such as rheumatoid arthritis rheumatic fever, multiple myeloma and ulcerative colitis (21).

b. Shigellosis

General Characteristics of the causative agents

Shigellae are:

- Gram negative
- Non-sporing non-capsulated rods
- Unlike salmonellae and many other enterobacteria, shigellae are non-motile.

Diagnostic laboratory tests

Specimen: fecal specimen for culture and blood for antibody detection

A. Microscopy

Fecal specimens from patients with shigellosis may be watery and contain little blood and mucus in the early stages of infection, but, consists almost entirely of pus and blood mixed with mucus in the later stages of infection. When examined microscopically, red cells and large number of pus cells are usually found. Specimens from patients with amoebic dysentery contain red cell, and usually very few pus cells (21).

B. Culture

A fresh feces specimen is required to isolate shigella.

- The specimens are inoculated on different media e.g. macConkey. A selective media Deoxycholate citrate agar (DCA), Salmonella Shigella agar (SS), Xylose lysine deoxy cholate (XLD) agar is required to isolate shigallae from feces. But XLD is more preferable than the others.
- Biochemical reactions are used for differentiation of suspect shigella colonies. The differentiation of suspected shigellae colonies using Motility Indole Urea (MIU) and kligler Iron Agar (KIA) and other biochemical reaction is described on annex II.

C. Serology

Serological test can be performed since antibodies to somatic antigens develop early in the acute phase of disease. (9) Normal persons often have agglutinins against several shigella species, therefore serology is not used to diagnose shiggella infection.

c. Cholera

General Characteristics of the causative agent

- The main species of medically important is Vibro cholerae 01. It is strongly oxidase positive and non lactos fermenter
- V. Cholerae is an aerobe and facultative anaerobe
- Gram negative motile usually curved rod with a single flagellum at one end
- Highly motile with a distinctive rapid to and fro movement

Diagnostic Laboratory Test

Specimen: A fecal specimen is required to test directly for V. cholera antigen and to isolate V. cholera in culture.

A. Microscopy

If the specimen is obtained on the first day of the illness the vibros are likely to be present in enormous numbers , and it is then possible ,in urgent cases to make provisional diagnosis by direct microscopic examination of a film of the the feces ,preferably by dark ground illumination. The vibros should be seen darting about and to be immobilized when specific antiserums added to the film.(17)

B. Culture

- In alkaline peptone water *V. cholera* grows rapidly producing turbidity just below the surface of the medium usually occurs within 4-6 hrs. The organism will grow at room temperature as well as at 35-37 °C. To confirm that the organisms are vibrios, examine a wet preparation and gram stained smear. The use of dilute carbol fuchsin (1 in 10) is recommended as a counter stain in the gram technique
- Thiosulphate-citrate Bile Salt sucrose (TCBS) agar is an excellent selective medium for the primary isolation of *V. cholera*. When staining vibrio species *V. cholerae* O1 and other sucrose fermenting vibrio organism (non – O1 *V. cholerae*) produces 2-3 mm in diameter yellow colonies on TCBS agar after overnight incubation at 35-37°C. *V. Cholerae* can also be cultured at room temperature if an incubator is not available. Sub culture from TCBS to a non-selective medium such as nutrient agar is essential for differentiation of *V. cholerae* with other sucrose fermenting vibrios (non O1 *V. cholerae*). O1 *V. cholerae* agglutinates with *V. cholerae* O group 1 polyvalent antiserum but other non-O1 *V. cholerae* do not agglutinate.

C. Serology

Rapid detection of *V. cholera*

Using monoclonal antibody reagents we can detect *V. cholerae* O1 antigen in fecal specimens without the need to culture. Such tests are simple to perform and have particular value when investigating a cholera epidemic although they are expensive (21).

d. *Escherichia coli*

General Characteristics

- Gram negative motile rod
- Aerobe and facultative anaerobe
- Lactose fermenter

Diagnostic laboratory test

Specimen: Feces

A. Culture

Stool culture on blood and MacConkey agar. E.coli produces 1- 4 mm in diameter colonies on blood agar after over night incubation at 35 -37°C. The colonies may appear pink on macConkey agar, due to production acid by fermenting acid.. An important bio chemical feature of E.coli is the production of indole from peptone water containing tryptophan, which differentiate the E.coli from other most entrobacteria.. The basic biochemical reaction of E.coli compaird with other entro bacteri is shown on annex II.

e. Brucellosis

General Characteristics

- Brucella are Gram negative coccobacilli (Short rods) and obligate parasite of human and animal
- Non-capsulated and non motile
- An intracellular bacteria, strict aerobic
- Requires a carbon dioxide enriched atmosphere in which to grow.

Diagnostic laboratory test

Specimen: Blood , bone marrow and lymph gland fluid for culture

A.Culture

- Blood or bone marrow specimens needed for culture in the acute stage of infection.. Triptone soya diphasic medium is recommended for Isolation of Brucella species from blood sample. A varity of colonial forms are produced by brucella strains including smooth, and rought colonies. The may be colorless or gray white.
- The inoculated diphasic media should be incubated at lest for 3 to 8week before reporting no brucella is isolated.
- When the organism is sub cultured on solid agar, for further identification, it needs 2 to3 days of incubation to see visible colonies. Only a few routine biochemical tests are helpful in differentiating Brucella species.

These include urase and hydrogen sulphide production. All Brucella strains are catalase positive.

B. Serology

Infection with Brucella organisms produces an antibody response. Measuring the titer of brucella antibodies in serum is an important method of diagnosing Brucellosis. Rapid slid screening agglutination test and tube or micro plate agglutination test can be used to test serum for Brucella antibodies (21).

N.B: Brucella species are highly infectious. Specimen must be marked **HIGH RISK**.

j. Bacillus cereus

Morphology

- Gram positive, rod shaped.
- Motile,
- Non- capsulated and non-lactose fermenter.

Diagnostic laboratory test

Specimen: feces, vomits or food.

A. Culture

- Mannitol egg-yolk phenol red polymyxin agar (MYPA) is recommended as a selective medium for the isolation of B.cereus from feces, vomit of food.
- The organism is non-lactose fermenter producing pale colonies on macConkey agar. After aerobic incubation for 18-24hrs at 37°C, look for large rough pale colonies on macConky agar. On egg yolk agar, B.cereus gives a strong lecithinase reaction

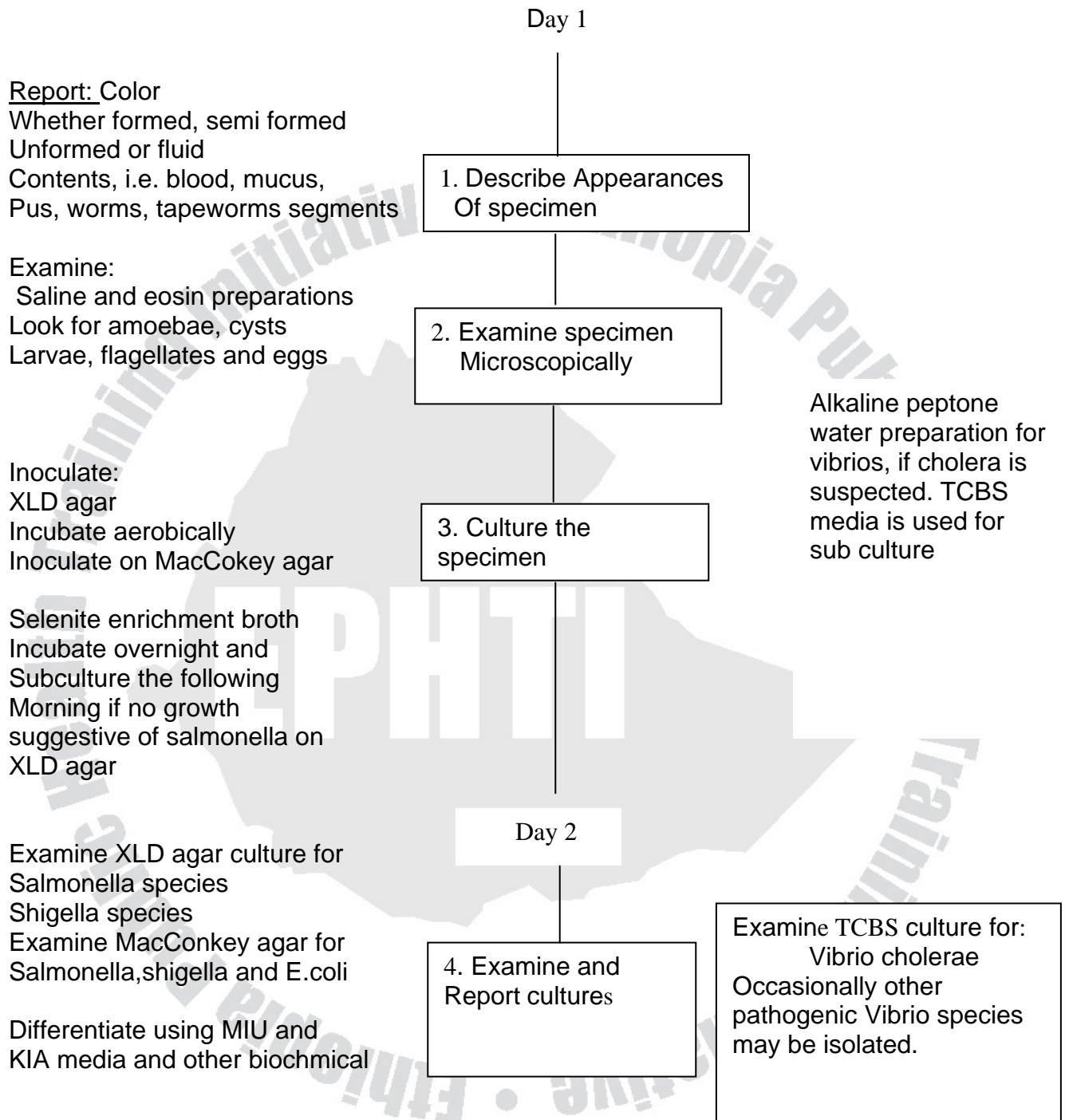


Figure 3.4.1 Summary of the laboratory examination of fecal specimens

iii. Viral food borne infection

A. Viral Hepatitis

Hepatitis A virus is a non-enveloped, heat, acid and ether-resistant RNA virus in the picorna virus family. A diagnosis of hepatitis can be made on the basis of characteristic presentation and the presence of liver function test (18). In case of hepatitis the serum aspartate aminotransferase (AST) and alanin amino transferase (ALT) show a variable increase during the prodromal phase of acute viral hepatitis and precede the rise in bilirubin level. . Antibodies to hepatitis A virus can be detected during acute illness when serum aminotransferase activity is elevated. This early antibody response is predominately of the IgM class and persists for several months. During convalescence, however anti-HAV of IgG class become predominate antibody (9).

B. Viral Gastroenteritis

Because rotavirus is shed in large amounts in the stool, detection is relatively easy. Various specific and highly sensitive commercial immunoassays are available to detect rotavirus antigen in fecal specimens.

DNA probe diagnosis appears to be sensitive and specific, as do polymerase chain reaction (PCR) – based assays, but this detection method have been used for research purpose (9).

Food poisoning/ intoxication

i .Bacterial food poisoning/ Intoxication

a. Staphylococcal food – poisoning

S. aureus food poisoning is caused by the ingestion of preformed toxin in contaminated food. Staphylococcus species are:

- Non motile
- Non capsulated
- Gram-positive cocci of uniform size; they occur characteristically in-groups but also single and in pair.
- Grow well aerobically and in carbon dioxide enriched atmosphere

In an outbreak plat out few loopfull of a saline suspension of faeces on plates of blood agar, macConky agar and a selective medium, eg. 6% Nacl nutrient agar and manitol salt agar. Incubate aerobically for 18 – 24 hrs at 37° C and examine for colonies of S.

aureus. On blood agar produces yellow to creamy 1-2 mm diameter colonies, on macConkey agar smaller (0.1-0.5 mm) colonies are produced after overnight incubation at 35-37°C. In an outbreak send sub-culture for phage typing and tests for enterotoxin production.

S. aureus may often be isolated from the faeces of a healthy person, so that its isolation from the feces of a patient with diarrhea is not proof of a casual role. Identification of phage type among the isolate from suspected food staff is fair evidence that the outbreak is due to staphylococcal food poisoning.

Some staphylococcal food poisoning outbreaks; however, are caused by foodstuff that has been heated at a temperature sufficient to kill the staphylococci though insufficient to inactivate the more thermostable enterotoxin. In such cases the diagnosis requires the demonstration of staphylococcal enterotoxin in the feces or food. Kits for the detection of staphylococcal enterotoxins A, B, C, and D by reversed passive latex agglutination are available commercially (17).

b. Botulism

The rapidity with which death can occur with the food-borne type of botulism necessitates an immediate diagnosis based up on history and clinical manifestations. For epidemiological purposes, serum specimens and food are analyzed for neurotoxin by mouse virulence assays. (22)

c. Clostridium Perfringens

Food poisoning may be caused by heat-sensitive strain or heat resistant strains of this organism. Heat resistant strains are more likely to be cause of outbreaks due to well cooked food stuffs as their spore are more likely to survive the cooking. The mere finding of the organism in the feces, even it is heat resistant, that not indicate that it has casual role in the illness, for normal subjects commonly have 100-10,000 Culture bacilli or spore of *C. perfringens* per gram of feces. Culture should be therefore done by a semi-quantitative method; for feces collected from patients at the height of the illness commonly contains 1,000,00 or more *C. perfringens* per gram and it is only the finding of such high counts that should be reported as probably significant.

It may also be helpful to examine specimens of feces for *C. Perfringens* enterotoxin. The examination of toxin may be made in a reference laboratory on feces transmitted by post but reversed passive latex agglutination kits are now available commercially (17).

d. Entero toxigenic Escherichia coli

Strains of *E. coli* producing heat-stable or heat-labile enterotoxin may cause diarrhea in adults and children. They may be cultured from feces on macConky and blood agar media and serotyped. Not all strains of commonly toxigenic serotypes produce enterotoxins and cultures may be examined for toxin production in a reference laboratory for or heat labile enterotoxin with a reversed passive latex agglutination kit (17).

ii. Chemical Food Poisoning

1. Metals

Occasionally, the toxicology laboratory is asked to aid in the diagnosing possible heavy metal (mercury, arsenic, lead) toxicity, and if the diagnosis proves positive, quantitative determination of blood or urine levels is very helpful in following the course of therapy (23).

a. Lead

Lead accumulates in the body because elimination is very slow. This deposits are primarily phosphate bind down in bone tissue. Since lead is found primarily in red blood cells, whole blood is the specimen of choice for detecting lead poisoning. Urine lead levels are very helpful in monitoring the effectiveness of therapy. The method of choice for measuring lead level in blood and urine are atomic absorption spectroscopy using a heated graphite furnace and electrochemical methods, specifically anodic stripping voltammetry and induction coupled plasma (23).

b. Mercury

Typical mercury level in blood is 0 to 5µg/100ml and urine level of 5 to 25µg/l is considered normal. The method most commonly used for both blood and a urine mercury determination is cold vaporization atomic absorption spectroscopy. A very simple but crude test to detect large amounts of mercury in urine is the Reinsch test. This test will also detect antimony, selenium, and arsenic but is not very sensitive to any

of these metals. The principle of the test is clean copper wire is submerged in boiling urine to which has been added a small amount of HCl. Mercury, if present will be deposited on the copper wire (23).

c. Arsenic

Because arsenic quickly cleared from the blood, urine is the specimen of choice for diagnosing arsenic poisoning. Arsenic will persist in the urine for about a week after an acute poisoning and for as long as a month following chronic exposure. Occasionally, hair and nails are analyzed to detect the long-term effect of arsenic poisoning. The recommended method of analysis is flame atomic absorption (23).

2. Pesticide Poisoning

Organophosphates represent the largest single group of pesticides used and causes approximately one-third of pesticide poisonings. The laboratory may help identify individuals who have become toxic with organophosphates. The method of screening is to measure serum pseudocholinesterase activity, which will be depressed in the presence of organophosphates. Individual pesticide testing is well developed but not warranted in a clinical toxicology laboratory because of infrequency of pesticide poisoning seen in the average emergency room and the expense of such testing.

Several analytical techniques have been applied to measuring pseudocholinesterase, including manometry, electrometric titration, and colorimetry. The photometry uses acetylcholine as a substrate. Acetylcholine is converted to thiocholine and acetate. Thiocholine then reacts with dithiobisnitrobenzoic acid to form the yellow-colored 2-nitro-5-mercaptobenzoate (23).

Now you are through the core and satellite modules, but there are still some activities remaining as stated below:

- 1. Read the task analysis of the different categories of the health team on Unit 4.**
- 2. Do the questions of pretest as posttest.
N.B. Use a separate answer sheet.**
- 3. Compare your answers of the pre and posttests with the answer keys given on ANNEX I and evaluate your progress.**

3.5. SATELLITE MODULE FOR HEALTH EXTENSION WORKERS

3.5.1 Purpose and Use of the Module:

Health Extension Package Workers, as they will be playing vital roles in the betterment of the health of the community, are expected to have basic information about the most important health problems of the country, common among which are food-borne diseases. This module aims at providing them with some of this information so as to enable them to recognize food-borne illnesses and outbreaks, refer cases for proper therapy (in the mean time providing basic treatment), and to prevent them from occurring.

3.5.2 Directions for Using the Module:

- Do the pre-test given below using a separate answer sheet for your answers.
- Read the material thoroughly including the section on task analysis for health extension package workers presented in section 3.4.15.

3.5.3 Pre-test:

Write the letter of your best choice for each of the following questions on a separate answer sheet.

1. One of the following is not a food-borne disease:
 - A. Typhoid fever
 - B. Typhus
 - C. Cholera
 - D. Shigellosis
 - E. None of the above.
2. Some food items which may lead to food-borne diseases include:
 - A. Raw vegetables not properly washed or washed with contaminated water
 - B. Raw meat (e.g., “kurt”)
 - C. Undercooked meat (e.g., “kitfo lebleb”)
 - D. Well-cooked meal kept in open overnight and eaten for breakfast in the next morning
 - E. All of the above can be sources for food-borne diseases.

3. Identify the false statement:
- A. All food-borne diseases can be prevented only by cooking all foods adequately.
 - B. The presence of latrine helps to reduce the transmission of food-borne illnesses.
 - C. Flies and cockroaches can be very important vectors in the transmission of food-borne diseases.
 - D. Early and proper treatment of patients with food-borne diseases helps to reduce the spread of the diseases.
 - E. None of the above
4. Which one of the following statements is true regarding the management of patients with food-borne diseases?
- A. All patients with diarrhea should be advised to take more fluid diets than usual.
 - B. All patients with diarrhea need to be given antibiotics such as tetracycline.
 - C. Diarrheal stools have to be disposed of carefully as they may also transmit HIV/AIDS.
 - D. An ill patient without diarrhea cannot be having a food-borne disease.
 - E. All of the statements are true.
5. If all patients who ate from a similar dish or in similar ceremony got ill with a similar kind of illness, then the problem has high likelihood of being related to:
- A. The hygienic practices of the individual(s) who prepared the food
 - B. The environment in which the food was prepared
 - C. The conditions in which the food was stored after preparation but before being served
 - D. The hygienic practices of the individuals who got sick
 - E. All except D
6. Patients who are infected with worms but are not excreting worms in their stools cannot be sources of infection for other individuals.
- A. True
 - B. False

7. Proper disposal of human excrement helps to reduce the transmission of food-borne diseases by flies to prepared food and also by preventing contamination of soil and vegetations with infective organisms.
- A. True
 - B. False

3.5.4 Learning Objectives:

- Define food-borne diseases
- List some common food-borne diseases in Ethiopia
- Describe the most important manifestations of some food-borne diseases
- Refer patients with food-borne diseases and, in the mean time, provide basic treatment
- Educate the public and advise individuals on how to prevent food-borne diseases

3.5.5 Definition:

Food-borne diseases are those diseases acquired following the ingestion of infective organisms, toxins, or chemicals together with food items or following the ingestion of poisonous plant or animal tissues or products.

3.5.6 Epidemiology

The different types of food-borne diseases are among the major causes of sickness, being responsible for large numbers of outpatient visits, hospital admissions and deaths. There are many factors that contribute to this condition, some of which are poor personal hygiene and environmental sanitation, grossly inadequate safe water supply, poor food preparation and storage of food items, and others.

3.5.7 Causes:

1. **Bacteria:** e.g., typhoid fever, shigellosis, E. coli infection, cholera
2. **Parasites** (the infective stages are microscopic cysts, eggs or larvae): e.g., amebiasis, giardiasis, ascariasis, tapeworm infection
3. **Viruses:** e.g., hepatitis, viral diarrhea
4. **Fungal toxins** such as aflatoxin

5. **Chemicals:** e.g., insecticides (malathion, etc.), heavy metals (lead, mercury, etc.)
6. **Poisonous plants:** e.g., mushrooms, “guaya”

3.5.8 Transmission (Modes of Acquisition):

The most important modes of transmission of food-borne diseases are:

1. Ingestion of raw or undercooked meat and meat products
2. Ingestion of raw milk
3. Ingestion of food contaminated with human feces (directly or indirectly)
4. Ingestion of raw vegetables contaminated with soil, human feces, etc.
5. Accidental ingestion of chemicals such as malathion together with food
6. Ingestion of poisonous plants intentionally as food items (“guaya”, mushrooms) or unknowingly (mushrooms, etc.)
7. Ingestion of food prepared using contaminated water, e.g., for washing vegetables
8. Ingestion of food kept in an unsuitable condition for long time after preparation (this creates conducive environment for the flourishing of micro-organisms on the food), especially if it has remained exposed to flies, roaches, etc.

Some common contaminants of foods

Figure 3.5.1 below illustrates some of the various biological or chemical contaminants of foods (2,19).

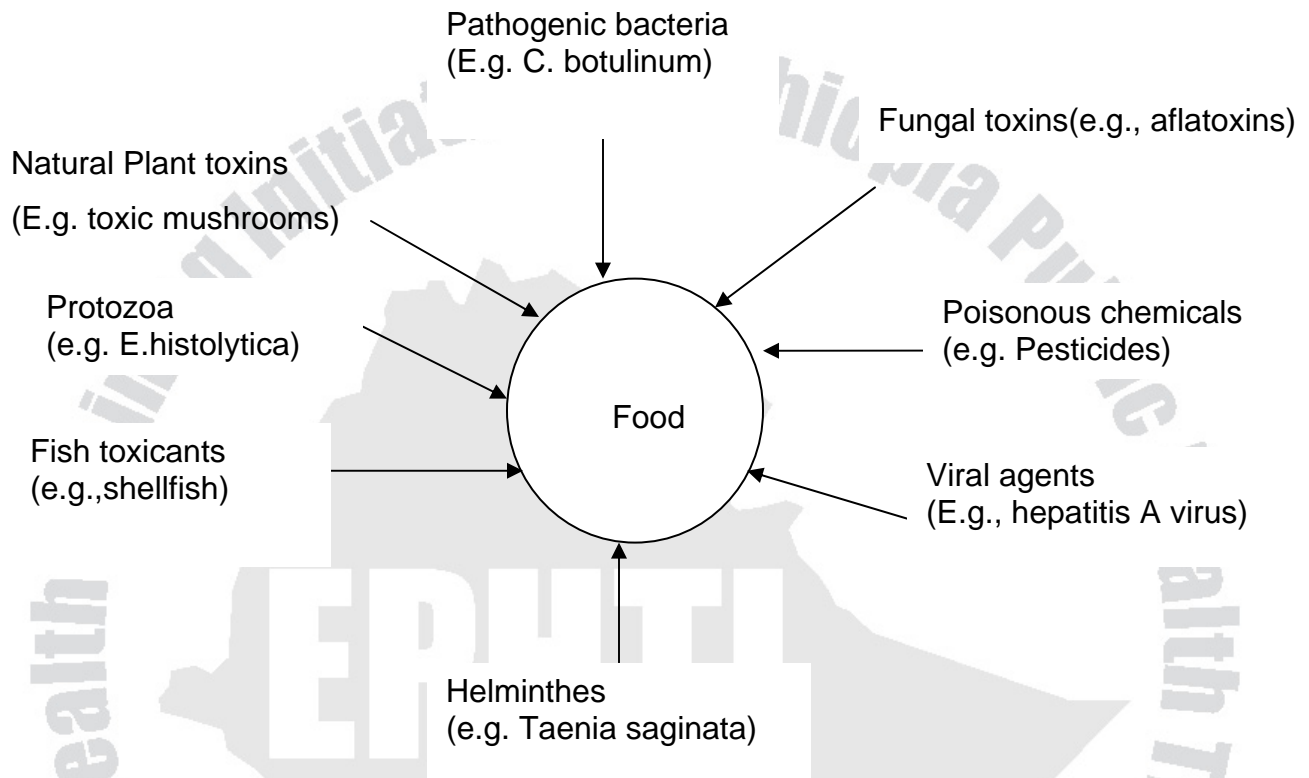


Figure 3.5.1 Contaminants of food

3.5.9 Sources of contamination of food:

Food can be contaminated all the way from the source of production (farm) until consumption, i.e., Farm ----Harvest -----Transportation-----Storage -----Distribution ---- --Processing -----Preparation -----Serving ----- Consumption. Food products are rich in nutrients required by microorganisms, which may lead to multiplication of the organisms to great extent if contaminated. Major contamination sources for foods include (4,7,19):

- **Water:** If a safe water supply is not used in processing and preparation of food it then becomes a source of contamination of the food (chemical or biological agents).

- **Sewage:** Raw, untreated sewage can contain pathogens that have been eliminated from the human body, as well as other materials including toxic chemicals from the environment. If raw sewage is used to irrigate vegetable farms, it can be a source of food contamination.
- **Equipment:** contamination of equipments used for processing, preparing or serving food occurs during production (manufacture) and when the material is not properly cleaned.
- **Food handlers:** The hands, hair, nose, and mouth harbor microorganisms that can be transferred to food during processing, packaging, preparation, and service by touching, breathing, coughing, or sneezing. Of all the viable means of exposing microorganisms to food, employees are the largest contamination source.
- **Insects and rodents:** Flies, cockroaches and rodents are associated with living quarters, eating establishments, and food processing facilities, as well as with toilets, garbage, and other filth. These animals transfer contaminants to food through their waste products; mouth, fur, intestinal tract, feet, and other body parts; and during regurgitation onto clean food during consumption.
- **Soil:** Soil may contain microorganisms as well as poisonous chemicals. These agents may get access to food either due to direct contamination or through dusts.
- **Other animals' bodies:** From the intestinal tracts of animals, microorganisms find their way directly to the soil and water. From there, they may find their way into plants, dust, utensils and/or food. Meat of animals can get contaminated during slaughtering, cutting, processing, storage, and distribution. Other contamination can occur by contact of the carcass with the hide, feet, manure, dirt, and visceral contents. Like wise drugs used to prevent disease and promote growth in animals may also become potential risk for human health due to persisting of these drugs in the meat or milk products.
- **Others:**
 - Mistaken use of a toxic chemical in the preparation seasoning or sweetening of food or by children believing it is a drink.

- Deliberate and malicious contamination of food by a person for some irrational reason.
- Water polluted by chemicals from farm and or spraying food trees (4,6).

3.5.10 Factors most commonly contributing to food-borne disease outbreaks

There are a number of factors that may lead to the occurrence of food-borne illness outbreaks. The major ones are:

- Preparation of food more than half a day in advance of needs
- Storage at ambient temperature
- Inadequate cooling
- Inadequate reheating
- Use of contaminated processed food (cooked meats and poultry, and the like)
- Undercooking
- Cross contamination from raw to cooked food from utensils, and unhygienic kitchen environment
- Infected food handlers or poor personal hygiene of food handlers
- Unsanitary dishware, utensils and equipment
- Improper food handling procedures such as unnecessary use of the hands during preparation and serving of food
- Improper food storage that may lead to cross contamination by agents of diseases (micro-organisms, poisonous chemicals), or exposure to moisture that may facilitate microbial growth
- Insects and rodents (4,13).

3.5.11 Some common food-borne diseases: their etiology and foods involved

1. Food infections

| Etiologic Category | Diseases | Causative organisms | Foods commonly involved |
|---------------------------|-------------------|--|--|
| 1. Bacterial | Typhoid fever | Salmonella typhi and paratyphi | Raw vegetables and fruits, salads, pastries, un-pasteurized milk and milk products. |
| | Shigellosis | Shigella species | All foods handled by unsanitary workers, potato or egg salad, lettuce, raw vegetables |
| | Cholera | Vibrio cholerae | Fruits and vegetables washed with contaminated water |
| | Bovine TB | M. Bovis | Un-pasteurized milk or dairy products from tuberculous cows. |
| | E.coli infections | E.coli | Beef, dairy products, fresh products, raw produce (potatoes, lettuce, sprouts, fallen apples), salads. |
| Etiologic Category | Diseases | Causative organisms | Foods commonly involved |
| 2. Viral | Viral GE | Rota virus, Norwalk virus, calici virus, astro virus | Any food of daily use with poor hygiene |
| | Viral hepatitis | Hepatitis A & E | Raw shellfish from polluted water, sandwich, salad, and desserts. |
| | Poliomyelitis | Polio virus | Any food of daily use with poor hygiene |
| 3. Parasitic | Taeniasis | Taenia species | Raw beef, raw pork |
| | Amoebiasis | <i>Entameba histolytica</i> | Any food soiled with feces |
| | Ascariasis | <i>Ascaris lumbricoides</i> | Foods contaminated with soil, specially foods that are eaten raw such as salads, vegetables |
| | Giardiasis | <i>Giardia lamblia</i> | Foods contaminated with feces |

2. Food poisonings/intoxications

| Etiologic Category | Disease | Causative agent | Foods commonly involved |
|-----------------------------------|---|--|---|
| A. Natural toxins in Foods | 1. Neurolathyrism | Beta oxalyl amino–alanine | “Guaya” (<i>Lathyrus sativus</i>) |
| | 2. Mushroom poisoning | Phalloidine and alkaloids found in some poisonous mushrooms. | Poisonous mushrooms such as species of <i>Amanita phalloides</i> and <i>Amanita muscaria</i> |
| B. Bacterial toxins | 1. Staphylococcal food poisoning | Enterotoxin from <i>Staphylococcus aureus</i> | Milk and milking products, sliced meat, poultry, potato salad, cream pastries, egg salad |
| | 2. Perfringens food poisoning | Strains of <i>Clostridium welchii</i> / <i>C. perfringens</i> | Inadequately heated or reheated meat, poultry, legumes |
| | 3. Botulism food poisoning | Toxin of <i>Clostridium botulinum</i> | Home-canned foods, low acid vegetables, corn and peas. |
| | 4. <i>Escherichia coli</i> food poisoning | Enterohemorrhagic <i>Escherichia coli</i> O157:H7 | Ground beef, dairy products, raw beef. |
| | 5. <i>Bacillus cereus</i> food poisoning | Enterotoxin of <i>Bacillus cereus</i> | Cereals, milk and dairy products, vegetable, meats, cooked rice. |
| C. Fungal toxins | 1. Ergotism | A toxin (ergot) produced by a group of fungi called <i>Clevises purpurea</i> | Rye, wheat, sorghum, barley |
| | 2. Aflatoxin food poisoning | Aflatoxin produced by some groups of fungus (e.g. <i>Aspergillus flavus</i> , <i>Aspergillus parasites</i>) | Cereal grains, ground nuts, peanuts, Cottonseed, sorghum. |
| D. Chemical food poisoning | Chemical poisoning | Heavy metals (e.g. Lead, mercury, cadmium) | - Fish, canned food - Foods contaminated by utensils made or coated with heavy metals |
| | | Pesticides and insecticides | - Residues on crops, vegetables, fruits. - Accidental poisoning where some chemicals may be mistaken with food ingredients. - When contaminated containers are used to hold or store foods. |

| Etiologic Category | Disease | Causative agent | Foods commonly involved |
|--------------------|---------|--------------------------|---|
| | | Additives (unauthorized) | Various food items where unauthorized additives may be added as coloring agents, sweeteners, preservatives, flavoring agents etc. |

3.5.12 Common signs and symptoms of food borne diseases

- ◆ Individuals with food-borne diseases can have many different kinds of manifestations.
- ◆ Some of these manifestations are listed below:
 1. Diarrhea (watery/mucoid/bloody), tenesmus (painful straining at defecation with sensation of inadequate emptying), abdominal pain, nausea, vomiting, bloating, belching, flatulence, abdominal distention
 2. Loss of appetite, loss of general sense of well-being, weakness, unusual hunger sensation, altered taste sensation
 3. Fever, chills, headache, muscle and joint pains,
 4. Paralysis
 5. Symptoms of fluid loss like thirst, weakness, dizziness, low blood pressure, fast pulse rate, poor skin turgor, sunken eyeballs,
 6. Yellowish discoloration of the eyes and skin, weight loss,
 7. Passage of worms in the stool and sometimes through the mouth, itching and discomfort in the perianal area
 8. Growth failure in children

3.5.13 Management:

- ◆ All patients suspected of having a food-borne disease should be immediately referred to the nearby health facility for determination of the specific cause and proper treatment.
- ◆ However, in the meantime, there are lots of supportive and other interventions that Health Extension Package Workers can do to help the patient and his/her family. For example:

1. Assess the level of dehydration and the presence or absence of visible blood in the stool in all patients with diarrhea; if there are evidences of significant fluid loss or if there is visible blood in the stool, refer the patient immediately to the nearby health center for proper treatment.
2. If a patient has diarrhea, advice him/her to take more of the fluid diets prepared at home such as gruel (“atmit”), tea, soup, boiled milk, etc. as long as the diarrhea is there. In addition, if there is ORS at hand provide the individual with some sachets and instruct him/her carefully on how to prepare and use the solution.
3. If a patient has fever, advice him/her and the family to use mechanical means of cooling the body such as tepid sponging;
4. Advice patients and their families on the importance of proper personal hygienic measures at home, particularly during food preparation, in order to prevent the infection from disseminating to other individuals

3.5.14 Prevention and Control:

The roles that Health Extension Package Workers can and should play in the prevention and control of food-borne diseases in particular and infectious diseases in general, are many.

Some of these roles are:

1. Provision of information and education on the means of transmission of food-borne diseases and their methods of prevention at household levels such as
 - Proper disposal of human excrement and other wastes,
 - Proper hand washing always after using the toilet and before and during food preparation and serving,
 - Keeping compound sanitation so as to prevent the breeding of flies, rats and roaches,
 - Keeping already prepared food items in the proper place and environmental conditions,
 - Proper cooking of animal foods before consumption,
 - Boiling of milk,

- Proper washing and cooking of vegetables
- Other important methods that facilitate the safety of food include the following:
 - Health education
 - Good personal and environmental hygiene
 - Availability of safe, ample and convenient water supply
 - Training of food handlers and managers on hygienic food preparation and handling
 - Stringent inspection and control actions
 - Legislative support (ordinances and codes), licensing
 - Good-house keeping practices including separate storage and care of toxic chemicals.
 - Understanding about additives and restrictions of unauthorized use.
 - Food equipment selection to avoid chemical poisoning arising from the material constituency and or coatings of some food utensils.
 - Avoidance and care of insecticide use in food processing and preparation areas.
- 2. Education of the public at large on the above issues as well as avoidance of consumption of potentially harmful plants
- 3. Advising patients and families to seek immediate medical help in the event of any food-borne illness
- 4. Searching for cases and referring to nearby health institution for proper management; this is particularly so when there is anyone with some form of food-borne illness in the community since there may be several others with the same problem who may have manifestations or may have not started to show them yet.

This brings you to the conclusion of the satellite module prepared for Health Extension Workers. What remains now is to:

- **Read the task analysis for Health Extension Package Workers in section 3.5.15**
- **Do the pre-test on section 3.5.3 as a post-test**
- **Compare your responses against the keys given on section 3.5.16.**

3.5.15: Task Analysis for Health Extension Workers

Table3. 5.1. Knowledge Objectives and Learning Activities

| No | Learning Objectives | Learning Activities |
|----|---|--|
| 1 | To define food-borne diseases | Define food-borne diseases |
| 2 | To classify food-borne diseases | Classify food-borne diseases |
| 3 | To describe the epidemiology of common food-borne diseases | Describe the magnitude of common food-borne diseases |
| 4 | To identify the etiologic agents of common food-borne diseases | Identify the etiologic agents of common food-borne diseases |
| 5 | To describe the clinical features of common food-borne diseases | List the major symptoms and signs of common food-borne diseases |
| 6 | To describe the management approach for common food-borne diseases | Describe the general management approaches for food-borne diseases |
| 7 | To explain the preventive and control measures for common food-borne diseases | <ul style="list-style-type: none"> -Discuss the general preventive and control measures for food-borne diseases -List environmental measures used in prevention and control of food-borne diseases |
| 8 | Outline the steps in the investigation of food-borne disease outbreaks | -Identify the most common factors responsible for food-borne disease outbreaks |

Table 3.5.2. Attitude Objectives and Learning Activities

| No | Learning Objectives | Learning Activities |
|----|--|---|
| 1 | Consider that food-borne diseases are a major public health problem | Recognize food borne diseases are one of the major public health problems in Ethiopia |
| 2 | Believe that improper handling of food can result in food borne diseases. | Believe increasing public awareness improves food handling practices |
| 3 | Appreciate preventive measures are more important than treatment in food borne disease. | Emphasize on preventive measures |
| 4 | Believe that food borne diseases can occur in the form of outbreak. | Emphasize on health education to prevent outbreak occurrence |
| 5 | Believe that the causes are not attributed to only microbial agents | Consider possibilities of non-microbial causes of food borne diseases |
| 6 | Consider that the role of food handlers is crucial in food borne diseases. | Emphasize on training of food handlers |
| 7 | Believe that some food borne diseases are fatal thus need immediate intervention | Emphasize on timely intervention |
| 8 | Believe that food borne diseases are preventable | Emphasize on prevention |
| 10 | Appreciate the role of different category of the health team in the prevention, control and management of food born diseases | Believe on team approach |

Table 3.5.3. Practice Objectives and Learning Activities

| No | Learning Objectives | Learning Activities |
|----|--|---|
| 1 | To identify a case of food borne disease | <ul style="list-style-type: none"> -Assess the environmental risk factors -search for the possible source of the disease |
| 2 | To manage a case of food borne disease appropriately | <ul style="list-style-type: none"> -Eliminate/minimize the environmental risk factors -Advise to visit health institutions promptly -Provide health information |
| 3 | To apply proper preventive and control measures | <ul style="list-style-type: none"> -Deliver health information -Search for possible common source of the disease -Organize and mobilize the health team and the community to participate in preventive and control measures - Apply appropriate control measures based on the assessment of the environment |
| 4 | To manage outbreaks of food borne diseases | <ul style="list-style-type: none"> -Apply appropriate interventions -Notify concerned authorities |
| 5 | Implement disease surveillance | <ul style="list-style-type: none"> -Report food borne disease outbreaks in time |

3.5.16 Keys to Pre and Post-tests (for health extension workers).

1. B
2. E
3. A
4. A
5. E
6. B
7. A



UNIT FOUR

TAKE HOME MESSAGES FOR CARE GIVERS/ SELF-CARE

- Food-borne diseases are caused by ingestion of food contaminated with poisonous substances or germs. Germs are tiny organisms that cannot be seen with the naked eye.
- If several people consume contaminated food from the same source, they may be affected by a similar type of sickness.
- The manifestations of food-borne sicknesses are many in type, e.g. diarrhea, vomiting, fever, abdominal pain, bloody stool, muscle weakness, etc.
- If an individual is possibly affected by a food-borne illness, he/she should seek medical help from health facilities as soon as possible.
- If not treated in time, food-borne illnesses can lead to serious and sometimes life-threatening problems.
- There are different measures that can be taken at home to prevent food-borne diseases:
 - Proper personal hygiene including washing the hands with ample water and soap every time after using the toilet, before preparing, serving or eating any food.
 - Construction and proper utilization of sanitary latrines and waste disposal pits.
 - Proper storage and preservation of food kept for long periods without consumption. Store foods off floor, in dry and well ventilated room. Perishable foods need to be kept in cool areas and preferably in refrigerators.
 - Consuming adequately cooked foods, and while hot.
 - Avoiding foods known to have harmful effects due to the presence of toxins, etc.
 - Using clean water from protected sources for preparation of food, washing dishware, and drinking.

- Wash food utensils preferably in three compartments: the first with warm water and detergent for washing, the second with warm clean water for rinsing, and the third with very hot water for sanitizing (disinfecting). Finally dry the utensils in air without the need for swabbing with a cloth to dry them. Swabbing may cross contaminate the utensils.
- Getting rid of flies, cockroaches, and rats
- Keeping cooked foods always properly covered if not immediately consumed so that they will not be contaminated with dust or by house flies
- Maintain good house keeping practice.



UNIT FIVE

TASK ANALYSIS FOR THE DEGREE TEAM MEMBERS

TABLE 5.1. KNOWLEDGE OBJECTIVES AND ACTIVITIES

| No | Learning Objectives | Learning Activities | | | |
|----|---|---|---|---|--|
| | | HO | NURSE | EHO/ | MLT |
| 1 | To define food-borne diseases | Define food-borne diseases | Define food-borne diseases | Define food-borne diseases | Define food-borne diseases |
| 2 | To classify food-borne diseases | Classify food-borne diseases | Classify food-borne diseases | Classify food-borne diseases | Classify food-borne diseases |
| 3 | To describe the epidemiology of common food-borne diseases | Describe the magnitude of common food-borne diseases | Describe the magnitude of common food-borne diseases | Describe the magnitude of common food-borne diseases | Describe the magnitude of common food-borne diseases |
| 4 | To identify the etiologic agents of common food-borne diseases | Identify the etiologic agents of common food-borne diseases | Identify the etiologic agents of common food-borne diseases | Identify the etiologic agents of common food-borne diseases | Identify the etiologic agents of common food-borne diseases |
| 5 | To explain the pathogenesis of common food-borne diseases | Explain the pathogenesis of common food-borne diseases | Explain the pathogenesis of common food-borne diseases | Indicate the most important pathogenic factors for common food-borne diseases | Indicate the most important pathogenic factors for common food-borne diseases |
| 6 | To describe the clinical features of common food-borne diseases | Explain the clinical features and disease course of common food-borne diseases | Explain the clinical features and disease course of common food-borne diseases | List the major symptoms and signs of common food-borne diseases | List the major symptoms and signs of common food-borne diseases |
| 7 | To state the diagnostic methods for common food-borne diseases | Describe the diagnostic methods (clinical, laboratory, etc.) for common food-borne diseases | Describe the diagnostic methods (subjective and objective assessments) for common food-borne diseases | Mention the laboratory diagnostic methods of common food-borne diseases | Explain detailed laboratory diagnostic procedures for common food-borne diseases |

TABLE 5.1. KNOWLEDGE OBJECTIVED AND ACTIVITIES (CONTINUED)

| No | Learning Objectives | Learning Activities | | | |
|----|---|--|--|---|--|
| | | HO | NURSE | EHO | MLT |
| 8 | To describe the management approach for common food-borne diseases | Describe the general and specific management measures for common food-borne diseases | Describe the nursing management approaches for common food-borne diseases | Describe the general management approaches for food-borne diseases | Describe the general management approaches for food-borne diseases |
| 9 | To explain the preventive and control measures for common food-borne diseases | Discuss the general and specific preventive and control measures for food-borne diseases | Discuss the general and specific preventive and control measures for food-borne diseases | <ul style="list-style-type: none"> ➤ Discuss the general and specific preventive and control measures for food-borne diseases ➤ Describe environmental measures used in prevention and control of food-borne diseases | Discuss the general preventive and control measures for food-borne diseases |
| 10 | Outline the steps in the investigation of food-borne disease outbreaks | Outline the steps in the investigation of food-borne disease outbreaks | Outline the steps in the investigation of food-borne disease outbreaks | <ul style="list-style-type: none"> ➤ Outline the steps in the investigation of food-borne disease outbreaks ➤ Identify the most common factors responsible for food-borne disease outbreaks ➤ List sample collection procedures for food-borne disease outbreaks | <ul style="list-style-type: none"> ➤ Outline the steps in the investigation of food-borne disease outbreaks ➤ Identify the laboratory procedures used for investigation of common food-borne disease outbreaks |

TABLE 5.2. ATTITUDE OBJECTIVES AND ACTIVITIES

| No | Learning Objectives | Learning Activities | | | |
|----|--|---|---|---|---|
| | | HO | NURSE | EHO | MLT |
| 1 | Consider that food-borne diseases are a major public health problem | Recognize food borne diseases are one of the major public health problems in Ethiopia | Recognize food borne diseases are one of the major public health problems in Ethiopia | Recognize food borne diseases are one of the major public health problems in Ethiopia | Recognize food borne diseases are one of the major public health problems in Ethiopia |
| 2 | Believe that improper handling of food can result in food borne diseases. | Believe increasing public awareness improves food handling practices | Believe increasing public awareness improves food handling practices | Believe increasing public awareness improves food handling practices | Believe increasing public awareness improves food handling practices |
| 3 | Appreciate preventive measures are more important than treatment in food borne disease. | Emphasize on preventive measures | Emphasize on preventive measures | Emphasize on preventive measures | Emphasize on preventive measures |
| 4 | Believe that food borne diseases can occur in the form of outbreak. | Emphasize on health education to prevent outbreak occurrence | Emphasize on health education to prevent outbreak occurrence | Emphasize on health education to prevent outbreak occurrence | Emphasize on health education to prevent outbreak occurrence |
| 5 | Believe that the causes are not attributed to only microbial agents | Consider possibilities of non-microbial causes of food borne diseases | Consider possibilities of non-microbial causes of food borne diseases | Consider possibilities of non-microbial causes of food borne diseases | Consider possibilities of non-microbial causes of food borne diseases |
| 6 | Consider that the role of food handlers is crucial in food borne diseases. | Emphasize on training of food handlers | Emphasize on training of food handlers | Emphasize on training of food handlers | Emphasize on training of food handlers |
| 7 | Believe that some food borne diseases are fatal thus need immediate intervention | Consider the timely management of a food borne disease | Consider the timely management of a food borne disease | Emphasize on timely intervention | Consider the timely reporting of lab. findings |
| 8 | Consider the importance of laboratory investigations in diagnosis and management of food borne diseases | Think of the appropriate lab. tests | Think of the appropriate lab. Tests | Think of the appropriate lab. Investigations in outbreaks | Emphasize on appropriate lab. tests and procedures |
| 9 | Believe that food borne diseases are preventable | Emphasize on prevention | Emphasize on prevention | Emphasize on prevention | Emphasize on prevention |
| 10 | Appreciate the role of different category of the health team in the prevention, control and management of food born diseases | Believe on team approach | Believe on team approach | Believe on team approach | Believe on team approach |

TABLE 5.3. PRACTICE OBJECTIVES AND ACTIVITIES

| No | Learning Objectives | Learning Activities | | | |
|----|--|--|--|---|--|
| | | HO | NURSE | EHO | MLT |
| 1 | To identify a case of food borne disease | -Take appropriate history and carryout physical examination -request and interpret necessary laboratory tests | make a subjective and objective assessment, interpret the data and reach on specific diagnosis | -assess the environmental risk factors -search for the possible source of the disease | Perform specific diagnostic laboratory procedures and identify the organisms |
| 2 | To manage a case of food borne disease appropriately | -Prescribe appropriate treatment -advise the importance of drug compliance -follow up the response to treatment | -administer the appropriate drugs -carryout the nursing interventions -evaluate the response of the patient | Eliminate/minimize the environmental risk factors -Advise to visit health institutions promptly -provide health information | -conduct laboratory tests to diagnose and monitor response to treatment as required -carryout antimicrobial drug sensitivity test (as applicable) |
| 3 | To apply proper preventive and control measures | -Deliver health information -Trace contacts -Search for possible common source of the disease -Organize and mobilize the health team and the community to participate in preventive and control measures -Undertake mass treatment and/or chemoprophylaxis if necessary - | -Deliver health information -Trace the exposed -Search for possible common source of the disease -Organize and mobilize the health team and the community to participate in preventive and control measures -Undertake mass treatment and/or chemoprophylaxis if necessary | -Deliver health information -Trace the exposed -Search for possible common source of the disease -Organize and mobilize the health team and the community to participate in preventive and control measures -Design appropriate control measures based on the assessment of the environment | -Deliver health information - Participate in mass treatment and/or chemoprophylaxis if necessary |
| 4 | To manage outbreaks of food borne diseases | -Verify the diagnosis of the outbreak -Design and apply appropriate interventions -Notify concerned authorities -Devise and implement monitoring tools | -Verify the diagnosis of the outbreak -Design and apply appropriate interventions -Notify concerned authorities -Devise and implement monitoring tools | -Verify the diagnosis of the outbreak -Design and apply appropriate interventions -Notify concerned authorities -Devise and implement monitoring tools | -Identify the causative agent of the outbreak -Participate in designing and applying of appropriate interventions -Participate in the monitoring of the outbreak |
| 5 | Implement disease surveillance | -Report notifiable diseases in time -Monitor outbreaks | -Report notifiable diseases in time -Monitor outbreaks | -Report notifiable diseases in time -Monitor outbreaks | -Report notifiable diseases in time -Monitor outbreaks |

UNIT SIX

ABBREVIATIONS AND GLOSSARY

A. ABBREVIATIONS

| | |
|-------|--|
| BID | Bis In Die (twice a day) |
| CNS | Central Nervous System |
| ELISA | Enzyme-Linked ImmunoSorbent Assay |
| EPHTI | Ethiopia Pulbic Health Training Initiative |
| HACCP | Hazard Analysis and Critical Control Point |
| IM | Intra <u>m</u> uscular |
| IV | Intra <u>v</u> enous |
| KIA | Kliger Iron Agar |
| MIU | Motility Indole Urea |
| ORS | Oral Rehydration Salts |
| PO | Per Os (through the mouth) |
| PT | Prothrombin Time |
| PTT | Partial Thromboplastin Time |
| QID | Quater In Die (four times a day) |
| SC | Sub <u>c</u> utaneous |
| TID | Ter in Die (three times a day) |

B. GLOSSARY

- Antidote:** a drug or other substance that antagonizes or abolishes the effect of a poison or toxin.
- Blanching:** treating vegetables, etc. with heat, e.g. steam or boiling water, briefly before freezing; it inactivates enzymes altogether and reduces discoloration and nutrient loss
- Canning:** a process of preserving food by heating and sealing it in airtight container. The can is filled with food, and air is pumped out of the space remaining at the top of the can to form a vacuum. the container is sealed, heated in a cooker, and then cooled to prevent overcooking of the food inside. It is used to preserve a wide variety of foods, including soups, sauces, fruits, vegetables, juices, meats, fish, and some dairy products.
- Cathartic:** a substance that aids bowel movement by exciting intestinal waves (peristalsis), increasing the bulk of feces, making the feces soft, or adding slick fluid to the wall of the intestines.
- Caustic substances:** any substance that destroys living tissue, or causes burning or scarring, as silver nitrate, nitric acid, or sulfuric acid
- Cholestasis:** stasis or interruptio of the flow of bile through any part of the biliary system, within and from the liver to intestine
- Cyanosis:** bluish discoloration of the skin and mucous membranes from lack of oxygen
- Defervescence:** dropping or disappearance of a fever
- Endotoxin:** a toxin produced within a micro-organism and liberated when the micro-organism disintegrates.
- Enterotoxin:** an exotoxin that acts on the intestine
- Epidemic:** the occurrence of a disease or diseases with a greater than normal (usual) rate of occurrence in a population
- Exotoxin:** a toxin excreted by a microbe into the surrounding medium.

- Hazard:** a situation or thing that increases the chance of a loss from some danger that may cause injury or illness
- Hazardous waste:** solid, liquid, or gas wastes that can cause death, illness, or injury to people or destruction of the environment if improperly treated, stored, transported, or discarded. Substances are considered to be hazardous wastes if they are ignitable, corrosive, reactive, or toxic.
- Hygiene:** practices necessary for establishing and maintaining good health.
- Intussusception:** the sinking of one part of the bowel into the next, like a telescope effect.
- Leukemoid reactions:** an abnormal condition resembling leukemia in which the white blood cell count rises in response to an allergy, inflammatory disease, infection, poison, hemorrhage, burn or other causes of severe physical stress.
- Mycotoxins:** compounds or metabolites produced by a wide range of fungi that have toxic or other adverse effects on humans and animals
- Outbreak:** an epidemic referring to a more localized situation.
- Pasteurization:** the process of applying heat at certain degree for a specified period, usually immediately followed by cooling, most often to milk or cheese to kill or slow the growth of harmful bacteria
- Sanitation:** the creation and maintenance of hygienic and healthful conditions
- Sitz bath:** also called *hip bath*, literally (German) "seat" bath, a bath in which only the hips and buttocks are soaked in water, saline or other solution
- Spore:** an inactive, resistant, resting, or reproductive body that can produce another vegetative individual under favorable conditions
- Syndrome:** a constellation of symptoms and signs
- Toxin:** a chemical produced by living organisms that is poisonous to humans and animals

UNIT SEVEN

ANNEXES

ANNEX I: Answer Keys to Pre-Test and Post-Tests

I. Answer Keys to Pre-test and Post-test for All Degree Categories

1. The term “food borne disease” is defined as a disease usually either infections or toxic in nature, caused by agents that enter the body through the ingestion of food (1).
- 2
 - a. **Food borne infections:** are diseases whose etiologic agents are viable pathogenic organisms ingested with foods and that can establish infection. E.g. Shigellosis
 - b. **Food borne poisonings/ intoxications:** diseases arising from the ingestion of toxins released by microorganisms, intoxications from poisonous plants or toxic animal tissues: or due to consumption of food contaminated by chemical poisons. E.g. Botulism
3. The spectrum of food borne diseases is constantly changing. New and re-emerging food born illnesses have resulted from recent changes in human demographics, international travel and commerce, microbial adaptations, economic development, technology and industry, eating behavior and land use. In the last couple of decades a number of diseases thought to be of unknown causes have been proven to result from food borne infections.

In developing countries like Ethiopia in particular, the problem attains great proportions due to many reasons; basic among which are poverty, poor environmental sanitation and lack of public health awareness. In these modern day times in which food is usually not consumed immediately following and/or at the site of production, the risks of food-borne diseases are becoming increasingly important; the concern is obviously much more in areas where food storage and preparation safety measures are far from optimum.

4. The extent of diagnostic evolution of food borne diseases can be based on history, clinical features, environmental assessment and laboratory investigations. If applicable, radiological examinations may be implemented.
5. Prevention and control of food–borne diseases, regardless of the specific cause, are based on the same principles:
 - a. Avoidance of food contamination
 - b. Destruction or prevention of contaminants
 - c. Prevention of further spread or multiplication of contaminants.

Specific modes of intervention vary from area to area depending on environmental, economic, political, technology and socio cultural factors.

6. C
7. Refer to Section 2.8.1 A-i to iii and B-i to ii
8. A
9. Refer to Section 2.11
10. Refer to Section 2.12 for the details.

Summary of steps in the investigation of food-borne disease outbreak investigation

1. Verify the existence of an outbreak
2. Compare the current number of cases with the past
3. **Note:** consider seasonal variations
4. Verify the diagnosis
5. Review clinical and laboratory findings
6. Describe the outbreak with respect to time, place and person'
7. Construct an epidemic curve
8. Calculate food-specific attack rates
9. Formulate and test hypotheses
10. Search for additional cases
11. Analyze the data
12. Make a decision on the hypotheses tested
13. Intervene and follow-up
14. Report the investigation.
15. Inform the public on the control and prevention of the outbreak

II. Answer Keys to Pre-test and Post-test for Specific Categories of the Health Team

A. Health Officers

1. D
2. E
3. D
4. B
5. D
6. E

B. Nurses

1. C
2. C
3. D
4. A
5. B
6. D
7. D
8. C

C. Environmental Health Officers

1. D
2. C
3. E
4. B
5. E
6. B
7. E
8.
 - Official samples
 - Informal samples
 - Standard samples
 - Post-seizure samples
 - Documentary sampling.

D. Medical Laboratory Technologists

1. A
2. E
3. B
4. D
5. B
6. D
7. C



ANNEX II: Laboratory Identification of Causes of Food-Borne Diseases

Direct Examination of stool specimen

Direct microscopic examination of stool specimen with physiological saline and Dabell's iodine solutions

Procedure

1. Place a drop of physiological saline in the center of the left half of the slide and place a drop of Dobell's iodine solution in the center of the right half of the slide.
2. With an applicator stick, pick up a small portion of the feces (about 2mg which is as much as the size of a match head) and put on the drop of saline.
3. Mix the feces with the two different drops using the applicator stick to form a homogeneous suspension.
4. Cover each drop with a cover slip. Touch the edge of the drop and gently lower the cover slip onto the slide. Avoid air bubble formation.
5. Examine the saline preparations using the 10x objective for trophozoites (vegetative/motile forms) and cysts as well as oocyst of intestinal protozoa and for any ova or larva of helminthes.
6. Examine the iodine suspension with 40 objective for better identification of the cyst stages of protozoa (iodine will stain the nuclei and the glycogen mass of the cyst).

Features Used To Assist In the Laboratory Identification of Enterobacteria

| Species | Lactose | Oxidase | Citrate | MIU Medium | | | KIA medium | |
|-----------------------|---------|---------|---------|------------|--------|------|------------------|-----|
| | | | | Motility | Indole | Urea | H ₂ S | Gas |
| Escherichia coli | + | - | - | + | + | - | - | + |
| Shigella species | - | - | - | - | d | - | - | - |
| Salmonella typhi | - | - | - | + | - | - | + | - |
| Salmonella partyphi A | - | - | - | + | - | - | - | + |
| Vibrio cholerae | - | + | d | + | + | - | - | - |

d: different strains give different results

H₂S: hydrogen sulphide (blackening)

ANNEX III: Bacterial Food Infections and Poisonings

| Incubation Period, Organisms | Signs and Symptoms |
|----------------------------------|---|
| 1 to 6 hours | |
| <i>Staphylococcus aureus</i> | Nausea, vomiting, diarrhea |
| <i>Bacillus cereus</i> | |
| 8 to 16 hours | |
| <i>Clostridium perfringens</i> | Abdominal cramps, diarrhea, vomiting rare |
| <i>Bacillus cereus</i> | |
| More than 16 hours | |
| Enterotoxigenic <i>E.coli</i> | Watery diarrhea |
| <i>V. cholerae</i> | Watery diarrhea |
| <i>Shigella</i> spp. | Dysentery |
| Enterohemorrhagic <i>E. coli</i> | Dysentery |
| <i>Salmonella</i> spp. | Inflammatory diarrhea |

The above table shows bacterial food infections and poisonings with predominant gastrointestinal manifestations (Modified from Harrison's Principles of Internal Medicine, 15th Edition, 2001).

ANNEX IV: Gastrointestinal Pathogens Causing Acute Diarrhea

| Mechanism | Location | Illness | Stool Findings | Examples Of Pathogens Involved |
|---|-----------------------------|------------------------------------|-----------------------------------|--|
| Non-Inflammatory (Enterotoxin) | Proximal small bowel | Watery diarrhea | No fecal leukocytes | <i>V. cholerae</i> , <i>Enterotoxigenic E. coli</i> , <i>Clostridium perfringenes</i> , <i>Bacillus cererus</i> , <i>Staph. aureus</i> , viral |
| Inflammatory (Invasion Or Cytotoxin) | Colon or distal small bowel | Dysentery or inflammatory diarrhea | Fecal polymorphnuclear leukocytes | <i>Shigella spp.</i> , <i>Salmonella spp.</i> , <i>Enterohemorrhagic E.coli</i> , <i>E. histolytica</i> |
| Penetrating | Distal small bowel | Enteric fever | Fecal mononuclear leukocytes | <i>Salmonella typhi</i> |

The above table shows gastrointestinal pathogens causing acute diarrhea (modified from Harrison's Principles of Internal Medicine, 15th Edition, 2001)

ANNEX V: Chemotherapy of Some Food-Borne Diseases

| No | Food-borne Disease | Antimicrobial Therapy | |
|----|-----------------------------|---|--|
| | | Adults | Children |
| 1 | Typhoid Fever | Chloramphenicol 500 mg po/iv qid for 14 days | 50-100 mg/kg/day po/iv in 4 divided doses for 14 days |
| | | Ampicillin 1 gram po/im/iv qid for 14 days | 100 mg/kg/day po/im/iv in 4 divided doses for 14 days |
| | | Amoxicillin 500 mg po tid for 14 days | 20-40 mg/kg/24 hour in 3 divided doses for 14 days |
| | | Trimethoprim-sulfamethoxazole 160/800 mg po bid for 14 days | 8 mg trimethoprim and 40 mg sulfamethoxazole per kg per day po in 2 divided doses for 14 days |
| | | Ciprofloxacin 500 mg po bid for 10-14 days | Not recommended for children less than 17 years [#] |
| | | Ceftriaxone 1-2 gram IM or slow IV once or in 2 divided doses daily for 5-7 days | 50-75 mg/kg/day IM or slow IV in single or 2 divided doses for 5-7 days |
| 2 | Shigellosis | Ampicillin 500 mg po qid for 5-7 days | 50-100 mg/kg/day in 4 divided doses for 5-7 days |
| | | Chloramphenicol 500 mg po qid for 5-7 days | 20-50 mg/kg/day po in 4 divided doses for 5-7 days |
| | | Trimethoprim-sulfamethoxazole 160/800 mg po bid for 5-7 days | 8 mg trimethoprim and 40 mg sulfamethoxazole per kg per day po in 2 divided doses for 5-7 days |
| | | Nalidixic acid 1 gram po qid for 5-7 days | 50-60 mg/kg/day in 4 divided doses for 5-7 days |
| | | Ciprofloxacin 500 mg po bid for 5-7 days | Not recommended for children younger than 17 years of age [#] |
| | | Ceftriaxone 1-2 gram daily IM or slow IV in single or 2 divided doses for 5 days | 20-50 mg/kg per day IM or slow IV in single for 2 divided doses for 5 days |
| 3 | Cholera | Tetracycline 500 mg po qid for 3-5 days OR Tetracycline 2 gram po stat | Tetracycline 250 mg po qid for 3-5 days for children older than 8 years of age |
| | | Doxycycline 100 mg po bid for 3 days OR Doxycycline 300 mg po stat | 100 mg po stat for children older than 8 years |
| | | Ciprofloxacin 30 mg/kg po stat (maximum 1 gram) | 15 mg/kg bid for 3 days (maximum 500 mg po bid) [#] |
| | | Erythromycin 500 mg po qid for 3 days | 40-50 mg/kg/day in 4 divided doses for 3 days |
| | | Trimethoprim-sulfamethoxazole 160/800 mg po bid for 3-5 days | 8/40 mg/kg/day in 2 divided doses for 3-5 days |
| | | Furazolidone 100 mg po qid for 5-7 days | 1.25 mg/kg po qid for 5-7 days Avoid the drug in infants less than 1 year of age because of the possibility of hemolytic anemia |
| 4 | Acute amebic colitis | Metronidazole 500-750 mg po tid for 5-10 days OR Tinidazole 2 gram PO daily for 3 consecutive days FOLLOWED BY Diloxanide furoate 500 mg po tid for 10 days OR Iodoquinol 650 mg po tid for 20 days OR | Metronidazole 30-50 mg/kg/daily divided into 3 doses for 10 days OR Tinidazole 50-60 mg/kg daily for 3 consecutive days FOLLOWED BY Diloxanide furoate 20 mg/kg/day in 3 divided doses for 10 days OR |

| No | Food-borne Disease | Antimicrobial Therapy | |
|----|-----------------------------|---|--|
| | | Adults | Children |
| | | Paromomycin 500 mg po tid for 10 days Tetracycline 500 mg po qid | Iodoquinol 10-13.3 mg/kg or 333.3 mg/m ² body surface area, tid for 20 days (maximum 1.95 grams in 24 hours) |
| 5 | Amebic liver abscess | As above | As above |
| | | Metronidazole IV infusion 500-750 mg tid until patient is able to complete course with oral drugs | 30 mg/kg daily in 3 divided doses until patient is able to complete course with oral drugs |
| | | Dehydroemetine 1 mg/kg/24 hours in a single IM or SC dose for 8-10 days PLUS Chloroquine 500 mg po bid or 250 mg po qid for 2 days FOLLOWED BY 500 mg po daily or 250 mg po bid for 14-21 days PLUS Diloxanide furoate as above | 1-1.5 mg/kg/24 hours in a single IM or SC dose for 8-10 days PLUS Chloroquine 10 mg/kg po bid or 5 mg/kg po qid for 2 days FOLLOWED BY 10 mg/kg po daily or 5 mg/kg po bid for 14-21 days PLUS Diloxanide furoate as above |
| 6 | Giardiasis | Metronidazole 250-500 mg po tid for 5-7 daysn OR Metronidazole 2 grams once a day for 3 days | 25 mg/kg/24 hour in 3 divided doses for 5 days |
| | | Tinidazole 2 gram po stat | 50-75 mg/kg po stat |
| | | Quinacrine 100 mg po tid for 5-7 days | 2 mg/kd po tid for 5-7 days |
| | | Mepacrine 2 mg/kg/24 hour in 3 divided doses for 5-7 days | Mepacrine 2 mg/kg/24 hour in 3 divided doses for 5-7 days |
| | | Furazolidone 100 mg po qid for 7-10 days | 1.25 mg/kg po qid for 7-10 days Avoid the drug in infants less than 1 year of age because of the possibility of hemolytic anemia. |
| | | Albendazole 400 mg daily for 5 days may be effective | Albendazole 200 mg po once daily for 5 days for children up to 2 years of age; for children 2 years and older, similar dose as for adults |
| 7 | Ascariasis | Piperazine citrate 3.5 grams per day for 2 consecutive day Treatment may be repeated after one week for heavy infection. | 75 mg per kg per day for two consecutive days. Treatment may be repeated after one week for heavy infection. |
| | | Levamisole 120-160 mg po stat | 2.5 mg/kg po stat |
| | | Albendazole 400 mg po stat Treatment may be repeated in three weeks. | Children up to 2 years of age: 200 mg po stat Children 2 years of age and over: as for adults |
| | | Mebendazole 100 mg po bid for 3 consecutive days OR 500 mg po stat Dose may be repeated in 2-3 weeks if required. | Mebendazole 100 mg po bid for 3 consecutive days |
| | | Pyrantel pamoate 10 mg /kg po stat (maximum 1 gram base) May be repeated in 2-3 weeks if required. | Pyrantel pamoate 10 mg /kg po stat (maximum 1 gram base) May be repeated in 2-3 weeks if required. |

| No | Food-borne Disease | Antimicrobial Therapy | |
|----|--------------------|--|--|
| | | Adults | Children |
| 8 | Taeniasis | Niclosamide 2 gram po stat May be repeated in 1 week if required. | <2 years: 500 mg po stat 2-8 years: 1 gram po stat 8 years: 1.5 gram po stat (For both, the dose may be repeated in 1 week if required) |
| | | Albendazole 400 mg po once a day for 3 consecutive days | For children up to 2 years of age: 200 mg po once a day for 3 consecutive days For children 2 years and older: as for adults. |
| | | Praziquantel 600 mg po stat | 5-10 mg/kg po stat for children over 4 years |
| | | Mebendazole 100 mg po bid for 3 days | Same as for adults |
| 9 | Brucellosis | Doxycycline 100 mg po bid combined with rifampicin 600 mg to 900 mg daily given for 8-12 weeks | Children < 7 years rifampicin 15mg/kg combined with TMP+SMZ 8/40 mg /kg for 8-12 weeks |

N.B.

- *The tetracyclines are generally not recommended for children younger than 8 years of age.
- #Use of quinolones in children and adolescents is not generally recommended (they cause arthropathy in weight-bearing joints in young animals) although they may in some circumstances be used for short-term.

ANNEX VI :

A. Principles of the HACCP System

| Principles of the HACCP System | Descriptions/Examples |
|---|---|
| Identify potential hazards | <ul style="list-style-type: none">▪ Select foods to study▪ Construct flow diagram for all product/process operations▪ List all hazards associated with each process step▪ Rank the hazards according to the severity▪ List measures that will eliminate or reduce hazards |
| Determine the critical control points (CCPs) for identified hazards | <p>E.g.</p> <ul style="list-style-type: none">▪ Good personal hygiene▪ Avoidance of cross contamination▪ Cooking and cooling |
| Establish the target levels/tolerance for controlling the CCPs | <ul style="list-style-type: none">▪ Establish requirements that must be met at each critical control point. These requirements should be observable and measurable, using such factors as time, temperature and sensory measures. |
| Establish/Implement monitoring systems for controlling CCPs | <ul style="list-style-type: none">▪ Set out a planned sequence of measurements and observations to assess the degree of control on identified CCPs |
| Take corrective actions | <ul style="list-style-type: none">▪ Identify a predetermined action for whenever the CCP indicates a loss of control▪ This can be achieved by careful examination of the data collected during monitoring and control |
| Verify that the HACCP system is working | <ul style="list-style-type: none">▪ Establish and apply methods to ensure that the HACCP system is working |
| Establish a documentation system for procedures and records | <ul style="list-style-type: none">▪ Develop and maintain procedures and practices for record-keeping |

B. Types of Hazards

| Types of Hazards | Microbiological | Examples |
|------------------|--------------------------|--|
| Biological | Bacteria | <i>Clostridium botulinum</i> <i>Salmonella choleraesuis</i> <i>Salmonella paratyphic A-C</i> <i>Shigella dysenteriae</i> <i>Vibrio cholerae</i> <i>Salmonella spp.</i> <i>Enteropathogenic E. coli</i> <i>Listeria monocytoenes</i> <i>Bacillus cereus and other spp.</i> <i>Campylobacter jejuni</i> <i>Clostridium perfringens</i> <i>Staphylococcus aureus</i> <i>Vibrio parahemolyticus</i> <i>Aeromonas hydrophila</i> |
| | Viruses | <i>Norwalk and Norwalk-like viruses</i> <i>Rotavirus</i> <i>Hepatitis A virus</i> |
| | Parasites | <i>Anisikiasis simplex</i> <i>Ameba</i> <i>Giardia</i> <i>Taenia spp.</i> <i>Trichinella spiralis</i> |
| Chemical | Raw materials | Heavy metals Pesticide/Insecticide residues Antibiotic residues Histamine Toxins |
| | In the process | Refrigerants Lubricants/Hydrocarbons from the process Pest control agents Sanitizing agents Water additive Paints |
| | From packaging materials | Plasticizers Printing code inks Adhesives Lubricants |
| Physical | Natural materials | Bone Skin Connective tissue Contaminating ingredients |
| | Foreign bodies | Insect infestation Glass Metal Plastic Wood paper |

C. Control Measures of Hazards

| Type of Hazard | Control Measures |
|-------------------|--|
| Biological Hazard | <p>Thorough cooking:</p> <ul style="list-style-type: none"> ▪ Holding above 60°C ▪ Refrigeration below 8°C ▪ Heat or cool rapidly <p>Avoid contamination:</p> <ul style="list-style-type: none"> ▪ Good manufacturing practice ▪ Sanitation of plant ▪ Personal hygiene and health ▪ Hand washing ▪ Separation of raw and cooked foods ▪ Protect food from pests ▪ Avoid handling foods that will not be further processed |
| Chemical Hazard | <p>Good agriculture practice Good aquaculture practice Good hygienic practice Good storage practice Prevention of microbial growth</p> |
| Physical Hazard | <p>Detection and removal Separation Air Liquid Sieves and filters Metal detection Magnetic grids/permanent magnets X-ray systems Vision systems Color sorting Human inspection and sorting</p> |

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