Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

NON-MICROBIAL FOOD POISONING

Methodical recommendations
to prepare 3rd year students for practical classes
Discipline "Hygiene and ecology"

Electronic resource

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The methodical recommendations set out the main aspects of the diagnosis and prevention non-microbial food poisoning. For 3rd year students to prepare for practical classes in the discipline "Hygiene and ecology".

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CONTENTS

1. PLANNED LEARNING OUTCOMES

According to the requirements of the educational and professional program, students must

know:

- causes and signs of non-microbial food poisoning;
- migration of toxic and radioactive substances via food chains;
- prevention of non-microbial food poisoning;
- effect of residual amounts of chemicals in food on public health;

be able to:

- identify the products which are toxic by nature; products which become toxic due to storage conditions; products, contaminated with toxic substances (xenobiotics) – heavy metals, pesticides etc.
- demonstrate mastery of methods of hygienic assessment of the impact of foods on public health;
- substantiate the implementation of preventive measures to predict non-microbial food poisoning.

Studying this topic will ensure that students acquire the following **program learning outcomes (PLO):**

- PLO 1. Have thorough knowledge of professional activity structure. To be able to carry out professional activities that require updating and integration of knowledge. To be responsible for professional development, the ability for further professional training with a high level of autonomy.
- PLO 2. Understanding and knowledge of fundamental and clinical biomedical sciences at a level sufficient for solving professional tasks in the health care area.
- PLO 3. Specialized conceptual knowledge, which includes scientific achievements in the field of health care and is the basis for conducting research, critical understanding of problems in the medicine field and related interdisciplinary problems.

- PLO 4. Determinate and identify leading clinical symptoms and syndromes; according to standard methods, using preliminary data of the patient's history, data of the patient's examination, knowledge about the person, his/her organs and systems, establish a preliminary clinical diagnosis of the disease.
- PLO 19. Plan and implement a system of anti-epidemic and preventive measures regarding the occurrence and spread of diseases among the population.
- PLO 21. Search for the necessary information in the professional literature and databases of other sources, analyze, evaluate and apply this information.
- PLO 25. It is clear and unambiguous to convey one's own knowledge, conclusions and arguments on health care problems and related issues to specialists and non-specialists.
- PLO 27. Communicate freely in the official language and in English, both orally and in writing to discuss professional activities, research and projects.

2. INTRODUCTION

Food poisoning is characterized by consumption of common meal, sharing of symptoms and affliction of a sizeable proportion of population involved in a short interval of time. Food poisoning can be caused by bacteria, bacterial toxins, inorganic poisons, plant and animal poisons. Most of the reported outbreaks are bacterial in origin and was discussed in previous topic. The present topic focuses on non-bacterial food poisoning.

For many years, food poisoning of a non-microbial nature occupied a small specific weight among food poisoning. Their share accounted for slightly more than 1-2% of these diseases. However, since the 90s in the last century, the number of non-microbial poisonings began to grow progressively and remains at a high level.

As a rule, non-microbial food poisoning is characterized by a severe course and high mortality, which attracts the attention of health authorities

Non-microbial food poisonings include food poisonings by products of the animal origin (liver of certain fish), food poisonings with the plant origin (mushroom poisonings), food poisonings with chemical substances (for example, poisonings with salts of metals). Mushroom poisonings take the first place among non-microbial food poisonings especially in Ukraine.

Non-microbial toxins or toxic substances in foods for human consumption can be "natural" in that they occur in nature. In certain fungi, lathyrus, cassava and fish, for example, these are the most common toxins. Less common but of great importance are toxins that are artificially added to food, such as various chemicals used to assist in food production, including fertilizers, weed killers, insecticides and fungicides.

Contamination by chemicals from the environment is a major global food safety issue, posing a serious threat to human health. These chemicals belong to many groups, including metals/metalloids, polycyclic aromatic hydrocarbons (PAHs), persistent organic pollutants (POPs), perfluorinated compounds (PFCs), pharmaceutical and personal care products (PPCPs), radioactive elements, electronic waste, plastics, and nanoparticles. Some of these occur naturally in the environment, whilst others are produced from anthropogenic sources. They may contaminate our food—crops,

livestock, and seafood—and drinking water and exert adverse effects on our health. (Figure 1 shows the pathway of contaminants through the environment).

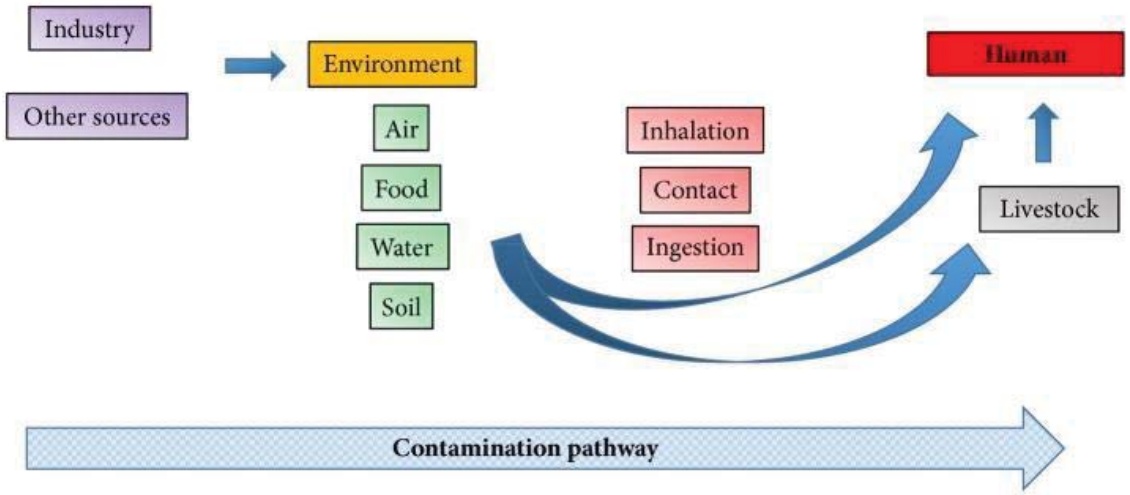


Figure 1. Sources of environmental contaminants in human foods [by Thompson L.A. et al., 2019]

In certain instances, the source of contaminants may be the environment. This is the case for metals such as lead and mercury, dioxins, and polychlorinated biphenyls (PCBs). Agricultural use of pesticides may lead to food contamination. Similarly, drugs used in both people and animals may contaminate waterways and pose a health risk to consumers. Additionally, food packaging methods may be a source of contamination, so-called “migrants” leaching from packing materials. These contaminants may cause acute or chronic toxic effects. Toxicity may relate to the route of exposure and dose, and personal characteristics such as age and health condition may affect the individual's susceptibility.

Due to the nature of contamination, some food products may be more contaminated than others. This may be due to several factors such as varying exposure to pesticides, differences in plant uptake mechanisms from the environment, or contaminants from food packaging. Dietary make-up will affect an individual's exposure to these contaminants. For example, nursing neonates have a high intake of contaminants that are excreted in breast milk. Exposure at different life stages may result in different toxic effects as well. For example, prenatal exposure to persistent organic pollutants has been linked to an increase in childhood obesity and increased

blood pressure.

For many food items – including vegetables, fish, and other seafood – human health risk assessment data is available after analysis of available food. Urban farms and gardens may pose additional risks due to contaminants such as metals. Furthermore, drinking water may become contaminated. Xenoestrogenic compounds have even been detected in rainwater. Water contamination may also result in pollution of marine biota, affecting suitability for consumption of seafood. Consequently, individuals with high consumption of seafood will intake higher levels of such contaminants.

Each of the possible contaminants in food can be linked to a variety of toxic effects. Any adverse effects seen depend on multiple factors, including whether exposure is acute or chronic, the dose received, the route of exposure, and details of the individual person such as age and health. As an example, lead toxicity affects almost all organs, but the most severely affected is the nervous system. In adults, long-term exposure results in reduced cognitive performance. More severe signs such as learning difficulties and behavioural problems are seen in infants and young children as they are more sensitive during this phase of neurodevelopment. High levels of contamination with lead may also cause kidney damage; chronic exposure may cause anaemia and hypertension, and reduced fertility in males. In pregnant women, high blood lead levels are associated with premature birth or babies with a low birth weight, and this risk is increased in emaciated women. On an individual level, blood sampling is a quick and easy method of assessing circulating levels of lead and can be used to indicate recent or current exposure. However, this does not account for lead stored elsewhere in the body, particularly in bones.

On the other hand, at a community level, it may be more important to identify contaminated sites and assess health risks to the general population and thereafter aim to reduce or remove exposure to contaminants such as lead. Thus, monitoring plays a vital role in food safety. Such monitoring has identified contamination of many foods (Table 1). In order to monitor effectively, samples should be analysed from a variety of sources: human samples to detect levels after exposure, diverse foods from the total

diet and drinking water sources, and also the environment itself (to identify the source of food contamination). Samples from people frequently include blood, urine, feces, breast milk, hair, and/or semen. Human biomonitoring is notably useful to facilitate risk assessment. A combination of environmental monitoring and biomonitoring may identify risk factors, such as detection of higher levels of cadmium in umbilical cord blood from mothers consuming more than two portions of fish each week. In the case of metals, environmental sampling has shown hotspots of contamination around mining (such as gold, lead, and zinc), electronic waste sites, and industrial areas. Contamination in soils at these sites has been linked to bioaccumulation in agricultural crops and associated increase in human health risk.

Table 1 – Examples of food contamination with different chemicals around the world.

Food contaminants	Foods	Country
Metals/metalloids		
Pb, Hg	Grains and vegetables	China
Pb, Cd	Livestock organs	Morocco
Pb	Sheep livers	Spain
Pb, Cd, Cu, Zn	Agricultural crops	Romania
Cd	Locally produced foods	Belgium
Tl	Lettuce and chard	Germany
Pb, Cd	Soybeans	Argentina
Cu, Zn, Mn, Fe	Fish	Turkey
Pesticides and polychlorinated biphenyls (PCBs)		
Chlorpyrifos	Catfish	Australia
	Vegetables	China
	Food plant	Algeria
DDTs and other OCPs	Edible offal	Egypt
	Milk	
	Chicken products	South Africa
	Milk	Ethiopia
	Fish	Mozambique

Food contaminants	Foods	Country
PCBs and OCPs	Baby foods	Korea
OCPs and pyrethroids	Honey	Egypt
PCBs and OCPs	Cereals	Poland
PCBs and OCPs	Milk, yak muscle and liver	Tibet Plateau
Radioactive substances		
Radioactive substances	Water and food	Japan
Radionuclides	Seafood	India
Uranium isotopes	Food	Balkans
Radioactive substances	Water and food	Switzerland

Heavy metals are transferred along the plant pathway in various ways. The most common is through soil spores to plants in ionic forms. The capillarity of plants enhances the translocation of heavy metals to the rest of the plant. Therefore, the concentration of heavy metals in plants may vary according to the different parts of the crop plants i.e., roots, leaves, and fruits. The roots of the plants play a vital role in the rate of metal absorption and translocation to the rest of the plant. Different crop species have membranes that may be more adapted to rapid absorption than others

One important factor to consider is the significance of crops such as wheat and maize in developing nations such as South Africa. These crops form the country's staple foods or national diet. They are distributed and sold to communities that may already be immuno-compromised or already under community or household level of food insecurity. The populations likely to be affected include schools under national school feeding and nutrition programs, hospitals, prisons, and indigents receiving food parcels. This is because the above-mentioned population groups do not have a range of options regarding what they can consume.

3. MUSHROOM POISONING

Mushrooms are a great source of nutrition. They are low in calories, fat free, and high in protein, making them an ideal food except for the fact that some are highly toxic if ingested. It is known more than 200 species potentially poisonous

mushrooms, most of them poisonous 20-25. Picking and consumption of wild mushrooms are increasingly popular. This rise in popularity has led to increased reports of severe and fatal mushroom poisonings.

The eight major toxins produced by mushrooms are categorized as cyclopeptides, monomethylhydrazine, muscarine, hallucinogenic indoles, isoxazole, coprine (disulfiram-like reaction), orellanine, and gastrointestinal tract–specific irritants.

All poisonous mushrooms cause vomiting and abdominal pain. Other symptoms vary greatly depending on mushroom species. Generally, mushrooms that cause symptoms early (within 2 hours) of ingestion are less dangerous than those that cause symptoms later (usually after 6 hours).

Depending from clinical picture conditioned trope toxins, **poisonous mushrooms are divided into 3 groups**:

- ***gastroenterotropic*** (false honey agaric gray-yellow (*Hypholoma fasciculare*), false honey agaric brick red etc.). Toxicity conditioned by the presence of *milky juice (latex*) in them, which irritates mucous membrane shell gastrointestinal tract;
- ***neurotropic*** (*Inocybe patouillardii*, *Clitocybe rivulosa*, *Amanita muscaria* (Fly Agaric), *Amanita pantherina* (Panther Cap) etc.), that contain *alkaloids (muscarine, muscaridine, hyoscyamine, scopolamine)* and 10 more biogenic amines, which have on the body diverse physiological impact;
- ***hepato-nephrotropic*** (*Amanita phalloides* (Death Cap) = pale toadstool green, pale toadstool white, pale toadstool yellow, common thread, orange-red cobweb *(Cortinarius orellanus),* umbrella mushrooms *(Lepiota brunneoincarnata*, *Lepiota reddish-brown*).

Toxicity everyone species pale toadstool is determined cyclical polypeptides *ama- and phallotoxins*, morel mushrooms - *gelvelic acid and gyromitrin*, spider mite - *orelanin*. In brick-gray-red umbrella mushrooms contain *phalloidin*, and brown-cherry mushrooms contain *cyanide*.

Despite on a variety of **clinical picture** in case of poisoning different in groups mushrooms, with each poisoning present latent period: in case of poisoning gastroenterotropic and neurotropic mushrooms - 2-3 hours, hepato-nephrotropic mushrooms - 5-6 hours. In any case the onset of the disease is always acute.

In case of poisoning with gastric enterotropic mushrooms in the clinic picture prevails acute gastroenteritis: acute pain in the upper half of the abdomen, nausea, vomiting, diarrhea, general intoxication.

In case of poisoning neurotropic mushrooms, diseases starts with abundant sweating and salivation, extremely strong secretion mucus from the nose and bronchi, lacrimation. In the future joins unbearable nausea, abdominal pain, often colic, vomiting, diarrhea. Then appear signs central nervous system injuries: dizziness, confused unconsciousness, excitement, delirium, hallucinations, convulsions. In case the predominance of mushrooms in the content muscarine in the clinic dominate phenomena from the gastrointestinal tract and vegetative nervous systems, including pupils constriction (miosis), bradycardia and bradypnea, arterial hypotonia. Contrarily, provided predominance in the mushroom muscaridine are observed excitement, delirium, hallucinations, pupils dilation (mydriasis), tachycardia.

In case of poisoning by hepato-nephrotropic mushrooms, diseases starts with an acute gastroenterocolitis: extremely abrupt abdominal pain, unrelenting vomiting, profuse diarrhea. Simultaneously appear signs of serious cardiovascular and central nervous system violations. It is developing acute hepatic-renal failure, death can occur on the 2-5th day of the disease, rarely on the 7th. In case of poisoning cobwebs (*Dactylium*) do not have diarrhea, in the clinic the picture dominate signs kidney injuries. The cause of death (after 2-3 weeks from the beginning of the disease) there may be an increase renal-hepatic failure. When favorable I will run process recovery long (weeks and months).

Early gastrointestinal symptoms

Mushrooms that cause early gastrointestinal symptoms (e.g., *Chlorophyllum molybdites* and the little brown mushrooms that often grow in lawns) cause

gastroenteritis, sometimes with headaches or myalgias. Diarrhea is occasionally bloody.

Symptoms usually resolve within 24 hours.

Treatment is supportive.

Early neurologic symptoms

Mushrooms that cause early neurologic symptoms include hallucinogenic mushrooms, which are usually ingested recreationally because they contain psilocybin, a hallucinogen. The most common are members of the *Psilocybe* genus, but some other genera contain psilocybin.

Symptoms begin within 15 to 30 minutes and include euphoria, enhanced imagination, and hallucinations. Tachycardia and hypertension are common, and hyperpyrexia occurs in some children; however, serious consequences are rare.

Treatment occasionally involves sedation (e.g., with benzodiazepines).

Early muscarinic symptoms

Certain mushrooms contain toxins that stimulate muscarinic cholinergic receptors, thus mimicking the effects of acetylcholine on these receptors (the toxins do not stimulate nicotinic cholinergic receptors). Mushrooms that cause early muscarinic symptoms include members of the *Inocybe* and *Clitocybe* genera.

Symptoms may include the SLUDGE syndrome, including miosis, bronchorrhea, bradycardia, diaphoresis, wheezing, and cramps. Symptoms are usually mild, begin within 30 minutes, and resolve within 12 hours.

Atropine may be given to treat severe muscarinic symptoms (eg, wheezing, bradycardia).

Delayed gastrointestinal symptoms

Mushrooms that cause delayed GI symptoms include members of the *Amanita, Gyromitra,* and *Cortinarius* genera.

The most toxic *Amanita* mushroom is *Amanita phalloides,* which causes 95% of mushroom poisoning deaths. Initial gastroenteritis, which may occur 6 to 12 hours after ingestion, can be severe; hypoglycemia can occur. Initial symptoms abate for a few days; then liver failure and sometimes renal failure develop. Initial care involves close

monitoring for hypoglycemia and possibly repeated doses of activated charcoal. Treatment of liver failure may require liver transplantation; other specific treatments (eg, *N*-acetylcysteine, high-dose penicillin, silibinin, IV fat emulsion) are unproved.

Amanita smithiana mushrooms cause delayed gastroenteritis, usually 6 to 12 hours after ingestion, and acute renal failure (usually within 1 to 2 weeks after ingestion) that often requires dialysis.

Gyromitra mushrooms can cause hypoglycemia simultaneously with or shortly after gastroenteritis. Other manifestations may include central nervous system toxicity (eg, seizures) and, after a few days, hepatorenal syndrome. Initial care involves close monitoring for hypoglycemia and possibly repeated doses of activated charcoal. Neurologic symptoms are treated with pyridoxine 70 mg/kg slow IV infusion over 4 to 6 hours (maximum daily dose of 5 g); liver failure is treated supportively.

Most *Cortinarius* mushrooms are indigenous to Europe. Gastroenteritis may last for 3 days. Renal failure, with symptoms of flank pain and decreased urine output, may occur 3 to 20 days after ingestion. Renal failure often resolves spontaneously, but dialysis may be required for a brief period if renal function does not improve in 3 to 5 days and chronic renal damage may be permanent.

Delayed muscular symptoms

Several mushroom species cause a delayed myotoxicity with resultant rhabdomyolysis that is sometimes fatal. *Tricholoma* spp can cause fatigue, muscle weakness, myalgias, and rhabdomyolysis 24 to 72 hours after ingestion. *Russula* spp have caused similar myotoxicity affecting myocardial tissue resulting in tachycardia, hypotension, arrhythmias, and death. There is no specific treatment other than supportive care.

Delayed neurologic syndromes

Some *Clitocybe* mushrooms cause erythromelalgia, which is paresthesias and severe burning dysesthesias with edema and erythema of the distal extremities, 24 hours after ingestion. IV nicotinic acid may be an effective treatment.

Hapalopilus rutilans causes a delayed (more than 12 hours after ingestion) central nervous system syndrome of vertigo, ataxia, visual disturbances, somnolence, and encephalopathy.

Pleurocybella porrigens causes a delayed (between 1 to 31 days) syndrome of altered consciousness, convulsions, myoclonus, dysarthria, encephalopathy, respiratory failure and death.

Prevention mushrooms poisoning:

- The prevention of mushroom poisoning is reduced to organizing the mushrooms hunting, their processing and sale.
- Only edible mushrooms can be collected.
- Only sorted mushrooms should be accepted from the pickers at procurement points.
- The processing of mushrooms and the production of mushroom semi-finished products at the enterprises of agro-industrial complexes should be carried out according to approved standards and rules.

3.1. AMANITA POISONING

Poisonings by species of *Amanita phalloides* (Death Cap) account for 95% of the fatalities due to mushroom intoxication; the mortality rate for this group is 5-10%. It produces two classes of cyclopeptide toxins: (1) phallotoxins, which are heptapeptides believed to be responsible for the early symptoms of Amanita poisoning, and (2)

amanitotoxin, an octapeptide that inhibits RNA polymerase and subsequent production of messenger RNA.

Cells with high turnover rates, such as those in the gastrointestinal mucosa, kidneys, and liver, are the most severely affected.

Amanita poisoning causes cellular necrosis, which may occur throughout the gastrointestinal tract, the most heavily exposed site. Acute yellow atrophy of the liver and necrosis of the proximal renal tubules are found in lethal cases.

The **clinical course** of poisoning with *Amanita phalloides* is biphasic. Nausea, vomiting, and severe abdominal pain ensue 6-24 hr after ingestion. Profuse watery diarrhea follows shortly thereafter and may last for 12-24 hr. During this time, as much as 9 L of fluid may be lost. From 24-48 hr after poisoning, jaundice, hypertransaminasemia (peaking at 72 to 96 h), renal failure, and coma occur. Death occurs 4-7 days after the ingestion. A prothrombin time less than 10% of control is a poor prognostic factor.

Treatment

Treatment for *Amanita phalloides* poisoning is both supportive and specific. Fluid loss from severe diarrhea during the early course of the illness is profound, requiring aggressive therapy for correction of this loss. In the late phase of the disease, management of renal and hepatic failure is also necessary.

Specific therapy for *Amanita phalloides* poisoning is designed to remove the toxin rapidly and to block binding at its target site. Oral activated charcoal and lactulose combined with fluid and electrolyte replacement are recommended as part of the initial treatment for children with *Amanita phalloides* poisoning. Forced diuresis should be avoided, since this increases renal exposure. Intravenous penicillin G (400,000 U/kg/24 hr) administered as a continuous infusion and silybin dihemisuccinate, the water-soluble isomer of the flavolignone silymarin (in an intravenous dosage of 20-50 mg/kg/24 hr), act synergistically to inhibit binding of both toxins, to interrupt enterohepatic recirculation of amanitotoxin, and to protect from further hepatic injury from the toxins. Hemodialysis and hemoperfusion are also recommended as part of the

initial treatment for intoxicated children. Orthotopic liver transplantation is recommended for children in whom severe hepatic failure develops.

3.2. ISOXAZOLE INTOXICATION

The third place in terms of toxicity is occupied by **fly agaric mushrooms**, which contain muscarine, mycoatropin and other toxins that cause poisoning with a predominance of neurological symptoms.

Amanita muscaria

(Fly Agaric)

Amanita pantherina

*(*Panther Cap*)*

Although *Amanita muscaria* and *Amanita pantherina* may contain muscarine, the toxins responsible for the CNS symptoms after ingestion of these mushrooms are muscimol and ibotenic acid, the heat-stable derivatives of the isoxazoles. Muscimol, a hallucinogen, and ibotenic acid, an insecticide, have anticholinergic effects. From 30 min to 3 hr after ingestion, CNS symptoms appear: obtundation, alternating lethargy and agitation, and, occasionally, seizures. Nausea and vomiting are uncommon. If large amounts of muscarine are contained in the mushroom, symptoms of cholinergic crisis also may occur.

Mortality in these poisonings usually does not exceed 2-3%.

Panther cap poisoning occurs 1-2 hours after igestion and manifests by nausea, vomiting, diarrhea, dryness mucous membranes oral cavity, increase temperature, mydriasis, tachycardia (as atropine poisoning).

Fly agaric poisoning observed since July month to November. Symptoms occur 30-40 minutes after ingestion (sometimes after 2 hours): nausea, vomiting, abdominal

pain, increased sweating and salivation, lacrimation, dyspnoea. Miosis (constrited puppils) is a specific poisoning symptom. Later there are diarrhea, general weakness, decline arterial pressure, violation heart rate A severe form of the disease characterized by excitement, convulsions, collapse and coma. In severe poisoning psychomotor excitement, euphoria, hallucinations, muscle twitching and cramps.

Specific therapy must be carefully selected. If an exaggerated cholinergic response is observed, atropine should be administered. Because ingestions of *A. muscaria* often are associated with anticholinergic findings, the acetylcholinesterase inhibitor physostigmine is often used to reverse the delirium and coma. Benzodiazepines also are used for the agitation and delirium. Seizures can be controlled with diazepam. In most cases, however, early treatment with ipecac (if the patient is conscious) and close observation are all that is required.

4. PLANT POISONING

Plant poisoning is normally a problem of young children who unintentionally ingest small quantities of toxic plants with little resulting morbidity and few deaths. Such poisonings are usually accidental and occur during walks. Surveys of calls to Poison Information Centres in Germany and the USA show that ingestion of plants is responsible for a significant number of calls (10% of all inquiries), but that serious poisonings are rare.

The plant poisons or phytotoxins are found in angiosperms or flowering plants. These phytotoxins comprise a vast range of biologically active substances such as alkaloids, polypeptides, amines, glycosides, oxalates, resins, toxalbumins etc.

Any part of the plant can have poisonous properties. This group of poisonings is characterized by a very short latent period (from 15-20 minutes to 1 hour) and very high mortality (death can occur after 1.5-3 hours, and most often from respiratory paralysis). The symptomatology depends on the actual beginning of the plant.

A few commonly grown plants are highly poisonous, and many plants are moderately poisonous (table 2). Few plant poisonings have specific antidotes. Most plant ingestions, including the plants listed in the aforementioned table, result in

minimal symptoms unless the leaves and other components are concentrated into a paste or brewed into a tea.

Table 2. – Moderately Poisonous Plants

Plant	Symptoms	Treatment
Aconitine (eg, derived from monkshood)	Bradycardia, arrhythmias, paresthesia, weakness	Supportive care Sodium bicarbonate
Colchicine (autumn crocus, meadow saffron, glory lily)	Delayed gastroenteritis, multiple organ failure Bone marrow suppression	Supportive care and possibly, as a last resort, experimental colchicine-specific Fab fragments
Cyanogenic glycosides (eg, in *Prunus* spp [eg, peach, apricot, and wild cherry pits], *Malus* spp [eg, apple seeds], and other seeds)	Symptoms of cyanide poisoning	Hydroxocobalamin Cyanide antidote kit (includes amyl nitrate, sodium nitrite, and sodium thiosulfate)
Deadly nightshade	Anticholinergic symptoms, hyperthermia, seizures, hallucinations	Supportive care For severe hyperthermia or seizures, possibly physostigmine
Fava beans	In patients with G6PD deficiency, gastroenteritis, fever, headache, hemolytic anemia	Supportive care For severe anemia and poisoning, consideration of exchange transfusion
Green potato and potato sprout	Gastroenteritis, hallucinations, delirium	Supportive care
Holly berries	Gastroenteritis	Supportive care
Licorice	Hypokalemia, hypertension, and retention of water and sodium (pseudohyperaldosteronism)	Supportive
Lily of the valley	Hyperkalemia, gastroenteritis, confusion, arrhythmias	See Digitalis preparations
Mistletoe	Gastroenteritis	Supportive care
Nightshade, common or woody	Gastroenteritis, hallucinations, delirium	Supportive care
Poinsettia	Minor mucous membrane irritation	Unnecessary

Plant	Symptoms	Treatment
Yew	Gastroenteritis Rarely, seizures, arrhythmias, coma	Supportive care

Fab = fractionated antibodies; sp/spp = species.

Fig. 2. A selection of 12 native poisonous plants with flowers or fruits/seeds in risk categories 2 and 3

Highly toxic and potentially fatal plants include the following: castor beans and jequirity beans, oleander and foxglove, hemlock

Castor beans and jequirity beans

Ricinus communis

(castor beans)

Abrus precatorius

(jequirity bean or rosary pea)

Castor beans contain ricin, an extremely concentrated cellular poison. Jequirity beans contain abrin, a related and even more potent toxin. In both, the beans have a relatively impervious shell; thus, the bean must be chewed to release the toxin. However, the seed coating of the jequirity bean is often not intact, and simple bacterial digestion can release the abrin toxin.

Symptoms of either poisoning may include delayed gastroenteritis, sometimes severe and hemorrhagic, followed by delirium, seizures, coma, and death. Whole-bowel irrigation should be considered because it aims to remove all beans ingested.

Oleander and foxglove

Cascabela thevetia (yellow oleander)

Digitalis genus (foxglove)

Foxglove (including *D. purpurea and Digitalis lanata*) is one of many plants containing cardiac glycosides that exert potent inotropic and electrochemical effects on cardiac tissue. Hundreds of cardiac glycosides have been isolated from plants across 12 botanical families, including members of the *Digitalis genus* (foxglove), *Cascabela thevetia* (yellow oleander) and *Convallaria majalis* (lily of the valley). The cardiac glycosides of medical importance are extracted from D. purpurea and D. lanata, yielding digitoxin and digoxin, respectively.

Ingestion of *oleander* can affect the gastrointestinal system, the heart, and the central nervous system. The gastrointestinal effects can consist of nausea and vomiting, excess salivation, abdominal pain, diarrhea that may contain blood, and especially in horses, colic. Cardiac reactions consist of irregular heart rate, sometimes characterized by a racing heart at first that then slows to below normal further along in the reaction. Extremities may become pale and cold due to poor or irregular circulation. The effect on the central nervous system may show itself in symptoms such as drowsiness, tremors or shaking of the muscles, seizures, collapse, and even coma that can lead to death. Oleander poisoning can also result in blurred vision, and vision disturbances, including halos appearing around objects. Oleander sap can cause skin irritations, severe eye inflammation and irritation, and allergic reactions characterized by dermatitis

Foxglove *(Digitalis purpurea L.)* leaves are frequently confused with borage *(Borago officinalis L.),* which is traditionally used as a food ingredient. Due to the

presence of the cardiac glycosides, mostly digitoxin, foxglove leaves are poisonous to human and may be fatal if ingested. Foxglove toxicity includes gastroenteritis, confusion, hyperkalemia, and arrhythmias. The serum digoxin level can confirm ingestion but is not useful as quantitative information.

Potassium levels are closely monitored. Hyperkalemia may respond only to hemodialysis. Calcium is not recommended for arrhythmias. Digoxin-specific fractionated antibody fragments have been used to treat ventricular arrhythmias.

Hemlock (Conium Maculatum)

Hemlock poisoning (poison hemlock and water hemlock) can cause symptoms within 15 minutes. Poisonings are commonly seen during childhood and are associated with a high mortality rate.

Conium maculatum poisoning occurs due to some of its piperidine alkaloid contents which have nicotinic effects, such as coniceine, coniine, N-methyl coniine, conhydrine, and pseudoconhydrine. While every part of the plant is toxic, the highest alkaloid concentration exists in the seeds. Ataxia and headache are the symptoms observed in the early stage of poisoning. Increased salivation, tachycardia, and pupillary dilation develop due to the effects of the plant on the autonomic ganglia. Muscle weakness or paralysis, bradycardia, and central nervous system depression may develop in some of the patients due to increased cholinergic stimuli. Rhabdomyolysis and acute renal failure have also been reported in some cases as a consequence of

Conium maculatum poisoning. Each of these piperidine alkaloids is a kind of peripheral neurotoxin; the neurotoxins show curare-like effects in neuromuscular junctions and create nicotinic effects in the autonomic ganglia.

To sum up, poison hemlock has effects on the nicotinic cholinergic receptor, beginning with dry mouth and progressing to tachycardia, tremors, diaphoresis, mydriasis, seizures, and muscle paresis. Rhabdomyolysis and bradycardia may occur.

Water hemlock seems to enhance gamma-aminobutyric acid (GABA) activity. Symptoms may include gastroenteritis, delirium, refractory seizures, and coma.

Table 3 – Synoptic presentation of the toxicology of native poisonous plants

Poisonous plant	Relevant constituents	Common symptoms of poisoning	Treatment options
Wolfsbane *Aconitum spp.*	Aconitine, mesaconitine, hypaconitine, lycaconitine	(Peri-)oral paresthesia, numbness and pain (anesthesia dolorosa), dysgeusia, dry mouth, nausea, vomiting, dizziness, hypotension, cardiac arrhythmia, bradycardia, seizures,respiratory depression	PD; consider antiarrhythmic drugs (e.g., amiodarone,magnesium sulfate, lidocaine, flecainide) or atropine for bradycardia; consider pacemaker therapy, sedatives
Belladonna *Atropa belladonna*	L-Hyoscyamine, atropine (DL-hyoscyamine),scopolamine, atropamine	Anticholinergic syndrome: flushing, dry,warm skin, mydriasis, dry mouth, tachycardia, agitation, confusion, hallucination, inhibition of micturition, hyperthermia, nausea, vomiting	PD, physical cooling (no antipyretic agents!), consider benzodiazepines; antidotein marked anticholinergic or central symptoms (e.g., delirium): physostigmine
Angel's trumpet *Brugmansia spp.*	Scopolamine (L-hyoscine), L-hyoscyamine, atropine (DL-hyoscyamine)	Anticholinergic syndrome (see belladonna), especially seizures, hallucinations, drowsiness, coma	See belladonna; continuous monitoring, especially if there is risk of harm to self or others
Cowbane *Cicuta*	Cicutoxin, fool's parsley, cicutol	Burning pain in the mouth, severe recurrent	PD, benzodiazepines, muscle relaxants,

Poisonous plant	Relevant constituents	Common symptoms of poisoning	Treatment options
virosa		vomiting, muscle cramps, rhabdomyolysis, kidney failure, coma, hyperthermia, apnea	hemodialysis (in kidney failure)
Autumn crocus *Colchicum autumnale*	Colchicine	Furry feeling in mouth/throat, persistent nausea, watery-mucous, sometimes bloody gastroenteritis; electrolyte disorders,exsiccosis, confusion/delirium, bleeding, cardiogenic shock, multiorgan failure	PD (especially activated charcoal), consider cholestyramine (3 x 4 g/day) to interruptenterohepatic circulation; intensive care, hemodialysis (in kidney failure)
Poison hemlock *Conium maculatum*	Coniine, γ-coniceine	Irritation/burning in oral mucosa, numbness, sore throat, recurrent vomiting, diarrhea,dizziness, „heavy legs“, angina pectoris, seizures, rhabdomyolysis, kidney failure, apnea	PD, benzodiazepines,hemodialysis (in kidney failure)
Jimson weed *Datura stramonium*	L-Hyoscyamine, atropine (DL-hyoscyamine),scopolamine	Anticholinergic syndrome (see belladonna), CK elevation	See belladonna and angel's trumpet
Henbane *Hyoscyamus niger*	Scopolamine, L-hyoscyamine, atropine (DL-hyoscyamine)	Anticholinergic syndrome (see belladonna); predominantly central sedating symptoms (sleepiness) due to high scopolamine content	See belladonna and angel's trumpet
Castor bean *Ricinus communis*	Ricin, ricinine	Nausea, hemorrhagic gastroenteritis;exsiccosis, hypotension, tachycardia, hypoglycemia, liver and spleen necrosis, kidney failure, multiorgan failure	PD, close monitoring of liver, kidney, and coagulationvalues

Poisonous plant	Relevant constituents	Common symptoms of poisoning	Treatment options
False hellebore *Veratrum spp.*	Protoveratrines, jervine, germerine	Similar to wolfsbane: burning in the mucous membranes, (recurrent) vomiting, exsiccosis, bradycardia, hypotension, hypothermia	PD; consider antiarrhythmic drugs (magnesium sulfate,lidocaine, flecainide) or atropine in bradycardia
Foxglove *Digitalis spp.*	Digoxin, digitoxin, gitoxin, lanatoside C	Nausea, (recurrent) vomiting, abdominal pain, AV block, tachycardia, pallor, headache, dizziness, fatigue, color visiondeficiencies (xanthopsia, "yellow vision"), hallucinations	PD (especially activated charcoal), consider cholestyramine (3 x 4 g/day) to interrupt the enterohepatic circulation; electrolyte monitoring (in particular potassium, calcium), antidote: digitalis antibodies, ICU monitoring
European yew *Taxus baccata*	Taxine A, B, C; taxols,taxanes, baccatines, cyanogenic glycosides	Choking, nausea, (recurrent) vomiting,abdominal pain, diarrhea, erythema, tachycardia/bradycardia , hypotension, cardiac arrhythmias, seizures, coma, cardiogenic shock, respiratory paralysis	PD (even hours after ingestion of needles); consider antiarrhythmic drugs (lidocaine), consider digitalis antibodies; consider calcium gluconate i.v., ultima ratio: i.v. lipid emulsion

Common sources of ***deliriant plant poisons*** are *Dhatura stramonium, Atropa belladonna, Hyoscyamus niger* and *Cannabis indica.* The active principle contains the alkaloids levohyoscyamine, hyoscine, scopolamine and atropine. The clinical features due to its poisoning produce anti-cholinergic syndrome with features like dryness of throat, difficulty in talking and dysphagia. The flushed face, dilated pupils, dry and warm skin, vomiting and unsteady gait is often confused for drunkenness.

Prevention of poisoning by wild plants should be aimed at protecting children from the possibility of eating poisonous plants. To do this, it is recommended to regularly (2-3 times a week) inspect the land plots of children's institutions and places where children regularly go for walks. When poisonous plants are detected, the area is cleaned (mowing or uprooting of plants followed by their destruction), digging up the soil, etc.

Food poisoning by poisonous weed seeds

A number of toxic weed seeds have been identified as contaminants in food. These include cocklebur, morning glory, castor, pokeweed, cowcockle. corncockle, nightshade, crotalaria, and jimsonweed. Of the cited seeds, jimsonweed is without a doubt the most common and the most toxic. Occasionally, herbicides are not effective in controlling this weed. For example, in 1973, sorghum exported to India was badly contaminated with jimsonweed seed because the seeds are about the same size and color as sorghum and cannot be separated during harvest or by screening and cleaning. Weed seed contamination can result from the plants growing with the grain crops and subsequent pick-up during harvest. It has been suggested that amounts of foreign material permitted by USA Federal Grain Standards contribute to the problem. According to these standards, USA No. 1 grades of grain may contain up to I% foreign material, i.e., dirt, weed seeds, and trash. Other grades may contain up to 5%.

Heliotropic toxicosis is a disease that develops with long-term consumption of grain products contaminated with heliotrope seeds.

Heliotrope toxicosis develops slowly and has signs of toxic hepatitis: liver enlargement, jaundice, ascites. Mortality in severe cases reaches 20-30%.

Trichodosmotoxicosisis a disease that occurs when food products are made from grain contaminated with the seeds of gray *trichodesma* .

The **clinical picture** includes symptoms of encephalitis and meningoencephalitis. General weakness, fatigue, muscle pain, headache, dizziness, nausea, vomiting are observed. Sometimes there is a sudden loss of speech, arms and legs are withdrawn, epileptiform seizures are often observed. Blood pressure drops sharply (to

55 and 80 mm Hg). Severe forms of poisoning lead to fatal consequences in 35% of cases.

Prevention of food poisoning by poisonous weed seeds is possible thorough cleaning of the seed grain, timely weeding of crops from weeds, early and quick harvesting of crops, since weeds ripen later, ban on the use of crushed grain for food purposes, use.

Food poisoning by plant food that become toxic in special conditions

Lectins and phytohemagglutinins (PHA) are natural toxicants present in many foods, especially in beans and other dietary pulses, which can have toxic effects when consumed without adequate cooking, occasionally leading to an acute gastroenteritis, sickness and abdominal pain, which may be severe enough to require hospitalisation. The symptoms generally clear within 3–4 hrs and recovery is usually rapid and complete.

The common foods include Peanut, Kidney bean, Fava bean (*Vicia faba*), Soya bean, Lentil Lens, Winged bean (*Psophocarpus tetragonolobus*), Garden pea, Horse gram, Lima bean (*Phaseolus lunatus*) and Navy bean (*Phaseolus vulgaris).*

Beans (such as green beans, red kidney beans and white kidney beans) contain naturally a toxin known as *phytohaemagglutinin (fazin)***.** Food poisoning caused by this toxin in raw and inadequately-cooked beans has a short onset time (1-3 hours) with symptoms of nausea, vomiting and diarrhoea. However, this toxic substance can be destroyed by subjecting the bean to thorough soaking and then cooking thoroughly at boiling temperature. Tinned beans which have been subjected to thorough heat-treatment are safe to eat without further cooking.

Cassavas refer to the edible root of cassava plants. Cassavas contain cyanogenic

glycoside. The bitter type of cassavas has higher levels of toxins than the sweet type. When raw or inadequately-cooked cassavas are ingested, the toxin will be transformed into a chemical called hydrogen cyanide, which may result in food poisoning. Symptoms of cyanide poisoning occur within a few minutes and may include constriction of the throat, nausea, vomiting, headache, etc., and death has been reported in severe cases.

Fruit seeds and stones such as apples, apricots, pears, plums, prunes, cherries, peaches could be toxic. The flesh of these fruits is not toxic, but the seeds and stone contain cyanogenic glycoside (amygdalin). When the consumer chews the fresh seeds or stone, the cyanogenic glycoside in it can be transformed into hydrogen cyanide, which is poisonous to the consumer. Young children are more susceptible and swallowing only a few seeds/stone may cause cyanide poisoning. Symptoms of poisoning are same as those by cassavas.

Potatoes contain natural *glycoalkaloids (solanin)*. The levels of these toxins are usually low and do not pose adverse effects in humans. However, potatoes that show signs of greening, sprouting, physically damaged or rotting may contain high level of glycoalkaloids and the majority of the toxins are present in the green area, in the peel, or just below the peel of the potatoes. High levels of glycoalkaloids have a bitter taste and symptoms of poisoning may include a burning sensation in the mouth or severe stomach ache nausea and vomiting. Cooking and frying cannot destroy glycoalkaloids.

The risk of poisoning by natural toxins in fruits and vegetables can be avoided or significantly reduced by taking the following measures:

1. Do not buy green potatoes or potatoes which are sprouting.
2. Do not eat vegetables and fruits raw or undercooked if they are usually consumed cooked.
3. Cook beans such as green beans, red kidney beans and white kidney beans, cassavas, bamboo shoots thoroughly at boiling temperature after thorough soaking in clean water.

4. Do not use raw or inadequately-cooked green beans or other bean species in the preparation of salad dishes. Always bear in mind a few raw beans can cause food poisoning symptoms.
5. When eating fresh fruits, avoid eating seeds of fruits, such as apples, apricots, pears, etc., whereas the flesh of these fruits is nutritious and safe to eat.
6. Cook bitter apricot seeds thoroughly and eat them in strict moderation.
7. Store potatoes in a dark, cool and dry place and avoid eating potatoes that show signs of greening, sprouting or rotting.

5. POISONING BY PRODUCTS OF ANIMAL ORIGIN

Poisoning by poisonous animal tissues is quite rare. They are associated with the use of poisonous tissues of fish (marinka, fugu, pinbelly, barbel, Sevan chromul), molluscs and glands of internal secretion of slaughter animals, which contain toxic natural compounds.

Biotoxins are more often concentrated in individual organs and tissues of poisonous fish (milk, roe, skin, peritoneum, liver), less often they are evenly distributed in muscle thickness.

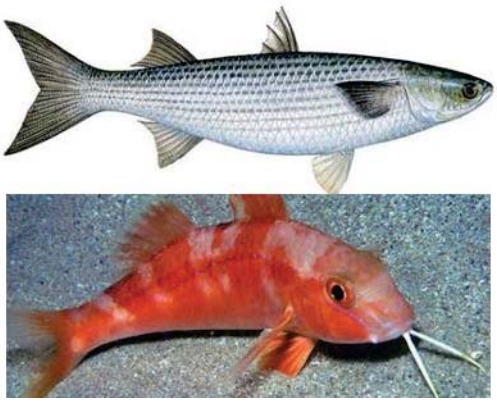
Poisoning by products of animal origin with natural toxicity.

Caviar, milk and black film peritoneum, gills Pisces **marinas** contain poisonous substances (especially in spring).

Lampreys produce toxic slime. For him removal lamprey necessary powder with and llyu, and then carefully to wash spring water

In the head mullet fish, sultanka (barabuli), " sleepy fish" the toxin is localized , which causes poisoning, sleepy _ the symptoms of which are hallucinations and nightmares, which are getting worse during sleep. With light intoxication occurs itching and burning in the throat at once after reception food , muscular weakness , partial paralysis legs. Symptoms appear after 0.5-2 hours . Recovery occurs after 12-24 hours depending from degree intoxication .

Poisoning this toxin can be consumed both raw and cooked fish.



Poisoning by products of animal origin, poisonous under certain conditions.

These poisonings are mainly observed when eating roe, milk and liver of some types of fish (tench, pike, perch, mackerel, etc.), which acquire toxic properties during the spawning period. Poisoning occurs like acute gastroenteritis.

Dorado , swordfish , marlin , grenadier , sea bass, shark and tuna : infected fish maybe contain 23 mg of mercury per kg of raw weight, i.e almost 100,000 times more concentration surrounding water Concentration mercury in filtered bivalves molluscs is an average of 50 mg per kilogram for mussels . Symptoms include numbness and weakness in the legs and arms, fatigue , ringing in the ears , narrowing of the field of vision , hearing loss , undivided speech and clumsy movements. Some of the heavy casualties of the disease Minamata (1956) went crazy , lost consciousness and died during month after the onset of the disease.

Bee honey poisoning occur if bees collected nectar from poisonous plants. Poisoning is characterized by a variety of symptoms, depending on the actual onset of the poisonous plant. For example, **grayanotoxins** are natural plant toxins found in rhododendrons and other plants of the family Ericaceae. They can be found in

honey made from the nectar produced by the flowers of these plants, and can cause a very rare poisonous reaction. Symptoms include dizziness, weakness, excessive perspiration, nausea, and vomiting shortly after the toxic honey is ingested. Other symptoms may include low blood pressure or shock, bradyarrhythmia (slowness of the heart beat associated with an irregularity in the heart rhythm) and other cardiac abnormalities. The disease proceeds acutely.

In order to prevent such poisonings, it is recommended to place apiaries in places free from the germination of poisonous plants. It is also not recommended to use honey from wild bees.

5.1. MARINE FOOD POISONING

PUFFERFISH POISONING

The poison found in pufferfish, blowfish, balloon fish, toads, sunfish, porcupine fish, toadfish, globefish, and swellfish is a *tetrodotoxin*. This is one of the most toxic poisons found in nature.

Tetrodotoxin (TTX), also known as anhydrotetrodotoxin 4-epitetrodotoxin, or tetrodonic acid, is a marine biotoxin associated with certain fish species, notably puffer fish. tetrodotoxin is mainly associated with fish of the order Tetraodontidae (pufferfish, balloon fish, fugu, globe fish, blowfish, toad fish) from the Pacific and Indian Oceans. The highest levels of tetrodotoxin are found in the viscera, particularly the liver and ovaries, and skin of the fish, but the muscle tissue does not usually contain dangerous levels of toxin.

Tetrodotoxin is a very potent neurotoxin, and operates in a similar way to the PSP toxin (saxitoxin) by selectively blocking the voltage-gated sodium channel – a large protein that extends across the plasma membrane of nerve and muscle cells.

Most people who eat pufferfish do so intentionally; pufferfish are considered an Asian delicacy, served in some types of sushi and sashimi. Unless the chef is specially trained to cut the meat in a particular fashion, the dish may contain a large amount of the toxin. Pufferfish poisoning is similar to paralytic shellfish poisoning.

Pufferfish Poisoning Symptoms

- Symptoms generally occur 10-45 minutes after eating the pufferfish poison and begin with numbness and tingling around the mouth, salivation, nausea, and vomiting.
- Symptoms may progress to paralysis, loss of consciousness, and respiratory failure and can lead to death.

Pufferfish Poisoning Treatment

- Vomiting should be induced if the poisoned person is awake and alert and has eaten the fish within 3 hours.
- The person may become paralyzed. Artificial respirations may keep the person alive until medical attention in a hospital's emergency department is possible.
- Rapidly turn the person onto their side if vomiting occurs.

CIGUATERA FISH POISONING

Ciguatera fish poisoning occurs after eating reef fish contaminated with toxins such as ciguatoxin or maitotoxin. These potent toxins originate from *Gambierdiscus toxicus*, a small marine organism (dinoflagellate) that grows on and around coral reefs. Dinoflagellates are ingested by herbivorous fish. The toxins produced by G. toxicus are then modified and concentrated as they pass up the marine food chain to carnivorous fish and finally to humans. Ciguatoxins are concentrated in fish liver, intestines, roe, and heads.

G. toxicus may proliferate on dead coral reefs more effectively than other dinoflagellates. The risk of ciguatera poisoning is likely to increase as coral reefs deteriorate because of climate change, ocean acidification, offshore construction, and nutrient runoff.

Vector fish of ciguatera toxins

Clinical Presentation

Ciguatera poisoning may cause gastrointestinal, cardiovascular, neurologic, and neuropsychiatric illness. The first symptoms usually develop within 3–6 hours after eating contaminated fish but may be delayed for up to 30 hours. Adverse health effects referable to the above-named organ systems include:

- Diarrhea, nausea, vomiting, and abdominal pain
- Bradycardia, heart block, hypotension
- Paresthesias, weakness, pain in the teeth or a sensation that the teeth are loose, burning or metallic taste in the mouth, generalized itching, sweating, and blurred vision. Cold allodynia (abnormal sensation when touching cold water or objects) has been reported as characteristic, but there can be acute sensitivity to both hot and cold. Neurologic symptoms usually last a few days to several weeks but may persist for months or even years.
- Fatigue, general malaise, insomnia

The overall death rate from ciguatera poisoning is <0.1% but varies according to the toxin dose and availability of medical care to deal with complications. The diagnosis of ciguatera poisoning is based on the characteristic signs and symptoms and a history of eating fish species known to carry ciguatera toxin. Fish testing can be done by the US Food and Drug Administration (FDA) in their laboratory at Dauphin Island. There is no readily available test for ciguatera toxins in human clinical specimens.

Prevention

Travelers can take the following precautions to prevent ciguatera fish poisoning:

- Avoid or limit consumption of reef fish.
- Never eat high-risk fish such as barracuda or moray eel.
- Avoid eating the parts of the fish that concentrate ciguatera toxin: liver, intestines, roe, and head.

Ciguatera toxins do not affect the texture, taste, or smell of fish, nor are they destroyed by gastric acid, cooking, smoking, freezing, canning, salting, or pickling.

Treatment

There is no specific antidote for ciguatoxin or maitotoxin poisonings. Symptomatic treatment may include gabapentin or pregabalin (neuropathic symptoms), amitriptyline (chronic paresthesias, depression, and pruritus), fluoxetine (chronic fatigue), and nifedipine or acetaminophen (headaches). Intravenous mannitol has been reported in uncontrolled studies to reduce the severity and duration of neurologic symptoms, particularly if given within 48 hours of the appearance of symptoms. It should only be given to hemodynamically stable, well-hydrated patients.

After recovering from ciguatera poisoning, patients may want to avoid consuming fish, nuts, alcohol, or caffeine for at least 6 months, as they may cause a relapse in symptoms.

SCOMBROID

Scombroid occurs worldwide in both temperate and tropical waters. One of the most common fish poisonings, it occurs after eating improperly refrigerated or preserved fish containing high levels of histamine and often resembles a moderate to severe allergic reaction.

Fish typically associated with scombroid have naturally high levels of histidine in the flesh and include tuna, mackerel, mahi mahi (dolphin fish), sardine, anchovy, herring, bluefish, amberjack, and marlin. Histidine is converted to histamine by bacterial overgrowth in fish improperly stored after capture. Histamine and other scombrotoxins are resistant to cooking, smoking, canning, or freezing.

Clinical Presentation

Scombroid poisoning resembles an acute allergic reaction, usually appearing 10–60 minutes after eating contaminated fish. Symptoms include flushing of the face and upper body (resembling sunburn), severe headache, palpitations, itching, blurred vision, abdominal cramps, and diarrhea. Untreated, symptoms usually resolve within 12 hours but may last up to 48 hours. Rarely, there may be respiratory compromise, malignant arrhythmias, and hypotension requiring hospitalization. There are no long-

term sequelae. Diagnosis is usually clinical. Clustering of cases helps exclude the possibility of true fish allergy.

Prevention

Fish contaminated with histamine may have a peppery, sharp, salty, taste or "bubbly" feel but will usually look, smell, and taste normal. The key to prevention is to make sure that the fish is properly iced or refrigerated at temperatures <38°F (<3.3°C) or immediately frozen after being caught. Cooking, smoking, canning, or freezing will not destroy histamine in contaminated fish.

Treatment

Scombroid poisoning usually responds well to antihistamines (H1-receptor blockers, although H2-receptor blockers may also provide some benefit).

OIL FISH POISONING

In the tropical waters of South Africa, South America, Australia , New Zealand are often caught big Pisces weighing 5-10 kg with white fat meat. They usually enter the trade network under by label "Oil fish": ruvetta, reksnya, gray delicate mackerel, promethean fish, seriolella, seriolla, bighead Atlantic.

Oilfish (*Ruvettus pretiosus*) and escolar (*Lepidocybium flavobrunneum*) belong to the family of Gempylidae; they are two of the more common fish species known to contain high levels of indigestible wax esters. According to literature, these fish species contain approximately 20% by weight of wax esters. As these fish species do not metabolise wax esters that occur naturally in their diet, these wax esters are accumulated in the fish body, including the skin and muscle meat. Geographical and seasonal variation may affect the level of wax esters in the fish.

Ruvettus pretiosus

Lepidocybium flavobrunneum

In humans, wax esters are not absorbed by the gut. These wax esters can have a laxative effect on some consumers: when used in food are possible gastrointestinal disorders which are caused by the fat of these fish. Gastrointestinal disorders occur during use fatty fish (more than 3-5% fat) with content unloved substances more than 20% to the total fat content and cause symptoms which range from mild and rapid passage of oily yellow or orange droplets to severe diarrhoea with nausea, vomiting and headache. Not all individuals who eat these fish species are affected. It seems that there is a variation in sensitivity among individuals, and some people seem to tolerate the fish and show no ill effect after consumption. Symptom onset time ranges from 30 minutes to 36 hours after ingestion. Recovery is expected within 24 to 48 hours in affected individuals. Preliminary review of 70 local food complaint cases of some 100 people with symptoms showed that almost all of them presented with oily diarrhoea and about one third with abdominal pain. Reported median latent period was 13 hours.

Wax esters are not broken down by cooking or freezing. There is no well-proven ways to reduce wax esters in the fish to guarantee a no-effect level, although certain cooking methods that separate a large proportion of the oil from the fish (such as grilling) coupled with discarding the cooking liquid may reduce the risk to some extent.

Most often in circulation is coming oilfish (ruvette) - big fish (mass from 3 to 12 kg), tissue fat of which contains 33-40% unsoaped substances , therefore during use fish for food , especially in smoked form , occurs " effect castor oil ".

5.2. SHELLFISH POISONING

Several forms of poisoning may occur after ingesting toxin-containing shellfish, including filter-feeding bivalve mollusks (mussels, oysters, clams, scallops, and cockles), gastropod mollusks (abalone, whelks, and moon snails), or crustaceans (Dungeness crab, shrimp, and lobster). Toxins originate in small marine organisms (dinoflagellates or diatoms) that are ingested and are concentrated by shellfish.

Clinical Presentation

Poisoning results in gastrointestinal and neurologic illness of varying severity. Symptoms typically appear 30–60 minutes after ingesting toxic shellfish but can be delayed for several hours. Diagnosis is usually one of exclusion and typically is made clinically in patients who have recently eaten shellfish.

Paralytic shellfish poisoning

Paralytic shellfish poisoning (PSP) is the most common and most severe form of shellfish poisoning. PSP is caused by eating shellfish contaminated with saxitoxins. These potent neurotoxins are produced by various dinoflagellates. A wide range of shellfish may cause PSP, but most cases occur after eating mussels or clams.

PSP occurs worldwide but is most common in temperate waters, especially off the Pacific and Atlantic Coasts of North America, including Alaska. The Philippines, China, Chile, Scotland, Ireland, New Zealand, and Australia have all reported cases.

Symptoms usually appear 30–60 minutes after eating toxic shellfish and include numbness and tingling of the face, lips, tongue, arms, and legs. There may be headache, nausea, vomiting, and diarrhea. Severe cases are associated with ingestion of large doses of toxin and clinical features such as ataxia, dysphagia, mental status changes, flaccid paralysis, and respiratory failure. The case-fatality ratio is dependent on the availability of modern medical care, including mechanical ventilation. The death rate may be particularly high in children.

Neurotoxic shellfish poisoning

Neurotoxic shellfish poisoning (NSP) is caused by eating shellfish contaminated with brevetoxins produced by the dinoflagellate K. brevis. Predominately an illness of the Western Hemisphere (southeastern coast of the United States, the Gulf of Mexico, and the Caribbean), there are also reports of the disease from New Zealand.

NSP usually presents as a gastroenteritis accompanied by neurologic symptoms resembling mild ciguatera or paralytic shellfish poisoning, 30 minutes to 3 hours after a shellfish meal. A syndrome known as aerosolized red tide respiratory irritation (ARTRI) occurs when aerosolized brevetoxins are inhaled in sea spray. This has been reported in association with a red tide (*K. brevis* HAB) in Florida. It can induce

bronchoconstriction and may cause acute, temporary respiratory discomfort in healthy people. People with asthma may experience more severe and prolonged respiratory effects.

Diarrheic shellfish poisoning

Diarrheic shellfish poisoning (DSP) is caused by eating shellfish contaminated with toxins such as okadaic acid. It occurs worldwide, with outbreaks reported from China, Japan, Scandinavia, France, Belgium, Spain, Chile, Uruguay, Ireland, the United States, and Canada.

Most cases result from eating toxic bivalve mollusks such as mussels and scallops. Symptoms usually occur within 2 hours of eating contaminated shellfish and include chills, diarrhea, nausea, vomiting, and abdominal pain. Symptoms usually resolve within 2–3 days. No deaths have been reported.

Amnesic shellfish poisoning

Amnesic shellfish poisoning (ASP) is a rare form of shellfish poisoning caused by eating shellfish contaminated with domoic acid, produced by the diatom *Pseudonitzchia* spp. Outbreaks of ASP have been reported in Canada, Scotland, Ireland, France, Belgium, Spain, Portugal, New Zealand, Australia, and Chile. Implicated shellfish include mussels, scallops, razor clams, and other crustaceans.

In most cases, gastrointestinal symptoms such as diarrhea, vomiting, and abdominal pain develop within 24 hours of eating toxic shellfish, followed by headache, memory loss, and cognitive impairment. In severe cases there may be hypotension, arrhythmias, ophthalmoplegia, coma, and death. Survivors may have severe anterograde, short-term memory deficits.

Prevention

Shellfish poisoning can be prevented by avoiding potentially contaminated shellfish. This is particularly important in areas during or shortly after algal blooms, which may be locally referred to as “red tides” or “brown tides.” Shellfish also carry a significant risk of infection from various viral and bacterial infections, for example hepatitis A virus, norovirus, *Vibrio vulnificus*, *Vibrio parahaemolyticus*, and several *Salmonella* and *Shigella* species. Ideally, travelers to developing countries

should avoid eating all shellfish. Marine shellfish toxins cannot be destroyed by cooking or freezing.

Treatment

Treatment is symptomatic and supportive. Severe cases of paralytic shellfish poisoning may require mechanical ventilation.

6. FOOD POISONING CAUSED BY CHEMICAL CONTAMINATION

Poisoning by impurities of chemical substances can be associated with their inclusion in the food chain and accumulation in food products as foreign substances. This type of poisoning can also be caused by the migration into food of chemicals from equipment, inventory, containers and packaging materials in the process of food processing and cooking.

Among poisonings with chemical impurities, the following are distinguished:

1. Poisoning with nitrates, nitrites.
2. Poisoning by salts of heavy metals
3. Poisoning by pesticides.
4. Poisoning by food additives.

6.1. TOXIC CONTAMINATION IN FOOD INTRODUCED BY PACKAGING, STORAGE AND COOKING

It is well documented that *food packaging* processes can introduce toxic contaminants in food, thus causing public health problems. Research shows that food contact chemicals can have harmful effects on humans. Therefore, stringent measures need to be devised to address this challenge. Furthermore, these food contact chemicals come in various ways, namely: food packaging, food storage containers, kitchen utensils, and food processing equipmen. The process of transferring and partitioning chemical compounds from food packaging into food through adsorption or diffusion is known as migration. Essentially, migrants from packaging have the potential to cause adverse effects on human health.

Food contact materials (FMCs) can release migrating substances. FMCs are

reportedly the leading source of chemical contamination in food and significantly contribute to chronic chemical exposure. Migration of contaminants into foodstuffs depends on factors such as the compositions of the material, package size, temperature, storage time, the nature of the food, and how it is exposed. As a result, food packaging is highly regulated internationally due to a vast range of literature on the carcinogenic effects of some of the chemical compounds used as ink and food protective membranes. In some instances, incorrect packaging methods, incorrect storage, and handling cause structural deficiencies in the packaging thus leading to bloating of canned foods and sealants leaking into the food product. Moreover, corrosion through oxidation can alter the structural integrity of metallic containers, thus posing a health hazard. For this reason, metallic containers are coated with epoxy resin varnishes to curb corrosion. Typical migrants from contact materials are chemicals such as benzene, especially in the flavored beverage industry. This chemical is used extensively in the manufacture of plastics and pesticides and it is known to have carcinogenic properties. Another chemical compound used in the food industry is bisphenol A (BPA) which is used in the lining of some food and beverages to extend their shelf life. It usually serves as a plasticizer in polymers like polyvinyl chloride (PVC).

Table 4 – Summary of selected food packaging and how contamination is introduced.

Type of Food	Packaging Type	Contamination Pathways
Rice	paperboard	Adhesives, coatings, inks
Maize	paper	Migration, moisture, inks
Meat, Fish	Polystyrene, corrugated fibreboard	Moisture in humid areas
Sugar	Paper, paperboard	Absorption of moisture and chemicals.
Raw and processed fruits / vegetables	Polystyrene, metals, vegetable parchment paper, moulded pulp packaging	Moisture absorption and migration
Dairy	Polystyrene Plastics, metals, folding cartons	Migration and leaching chemicals
Bakery	Polystyrene, greaseproof paper	Moisture absorption
Beverages	Metals, composite cans, foil wraps	Migration of Bisphenol A Blackening, corrosion, bulging, tin dissolution, leaching coatings

Food products such as sugar, maize, flour, rice, and most cereals are usually packaged in paper or board materials. For this reason, there are possibilities of chemical contamination by exposure to printing ink or, in extreme cases, absorption of moisture and chemical leaks when stored directly on the floor. Polymers are widely used in various applications in the food industry. Extensive research has progressed their usefulness in material design and general safety. Notably, polymers are used in food packaging and the most common are poly-ethylene, polyvinyl chloride, polystyrene, polycarbonate, and polyethylene terephthalate. Phenolic endocrine-disrupting chemicals in food through food packaging have been largely reported in canned food, disposable plastic bottles, polyethylene terephthalate, etc. This happens by the migration of chemicals from food packaging over time after being exposed to conditions such as heat and normal tear and wear.

The main source of **lead** in products is cookware with lead-containing coatings (coating, glaze, enamel), in which the lead content exceeds permissible standards. Lead poisoning is accompanied by the phenomena of general intoxication (weakness, dizziness, headache, unpleasant taste in the mouth). Among the specific phenomena are tremors of the limbs, loss of body weight, blue-gray "lead" border on the gums, lead colic, constipation, anemia. A characteristic sign of chronic lead poisoning is the gray color of the skin - lead color. Nervous system disorders in the form of paresis, paralysis, and convulsions are also noted.

Most often, these are tin metal cans, in which lead passes into products made of tin coating of the tin and lead solder in the seams of the can, as well as glazed ceramic dishes of artisanal manufacture .

Ceramic dishes. According to sanitary standards, the presence of lead is allowed in the manufacture of decorative dishes. It turns out that it is added to paint to give pottery smoothness and a beautiful shine. In the instructions for using such dishes, it must be written that food cannot be stored in it. It has been established that lead and cadmium are almost always present in ceramic products with excessively bright red and yellow painted paint, and copper with a bright green color.

Lead in cans. In addition to dishes as a source of lead poisoning some cans can

also become, as their elements are connected to each other one solder containing lead. Such banks are easy to distinguish by availability corrugated seam and connecting line of silver-gray color with irregular outlines Although the inner surface of the cans is usually covered with a special coating composition, but it does not always help.

Cases are known when up to 3 mg/kg accumulated during long-term storage lead, which is much higher than the permissible level. Especially high concentration it can be in canned acidic products: tomatoes, fruit juices and etc. In addition, they usually contain another toxic component – tin.

To protect yourself from danger, you should buy canned goods in tins cans with smooth welded seams that are between the sticker and the top or the lower end of the cans.

Unlike lead compounds, **copper and zinc** salts cause only acute poisoning. Such poisoning occurs when copper and galvanized dishes are used improperly.

Consumption food containing copper salts, patients experience colicky abdominal pain, bloody diarrhea, vomiting blue-green masses, presence of a coppery taste in the mouth, headache and weakness. The disease ends within the first day. Prevention of copper poisoning is based on the refusal to use non-tinned (tin) copper dishes in everyday life.

Brass. Copper dishes are well washed with hot water by adding a small amount of alkali. If the copper dishes are badly tinned, it should not be used because it produces harmful oxides. Store food in copper dishes are not allowed. Due to its high thermal conductivity, copper has an excellent quality for cooking - the heat is evenly distributed over the surface of the dishes. However, even a very small amount of this metal destroys ascorbic acid in berries and fruits, and food stored in copper dishes loses vitamins, polyunsaturated fatty acids are easily oxidized in it, forming free radicals that are dangerous for the body. In addition, copper easily oxidizes in a humid environment and a green or *blue-green film – patina* - appears on the dishes. When heated, it interacts with food acids, forming copper salts harmful to the body. Therefore, after washing, the plate or basin should be thoroughly wiped, preventing the formation of a film. If, after all, the patina has appeared, then before using the dishes, it must be

carefully removed from the entire surface. This can be done as follows: wipe with table salt dipped in vinegar, and immediately rinse first with warm, then cold water.

Galvanized dishes. Only galvanized dishes can be used for boiling and storing water, but in no case not for cooking, boiling or food storage, because zinc is easily oxidized and forms salts that can cause food poisoning.

The harm of zinc begins with a significant overdose of the metal in the body - 150-600 mg is already poison for a person, and 6 g guarantees a fatal result. In case of metal intoxication, weakness, nausea and other symptoms are observed poisoning. Of course, we rarely meet the metal in such doses, but the damage of zinc can be imperceptible to us due to indirect contact with the element. For example, it is not recommended to drink water from galvanized dishes, soluble zinc compounds have a negative effect on the gastrointestinal tract.

6.2. PESTICIDE POISONING

A pesticide is a common name for all plant growth regulators, fungicides, herbicides, insecticides, rodenticides, molluscicides, and nematicides. Pesticides are widely used globally due to their benefits in controlling the manifestation of pests. They can be applied throughout the food production chain i.e., farm, production, storage, transportation, distribution, processing, and at the consumer level. They are essentially chemicals used to mitigate against pests that cause plant diseases. They are known to affect “target as well as non-target species”. The growth of the agricultural sector has also increased the usage of pesticides over the years. The first-generation pesticides were manufactured in the 1860s and later discontinued due to their toxic effect. In the 1870s, synthetic organic compounds were introduced. However, it was not until the 1940s that pesticides were manufactured and used extensively. In addition, the rise of industrial production in both developed and under-developed nations increased the usage of pesticides in their forests and crop fields. The general long-range transportation of pesticides makes them pollutants that transcend local, regional, and national boundaries. Pesticide poisoning has a detrimental effect on human beings. As reported by Bhalla and colleagues, 250,000–370,000 people die every year due to the

direct or indirect ingestion of pesticides. Moreover, between the years 2010–2014, Japan was reported to use pesticides more than any country. This demonstrates a heavy reliance on pesticides in some countries due to a lack of cost-effective alternatives.

There are about 1400 known pesticides, approximately 80% of all pesticides globally are produced in developed nations annually. Some pesticides such as DDT have long been banned in some countries, however, bioaccumulation is still detected in some streams due to the lasting effects of the chemical compounds. DDT can last in the soil for years until it enters the food chain through adsorption and eventually contaminates the food.

Effects of exposure to pesticides generally fall into three categories: allergic, acute and delayed effects.

Allergic effects. Some people develop a reaction after being exposed to a certain pesticide, a process known as sensitization. Such effects include asthma, skin irritation and eye and nose irritation. Not all people develop allergies; however, certain people seem to be more sensitive than others to chemical irritants.

Acute effects. Acute effects appear immediately or within 24 hours of exposure. These are more accurately diagnosed than delayed effects because they tend to be more obvious. Often they are reversible if appropriate medical care is given promptly, but may be fatal if not treated. Acute effects of pesticides are classified according to the site of the exposure: oral, inhalation, dermal and eye exposures. Table 2 shows typical precautionary statements used on pesticide labels to describe both allergic and acute effects.

Delayed effects. Sometimes, the term "chronic effects" is used to describe delayed effects, but this is only one type of delayed effect. *Delayed effects* also include *developmental, reproductive* and *systemic effects.* Chronic effects are illnesses or injuries that persist over long periods and may not appear until several years after exposure to a pesticide. Chronic effects include production of tumors, malignancy or cancer and changes in the genes or chromosomes. Developmental and reproductive effects occur to the fetus in the womb or by exposure to the reproductive system in men as well as women. These effects include birth defects, miscarriage or stillbirth,

infertility or sterility in men or women and impotence in men. A delayed systemic effect is an illness or injury that does not appear within 24 hours of exposure. Such effects include blood disorders such as anemia or an inability to coagulate; nerve or brain disorders such as paralysis, tremor, behavioral changes and brain damage; skin disorders such as rash; lung and respiratory disorders such as emphysema and asthma; and liver and kidney disorders such as jaundice and kidney failure.

Prevention of pesticide poisoning:

1. Strict control over the production, transportation, storage and use of poisonous chemicals.
2. Strict adherence to agrotechnical methods of using pesticides, taking into account their toxicological characteristics.
3. Control of the content of residual amounts of pesticides in food products. The use of products containing residual amounts of pesticides exceeding the maximum permissible levels is prohibited.
4. Development of special methods of releasing food products from pesticide residues. Thus, it has been established that the most effective methods of freeing milk from pesticide residues are its defatting and drying, which allow almost complete removal of persistent pesticides from the product.

ORGANOCHLORINE (OCPs) POISONING

Chlorinated hydrocarbon (organochlorine) insecticides, solvents, and fumigants are widely used around the world. This class of chemicals comprises a variety of compounds containing carbon, hydrogen, and chlorine and are largely banned in North America and Europe, but are used extensively in many developing nations. In addition, these chemicals may still be found in storage in the United States; thus, exposure remains possible.

Organochlorines are almost everywhere, persistent, hydrophobic, and resistant to degradation. Organochlorine pesticides are chemically stable and semi-volatile, which implies that they last longer in environments and can be transported easily in the atmosphere through the wind, and they are known to be lipophilic. This trait makes

them attach easily to animal and human fatty tissues. OCPs are majorly found in fatty foods due to their fat solubility, e.g., fish, meat, and dairy products. African countries such as Togo, Nigeria, and Ghana still report figures higher than the maximum permissible limits due to their extensive use.

Organochlorines are known to bioaccumulate in organisms and this process can involve bioconcentration and biomagnification. Bioaccumulation means that OCPs enter the tissues of organisms, for example in fat tissues of aquatic organisms such as fish and crustaceans; another term for this is that they are *assimilated*. They may be found in higher concentrations in some tissues, such as liver or kidney than in others like muscle because of their lipophilic nature; this is bioconcentration.

The toxicity of organochlorine pesticides varies according to their molecular size, volatility, and effects on the central nervous system (CNS) by an interference with different ion channels. DDT and analogs interfere with sodium channels, causing both central and peripheral nervous effects. In general, they cause either CNS depression or stimulation, depending upon the agent and dose. Organochlorines are endocrine disruptors by suppressing male and female reproductive systems and acting by dysregulation of immune functions.

Acute OCPs poisoning appear salivation , bronchorrhoea , collapse , toxic damage liver and kidney (acute renal insufficiency, hepatargia). Death is coming from oppression cardiovascular activity and paralysis respiratory center.

Chronic OCPs poisoning cause damage nervous system and parenchymal organs (hepatitis, nephropathy , myocardiodystrophy).

Treatment provides introduction antioxidants (vitamins C, E), amino acids, cytochrome P-450.

ORGANIC MERCURY POISONING

Organic mercury toxicosis usually occurs through ingestion of contaminated food products, especially fish and seafood products due to their bioaccumulation of mercury from contaminated water. Minamata disease was a neurological syndrome described in humans, birds, and cats that were exposed to methylmercury-contaminated fish in the Minamata area of Japan in the 1960s. Organomercurials are also used as fungicides for

seeds and plants. In humans, sporadic outbreaks of neurological diseases related to ingestion of flour made from ethylmercury-treated seed grain were reported in Iraq in the 1950s through 1970s.

Organomercurials are well absorbed by inhalation or ingestion. In ruminants, methylmercury is demethylated to inorganic mercury, decreasing absorption. Dermal absorption, although slow, can be sufficient to cause toxicosis. Absorbed organomercurials are transported bound to red blood cells and are widely distributed throughout the body. Organomercurials readily cross the blood–brain barrier and placenta, where fetal blood levels can exceed maternal blood levels by up to 20%. Organomercurials also are secreted into milk. The biological half-life of organic mercury in humans is approximately 65 days.

Alkyl mercury compounds have stable mercury–carbon bonds that are slowly cleaved after absorption, making these products more persistent and more toxic. Aryl and alkoxyalkyl mercury compounds have mercury–carbon bonds that are more readily cleaved, and these compounds are subsequently catabolized to free mercury ions, which are then eliminated. Methylmercury is the most toxic of organomercurials, primarily due to its persistence (as demethylated inorganic mercury) within the central nervous system. Methylmercury that is not sequestered within the nervous system is demethylated and primarily excreted in the feces as inorganic mercury.

The primary target organ for organomercurials is the nervous system. Signs may begin days to weeks following exposure, depending on the amount and type of organic mercury involved. Neurological effects include visual disturbances, ataxia, paresthesia, hearing loss, dysarthria, mental deterioration, muscle tremor, sensory dysfunction, movement disorders, behavioral changes, and death. Pigs tend to develop a paralytic disease, while in cattle the syndrome resembles lead toxicosis. Rats and monkeys (*Macaca fascicularis)* develop ataxia, impaired motor function, and gait abnormalities upon exposure to dietary organomercurials. Neurologic lesions include cortical neuronal loss that may be manifested grossly as laminar cortical necrosis, with or without cerebellar or cerebral edema.

Non-neurologic lesions of organomercury toxicosis are less commonly reported. Renal lesions described in rats, cattle, and pigs include grossly swollen, wet, pale kidneys.

Initial manifestations of acute poisoning consist of general weakness , headache, dyspeptic disorders . In the mouth maybe to feel unpleasant " metallic " taste , bleeding gums, characteristic asthenovegetative syndrome, lumbosacral radiculitis , polyradiculoneuritis , toxic encephalomyeloneuritis , pain and paresthesias in the limbs , disorders superficial species sensitivities in the form of "mittens", " socks" or stocking. Distal parts of limbs acquire cyanotic coloring. Possible development diencephalon syndrome, toxic nephropathy , toxic myocarditis .

Chronic poisoning manifests mercury erythrism , mercurial tremor, diencephalic disorders with toxic encephalopathy (in late stages disease). Characteristic manifestations are toxic hepatitis, myocardiodystrophy, anemia (hypochromic . In case of mild and moderate intoxications degree in blood and urine mercury content is found which exceeds 0.01 mg/l.

Diagnostic value has definition content mercury in urine (from 0.01 to 0.5 mgl).

Treatment provides application antidote therapy (donors thiols groups): unitiol (5-10 ml of 5% solution intramuscularly every 3-6 hours), succimer (0.3 g intramuscularly the first two days after 6 hours, days 3 and 5 after 8 hours, on days 6 and 7 - after 12 hours).

ORGANOPHOSPHORUS PESTICIDES

The organophosphorus insecticides are the most widely used class of insecticides today. More than 40 of them are currently registered for use, and all pose the risk of acute toxicity. Examples of commonly used organophosphorus include chlorpyrifos, diazinon, malathion and methyl parathion. According to the American Association of Poison Control Centers, organophosphorus are the most commonly implicated class of all pesticides in symptomatic illnesses (1996 data). All apparently share a common mechanism of cholinesterase inhibition and can cause similar symptoms. Because they share this mechanism, exposure to the same organophosphate by multiple routes or to

multiple organophosphorus by multiple routes can lead to serious additive toxicity. It is important to understand that there is a wide range of toxicity in these agents and wide variation in their absorption capacities. Exposure by inhalation results in the fastest appearance of toxic symptoms, followed by the gastrointestinal route and finally the dermal route. The most commonly reported early symptoms include headache, nausea, dizziness, and increased secretions, such as sweating, salivation, tearing and respiratory secretions. Progressive symptoms include muscle twitching, weakness, tremor, incoordination, vomiting, abdominal cramps and diarrhea. Some victims may have altered vision, such as blurred or dark vision. Persons attending a victim should avoid direct contact with contaminated clothing and wear chemical-resistant gloves while washing the pesticide from the victim's skin and hair. Antidotes are available for treating organophosphate exposure, most notably atropine sulfate. In all cases, antidotes should be administered by a health professional.

Blood tests can be used to determine whether organophosphorus have accumulated in a person's body. One such test uses cholinesterase, an enzyme that occurs naturally in the blood at levels that vary from one person to another. "Baseline" levels of cholinesterase for an individual can be determined at a time of the year when pesticide handling is minimal. The baseline helps determine the normal level of cholinesterase in the body. Other tests throughout the year indicate if there is a reduction in the baseline level. If a reduction has occurred, the individual should not apply organophosphate insecticides. The body normally produces new cholinesterase continuously, and levels return to normal after several weeks.

Carbamates

Carbamate insecticides are widely used in homes, gardens and agriculture. Common examples of carbamates include aldicarb, carbaryl and carbofuran. Like organophosphorus, they inhibit cholinesterase enzymes and therefore share similar exposure symptoms, although carbamate poisonings tend to be of shorter duration (less than 24 hours). Toxic exposures to carbamates can occur via dermal, inhalational, and gastrointestinal exposures.

As with the organophosphorus, blood tests can determine whether carbamate insecticides are affecting cholinesterase levels.

The contamination from organophosphorus pesticides can be removed by using microorganisms in the process of bacterial degradation. Past studies as reported by researchers have shown that lactic acid bacteria such as *Lactobacillus bulgaricus* and *Streptococcus thermophilus* have the ability to degrade pesticides. Moreover, enzymes such as phosphodiesterases, methyl parathion hydrolases, and organophosphorus acid anhydrolases can degrade organophosphorus pesticides. Other studies have reported *Saccharomyces cerevisiae* yeast during fermentation as a potent option in the removal or reduction of some pesticides in food.

Table 5 – Summary of the classification of pesticides and human health effects.

Pesticide Name	Classification	Route of Exposure/ Pathway	Documented Health Effects
Dichlorodiphenyl-trichloroethane (DDT)	Organochlorine	Ingestion, inhalation-Crop fields	Parkinson's disease, neurotoxic effects
Hexachlorocyclohexane	Organochlorine	Ingestion of contaminated food	Birth defects in humans
Benzene hexachloride	Organophosphorus	Ingestion Locust control	Liver disease, skin lesion, loss of hair, thyroid damage, ulceration
Malathion, chlorpyrifos, diazinon, temephos	Organophosphorus	Ingestion	Neurologic toxic effects, impaired vision, headache, dizziness

Management pesticide contamination in food.

Therefore, to control the risk of pesticide exposure, risk assessment models need to be applied in food production industries and retailers to assess the level of pesticide residues.

Daily consumption of food comes with potential daily exposure to pesticides. Most countries have pre-determined maximum permissible limits as recommended by

FAO/WHO/Codex Alimentarius. In Europe, the European Food Safety Authority (EFSA) conducts independent research on risk assessments and further advice authorities on permissible levels. Essentially, the EFSA functions as a gatekeeper to the European Commission regarding the approval of new substances and setting new maximum residue levels after completing their rigorous peer review and due diligence on potentially harmful substances. In cases where pesticide residues exceed the maximum residue level, the exposure must be compared with the acceptable daily intake (ADI) and/or the Acute Reference Dose (ARfD) to assess the risk for the consumer. Researchers define the ADI as "the number of pesticides in mg/kg to which humans can be exposed to daily through ingestion during a lifetime without appreciating risks to the health on the bases of all known facts at the time of evaluation". On the other hand, the ARfD is established for the general population based on children and infants, including women who are considered to be of childbearing age. Essentially, it is the exposure level at which harmful effects are likely to occur in the most sensitive individuals in a population during a single day exposure, within 24 h. Member countries affiliated with the WHO have established threshold limits for food contaminants to comply with recommendations as defined in the Codex Alimentarius.

Table 6– Maximum allowable levels of pesticide residue that may be present in food according to the Codex Alimentarius International Food standards

Pesticide	**Foodstuff**	**Maximum Residue Levels (MRL) (mg/kg)**
Abamectin	Citrus fruits	0.02
	Soya beans (dry)	0.002
Acephate	Cabbages, head	2
	Meat (From mammals other than marine mammals)	0.05
DDT	Carrot	0.2
	Cereal grains	0.1
	Poultry meat	0.3
Azoxystrobin	Strawberry	10
	Sunflower seed	0.5
	Banana	2
	Soya bean (dry)	0.5

Pesticide	Foodstuff	Maximum Residue Levels (MRL) (mg/kg)
	Sorghum	10
	Sugar cane	0.05
	Poultry meat	0.01
	Rice	5
Tebuconazole	Apples	1
	Apricot	2
	Barley	2
	Broccoli	0.2
	Carrot	0.4
	Coffee beans	0.1
	Prunes, dried	3
	Tomato	0.7
	Wheat	0.15

7. TEST QUESTIONS FOR SELF-CONTROL

Choose the food contains solanine

A. apricot and peach kernels

B. potatoes

C. peas

D. beans

E. beech nuts

Typical organoleptic sign indicating the contamination of bread with weed impurities:

A. bread has a bitter aftertaste

B. bright red round spots on bread crust

C. bread crumb has mucous consistency

D. bread has a sickly sweet taste

E. white grains on bread crust

Toxic effect of poison caused by Amanita phalloides also known as 'death cap':

A. hepatotropic action

B. neurotropic action

C. local irritant effect
D. cardiotoxic action
E. nephrotoxic action

Toxic effect of poison caused by Amanita muscaria also known as 'fly agaric':

A. hepatotropic action
B. neurotropic action
C. local irritant effect
D. cardiotoxic action
E. nephrotoxic action

Organochlorine compounds are characterized by:

A. small cumulative properties, they are not excreted with the milk of lactating animals
B. pronounced cumulative properties, the ability to be excreted with milk
C. relatively low toxicity
D. carcinogenic, mutagenic and teratogenic properties
E. relatively quickly inactivated in the external environment

Organomercury compounds are characterized by:

A. small cumulative properties
B. high toxicity, significant stability in the external environment, long-term storage in food products
C. relatively low toxicity
D. carcinogenic, mutagenic and teratogenic properties
E. relatively quickly inactivated in the external environment

Source of food poisoning by tetrodotoxining:

A. mullet fish
B. lampreys

C. puffer fish

D. mackerel

E. oilfish (ruvetta)

During the examination, a blue-green film of patina was found on the bottom of the pan. Specify which metal caused its formation?

A. Copper.

B. Tin.

C. Lead

D. Iron.

E. Aluminum.

Why is it not recommended to use copper dishes for cooking and storing food?

A. Copper in a humid environment is easily oxidized and a patina appears on the dishes, which interacts with food acids and forms copper salts.

A. Even a small amount of copper destroys ascorbic acid in berries and fruits.

C. Food stored in copper dishes loses vitamins.

D. It easily oxidizes polyunsaturated fatty acids, forming free radicals.

E. All answers are correct.

Specify the substances that belong to the group of biogenic amines:

A. Histamine, serotonin, tryptamine, tyramine, cadaverine, putrescine.

B. Histamine.

C. Serotonin and histamine.

D. Tryptamine, tyramine.

E. Cadaverine, putrescine.

For what purposes can galvanized dishes be used?

A. For making compotes.

B. To prepare the first dishes.

C. For frying meat.

D. For boiling and storage

What toxic substances can migrate from a tin can into canned foods, mainly acidic: tomatoes and juices?

A. Lead and tin.

B. Copper and aluminum.

C. Aluminum and lead.

D. Zinc and iron.

E. Iron and mercury

8. SITUATIONAL TASKS

Task 1. In May, family, consisting of 5 people, left the city for the country to do agricultural work. Two dishes were prepared for lunch - mushroom soup and fried mushrooms from morels, which grow in large quantities in this spring period.

8-9 hours after dinner, the father and mother developed nausea, dizziness, general weakness, and poor health. A little later, about an hour later, vomiting appeared, which intensified during the day and was accompanied by abdominal pain. The condition of the parents did not improve and they were hospitalized.

On the 2nd day, both patients developed jaundiced staining of the sclera, skin, and a severe headache. On the 3rd or 4th day, the condition of the patients began to improve.

From the survey of the patients, it was found that fried mushrooms were eaten by all family members, and mushroom soup was eaten only by the father and mother. Before use, all mushrooms were washed, sliced, and then boiled. One part of the boiled mushrooms and their broth was used to prepare soup, and the other part was re-washed, thoroughly squeezed, and then fried.

Specify the diagnosis of food poisoning, the "suspicious product" (dish), the cause of the disease, measures to prevent similar diseases.

Task 2. An adolescent ate an apple from the garden for several hours after spraying trees with a solution of a substance with insecticidal action without washing eat. After a while, he developed increased secretion of saliva, sweat, tears, abdominal pain, and diarrhea. On examination, he was found to have miosis.

Which group of substances caused the symptoms? What is the pathogenetic mechanism? Which agent should be administered as the first aid in poisoning with organophosphate insecticides? How to prevent this poisoning?

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Електронне навчальне видання комбінованого використання
Можна використовувати в локальному та мережному режимі

Редька Ірина Василівна

НЕМІКРОБНІ ХАРЧОВІ ОТРУЄННЯ

Методичні рекомендації до практичних занять
для здобувачів вищої медичної освіти 3-го року навчання
з дисципліни «Гігієна та екологія»

(Англ. мовою)

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