Poisoning in Large Animals

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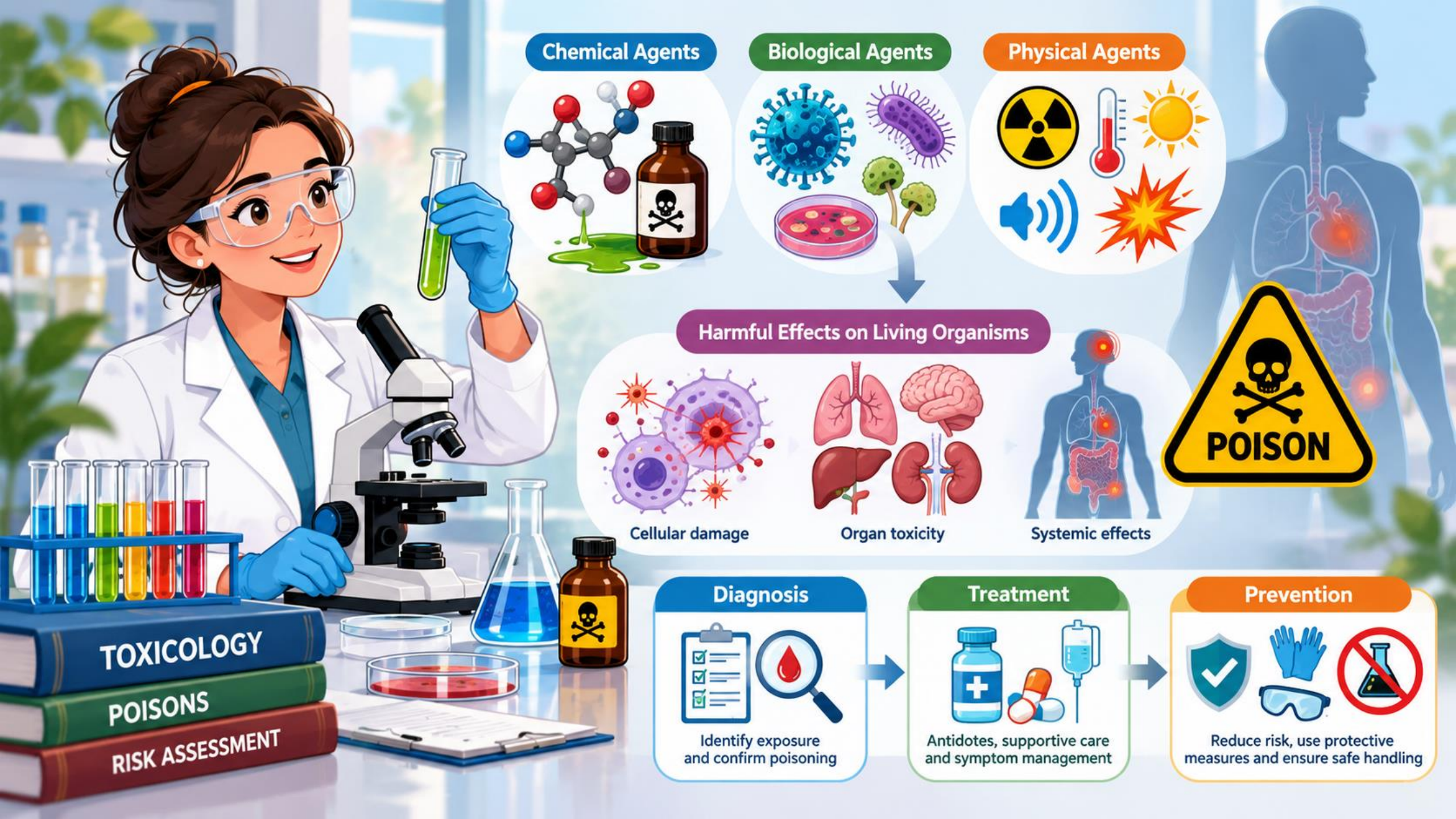
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Introduction


Chemical Agents
Biological Agents
Physical Agents
Harmful Effects on Living Organisms
Cellular damage
Organ toxicity
Systemic effects
POISON
TOXICOLOGY
POISONS
RISK ASSESSMENT
Diagnosis
Identify exposure and confirm poisoning
Treatment
Antidotes, supportive care and symptom management
Prevention
Reduce risk, use protective measures and ensure safe handling

Poison, Toxic Potential, and Radiation Toxicology

Poison

A **poison** is any substance or agent that, after entering a living organism or being produced within the body, causes disruption of normal cellular, tissue, organ, or physiological functions and may lead to illness, injury, or death depending on the dose and conditions of exposure.



Toxic Potential

Substances that have the potential to produce harmful effects under specific conditions, such as increased dose, prolonged exposure, altered route of administration, or increased susceptibility of the organism, are considered to have toxic potential.

Increased dose



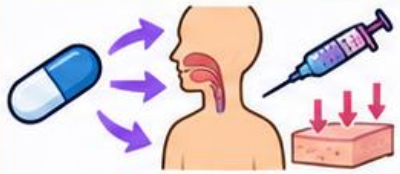
Higher amounts can increase harm.

Prolonged exposure



Longer exposure period can increase toxic effects.

Altered route of administration



Different routes can change the level of toxicity.

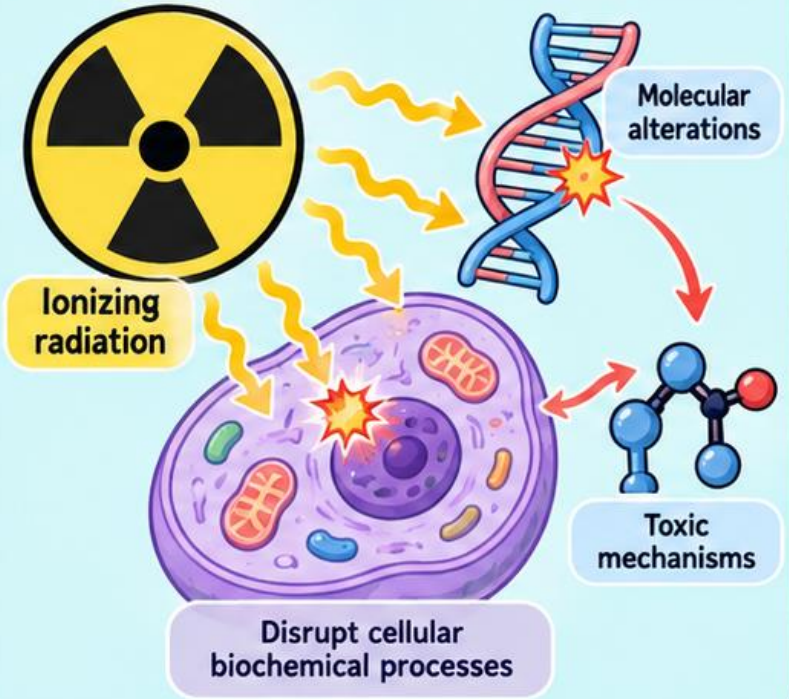
Increased susceptibility of the organism



Certain individuals may be more vulnerable to toxic effects.

Radiation Toxicology

Harmful physical agents, such as ionizing radiation, are also studied in toxicology because they can induce molecular alterations and disrupt cellular biochemical processes through toxic mechanisms, particularly within the field of radiation toxicology.



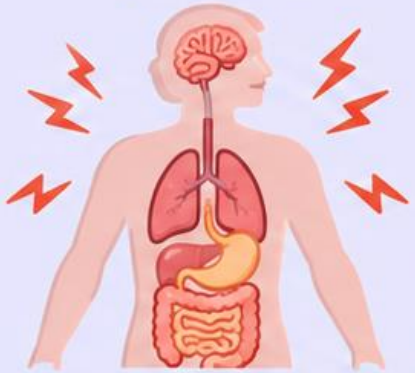


Classification of Poisons



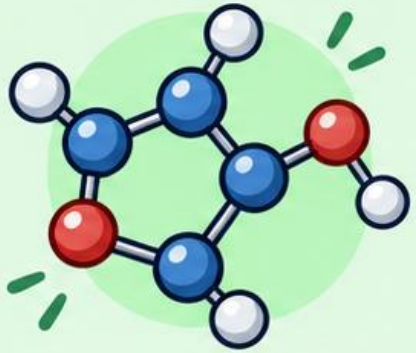
Poisons can be classified according to different criteria, including:

1 Toxic effects produced



Poisons can be grouped based on the specific harmful effects they produce in the body (e.g., on the nervous, respiratory or digestive systems).

2 Chemical structure and properties



Classification can be based on chemical structure (e.g., organic or inorganic compounds) and physicochemical properties such as solubility, volatility and reactivity.

3 Origin or source



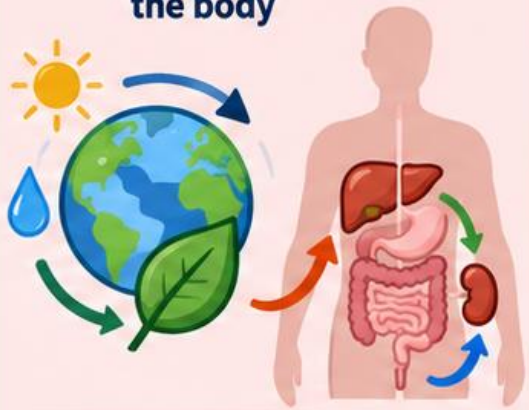
Poisons can be classified according to their natural or synthetic origin, such as plants, animals, microorganisms, minerals or industrial sources.

4 Uses and applications



Classification can be based on how the poison is used, such as pharmaceuticals, pesticides, industrial chemicals or household products.

5 Methods of degradation and their fate in the environment and within the body



Poisons can be classified according to how they are degraded in the environment (e.g., by sunlight, water or microorganisms) and their fate in the body (absorption, metabolism and elimination).



In conclusion:

In modern toxicology, poisons are mainly classified based on their **origin** and **chemical structure,** because these factors strongly influence toxic properties, mechanisms of action, absorption, metabolism, and elimination.



Clinical Forms of Poisoning

Classification of Poisoning in Animals by Onset and Duration

Sudden death



Death occurs without obvious clinical signs or after a very short period of clinical manifestations.

Peracute poisoning



A rapidly developing form of poisoning in which severe clinical signs or death usually occur within **a few hours to a maximum of 48 hours** after exposure.

Acute poisoning



Poisoning with a rapid onset and a clinical course lasting approximately from **48 hours to two weeks.**

Subacute poisoning



Poisoning with a clinical course generally lasting from **two to four weeks.**

Chronic poisoning



Poisoning resulting from prolonged or repeated exposure to toxic agents, with clinical signs persisting for **more than four weeks.**

Very short / sudden

A few hours – 48 hours

48 hours – two weeks

Two – four weeks

> 4 weeks

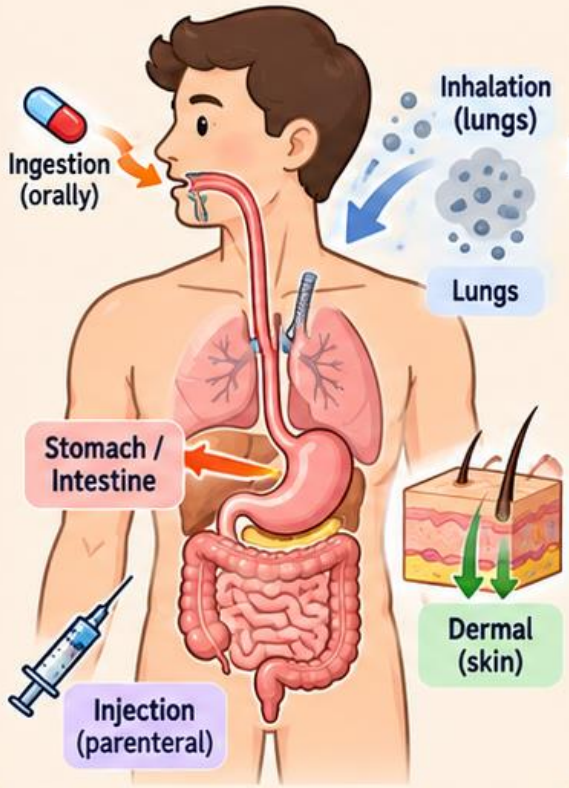
Toxicokinetic

Fate of Toxic Substances in the Body (ADME)

The fate of toxic substances in the body generally involves four main processes known as ADME:

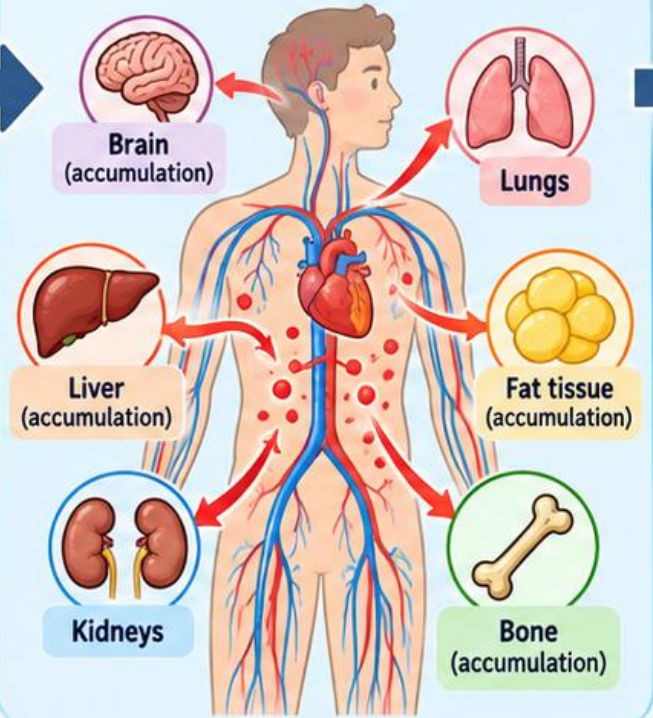
1 Absorption

Toxic substances enter the body through various routes such as the gastrointestinal tract, lungs, skin, or injection.



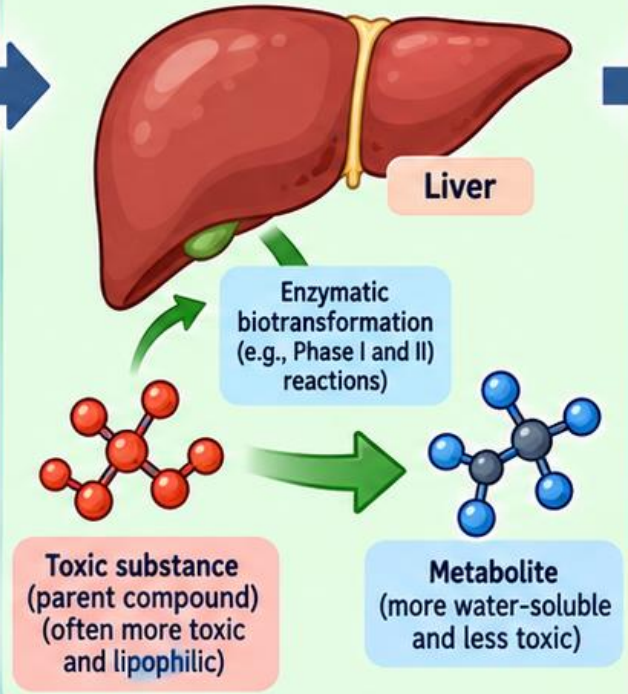
2 Distribution and Accumulation

After absorption, toxic substances are carried by the bloodstream to various organs and tissues, where they may distribute and accumulate, especially in fat, liver, kidneys, brain, or bones.



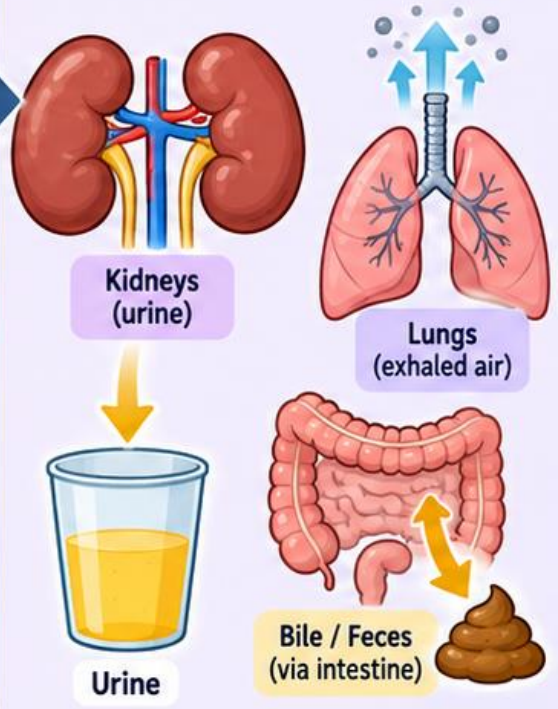
3 Biotransformation (Detoxification)

Toxic substances are chemically modified, mainly in the liver, into more water-soluble and less toxic metabolites, which makes them easier to excrete.



4 Excretion

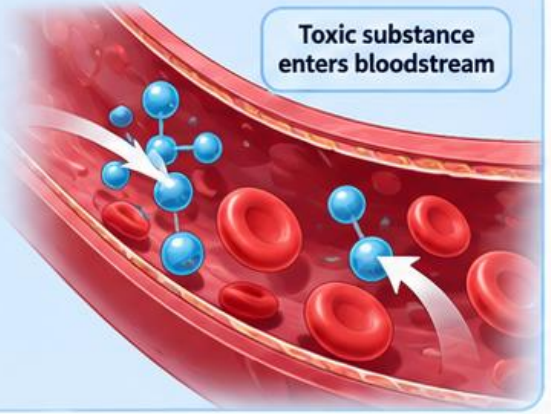
The parent compound and/or its metabolites are eliminated from the body, mainly via the kidneys (urine), but also through bile/feces or exhaled air (lungs).



Absorption

Absorption of Toxic Substances

For most toxic substances to produce systemic toxic effects, they **must be absorbed** from their site of entry and **enter the bloodstream.**



Exception

Some toxic agents produce their effects through direct tissue destruction without requiring systemic absorption, including:



Strong acids



Strong alkalis



Phenolic compounds

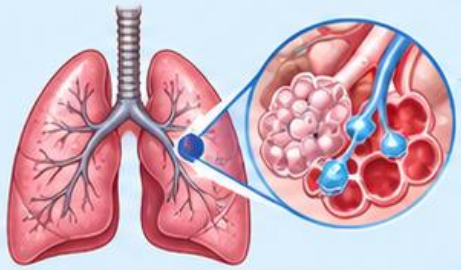
These substances cause toxicity mainly through **direct corrosive effects** and **tissue necrosis.**

Routes of Absorption of Toxic Substances

The major routes of absorption include:

1. Respiratory tract

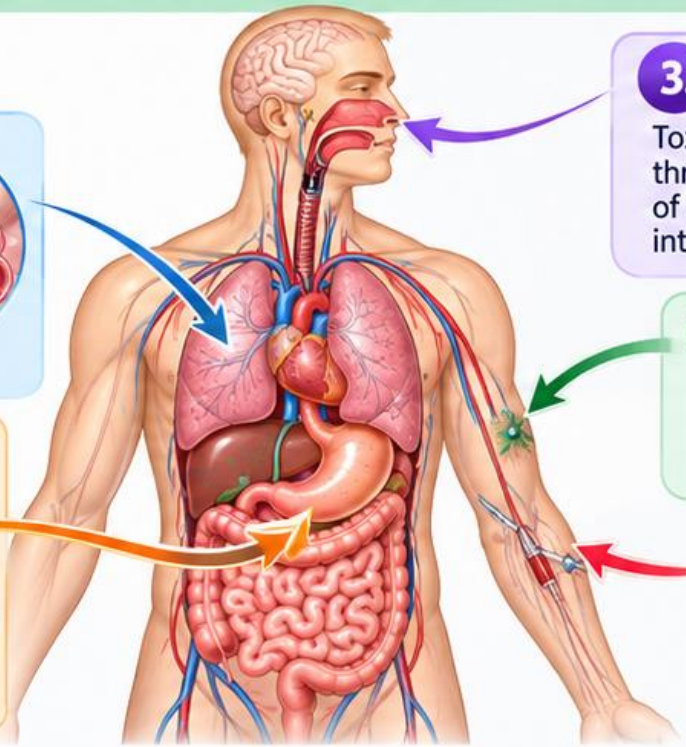
Toxic substances can be inhaled as gases, vapors, aerosols or dusts, and absorbed through the alveoli into the bloodstream.



2. Gastrointestinal tract

Toxic substances can be ingested and absorbed through the stomach and intestines into the bloodstream.





3. Mucous membranes

Toxic substances can be absorbed through the mucous membranes of the eyes, nose, and oral cavity into the bloodstream.



Eye (conjunctiva)



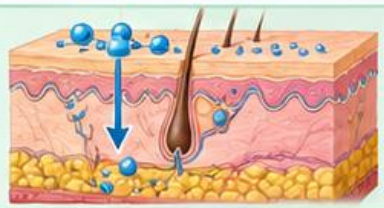
Nasal mucosa



Oral mucosa

4. Skin

Many toxic substances can be absorbed through intact or damaged skin into the bloodstream.



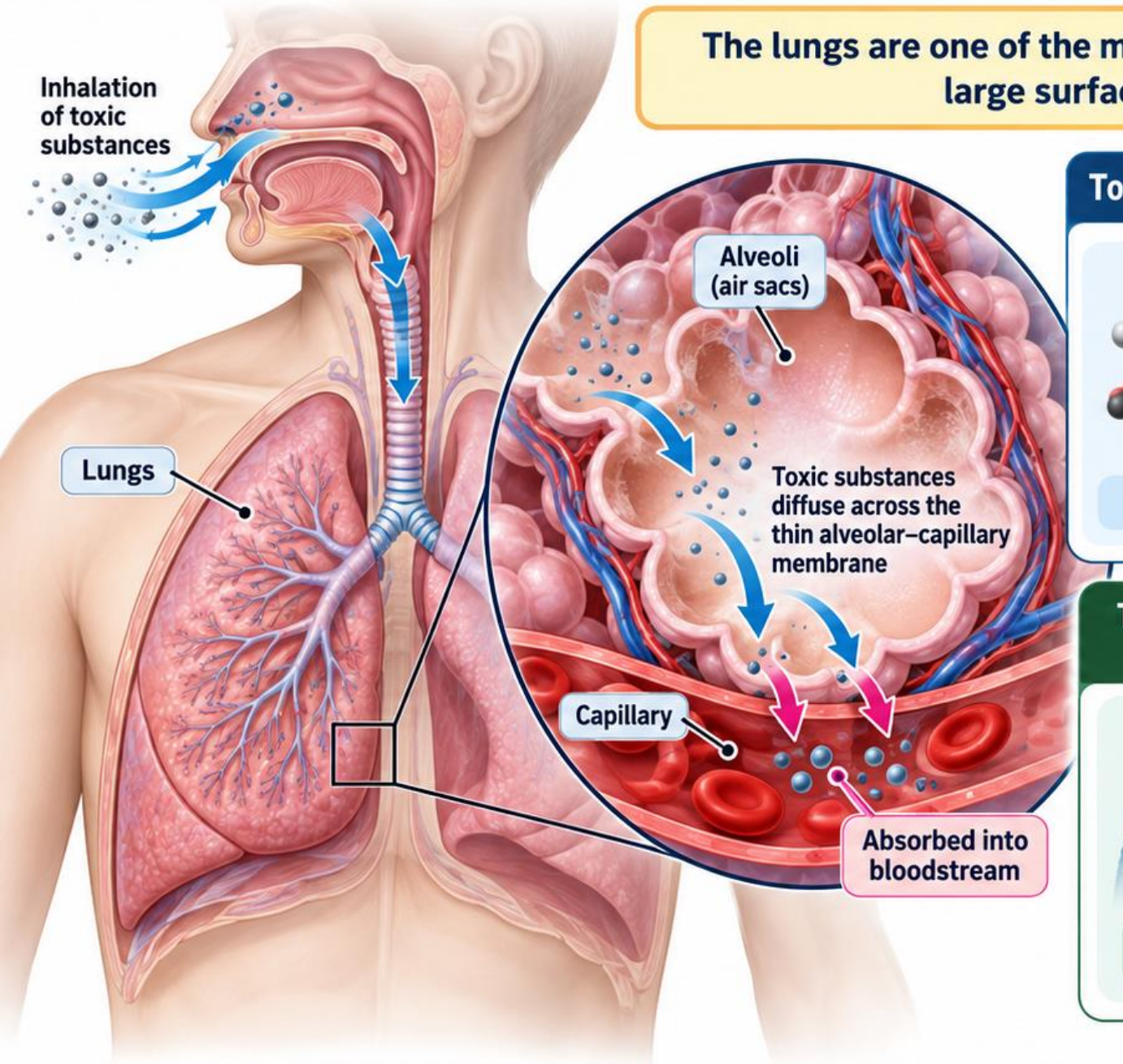
5. Injection (iatrogenic exposure)

Toxic substances can enter directly into the bloodstream via injection (e.g., accidental or therapeutic exposure).



Respiratory Absorption of Toxic Substances

The lungs are one of the most efficient routes for toxin absorption due to their large surface area and extensive blood supply.



Toxic substances absorbed through the respiratory system include:



Gases



Vapors



Dust particles

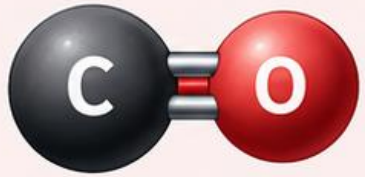


Aerosols and sprays

This route is more important in humans; however, it is also relevant in animals, including exposure to:



Inhalational anesthetic agents

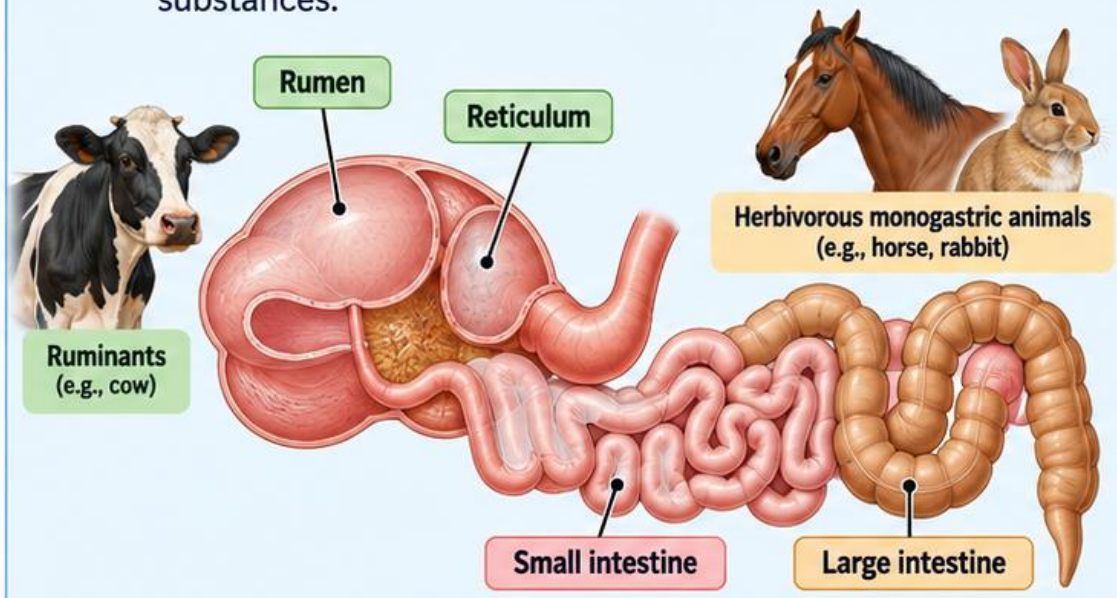


Carbon monoxide (CO)

Gastrointestinal Absorption of Poisons

1 Gastrointestinal Tract

- The gastrointestinal tract is the **most common route** of toxin entry in animals.
- In most species, the **small intestine** is the primary site of absorption.
- However:
  - In **ruminants**, the **rumen** and **reticulum** play important roles in toxin absorption.
  - In herbivorous monogastric animals, the **large intestine** may also contribute to absorption of certain toxic substances.



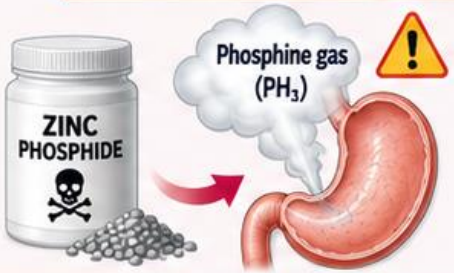
2 Factors Affecting Gastrointestinal Absorption of Poisons

A Fullness of the Stomach and Rumen

A full stomach or rumen generally:

1. Delays toxin absorption
2. Dilutes toxic substances
3. Allows ruminal microorganisms to degrade and inactivate some toxins

Exception: Zinc Phosphide



In a full stomach, gastric acid reacts with zinc phosphide and produces phosphine gas, a highly toxic compound, which may result in:

- **Severe poisoning**
- **Gastroenteritis**

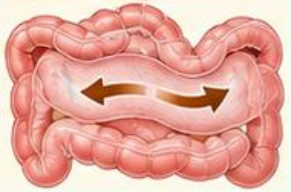


Conversely, when zinc phosphide enters an **empty stomach**, irritation of the gastric mucosa may **induce vomiting** and **promote removal** of part of the toxin.

B Intestinal Motility

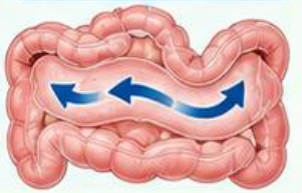
Stasis

- Reduced intestinal movement increases contact time and may **increase toxin absorption.**



Hyper-motility

- Increased intestinal movement causes **diarrhea**, reduces contact time, and **decreases toxin absorption.**



3 Summary

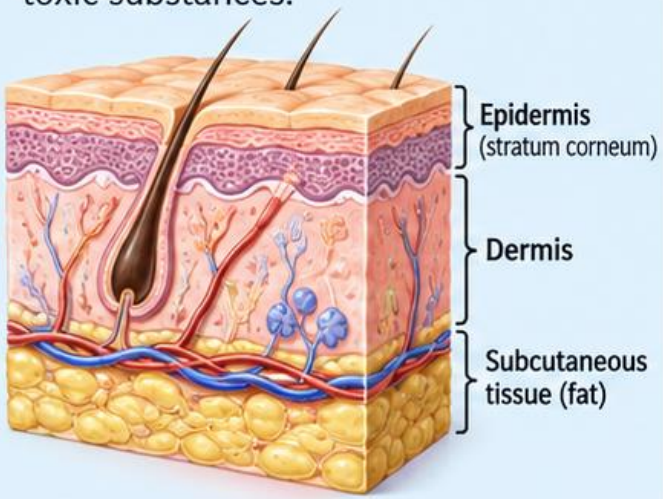
In summary, gastrointestinal absorption of poisons is influenced by:

1. Physical and chemical properties of the toxin
2. Animal species
3. Physiological and chemical conditions of the gastrointestinal tract

Dermal Absorption of Toxic Substances

1 Skin

Intact skin is generally not an efficient route for absorption of most toxic substances.



2 Factors affecting dermal absorption



Lipid solubility
More lipid-soluble compounds are generally absorbed more readily through the skin.



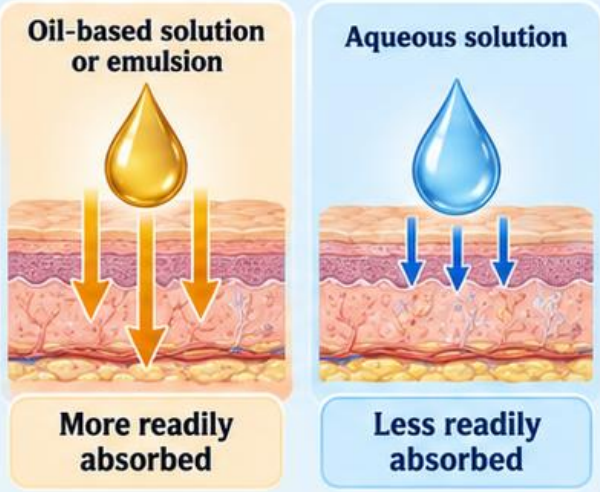
Chemical structure of the compound
Molecular size, polarity, and other structural features affect absorption.



Skin integrity
An intact stratum corneum limits penetration.

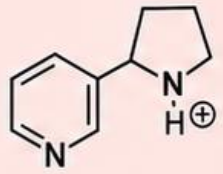
3 Type of formulation

Oil-based solutions and emulsions are absorbed more readily than aqueous solutions.

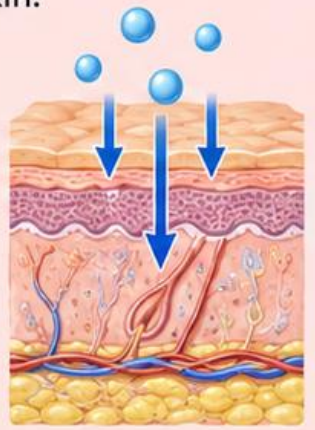


4 Exception

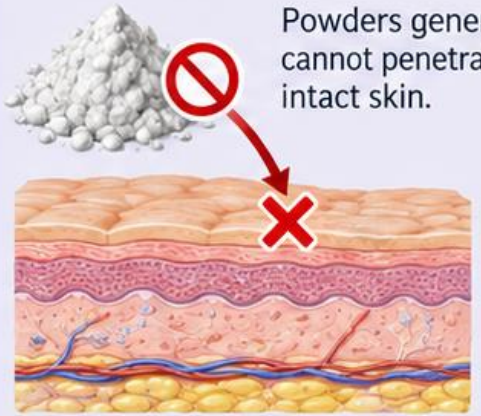
Aqueous solutions of **nicotine salts** can be absorbed effectively through the skin.



Nicotine
(salt form)

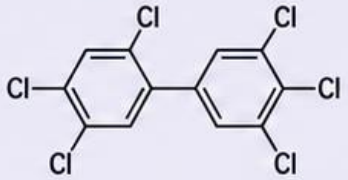


5 Powders and special compounds

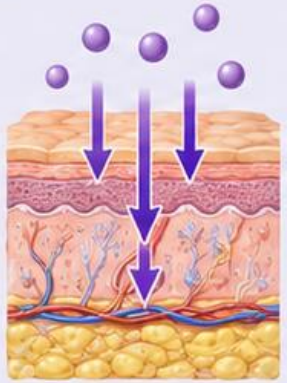


Powders generally cannot penetrate intact skin.

However, some compounds such as **organochlorine hydrocarbons** can pass through healthy skin.

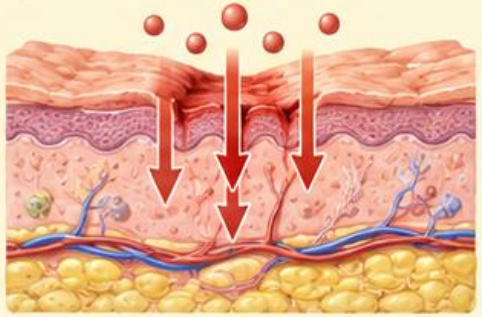


Organochlorine hydrocarbons
(e.g., DDT)



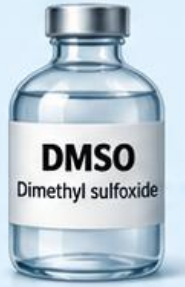
6 Damaged skin

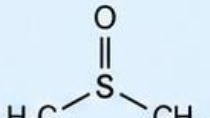
Damaged skin has much higher permeability, allowing easier penetration of toxic substances.

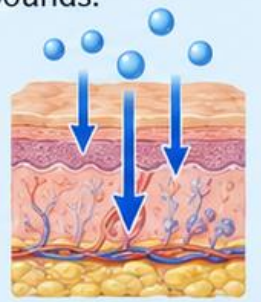


7 DMSO

Dimethyl sulfoxide (**DMSO**) may be used experimentally or therapeutically as a **penetration enhancer** to increase dermal absorption of certain compounds.





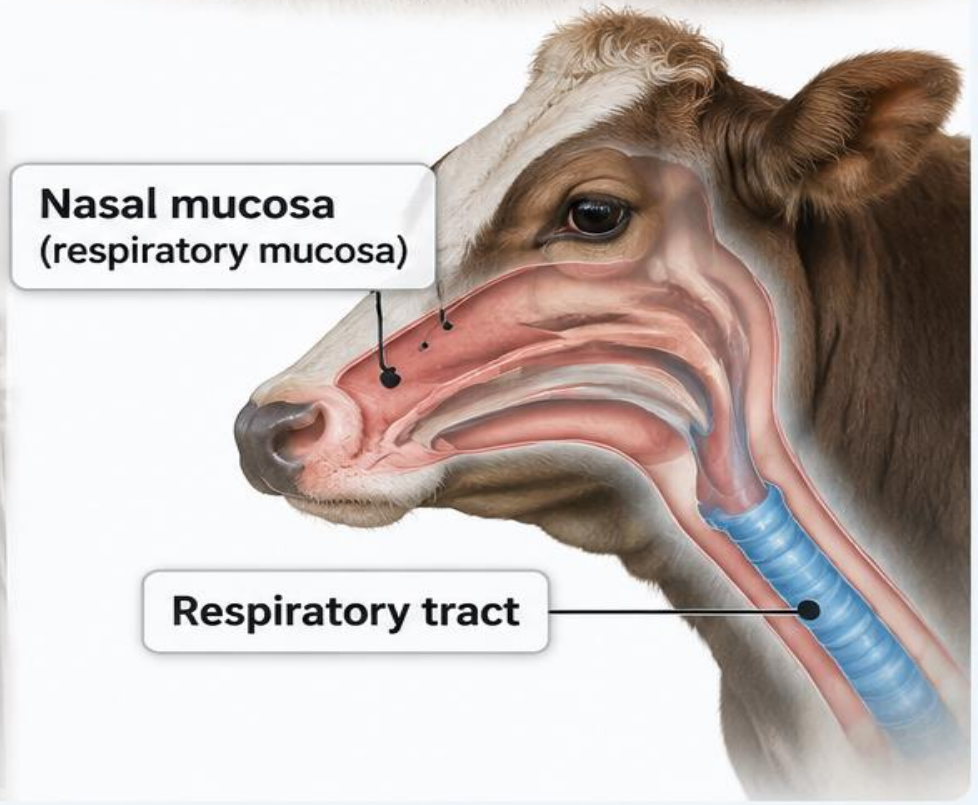


Mucous Membranes

- The **ocular mucosa** is an important route of toxin absorption.
- **Example:**
  Spraying organophosphate compounds in enclosed areas such as barns may result in toxin absorption through ocular and respiratory mucous membranes.



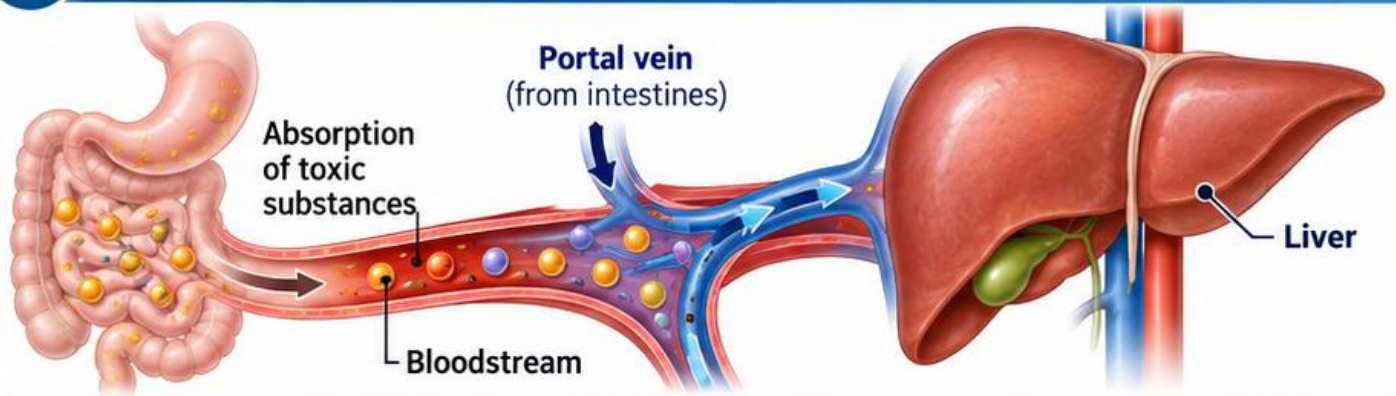




Distribution

Distribution and Selective Accumulation of Toxic Substances

1 Transport to the Liver

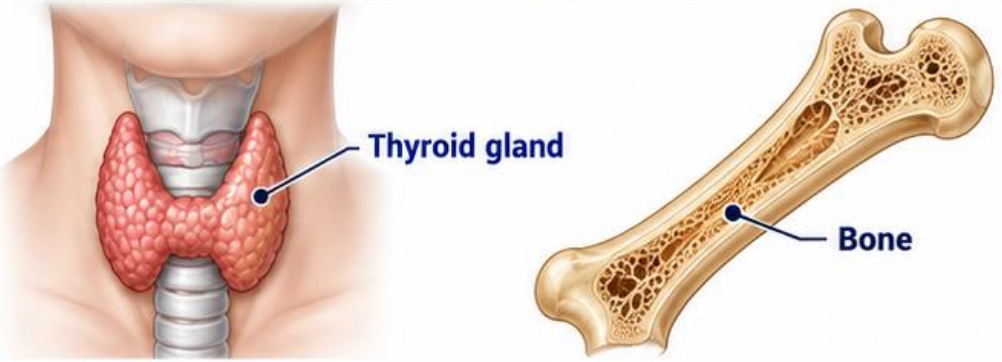


After absorption, toxic substances are transported through the **bloodstream**; many absorbed compounds reach the liver through the **portal vein**, where they undergo detoxification or, more accurately, **biotransformation** processes.

The **liver** is one of the major organs involved in metabolism of toxic substances and is frequently affected in poisoning cases; many toxic agents produce characteristic **liver lesions** because of its central role in **xenobiotic metabolism**.

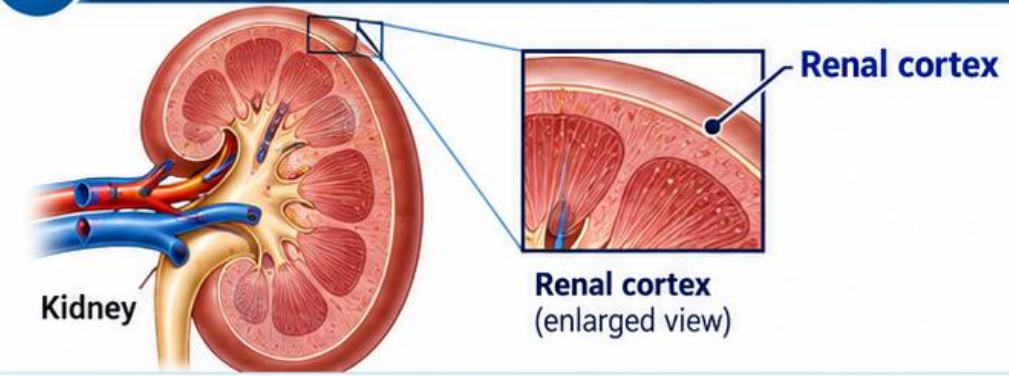
2 Selective Accumulation in Specific Organs

Toxic substance	Main site of accumulation
Iodine	Thyroid gland
Lead, Fluoride, Strontium	Bone



Accumulation of certain elements in bone may **temporarily reduce their toxicity** because they become relatively immobilized and less available to active tissues.

Protective Accumulation Example: Lead



- Sometimes, accumulation of a toxic substance in a specific tissue occurs as a **protective mechanism** to minimize damage during elimination.
- **Example: Lead**
- Lead may accumulate in the **renal cortex**, which reduces its penetration into deeper renal structures and limits damage to more sensitive areas.
- Therefore, the renal cortex is considered one of the appropriate tissues for sampling in suspected lead poisoning cases.

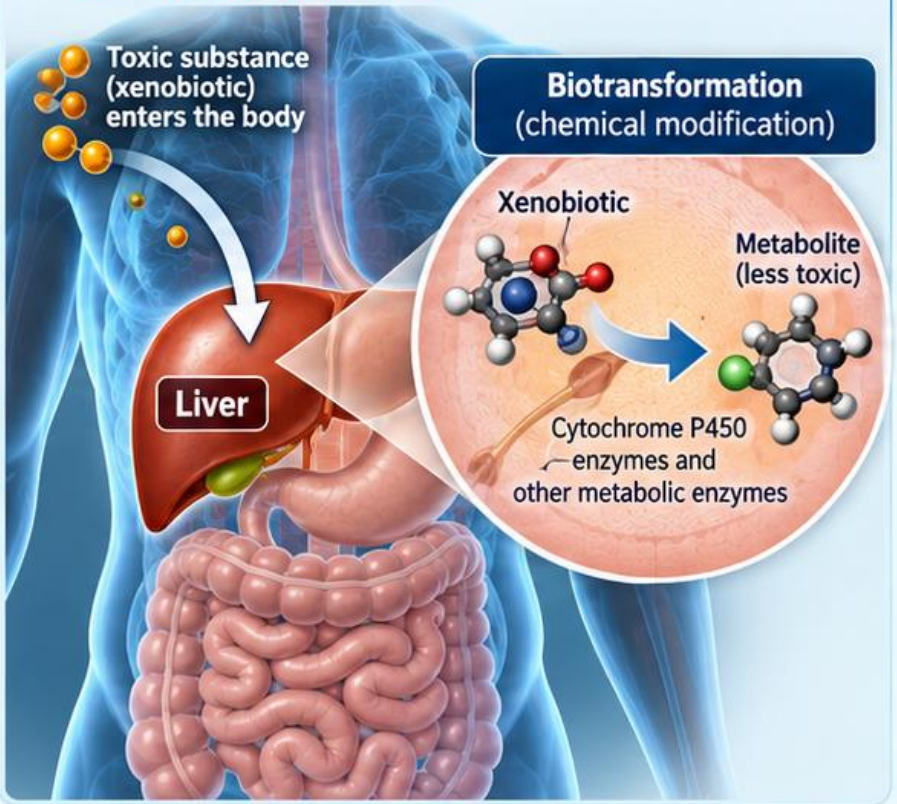
Biotransformation

Biotransformation of Toxic Substances

Biotransformation may lead to **detoxification**, **no change**, or **increased toxicity**.

1 Introductory concept

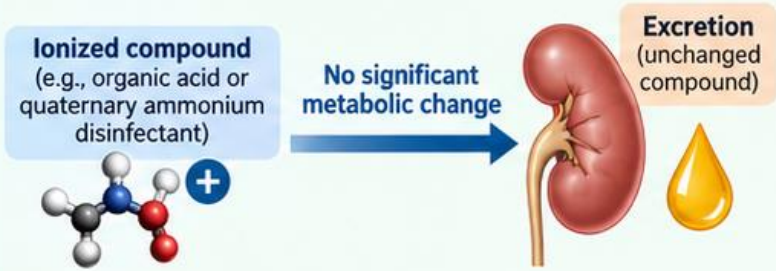
- Most toxic substances and foreign compounds (xenobiotics) undergo chemical modifications inside the body to **reduce toxicity** or convert them into **less harmful compounds**.
- **Biotransformation does not always result in detoxification.**



2 Exceptions

A Elimination without modification

- Some **highly ionized** chemical compounds, such as certain organic acids and quaternary ammonium disinfectants, may be **eliminated from the body without undergoing significant metabolic changes**.



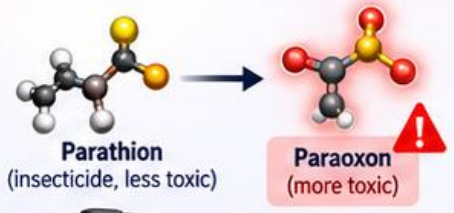
B Metabolic Activation (Lethal Synthesis)

- In some cases, biotransformation does not decrease toxicity; instead, it produces a more toxic metabolite.
- This process is called **metabolic activation**, historically called **lethal synthesis**.

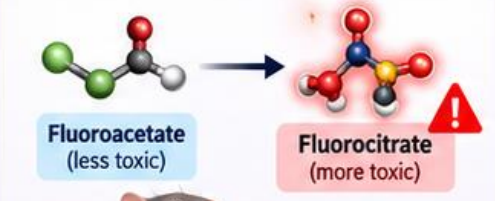


3 Examples of metabolic activation

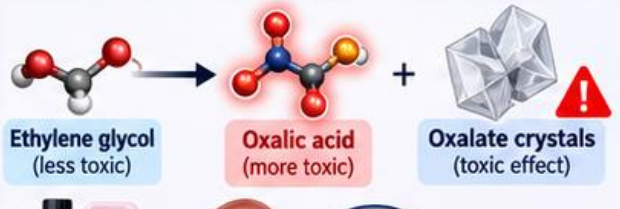
Parathion → Paraoxon



Fluoroacetate → Fluorocitrate



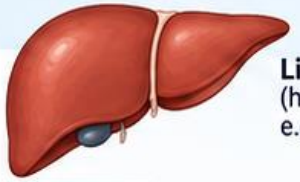
Ethylene glycol → Oxalic acid and oxalate crystals



Stages of Biotransformation and Species Differences

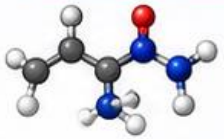
1 Phase I Reactions

- Biotransformation generally occurs in two major phases.
- Xenobiotics are modified by hepatic enzymes through:
  - Oxidation
  - Reduction
  - Hydrolysis
- Oxidation reactions are the most common in Phase I.
- Many ester-containing toxic compounds undergo hydrolysis during Phase I metabolism.

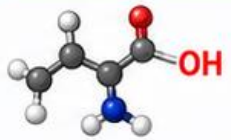


Liver
(hepatic enzymes, e.g., CYP450)

Xenobiotic
(lipophilic)



Phase I metabolite
(more polar)

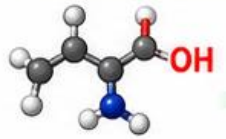


- **Oxidation** (e.g., hydroxylation)
- **Reduction** (e.g., dehalogenation)
- **Hydrolysis** (e.g., ester hydrolysis)

2 Phase II Reactions

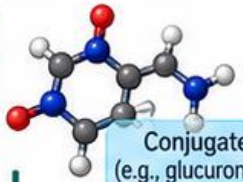
- During Phase II, metabolites produced in Phase I are converted into more water-soluble compounds through conjugation reactions.
- Major conjugation reactions include:
  - Glucuronidation
  - Acetylation
  - Glycine conjugation
- These reactions increase water solubility and facilitate elimination through urine or bile.

Phase I metabolite
(more polar)



Conjugation reactions

Phase II conjugate
(water-soluble)



Conjugate
(e.g., glucuronide)



Urine excretion
(renal elimination)



Bile excretion
(fecal elimination)



Xenobiotic
(lipophilic)



Phase I
(oxidation, reduction, hydrolysis)



Phase I metabolite
(more polar)



Phase II
(conjugation reactions)



Urine / Bile excretion
(water-soluble conjugates)

3 Species Differences in Biotransformation

- Chemically similar compounds may have different metabolic pathways in different animal species.
- This occurs because of:
  - Differences in enzyme activity
  - Genetic variations
  - Species-specific metabolic limitations
- These differences are especially important in veterinary toxicology because susceptibility to toxins varies greatly among animal species.



Cow | Horse | Dog | Cat | Sheep | Poultry



Enzyme activity
(e.g., CYP450, transferases)



Genetic variations
(e.g., polymorphisms)



Species-specific metabolic limitations
(e.g., absent or low enzyme activity)

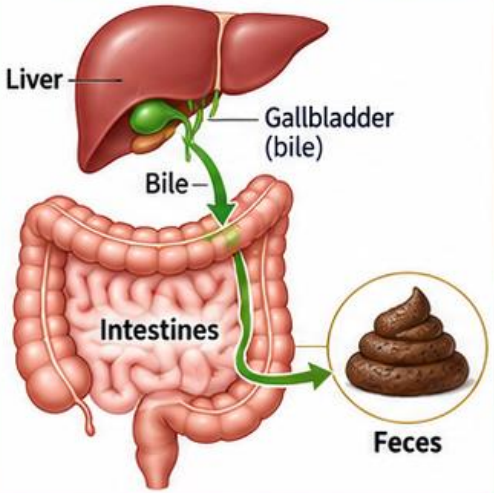
Excretion

Elimination of Toxic Substances from the Body

Toxic substances and their metabolites are eliminated through several routes.

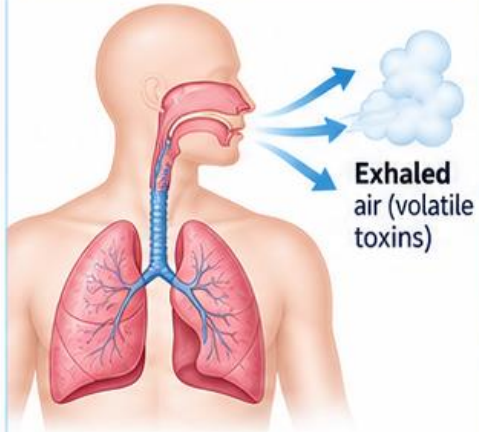
A- Gastrointestinal Tract

- Compounds with high molecular weight and low solubility may be directly eliminated through feces or secreted into bile and then excreted through the intestine.



B- Respiratory System

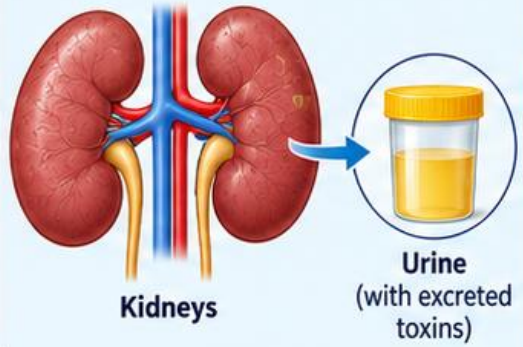
- Volatile toxic substances may be eliminated through exhaled air.
- **Examples:** Cyanide compounds, Phosphorus compounds.



C- Kidneys

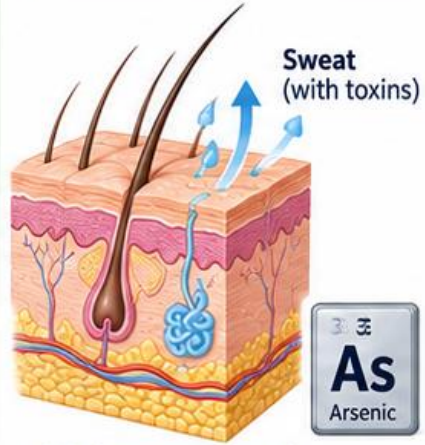
Most important route

- The kidneys are the **most important route** for elimination of many xenobiotics.
- Because of their role in toxin excretion, **renal damage is a common finding** in poisoning caused by nephrotoxic substances.



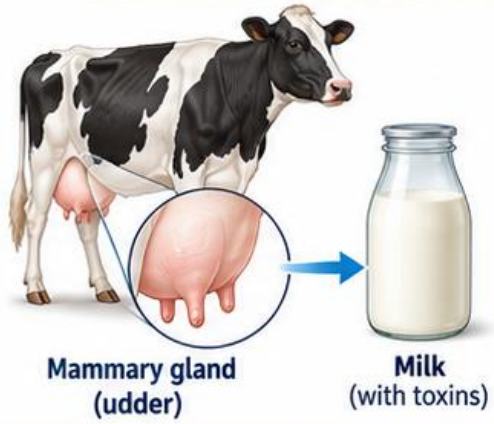
D- Skin

- The skin has a **minor role** in toxin elimination.
- **Recognized example:** Arsenic.



E- Milk and Mammary Gland

- In lactating animals, especially dairy cattle, secretion of toxic substances into milk is important because it may affect:
  - Food safety
  - Public health
  - Consumer exposure



Routes of Elimination (Summary)



Mechanisms of Action of Poisons

Mechanisms of Toxic Action (1/2)

Toxic substances produce their effects through different mechanisms, including:

1. **Direct Tissue Destruction**
2. **Interference with Cell Membrane Permeability**
3. Inhibition of Essential Enzymes

1 Direct Tissue Destruction

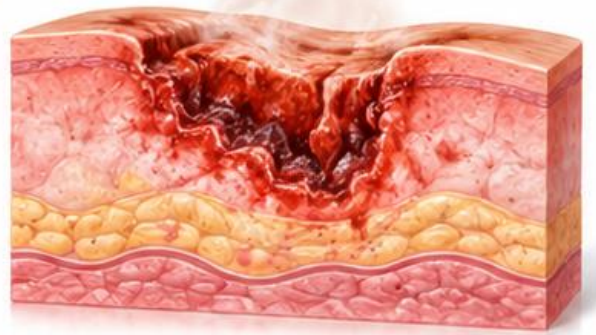
Some toxic agents act through direct corrosive effects and tissue damage.

These substances are called:
Corrosive agents



Examples:

- Strong acids
- Strong alkalis
- Phenolic compounds

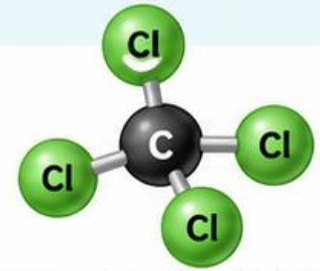


2 Interference with Cell Membrane Permeability

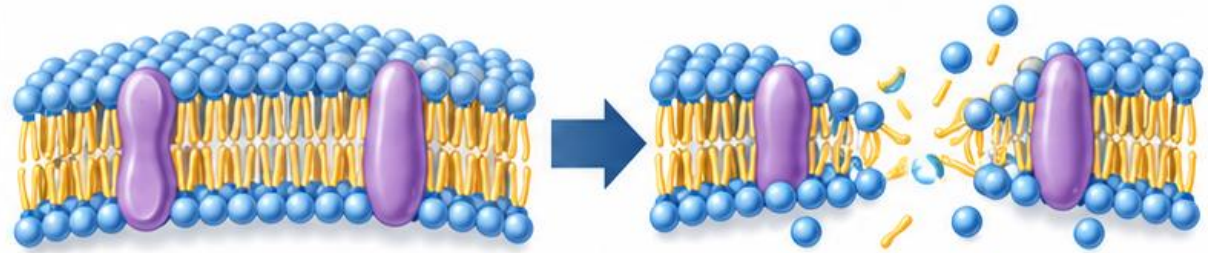
Some toxic substances disrupt the structure or function of biological membranes.

Example:

- Carbon tetrachloride (CCl_4)



Carbon tetrachloride (CCl_4)



Normal cell membrane

Disrupted cell membrane

Mechanisms of Toxic Action (2/2)

3 Inhibition of Essential Enzymes

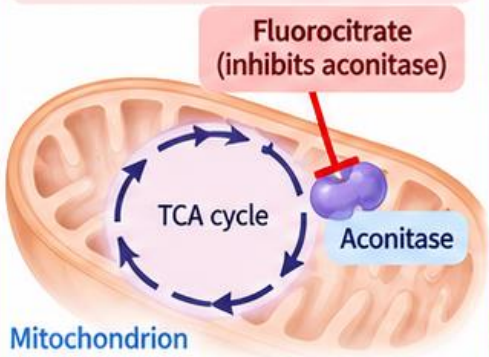
Many poisons act by inhibiting vital enzymes required for normal cellular metabolism.

1 Fluoroacetate (CH_2F-COOH)

Fluoroacetate
↓
Fluoroacetyl-CoA
↓
Fluorocitrate

Fluorocitrate strongly inhibits aconitase in the tricarboxylic acid (TCA) cycle, blocking cellular energy production.

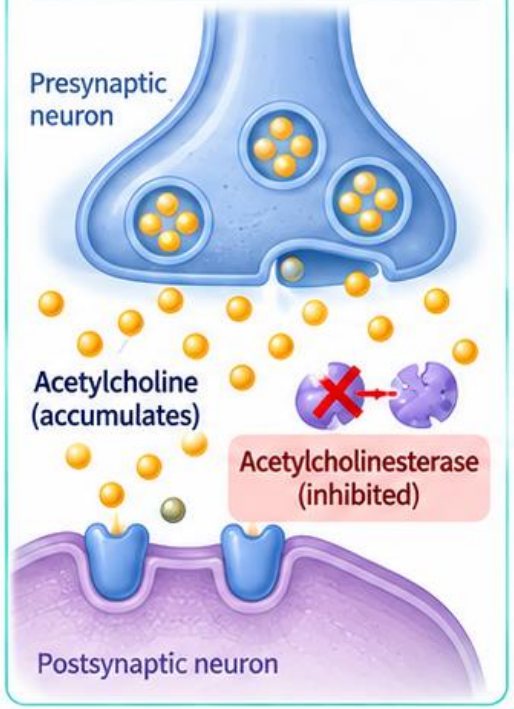
Example of a suicide substrate: the parent compound is less toxic, but metabolism converts it into a highly toxic product.



2 Organophosphate Compounds

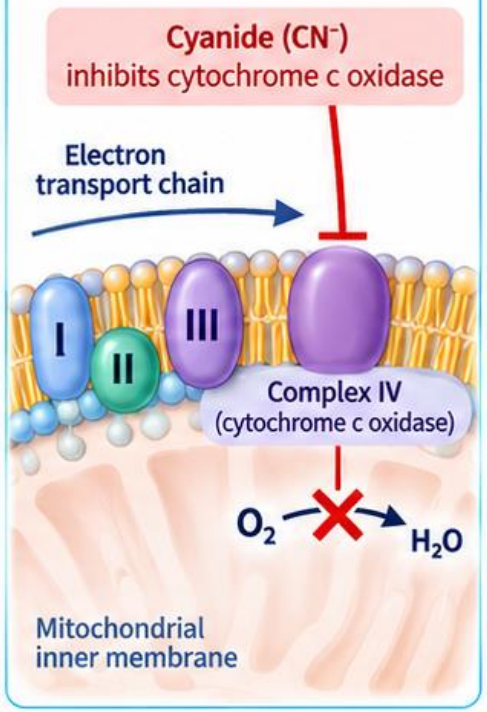
Inhibit acetylcholinesterase.

This causes accumulation of acetylcholine at synapses and excessive cholinergic stimulation.



3 Hydrogen Cyanide (HCN)

Inhibits cytochrome c oxidase in the mitochondrial electron transport chain, preventing cellular oxygen utilization.



4 Arsenic

Binds to sulfhydryl (-SH) groups of proteins and inhibits enzymes, especially the pyruvate dehydrogenase complex and dihydrolipoic acid-dependent enzymes.

This disrupts cellular energy metabolism.

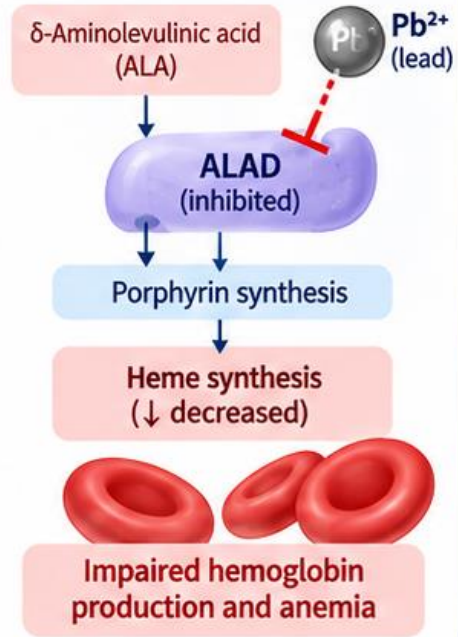


↓
Inhibition of enzymes (e.g., pyruvate dehydrogenase complex)
↓
Disrupted cellular energy metabolism

5 Lead

Inhibits enzymes involved in heme synthesis, including δ-aminolevulinic acid dehydratase (ALAD).

This impairs hemoglobin production and contributes to anemia.



Factors affecting toxicity

Factors Affecting Toxicity

1. Dose of the Poison

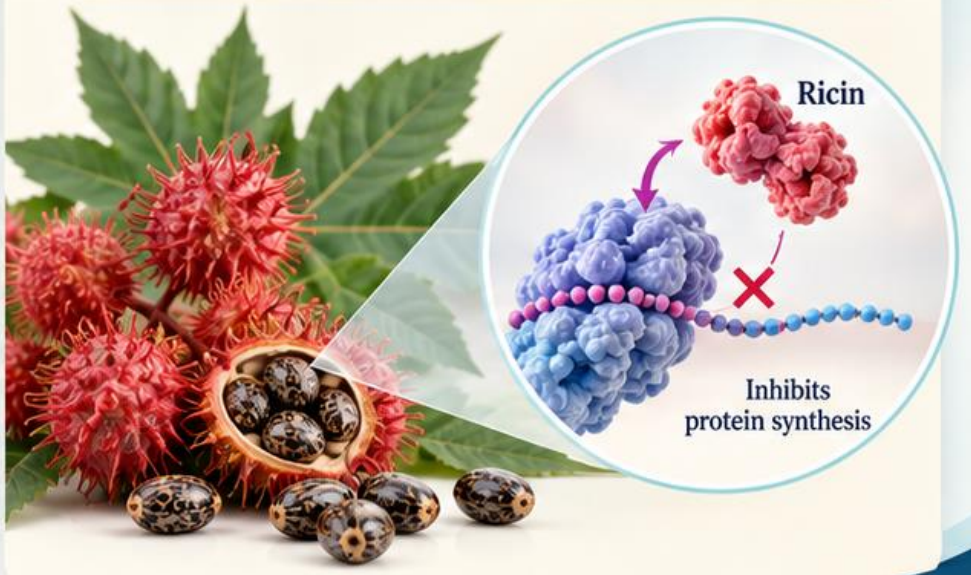
Why LD50 is of limited clinical value:

1. **Sensitivity can vary even among individuals of the same animal species.**
   Example: ricin toxicity in horses.

2. **Animals differ greatly in body size, which affects toxic response.**



Example:
Ricin is a potent plant toxin that inhibits protein synthesis after entering target cells.



Factors Affecting Toxicity

2. Frequency of Exposure

Key points about frequency of exposure:

1. In general, increasing the frequency of exposure increases toxicity and the likelihood of poisoning.
2. This may result from toxin accumulation or cumulative harmful effects.
3. Repeated exposure to doses below the toxic threshold may increase tolerance or resistance.
4. This adaptation is often described as habituation to the toxin.

Whether this effect is due to true immunity or acquired tolerance is still unclear. Rare cases of immunity to ricin and other plant toxins have been reported.



Factors Affecting Toxicity

3. Physical and Chemical Nature of Toxins

Physical Form

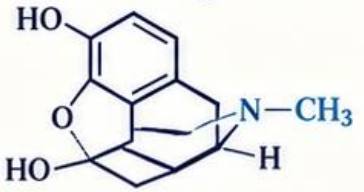
1. In solutions, the type of solvent influences penetration and absorption.
2. Oily solvents generally have greater penetrating power.
3. In solid form, particle size affects absorption and penetration.



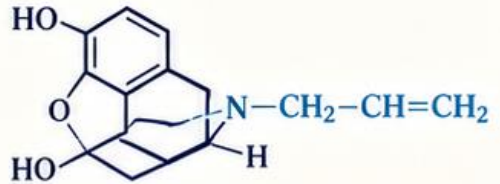


Chemical Nature

1. Small changes in chemical structure can markedly alter toxicity.
2. Example: Morphine vs. Nalorphine — replacing CH_3 with $-CH_2-CH=CH_2$ at the NH attachment changes biological activity.



Morphine
(lower antagonist activity)



Nalorphine
(higher antagonist activity)

3. **Example: Hyoscyamine and atropine.**

Factors Affecting Toxicity

4. Type of Animal

1. Different animal species show different sensitivities to the same chemical.
2. This principle is critically important in the development of pesticides and drugs.
3. Example: Malathion is highly toxic to insects but less toxic to mammals.



Causes of Species Differences

Anatomical differences:

- **Insects:** the chitinous body covering affects interaction with toxins such as DDT.
- **Monogastrics:** a short and efficient gastrointestinal tract may absorb compounds differently from herbivores.
- **Ruminants:** the presence of the rumen can alter toxic response.
- **Example:** bracken fern poisoning differs between monogastrics and ruminants.



Factors Affecting Toxicity

Physiological Differences, Body Size, Age, and General Health

1. Physiological differences also affect toxicity, including feeding behavior and enzymatic differences.
2. Examples among species: morphine in horses versus other animals; chronic copper poisoning in sheep.
3. Breed differences: sensitivity to gossypol differs between Holstein and Jersey calves.
4. Individual variation within the same breed is called idiosyncrasy.
5. Body size and age influence toxic effects.
6. Animals in poor general condition have lower resistance and detoxification capacity.
7. Predisposing conditions include liver disease, kidney disease, parasitic infestation, and malnutrition.



Common Causes of Poisoning

Common Causes of Poisoning

1. Overview: Natural and Human Causes

Accidental (Non-intentional) Poisoning

Common causes of accidental poisoning can be grouped into natural causes and human-related causes.

1 Natural causes

- Toxic minerals
- Poisonous plants
- Venomous reptiles and insects

2 Human-related causes

- Industrial pollution
- Pesticides
- Household toxicants
- Improper use of drugs or unusual sensitivity
- Contaminated food



Common Causes of Poisoning

2. Natural Causes of Poisoning

A. Toxic minerals



1 Direct exposure:

- Fluoride in soil, rocks, and drinking water.
- Nitrate in well water.



2 Indirect exposure:

- Copper accumulation in non-gramineous plants (enzootic jaundice / toxaemic effects).
- Molybdenosis (teart diarrhea).
- Selenium poisoning after consumption of indicator plants.

B. Poisonous plants

1 Poisonous plants are another major natural cause of accidental poisoning.



Common Causes of Poisoning

3. Human-Related Causes: Industry and Pesticides

A. Industrial pollution

1. Because agriculture and industry are often close to each other, industrial toxicants may affect animals.
2. Lead and arsenic are two prominent examples.
3. Factory emissions containing fluoride may also cause poisoning when chimney standards are not properly observed.



B. Pesticides

1. Pesticides include insecticides, herbicides, and rodenticides.



Insecticides

kill insects (e.g., organophosphates, pyrethroids)



Herbicides

kill unwanted plants (e.g., glyphosate, 2,4-D)



Rodenticides

kill rats and mice (e.g., anticoagulants such as warfarin)



Common Causes of Poisoning

4. Human-Related Causes: Household Toxicants and Drugs

C. Household toxicants

1. Lead is a major example in this group.
2. Sources include paints, linoleum, and golf balls.
3. Cattle are especially affected because of their curiosity.
4. Lead poisoning in cattle has been reported after ingestion of tractor paraffin, diesel oil, or machine oils.
5. Disinfectants and antifreeze (ethylene glycol) are also important household toxicants.



D. Drugs

1. Drugs are selective toxicants.
2. Poisoning may occur when they are given in excessive doses, too rapidly, or at short intervals.



Common Causes of Poisoning

5. Food, Water, and Summary

E. Food and water

1. Foods that are harmless under normal conditions may become dangerous if poorly harvested or stored.
2. Examples: solanine poisoning in green potatoes; dicoumarol poisoning from moldy sweet clover; leukoencephalomalacia in horses associated with moldy hay and feed.
3. Hyperkeratosis X in cattle may occur when feed is contaminated with motor-machine lubricants containing chlorinated naphthalene.
4. Water contamination is not a common source of poisoning, but lead, nitrate, and nitrite contamination may occasionally occur.

Summary

In most animal poisonings of human origin, the main causes are negligence, forgetfulness, or lack of awareness.



Green potatoes (solanine poisoning)



Moldy sweet clover (dicoumarol poisoning)



Moldy hay and feed (leukoencephalomalacia in horses)



Feed contaminated with motor-machine lubricants (hyperkeratosis X in cattle)



Water contamination (lead, nitrate, and nitrite)

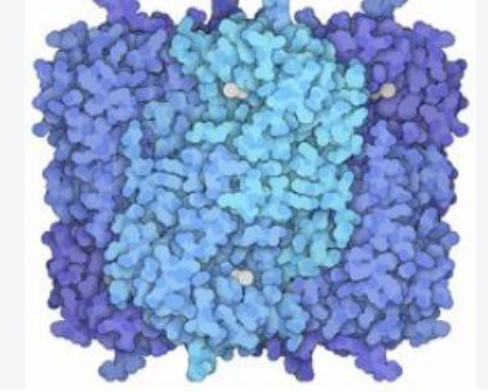


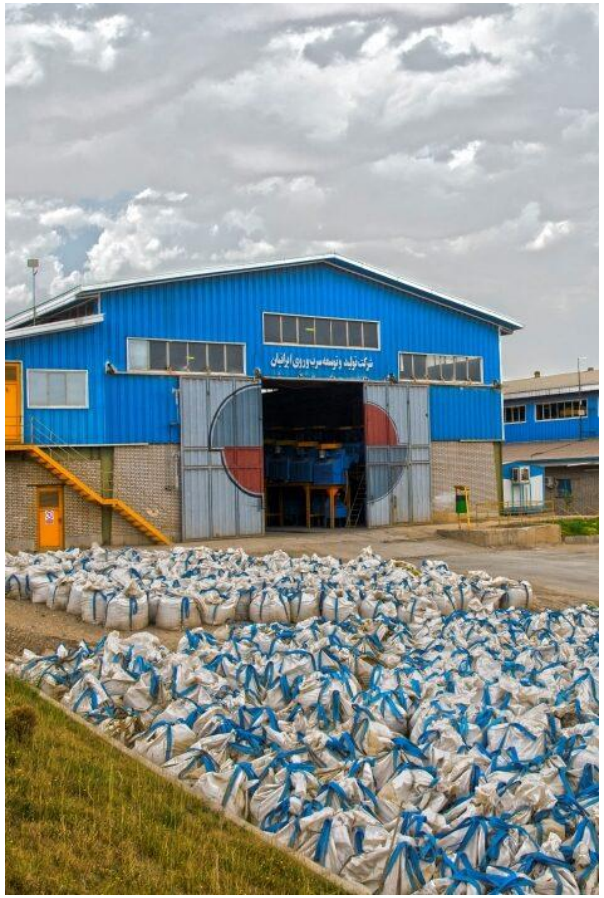

GREENSTAR
Copper Sulphate
1Kg






Sweetlix
MINERAL
SUPPLEMENT






بساعات باتری
GERMAK BATTERY




FlashLube
HIGH TEMPERATURE
GREASE




Fiberlock
LBC
LEAD BARRIER
COMPOUND


BATTERY








Vitamin E
+ Selenium
50 mL
For Veterinary Use Only
Vitamin E + Selenium

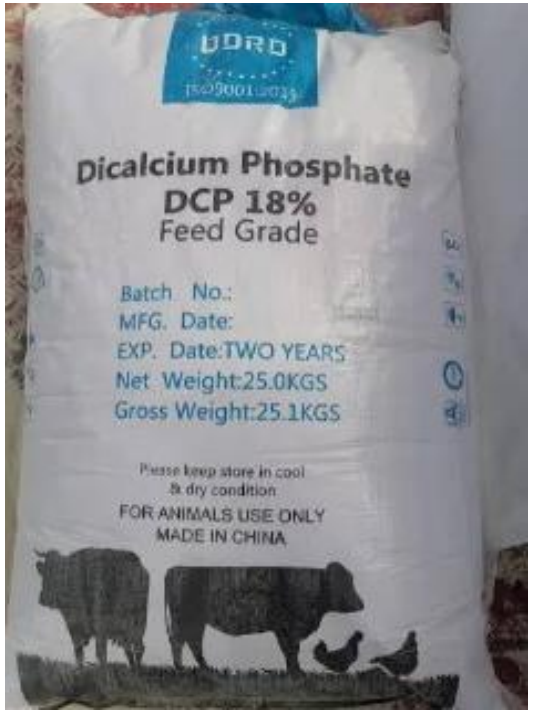
Flurine

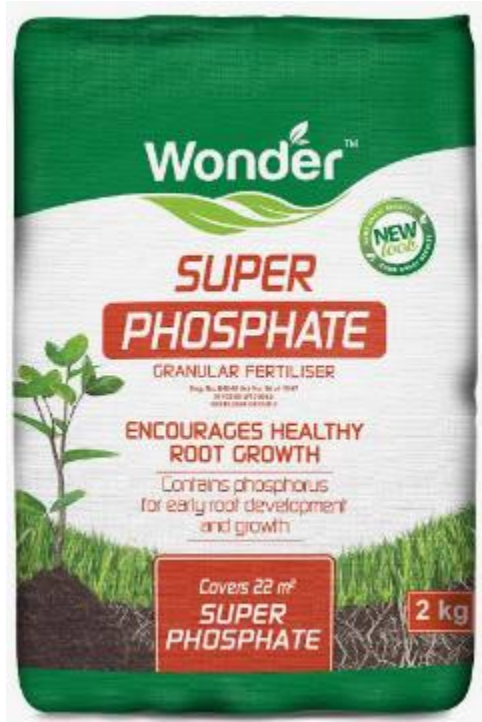



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IRANIAN ALUMINUM COMPANY

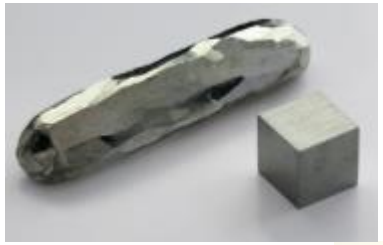





UDRD
Dicalcium Phosphate
DCP 18%
Feed Grade
Batch No.:
MFG. Date:
EXP. Date:TWO YEARS
Net Weight:25.0KGS
Gross Weight:25.1KGS
Please keep store in cool & dry condition
FOR ANIMALS USE ONLY
MADE IN CHINA


Wonder
SUPER
PHOSPHATE
GRANULAR FERTILISER
ENCOURAGES HEALTHY ROOT GROWTH
Contains phosphorus for early root development and growth
Covers 22 m²
SUPER PHOSPHATE
2 kg





Zn & Cd





Sulfur








VOERMOL
MELASSEMEEL
V1995
N-FF 0079
MASSA 40 kg MASS
VOERMOL FEEDS (PTY) LTD

چرخ ریسک یا پیرگیاه (Senecio spp): آلکالوئید پیرولیزیدین





آفتاب پرست یا گل عقرب (Heliotropium spp): آلکالوئید پیرولیزیدین







سگ زبان (Cynoglossum officinale): آلکالوئیدهای پرولیزیدین





خر زهره (Nerium oleander): گلیکوزیدهای قلبی



استبرق (Calotropis procera): گلیکوزیدهای قلبی







گل انگشتانه (Digitalis spp): گلیکوزیدهای قلبی







گون (Astragalus spp): تراتوژن





فرفیون / شیرسگ (Euphorbia spp.): تراتوژن









پیازهای وحشی همچون کنگر و لاله سرنگون: تراتوژن







تاتوره (Datura stramonium): متسع کننده مردمک و نوروتوکسیک





بنگ‌دانه / بذرالبنج (Hyoscyamus niger): متسع کننده مردمک و نوروتوکسیک





تاج‌ریزی سیاه (Solanum nigrum): متسع کننده مردمک و نوروتوکسیک







گل چای یا هوفاریقون (Hypericum perforatum): حساسیت به نور





سورگوم یا سودان گراس: اسید هیدروسیانیک





دانه بادام تلخ، دانه زرد آلو تلخ، دانه گیلاس وحشی: اسید هیدروسیانیک







علف شور (Atriplex): اگزالات بالا





اسفناج وحشی (Chenopodium): اگزالات بالا





ترشک (Rumex spp): اگزالات بالا









شوکران کبیر (Conium maculatum): نوروتوکسین سریع کشنده







افعی گرزه: مایوتوکسین + DIC + همولیز
{خطرناکترین مار ایران با بیشترین گزارش گزش}



افعی جعفری: مایوتوکسین + DIC + همولیز



افعی شاخ دار: مایوتوکسین + DIC + همولیز



افعی دم عنکبوتی: مایوتوکسین + DIC + همولیز



افعی دماوندی: مایوتوکسین + DIC + همولیز



افعی زنجانی / البرزی: مایوتوکسین + DIC + همولیز



افعی سوسن / پلنگی: مایوتوکسین + DIC + همولیز





کفچه‌مار / کبرا خراسانی: نوروتوکسین



کبرا سیاه / مار سیاه بیابانی: نوروتوکسین



مار دریایی خلیج فارس: مایوتوکسین + نوروتوکسین



تیرمار: تورم موضعی



یله مار: تورم موضعی



افعی پلنگی: تورم موضعی



سوسک دراکولا: در گیلان





عقرب گادیم: خوزستان



عقرب توسن یا دم تخت



عقرب زرد خوزستان



عنکبوت گوشه نشین



سوسک طاول زا: قزوین



بیوه سیاه: خراسان



زنبور سرخ



زنبور زرد



کرم ابریشم و بیدهای مودار





Diagnosis of Poisoning

1. Overview and Diagnostic Principles

Diagnosis of poisoning is based on four main principles:

1. Clinical signs
2. Evaluation of the animal's environment
3. Pathological findings
4. Laboratory findings

Experience as a fifth principle

A fifth principle, experience, is often omitted for two reasons:

- Clinical signs in large animals do not always match those seen in laboratory animals.
- Gaining experience in large animals is costly.

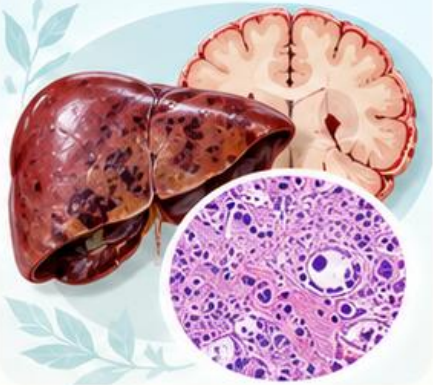
1. Clinical signs



2. Evaluation of the animal's environment



3. Pathological findings



4. Laboratory findings



5. Experience (often omitted)



Diagnosis of Poisoning

2. Clinical Signs

Clinical diagnosis is based on the veterinarian's observations in the live poisoned animal.

If the animal has died, diagnosis relies partly on the history provided by the owner.

Limitations of clinical signs

1. Different individuals of the same species may show different clinical signs.
2. Unusual or nonspecific signs may also occur.




Salivation


Lethargy


Neurological signs


Respiratory signs


Gastrointestinal signs



HISTORY FROM OWNER

- Exposure opportunity
- Access to toxic plants or chemicals
- Recent feed changes
- Timing of onset
- Other animals affected



Diagnosis of Poisoning

3. Evaluation of the Animal's Environment

Investigation of the animal's living environment is especially important in the diagnosis of poisoning.

Information is obtained by questioning the owner and, if possible, by inspecting the farm buildings and surrounding area.

Major categories of toxicants

1. Household
2. Food-related
3. Drug-related
4. Environmental

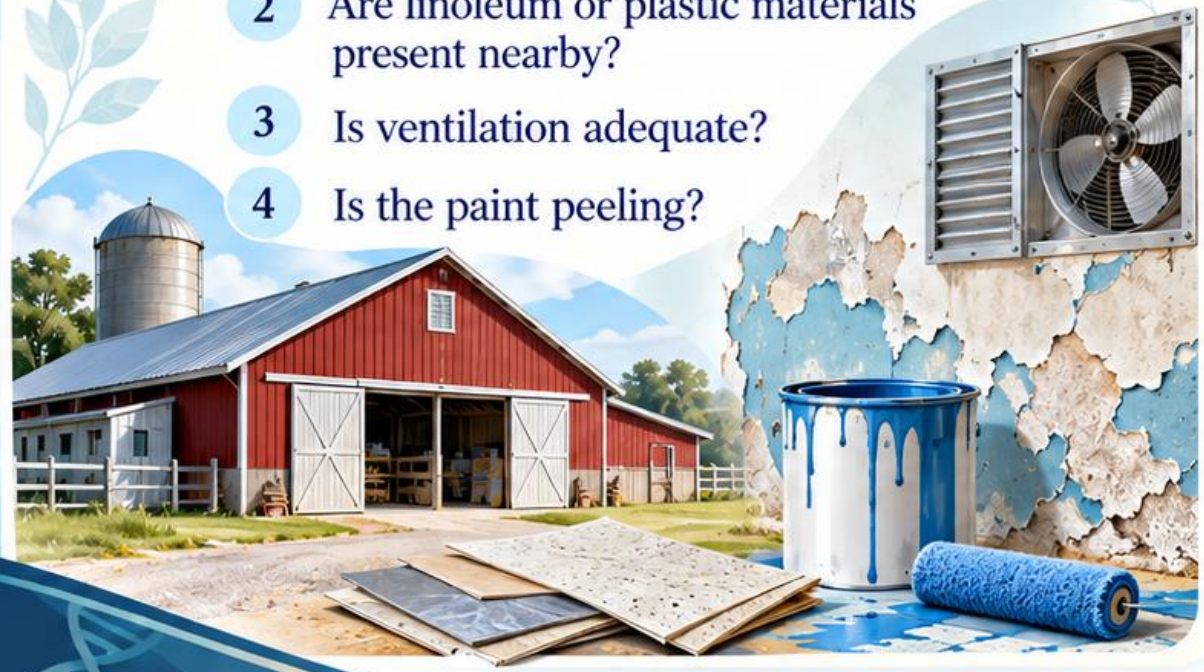


Diagnosis of Poisoning

4. Questions About Buildings and Feed

Building-related questions

1. Has the building been painted recently?
2. Are linoleum or plastic materials present nearby?
3. Is ventilation adequate?
4. Is the paint peeling?



Feed-related questions

1. Has the diet changed?
2. Has a new feed shipment arrived?
3. Is there anything unusual in the feed?
4. Ask about additives and drugs mixed into the feed.



Diagnosis of Poisoning

5. Water, Supplements, Pasture, and Air

1 Water

- Has drinking water been tested for minerals and other compounds?
- Water may contain lead, nitrate, or excessive salt.

2 Supplements

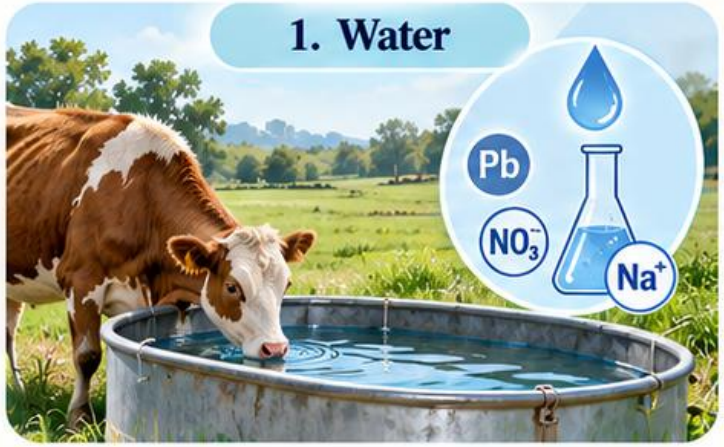
- Ask about the type and amount of supplements used.

3 Pasture

- Has the pasture been treated recently with pesticides?
- Have chemical fertilizers been used?

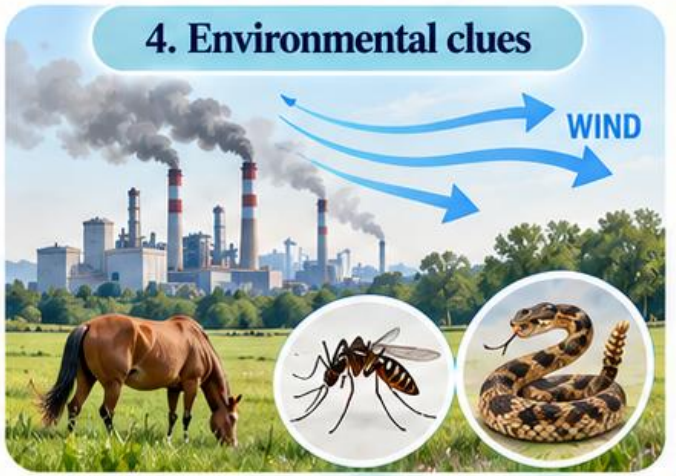
4 Environmental clues

- Consider air pollution, biting insects, and venomous reptiles.
- Wind carrying pollutants from factories may be relevant.









Diagnosis of Poisoning

6. Pathological Evidence: External Clues

Necropsy lesions are often less diagnostically specific than other findings.

Unpigmented skin and visible mucous membranes may change color.



Conjunctival mucous membrane



Oral mucous membrane



Third eyelid mucous membrane

Color clues



Yellow: suggests liver injury or hemolytic crisis, as in copper poisoning.





Brown: may be seen in nitrite or chlorate poisoning due to methemoglobin formation.





Cherry red: may be seen in cyanide poisoning.

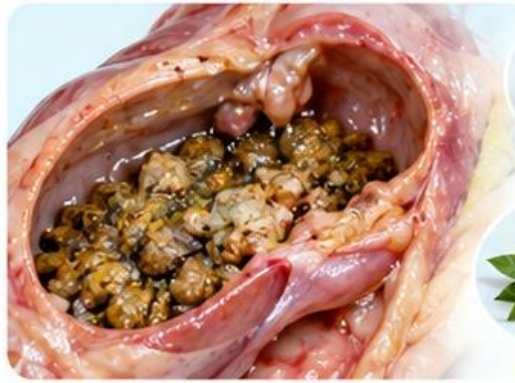


Diagnosis of Poisoning

7. Pathological Evidence: Stomach, Kidneys, and Muscles

1 Stomach contents

- Characteristic odors may suggest cyanide, phosphorus, or phenol poisoning.
- Visible evidence may include lead fragments, paint chips, poisonous plant leaves, or dead rodent remains.





Lead fragments



Paint chips



Poisonous plant leaves



Dead rodent remains

Color of stomach contents



Blue
copper salts



Orange-yellow
chromium salts



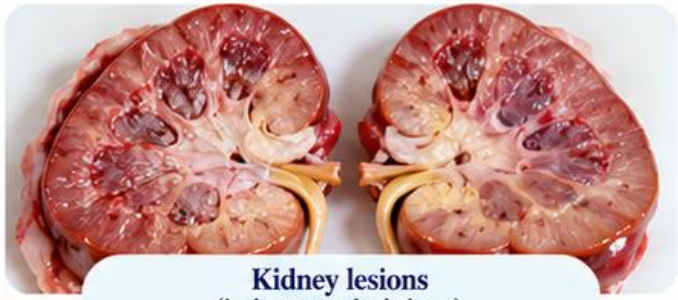
Yellow
nitric acid or picric acid



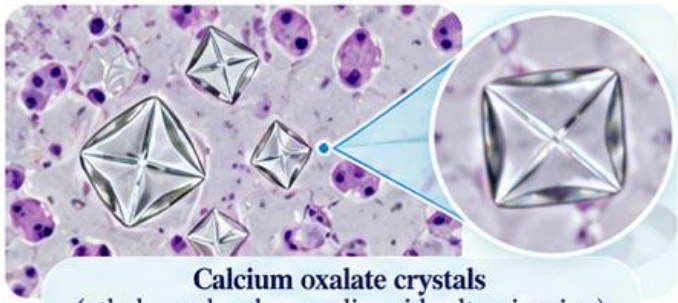
Black
sulfuric acid

2 Kidneys

- Kidney lesions may occur when irritant toxins are excreted through the kidneys.
- Oxalate crystals may be seen in ethylene glycol or oxalic acid salt poisoning.



Kidney lesions
(irritant toxin injury)



Calcium oxalate crystals
(ethylene glycol or oxalic acid salt poisoning)

3 Muscles

- Yellow discoloration may occur in lead poisoning.
- Hemorrhage may occur in bracken fern or sweet clover poisoning.



Yellow discoloration in muscle
(lead poisoning)



Hemorrhage in muscle
(bracken fern or sweet clover poisoning)

Diagnosis of Poisoning

8. Laboratory Findings and Sample Handling

1 Final confirmation

The final confirmation of poisoning is obtained by identifying the toxicant in the body through laboratory testing.

2 Samples commonly collected

- Liver, kidney, and body fat
- Stomach and intestinal contents
- Blood and urine
- Any other necessary tissue or fluid

3 Suggested amounts and handling

- About 250 g from each organ (or 100 g with newer methods)
- Blood: 10 mL
- Urine: all that is available in the bladder
- Stomach or rumen contents: 500 mL
- Glass containers are preferred
- Plastic containers are not recommended for many organic toxicants
- Keep samples chilled, not frozen
- Avoid preservatives whenever possible
- If refrigeration is impossible over long distances, alcohol may be used; send a separate bottle of the same alcohol with the sample.

Common Samples Collected



Liver, kidney, and body fat | Stomach and intestinal contents | Blood and urine | Any other necessary tissue or fluid

Sample Handling and Storage



Use appropriate containers (Glass preferred) | Keep samples chilled, not frozen | Use alcohol for long-distance transport if needed (send a separate bottle of the same alcohol) | Laboratory analysis to identify the toxicant

Treatment of Poisoning

1. Overview of Treatment Principles

Treatment of poisoning is based on three main principles:

1. **Prevent further absorption of the poison.**
2. **Symptomatic and supportive therapy.**
   - a Reduce the clinical effects of absorbed toxins.
   - b Maintain the animal's vital functions.
3. **Administer antidotes to neutralize the toxicant and promote its elimination from the body.**







Treatment of Poisoning

2. Prevent Further Absorption: Skin and Stomach

1. **If the poison is on the skin:** wash thoroughly with water.
2. **For poison still waiting to be absorbed from the stomach of non-herbivorous animals:** use emetics.
3. **Apomorphine:** 0.1 mg/kg.
4. **Oral emetics may include table salt** (1 teaspoon placed at the back of the tongue), mustard, and 1% copper sulfate.

1. Dermal Decontamination



2. Gastric Decontamination in Non-Herbivorous Animals



3. Apomorphine (Emetic)



4. Other Oral Emetics



Table salt (1 teaspoon placed at the back of the tongue)

Mustard (powder)

1% Copper sulfate solution

IV (0.1 mg/kg) or SC (0.2mg/kg) or Subconjuctival tab (0.25 mg/kg) or eye drop (1/5kg)









In cats (weak apomorphine respons)





Salt tablet on tongue [expired protocol]





Mustard in warm water (weak & expired)





CuSo4 %1 [Old & expired & Dangerous]





Treatment of Poisoning

3. Contraindications to Emetics

Emetics should NOT be used in the following situations:

1. Poisoning with volatile petroleum products such as gasoline or diesel
2. Corrosive substances such as acids and alkalis
3. Unconscious animals
4. Animals with collapse/fainting or seizures

1. Volatile petroleum products (such as gasoline or diesel)



2. Corrosive substances (such as acids and alkalis)



3. Unconscious animals



4. Animals with collapse/fainting or seizures



Treatment of Poisoning

4. Gastric Lavage, Adsorbents, and Intestinal Removal

1. **Gastric lavage is used in monogastric herbivores and in unconscious non-herbivorous animals.**

2. **Final lavage should be followed by an adsorbent suspension containing:** activated charcoal 10 g, magnesium oxide 5 g, kaolin 5 g, and tannic acid 5 g.

3. **In ruminants, the best method is rumen evacuation by rumenotomy (laparo-rumenotomy).**

4. **If toxins have entered the intestines, use saline cathartics.**

5. **Magnesium sulfate or sodium sulfate:** 0.6–1 g/kg body weight.

1. Gastric Lavage



2. Adsorbent Suspension (after lavage)



3. Rumen Evacuation (Rumenotomy)



4. Saline Cathartics (Intestinal Removal)



Treatment of Poisoning

5. Symptomatic and Supportive Therapy

Supportive therapy is used to maintain the animal's vital functions until the toxin is neutralized, eliminated, or a specific antidote is given.

Each observed clinical sign should be treated.

Seizures:

- Sedatives or anesthetics such as pentobarbital 10–30 mg/kg IV or intraperitoneal.

Coma:

- CNS stimulants such as amphetamine or methamphetamine 0.25 mg/kg subcutaneously.



Supportive Care and Monitoring

- Maintain airway, breathing, and circulation
- Provide fluid therapy and correct electrolytes
- Maintain body temperature
- Monitor vital signs and organ function

Seizures



Sedatives or anesthetics such as pentobarbital 10–30 mg/kg IV or intraperitoneal.

Coma (CNS Depression)



CNS stimulants such as amphetamine or methamphetamine 0.25 mg/kg subcutaneously.

Benz: Diaz (1/20 kg) Mida (1/50 kg)





1/Human


Methocarbamol
Injection, USP
1,000 mg per 10 mL
(100 mg per mL)


ریلاکسین®
تزریقی

Lower cardiac & respiratory suppress
60 mg/kg …. Slow IV
1/10 kg



1/10 kg (16-20 mg/kg) …. IM & Slow IV in 4 meal…. Long act







Only in horse (1-5 cc/100 kg 2%)




DANGER


Ketamine
Hydrochloride
Injection, USP
200 mg per 20 mL
(10 mg per mL)


50 mL
VETone
Acepromazine
(Acepromazine Maleate Injection, USP)
Injection
500 mg/50 mL (10 mg/mL)
CAUTION: Federal law restricts this drug to use by or on the order of a licensed veterinarian.
NDC 13985-587-50
V1 501075
Net Contents: 50 mL

Treatment of Poisoning

6. Supportive Measures for Common Problems

Common supportive measures for poisoning cases:

1. **Vomiting and diarrhea:** saline solutions, Ringer, or lactated Ringer.
2. **Rumen acidosis:** sodium bicarbonate, magnesium hydroxide, or magnesium oxide.
3. **Rumen alkalosis:** acetic acid or vinegar.
4. **Hemorrhage:** administer vitamin K.

1 Vomiting and diarrhea

Saline solutions, Ringer, or lactated Ringer.



2 Rumen acidosis

Sodium bicarbonate, magnesium hydroxide, or magnesium oxide.



3 Rumen alkalosis

Acetic acid or vinegar.



4 Hemorrhage

Administer vitamin K.



Treatment of Poisoning

7. Antidotes: Purpose and Classification

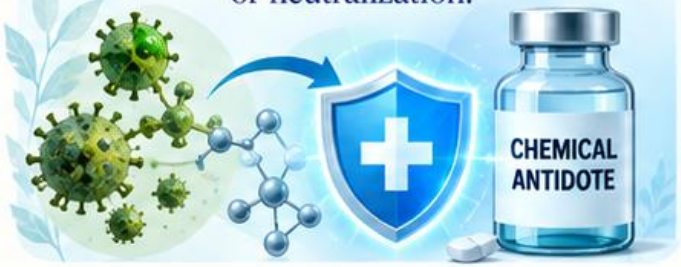
Antidotes provide the specific treatment of poisoning by neutralizing absorbed toxins and helping eliminate them from the body.

Classification of antidotes:

1. Chemical
2. Pharmacological
3. Physiological
4. By mechanism of action

1 Chemical Antidotes

Directly react with and inactivate the toxin through chemical binding or neutralization.



2 Pharmacological Antidotes

Antagonize the toxin's effects at the receptor or functional site.



3 Physiological Antidotes

Counteract the toxin's physiological effects by restoring normal body functions or providing essential components.



4 By Mechanism of Action

Classified according to how they prevent, neutralize, or enhance elimination of toxins (e.g., chelating agents, enzyme reactivators, antimetabolites, etc.).



Treatment of Poisoning

8. Antidote Mechanisms and Examples

Examples based on mechanism of action:

A Chelation:

Calcium disodium EDTA (Na_2Ca EDTA) binds lead and makes it excretable by the kidneys.

B Binding / reactivation:

Pralidoxime in organophosphate poisoning binds phosphorylated cholinesterase and helps release/reactivate the enzyme.

C Receptor-site occupation / competitive use:

Ethanol in ethylene glycol poisoning; vitamin K in coumarin poisoning.

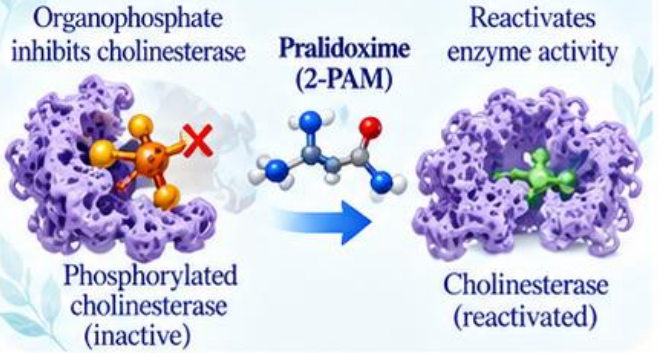
D Altering toxin metabolism:

Sodium thiosulfate in cyanide poisoning.

A. Chelation (Lead Poisoning)



B. Binding / Reactivation (Organophosphate Poisoning)



C. Receptor-site Occupation / Competitive Use

Ethanol
(Ethylene Glycol Poisoning)



Ethanol competitively inhibits alcohol dehydrogenase, preventing metabolism of ethylene glycol to toxic metabolites.

Vitamin K
(Coumarin Poisoning)



Vitamin K replenishes reduced vitamin K, allowing synthesis of functional clotting factors.

D. Altering Toxin Metabolism (Cyanide Poisoning)



Sodium thiosulfate donates sulfur to cyanide, forming the much less toxic thiocyanate, which is excreted in urine.