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THEME:

TOXICOLOGICAL RISK ASSESSMENT IN HEALTHCARE DISEASE DIAGNOSIS

TOXICOLOGY

- The science “that studies the **harmful effects of exposures to chemicals** including drugs, environmental contaminants and naturally occurring substances found in food, water, air and soil”.
- **Toxicant** a toxic substance introduced into the environment.

Xenobiotics

- Foreign chemicals *not* synthesized within the body are referred to as "**XENOBIOTICS"** (Gr. *Xenos* meaning 'stranger')
- Xenobiotics may be naturally occurring chemicals synthesized by plants, microorganisms or animals (including humans)
- Xenobiotics may be synthetic chemicals synthesized by humans
- Poisons are xenobiotics, but not all xenobiotics are poisonous

Pathways by which toxic substances travel

The six main pathways by which toxic substances now travel around the planet are:

rivers, lakes, groundwater, oceans and airborne droplets

pastures, livestock and the food chain

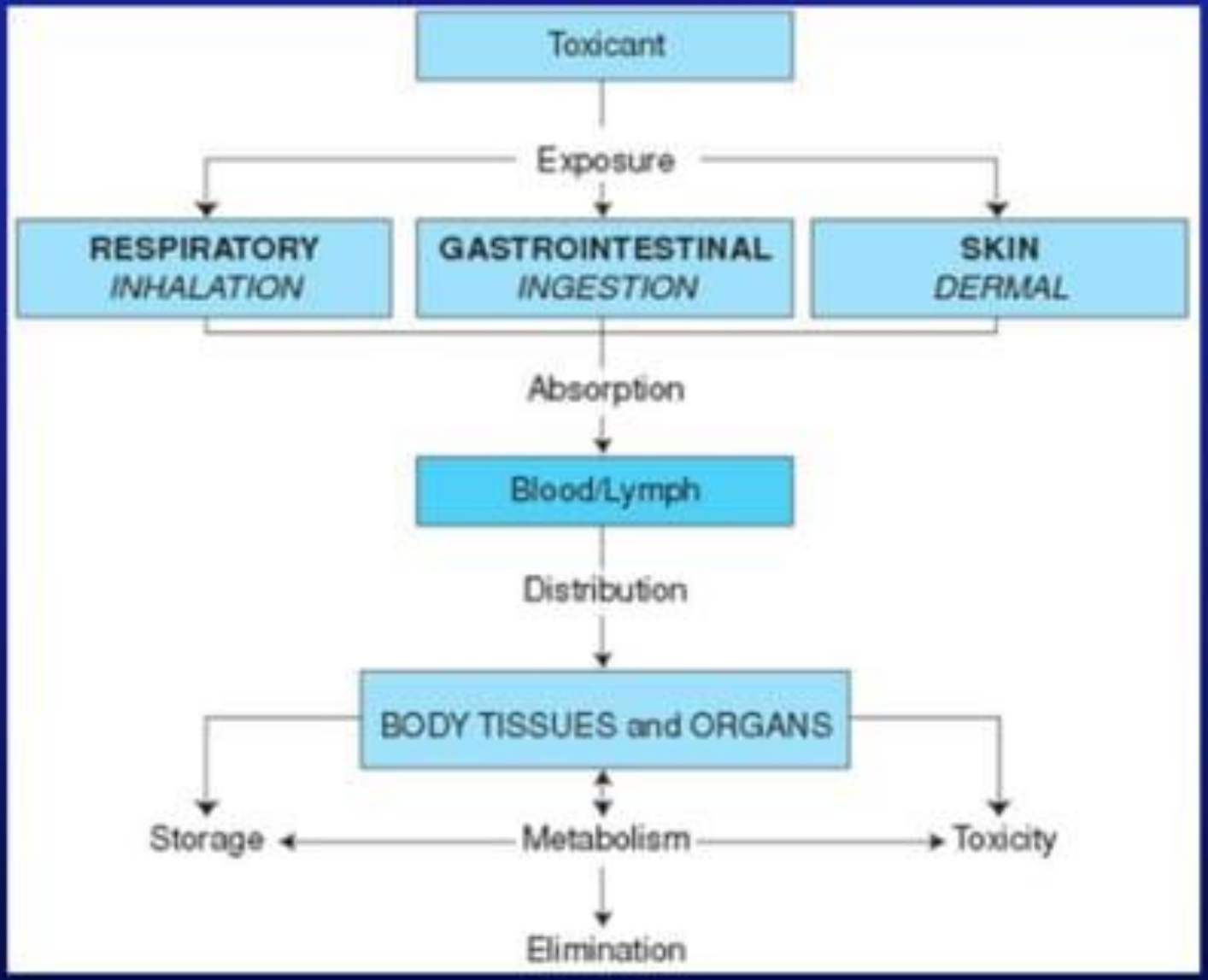
gases, dusts and aerosols

most humans now carry a lifelong burden of contaminants

manufactured goods and food, both legal and illegal

including birds, fish, animals, insects, plants and microbes crops,

Absorption and Fate of a Toxicant



Richards and Bourgeois, Principle and Practice of Toxicology in Public Healt

Toxico-Kinetics **describes the rate of a chemical enter the body and how the compound metabolize and excrete once it is in the body.**

Effects

Mechanism

EXPOSURE

"Absorption"
How the chemical enters the body

'DOSE'
(Plasma)

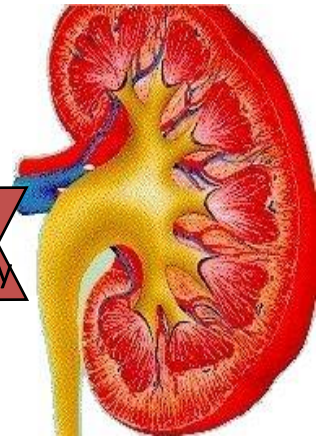
Distribution
Where the chemical goes in the body

TISSUES

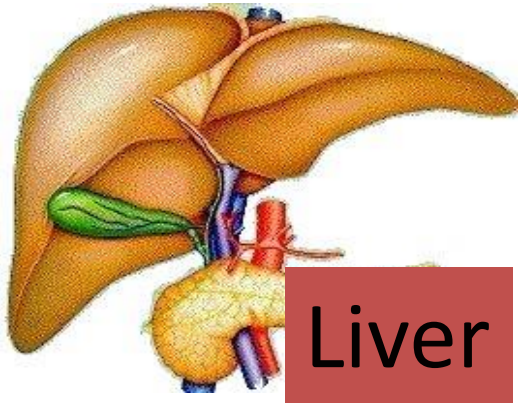
Excretion

Metabolism

Exit from body

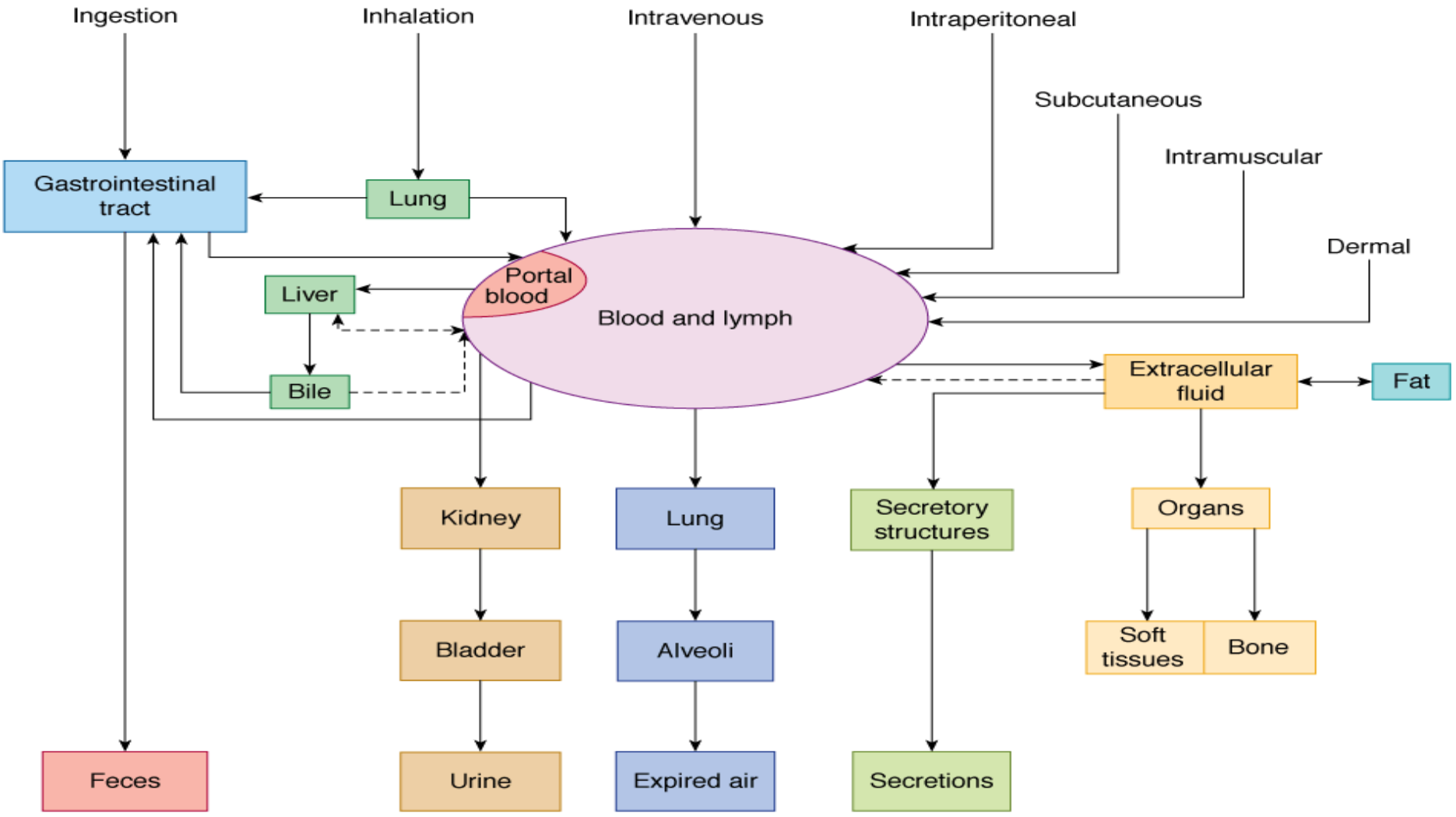


Excretion



Liver

DISTRIBUTION OF TOXICANTS AND STORAGE SITES



Source: Klaassen CD, Watkins JB: *Casarett & Doull's Essentials of Toxicology, 2nd Edition:* http://www.accesspharmacy.com

Storage of Toxicants

Location	Toxicant	Sources
Fat	Lipophilic Compound (eg. Some pesticides, dioxins, PCBs (polychlorinated biphenyls)	**Pesticides**(apples, pears); **dioxins (byproducts of herbicides(use to control unwanted plants))** (meat and dairy products, fish, and shellfish); **PCB** (catfish, buffalo fish, and carp usually have the highest PCB levels, plastics, rubber products, etc)
Bones	Chemical similar to calcium (eg. Fluorine, lead, strontium)	**Fluorine** (black tea, coffee, potatoes) Lead (paint, dust, jewelry, toys; food that contain high level of **Lead**: (chocolate, peas, sweet potatoes; candy, wrappers, pottery containers; 61% of lipstick contain lead) **Strontium** (seafood, milk, wheat, bran, meat, poultry, carrots, peas, beans, etc)
Blood		
Liver and kidney		
Other organs		

Metabolism of Toxicants

- **Metabolites:** substance produced during metabolism, often mediated by enzyme reactions.
- **Bioactivation (activation):** production of metabolites that are more toxic than the parent substance.
- **Detoxication:** production of metabolites that are less toxic than the parent substance.

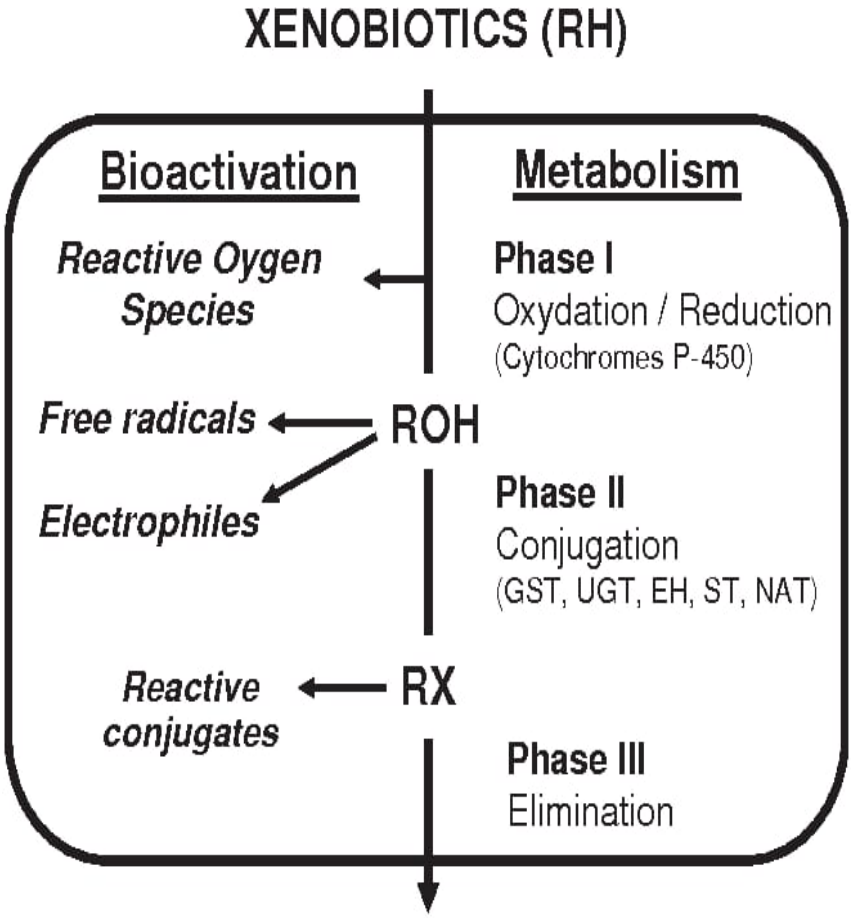
Metabolism of Toxicants

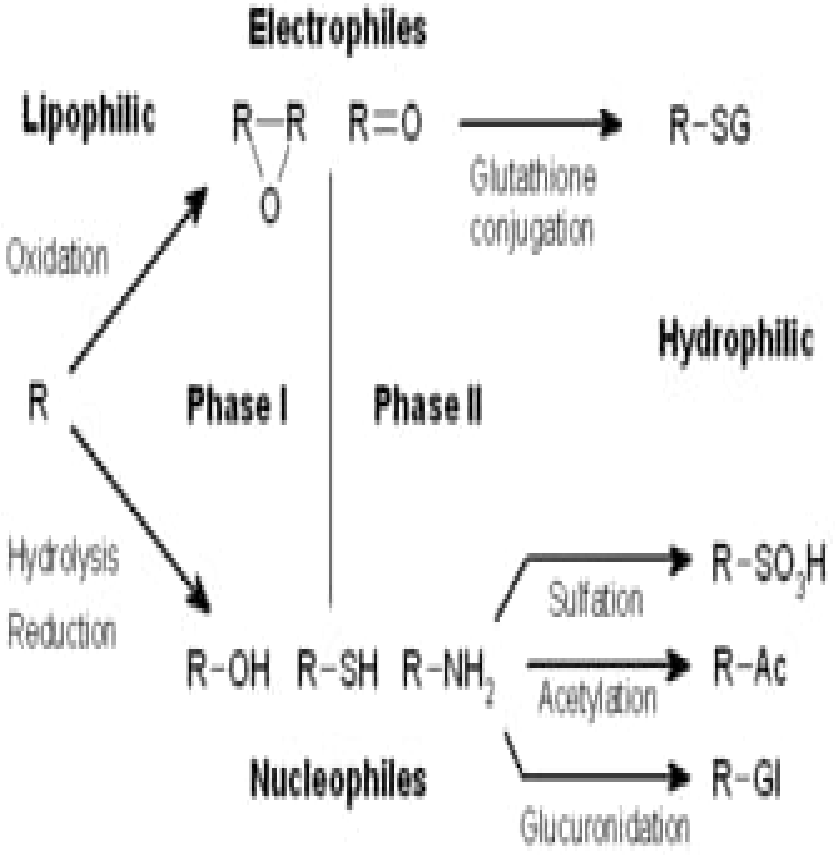
- **Biotransformation** – plays a significant role in the body.
- For example, it helps convert toxic compounds such as xenobiotics into less harmful substances freely excreted from the body.
- It also aids in modifying substrates such as drugs into a form that the body can easily use.

Metabolism of Toxicants

- It occurs mainly in the liver and also in lungs, kidney and intestine.
- It is catalyzed by enzymes including cytochrome P450.
- Increases water solubility for excretion via kidney.
- Sometimes biotransformation increases toxicity, because metabolites or mediators can be toxic.

Metabolism of Toxicants





Routes of Elimination

- Biliary, Renal, fecal
- Excretion: kidney, urine, water soluble compounds,
- Surplus or waste: molecules and ions flow out as urine,
- Reclaims useful materials
- Filters the blood of the small molecules and ions.

Routes of Elimination

- Excretion: Lugs (toxicants: volatile compounds, gaseous metabolites
- Liver: bile; fat soluble compounds
- Other routes: hairs, nails, skin, sweet

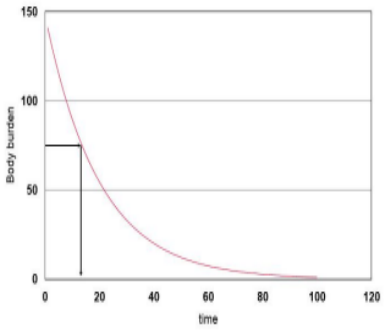
Biological Half Life

- Time taken for half the amount of the substance absorbed to be excreted

Some Half Lives

Toluene	~ 10 hours
Selenium	~ 10 days
Mercury	~ 6 weeks
Cadmium	~ 10 years or more

Biological Half Life



Half Lives

- Mineral dusts
- Metal compounds
- Organic solvents & inorganic gases

Amphibole asbestos is particularly biopersistent
Can remain in lungs for decades

- **Selenium** can cause stomach discomfort, headache, and rash. High does can cause hair loss, fatigue, nausea, vomiting, and weight loss. Extremely doses can lead to organ failure and death.
- **Sources**: seafood, organ meat, and brazil nuts, are the foods highest in selenium.
- **Toluene source**- crude oil (gasoline), paints, glues, nail polish, inks, and stain remover.
- **Health effects-** inflammation of the skin, liver and kidney damage, muscle fatigue, etc.

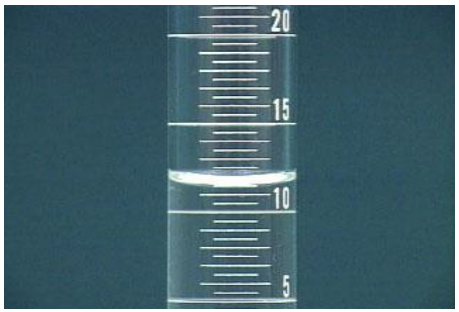
EXPOSURE

- **Exposure** – any condition which provides an opportunity for an external environmental agent to enter the body.
- **Agent** – any chemical, biological, or physical material capable of provoking a biological response different than the vector carrier (air, water, food)

EXPOSURE

In order for a chemical to produce a biological effect, it must first reach its target site within the body (**TOXICOKINETICS**)

Toxicity is a function of the effective DOSE (how much) of a xenobiotic at its target site, integrated over TIME (how long)



X

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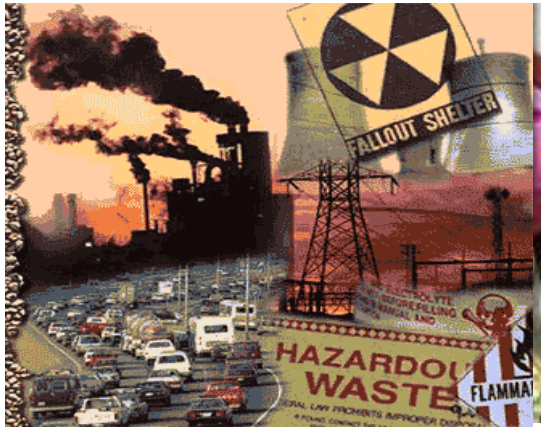
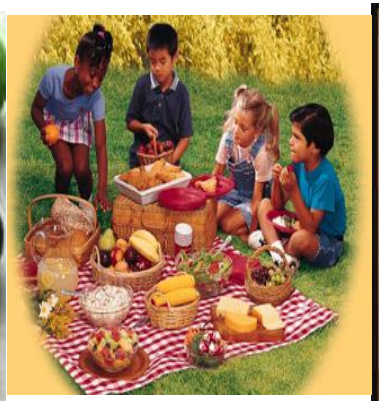
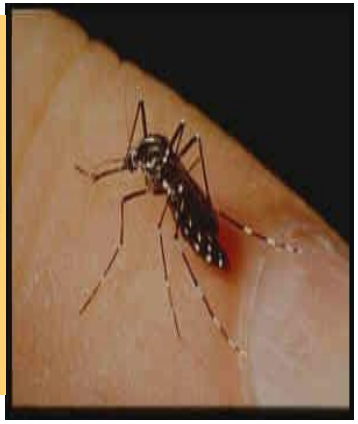
EXPOSURE Concepts: Source

- **Exposure to chemicals may come from many sources:**
  - ✓ Environmental
  - ✓ Occupational
  - ✓ Therapeutic
  - ✓ Dietary
  - ✓ Accidental
  - ✓ Deliberate







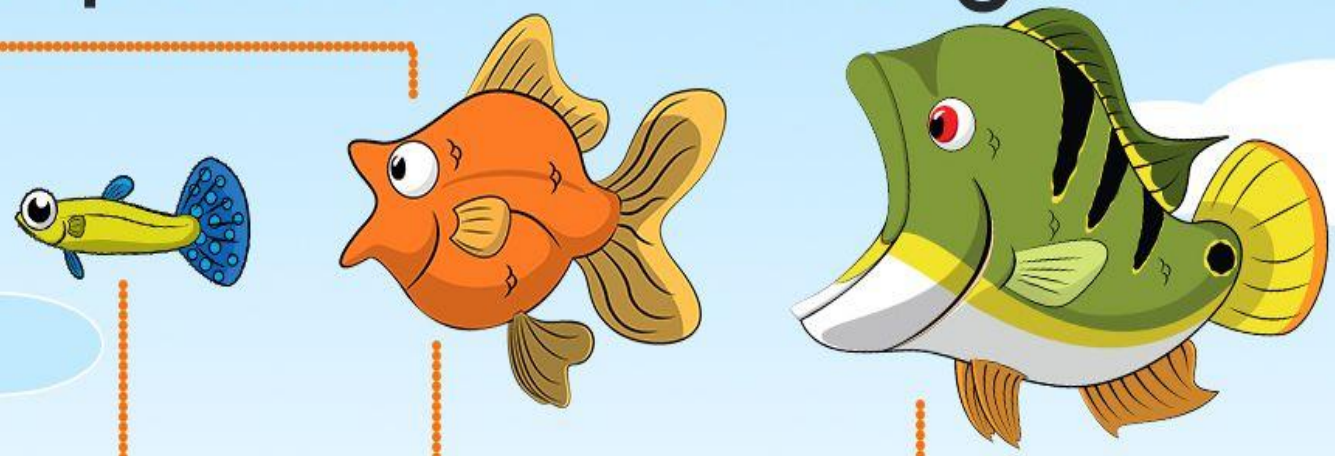
EXPOSURE Concepts –*Time*

- DURATION and FREQUENCY are also important components of exposure and contribute to DOSE. Both may alter toxic effects
- Acute Exposure = less than 24 hr; usually entails a single exposure
- Repeated Exposures (Frequency) are classified as:
  - ✓ Sub-acute = repeated exposures for up to 30 days
  - ✓ Sub-chronic = repeated exposures for 30 – 90 days
  - ✓ Chronic = repeated exposures for more than 90 days

Bioaccumulation

Buzzle.com

Increase in concentration of a pollutant in an organism.

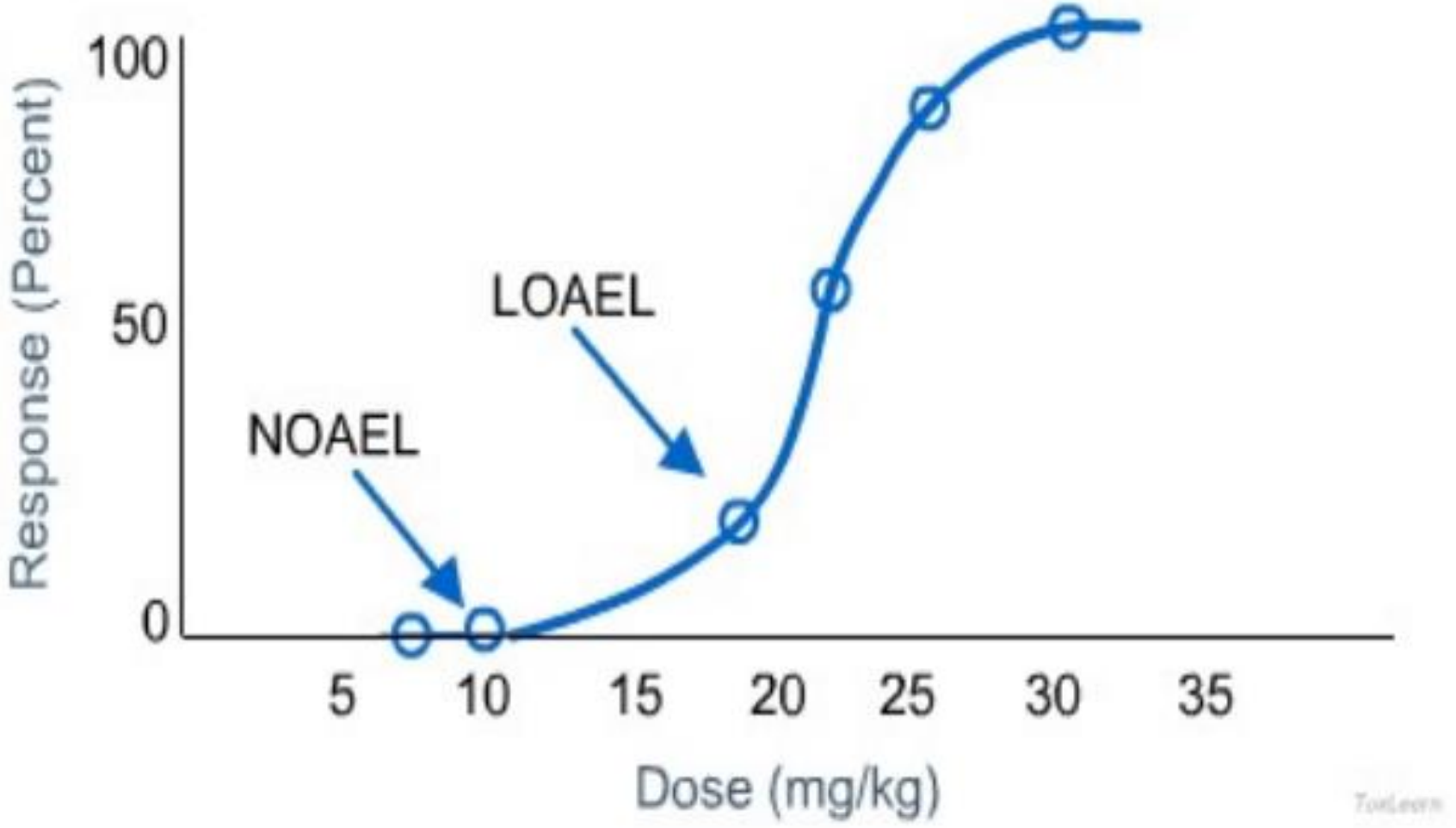


Biomagnification

Increase in concentration of a pollutant in a food chain.

TYPES OF DOSE

Exposure dose	the amount of a xenobiotic encountered in the environment
Absorbed dose	the actual amount of the exposed dose that enters the body
Administered dose	the quantity administered usually orally or by injection
Total dose	the sum of all individual doses

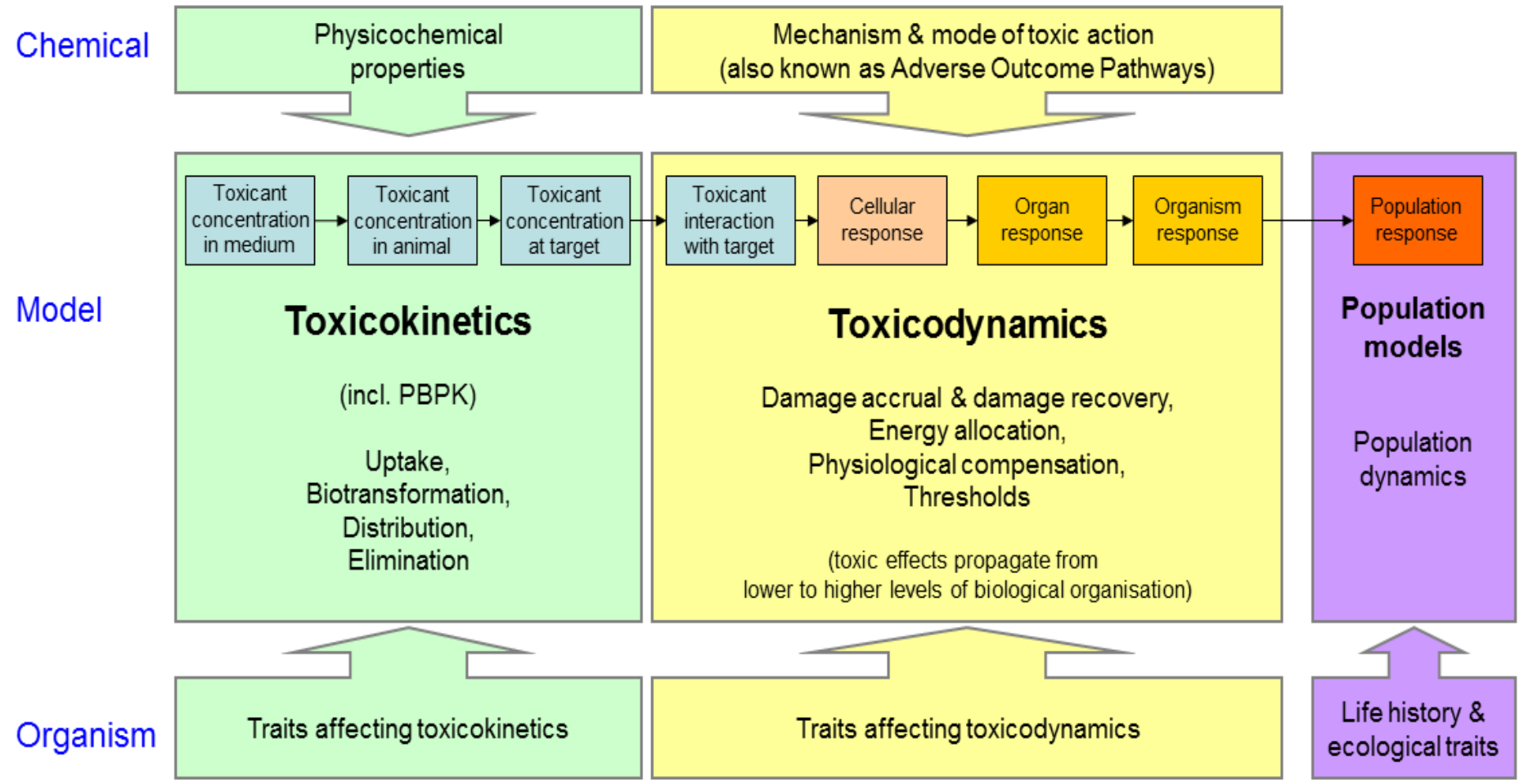


highest dose at which no observable adverse effect is seen (NOAEL)

lowest dose at which an adverse effect is observed (LOAEL).

Substance	Non-Toxic or Beneficial Dose	Toxic Dose	Lethal Dose
Alcohol *ETHANOL BLOOD LEVELS*	**0.05 %**	**0.1 %**	**0.5 %**
Carbon Monoxide *% HEMOGLOBIN BOUND*	**< 10 %**	**20 - 30 %**	**> 60 %**
Secobaraital *(sleep aid)* *BLOOD LEVELS*	**0.1 mg/dL**	**0.7 mg/dL**	**> 1 mg/dL**
Aspirin	**0.65 gm** *(2 tablets)*	**9.75 gm** *(30 tablets)*	**34 gm** *(105 tablets)*
Ibuprofin *E.G., ADVIL & MOTRIN*	**400 mg** *(2 tablets)*	**1,400 mg** *(7 tablets)*	**12,000 mg** *(60 tablets)*

Mechanistic effect models for ecotoxicology



→ Arrows indicate a causal relationship

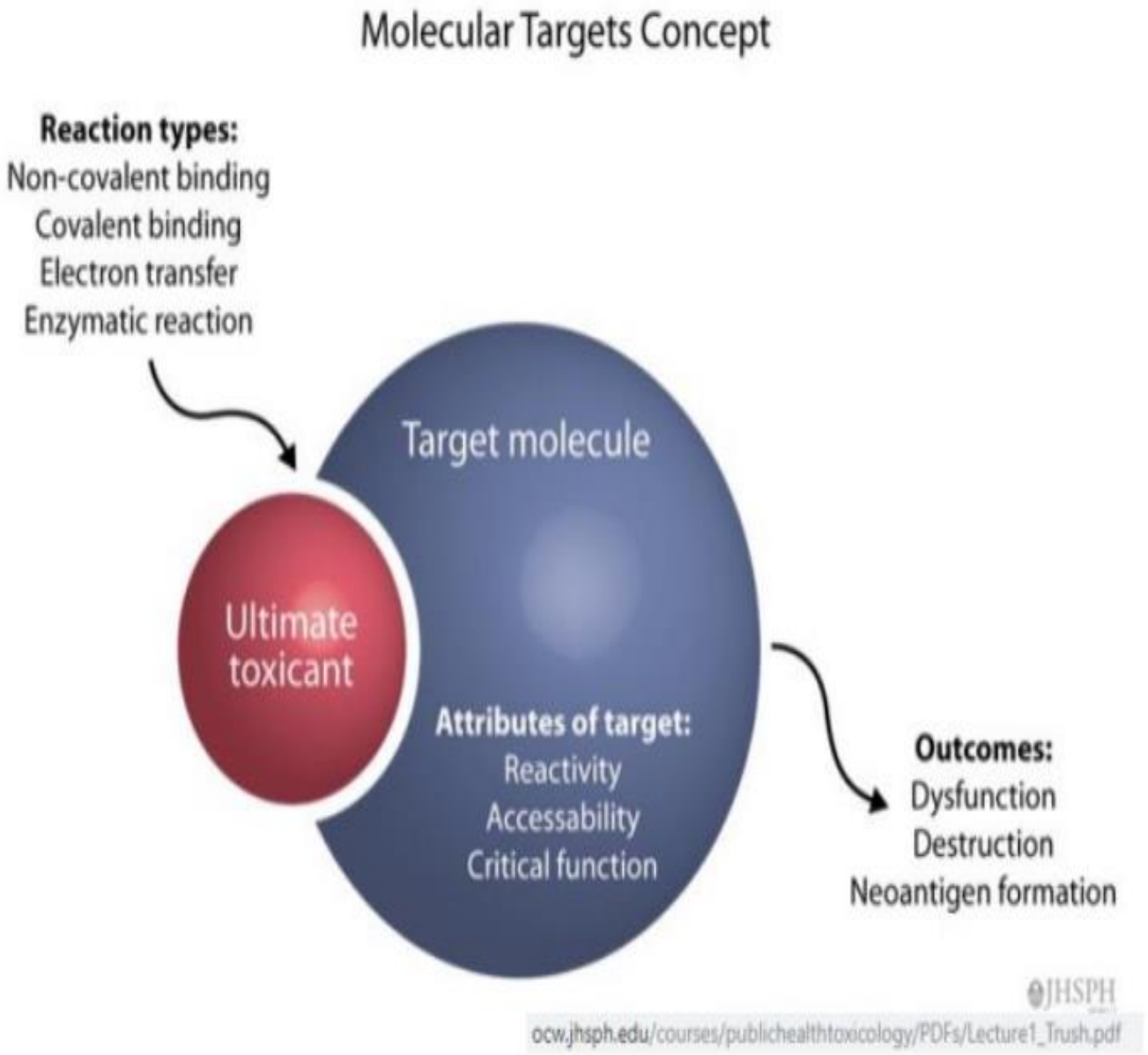
See also: Ashauer & Escher *JEM* (2010), Rubach *et al. IEAM* (2011), Jager *et al. ES&T* (2011), Ashauer *et al. ET&C* (2011)

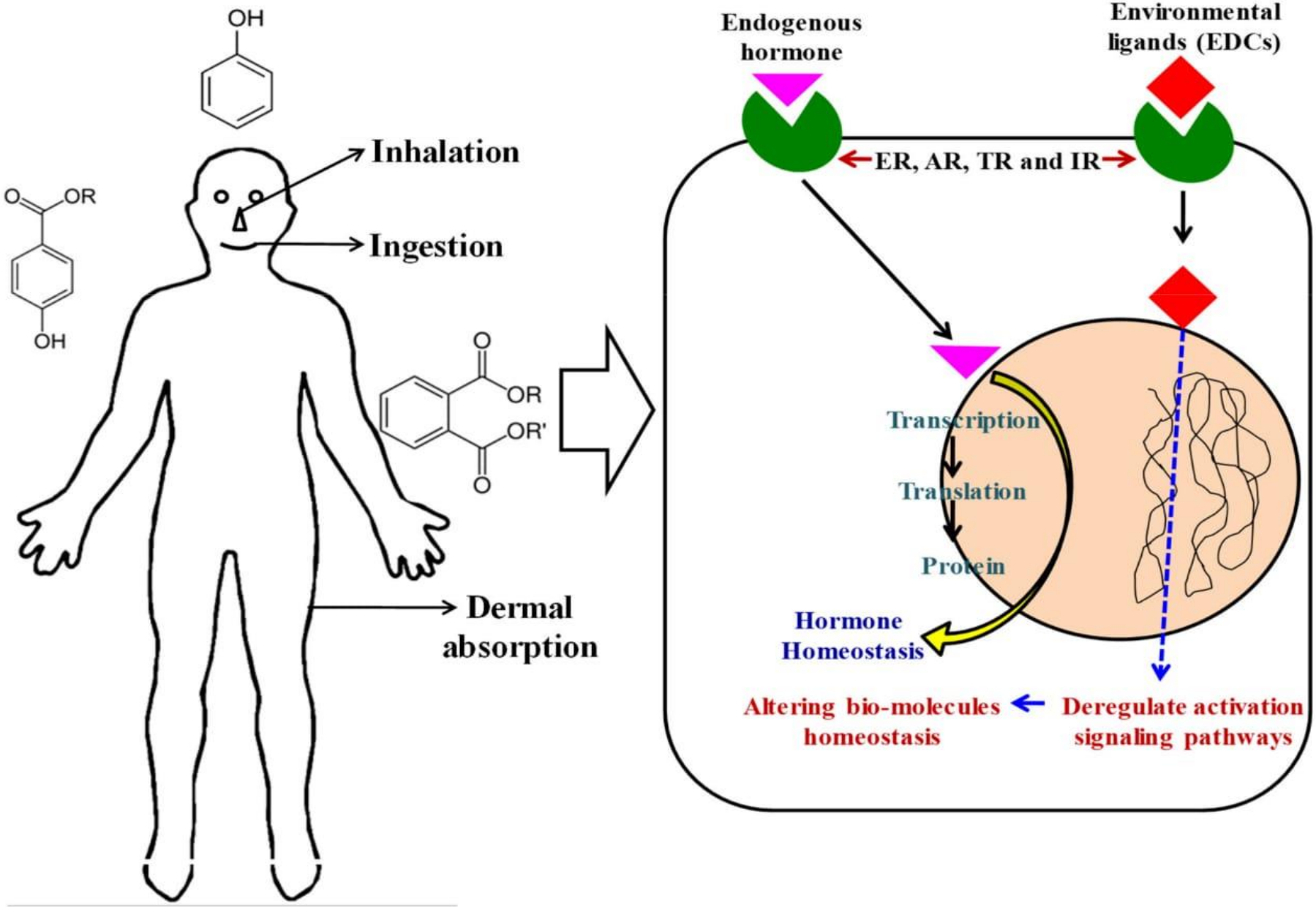
www.ecotoxmodels.org

Biological Target/Site of Action

- binding proteins
  - Arylhydrocarbon receptor-Dioxin
- Lipids (carbon tetrachloride)
- ion channels,
- DNA (aflatoxin)
- variety of other receptors (endocrine disrupting compounds-androgen/estrogen/thyroid receptor)

- interact with these receptors and produce structural or functional alterations.



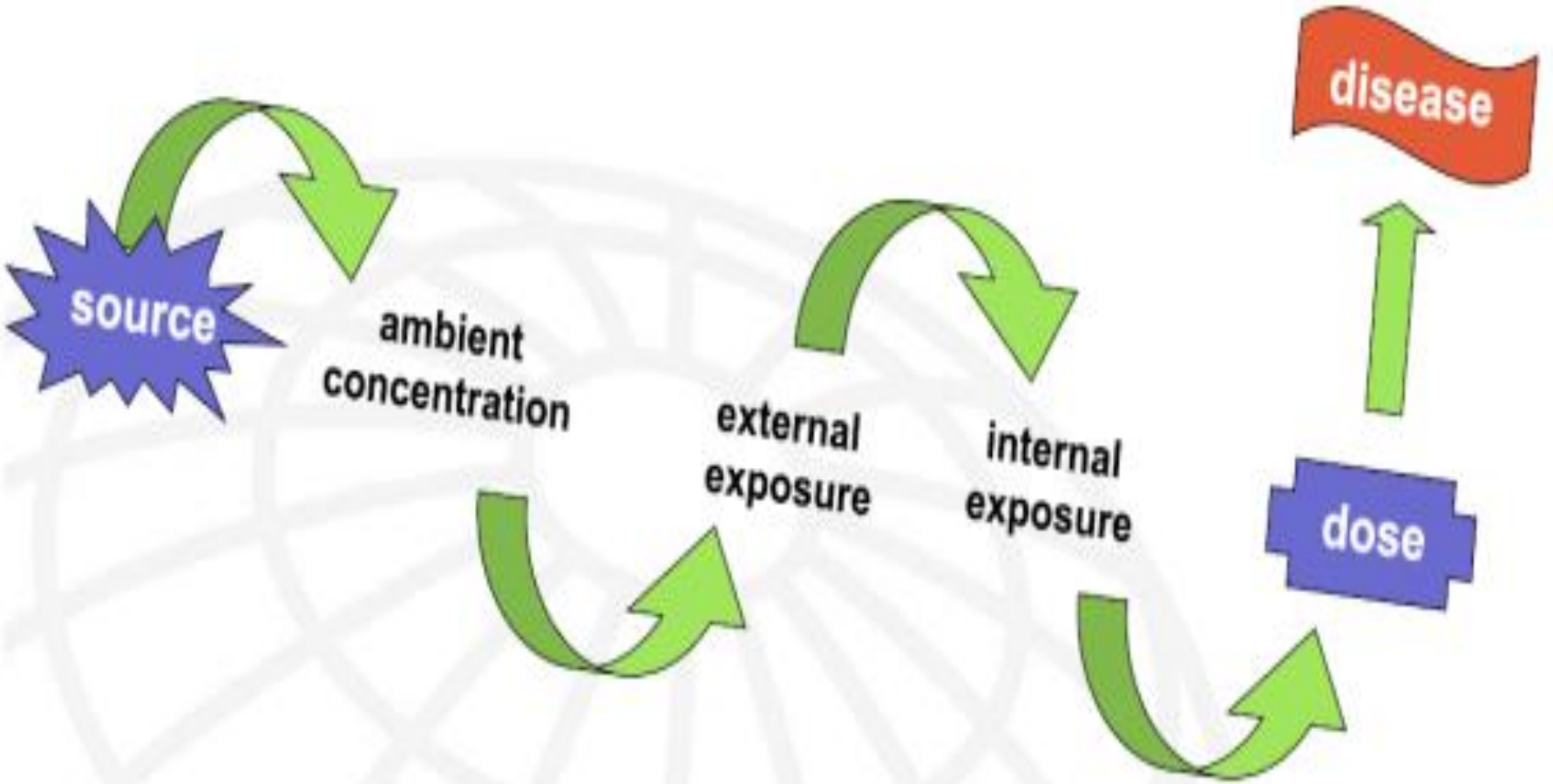

OH
Inhalation
Ingestion
OR
OH
OR
OR'
Dermal
absorption
Endogenous
hormone
Environmental
ligands (EDCs)
ER, AR, TR and IR
Transcription
Translation
Protein
Hormone
Homeostasis
Altering bio-molecules
homeostasis
Deregulate activation
signaling pathways

Transport & Deposition

- **Transport**
  - Blood
  - Lymph, haemolymph
  - Water stream in xylem
  - Cytoplamic strands in phloem
- **Deposition**

Toxicant	Target organs
Pb	Bone, teeth, brain
Cd	Kidney, bone
OC, PCB	Adipose tissue
OP (organophospahte)	Nervous tissue
Aflatoxin	Liver

From source to dose...



- Acute or chronic poisonings may occur following exposure through water, air, and food.
- Bioaccumulation of these heavy metals leads to a diversity of toxic effects on a variety of body tissues and organs.
- Heavy metals disrupt cellular events including growth, proliferation, differentiation, damage-repairing processes, and apoptosis.

- Research have shown that these metal induce toxicity including ROS generation, weakening of the antioxidant defense, enzyme inactivation, and oxidative stress.
- On the other hand, some of them have selective binding to specific macromolecules.

Key terms

- **Reactive oxygen species (ROS)** – are molecules capable of independent existence, containing at least one oxygen atom and one or more **unpaired electrons**.
- **Oxidant-** any substance that has the ability to oxidize other substance.
- **Antioxidant** – are compound that **inhibit oxidation**, a chemical reaction that can produce free **radicals**.
- **Oxidative stress** an imbalance between production and accumulation of oxygen reactive species (ROS) in cells and tissues and the ability of a biological system to **detoxify these reactive products**.

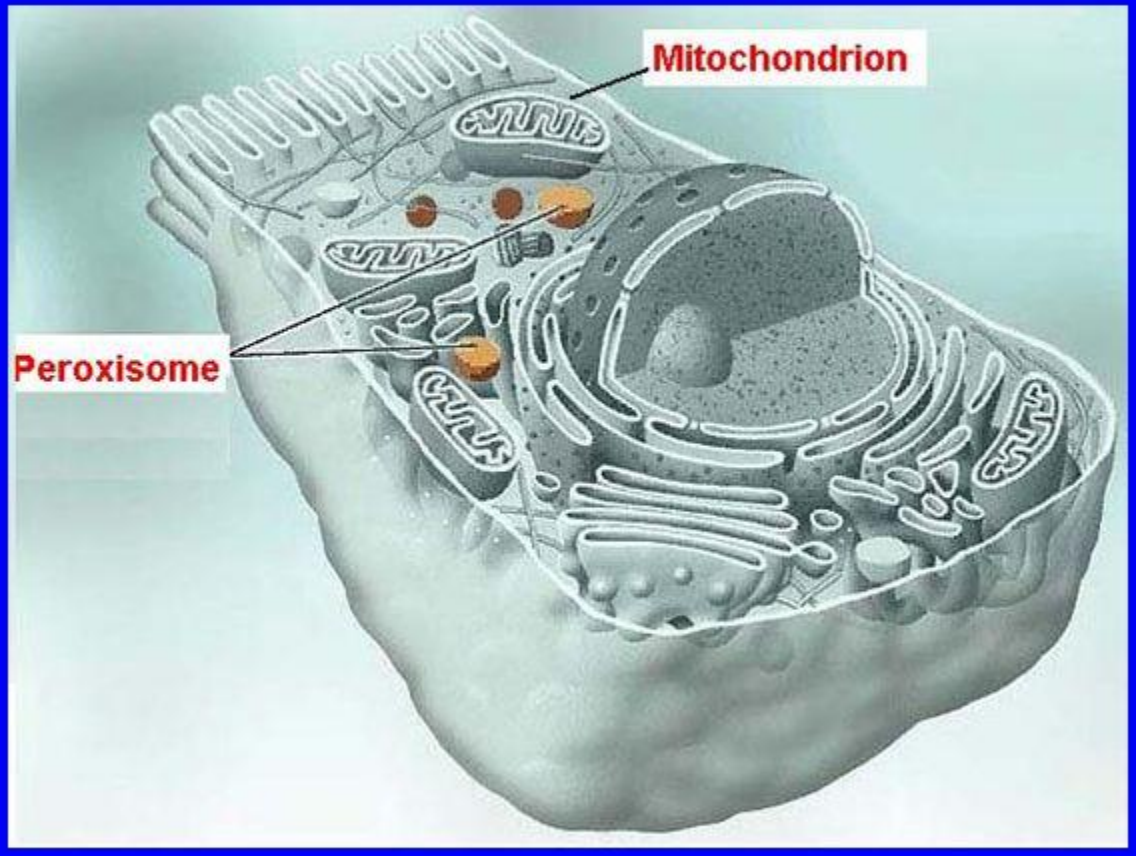
Reactive Oxygen Species

- **Benefits**: ROS are by-product of cellular oxidative metabolism and play important roles in the modulation (control influence on) of cell survival, cell death, differentiation (process by which dividing cells change their functional or phenotypical type), cell signaling and inflammation-related factor production.

Disease cause by ROS

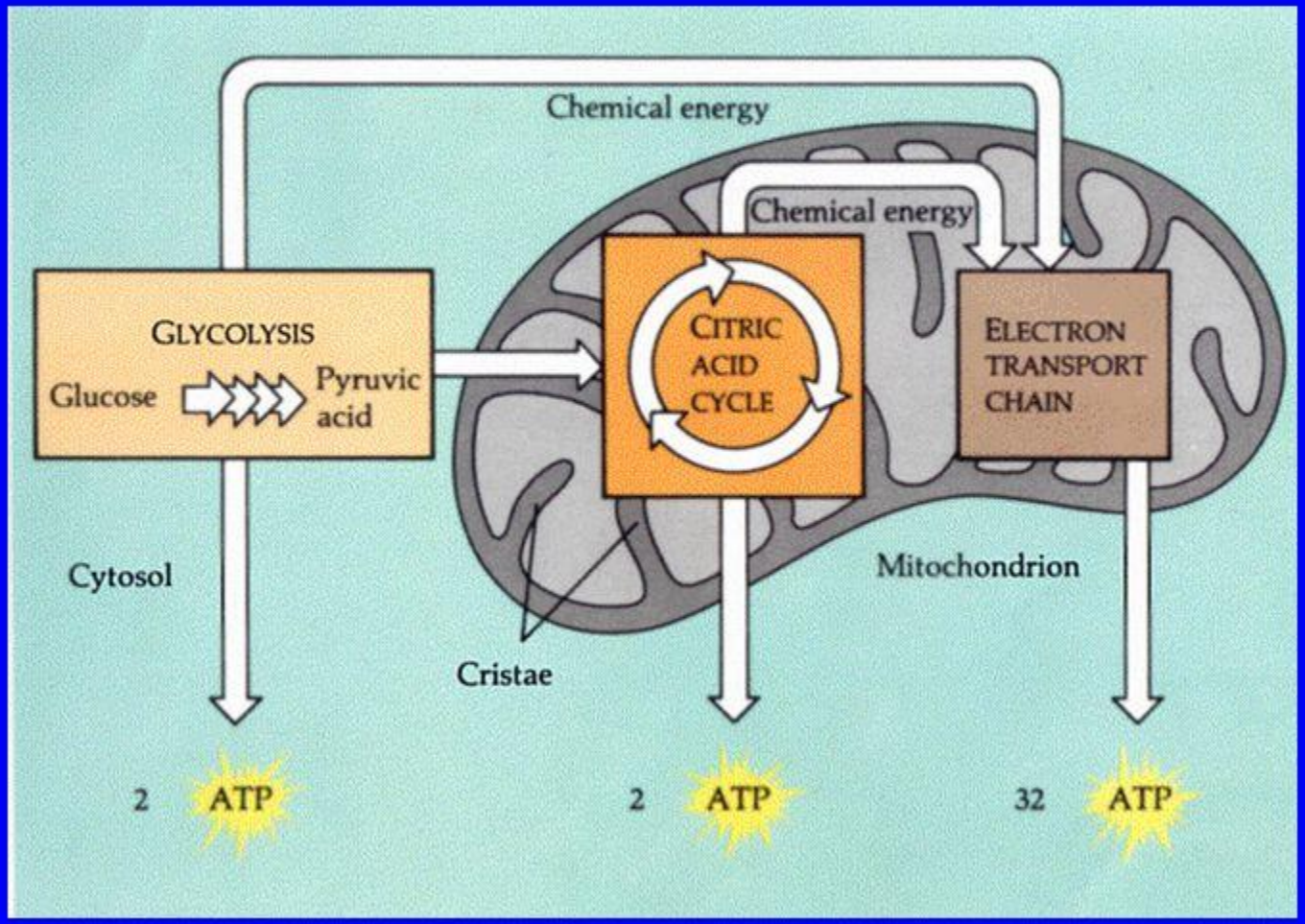
- **Harm**: when ROS overwhelm the cellular antioxidant defense system, oxidative stress occurs, which results in oxidative damage of nucleic acids, proteins, and lipids
- **Disease condition:** Cancer, Inflammatory diseases, Neurologic diseases, Vascular diseases, organ failure, diabetes, ageing.

BIOLOGICAL ROLE OF OXYGEN



- Carbohydrate metabolism for ATP Production (Mitochondria)
- Degradation of metabolic by-products by (Peroxisomes)

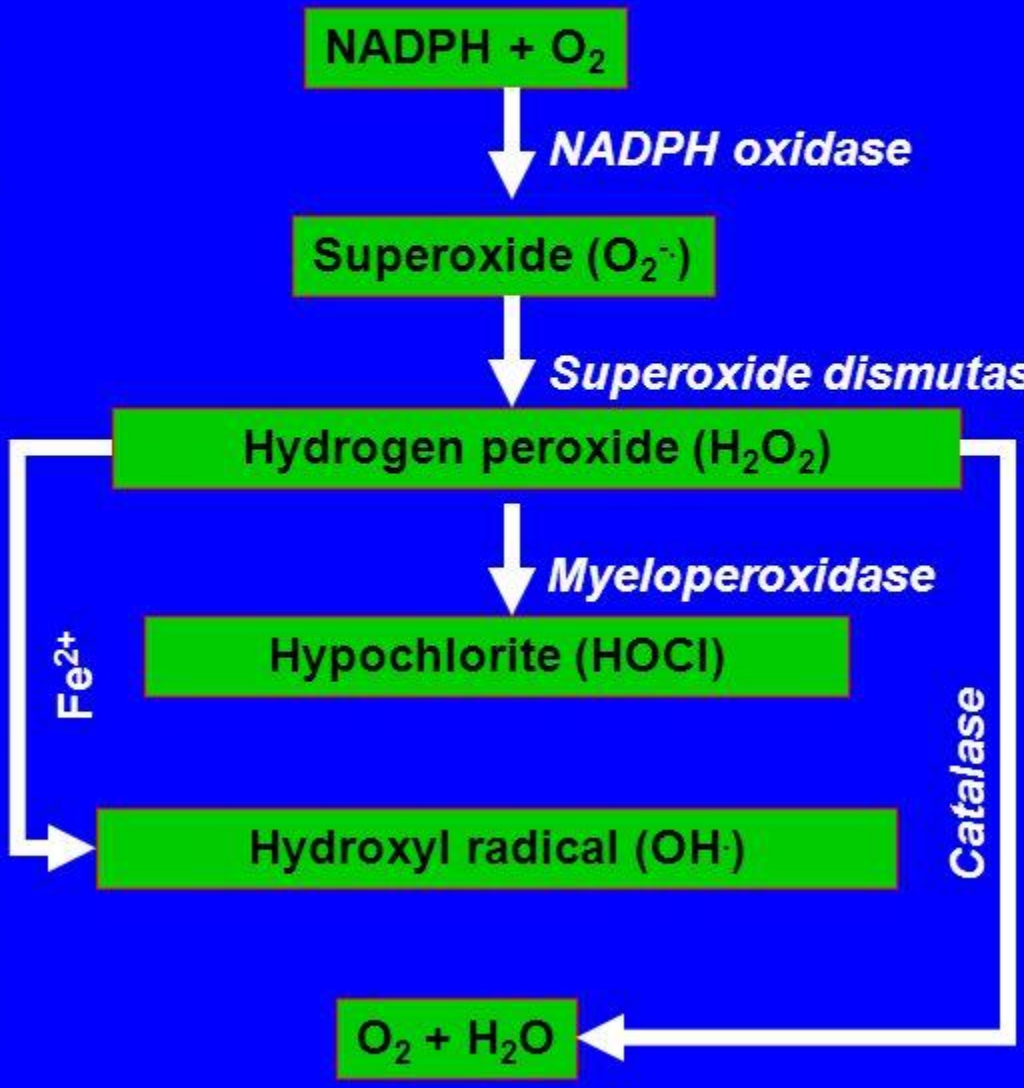
CELLULAR RESPIRATION



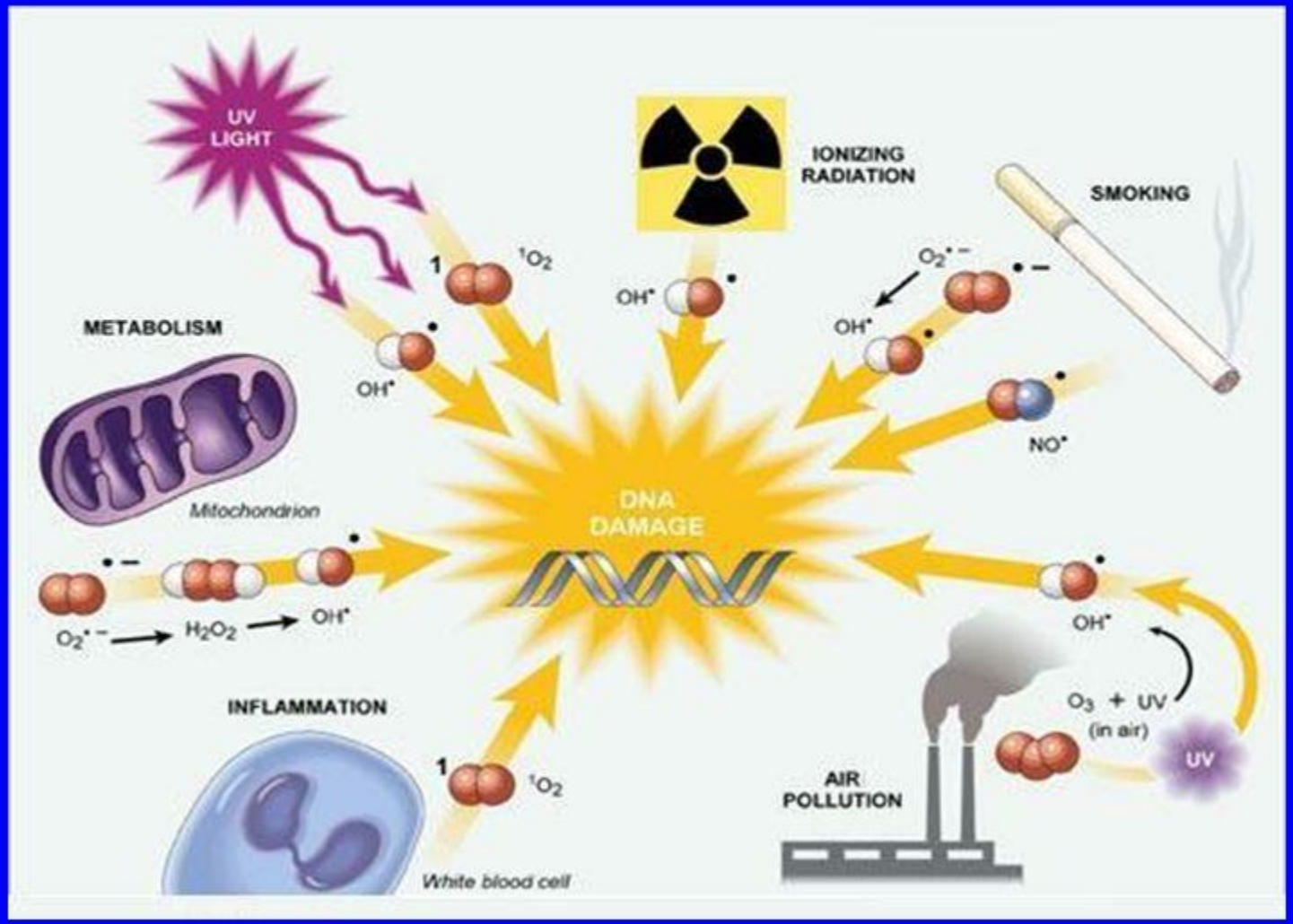
Production of Reactive Oxygen Species

REACTIVE OXYGEN SPECIES (ROS)

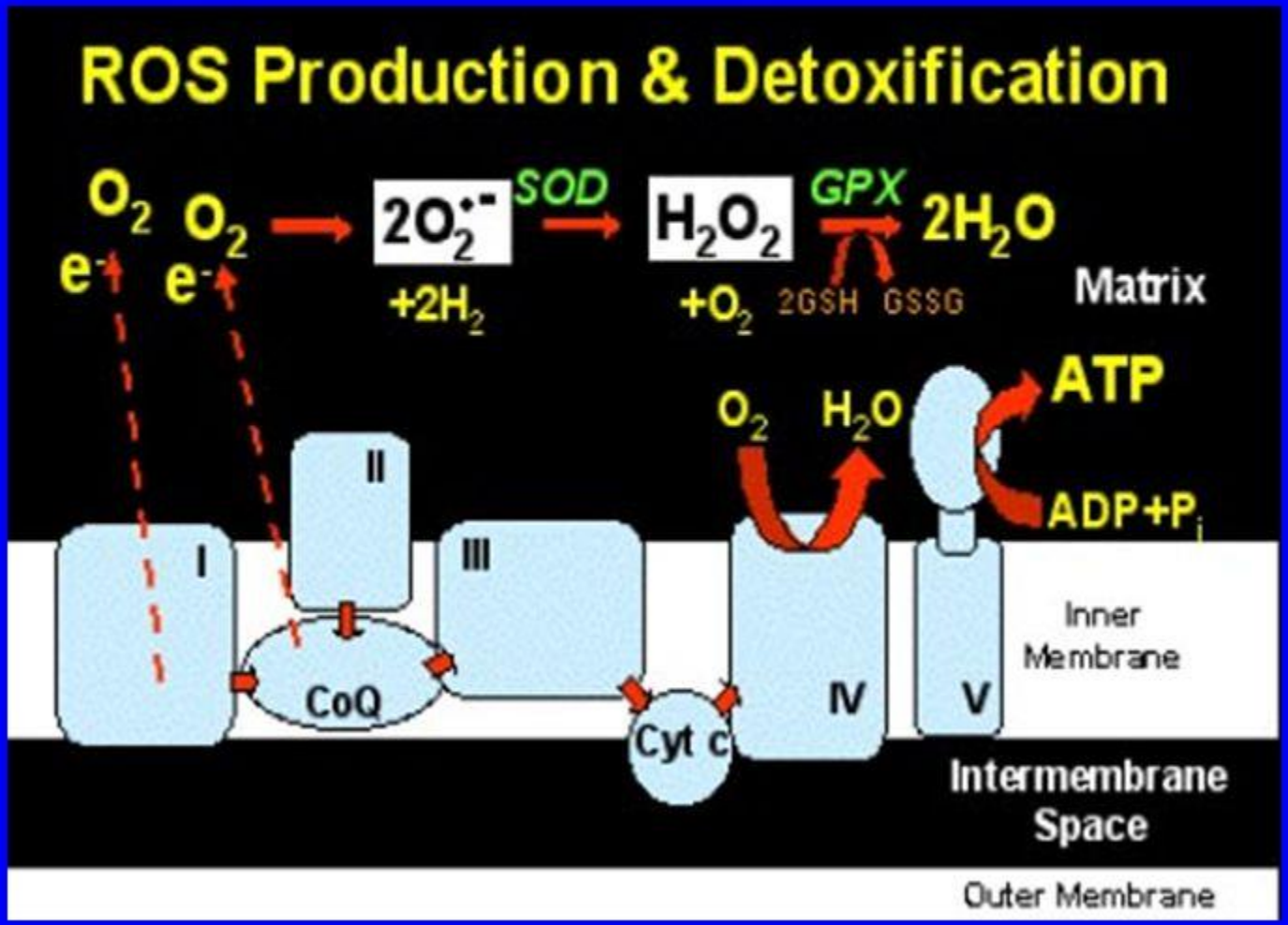
Reactive Oxygen Species	
$O_2^{\cdot -}$	Superoxide Radical
$OH\cdot$	Hydroxyl Radical
$ROO\cdot$	Peroxyl Radical
H_2O_2	Hydrogen peroxide
$^1O_2$	Singlet Oxygen
$NO\cdot$	Nitric Oxide
$ONOO^-$	Peroxynitrite



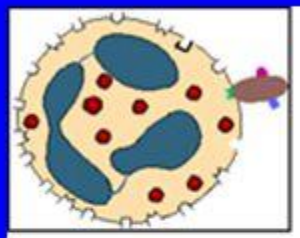
GENERATION OF REACTIVE OXYGEN SPECIES

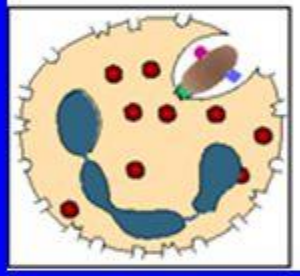


GENERATION OF ROS AS METABOLIC BY-PRODUCTS



GENERATION OF ROS IN IMMUNE DEFENSES



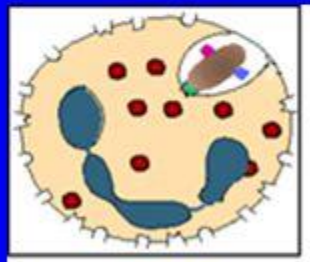


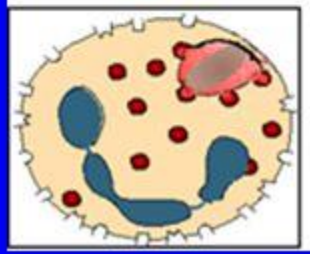
$$\text{Glucose} + NADP^{+} \xrightarrow[\text{G-6-P-dehydrogenase}]{} \text{Pentose-P} + NADPH$$

$$NADPH + O_2 \xrightarrow[\text{Cytochrome}_{b558}]{} NADP^{+} + O_2^{\cdot-}$$

$$2O_2^{\cdot-} + 2H^{+} \xrightarrow[\text{Superoxide dismutase}]{} H_2O_2 + {}^1O_2$$

$$2O_2^{\cdot-} + H_2O_2 \rightarrow {}^{\cdot}OH + OH^{-} + {}^1O_2$$





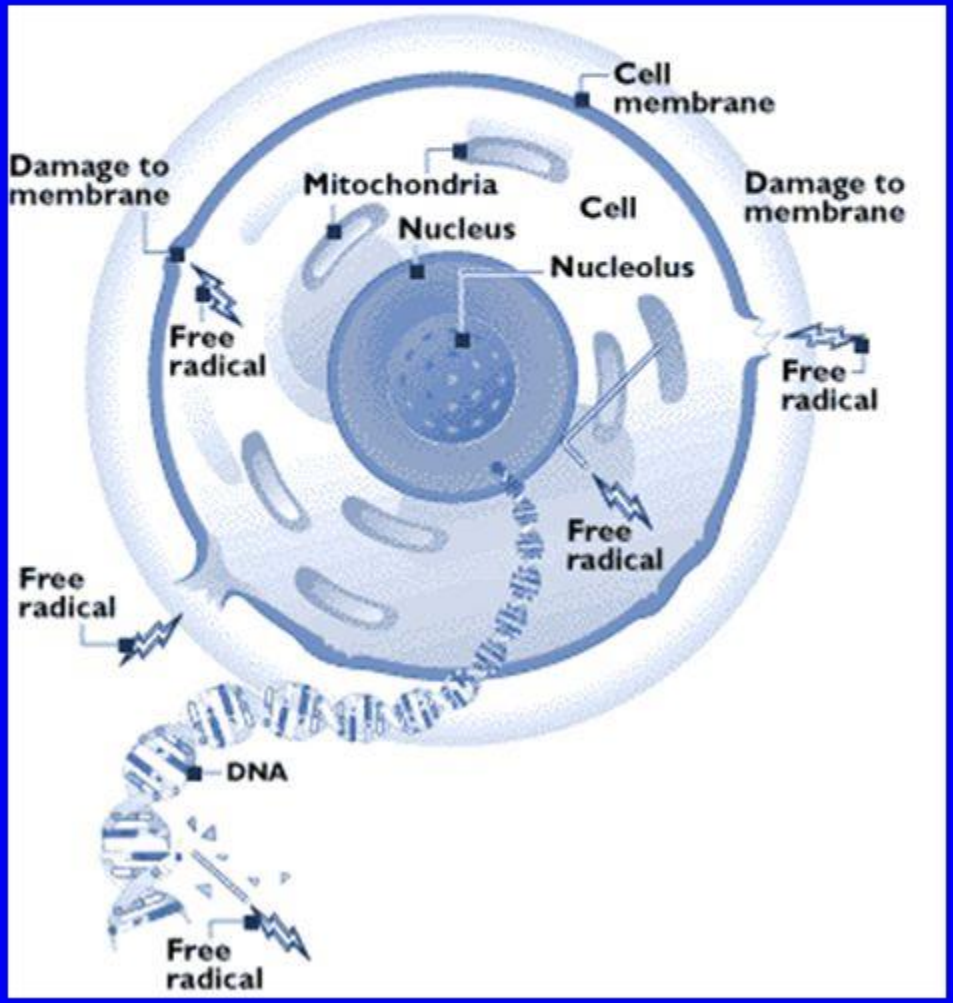
$$H_2O_2 + Cl^{-} \xrightarrow[\text{myeloperoxidase}]{} OCl^{-} + H_2O$$

$$OCL^{-} + H_2O \rightarrow {}^1O_2 + Cl^{-} + H_2O$$

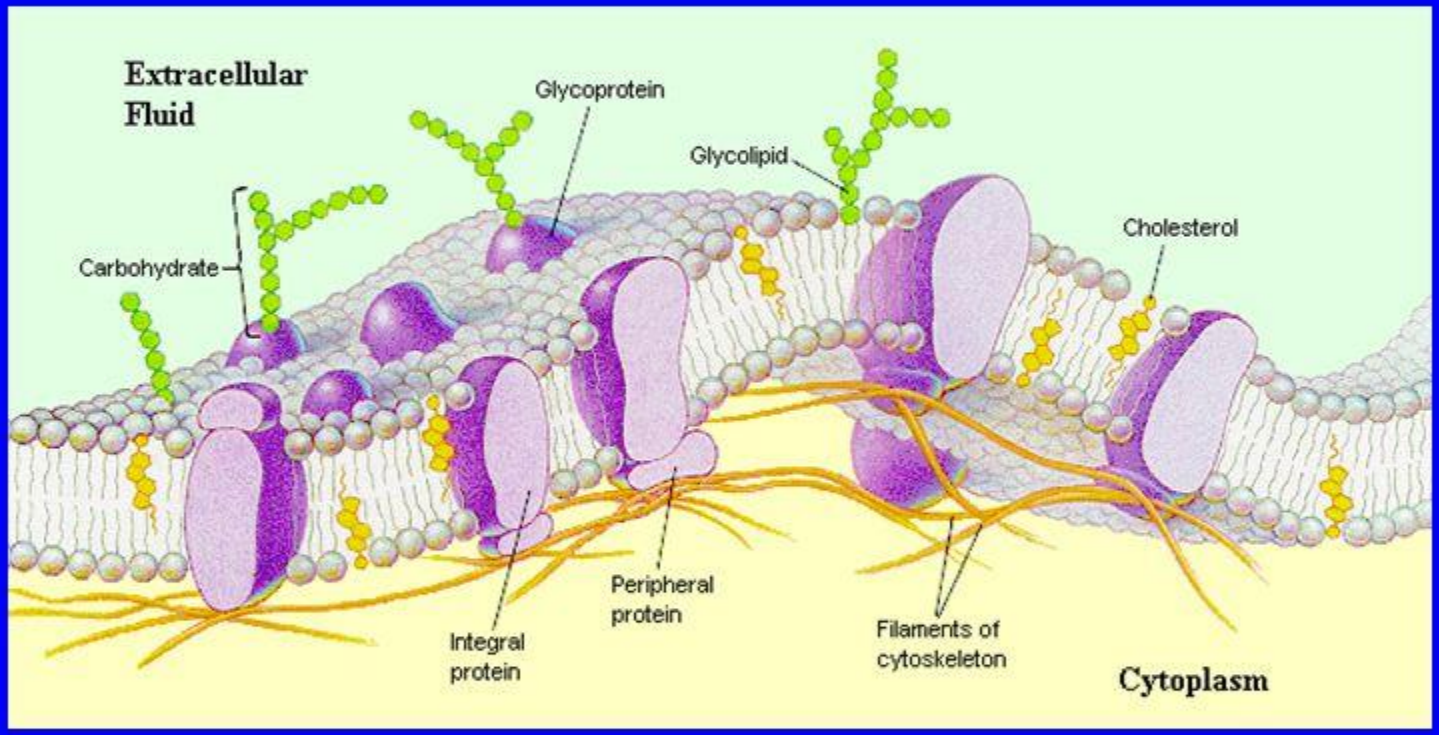
$$2O_2^{\cdot-} + 2H^{+} \xrightarrow[\text{Superoxide dismutase}]{} H_2O_2 + {}^1O_2$$

$$2\,H_2O_2 \xrightarrow[\text{catalase}]{} H_2O + O_2$$

OXIDATIVE DAMAGES

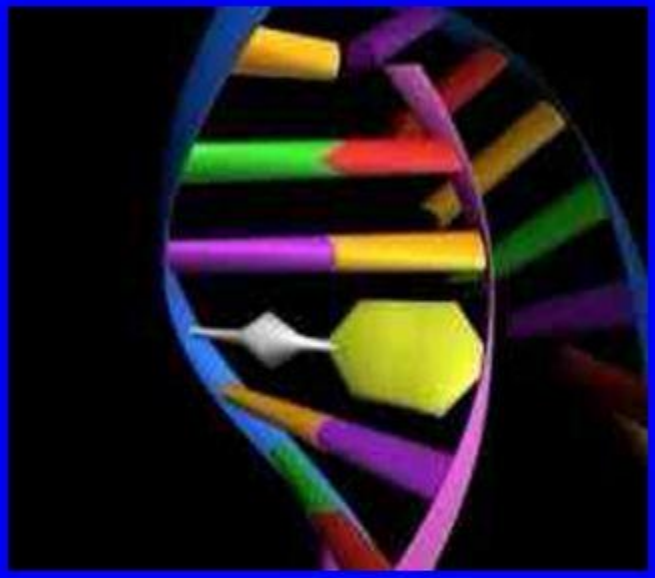


OXIDATIVE DAMAGE TO LIPIDS



- Increase membrane rigidity
- Reduce activity of membrane-bound enzyme
- Alter activity of membrane receptors
- Alter cell permeability

OXIDATIVE DAMAGE TO NUCLEIC ACIDS



- Mutation
- Single strand breakage
- Nucleotide degradation
- Cross-linking to protein

ANTIOXIDANTS

Enzymatic Antioxidants

Superoxide dismutase	Catalysis the dismutation of superoxide to hydrogen peroxide.
Catalase	Catalysis the dismutation of superoxide to water and oxygen.
Glutathione peroxidase	Degradation of hydrogen peroxide

Non-Enzymatic Antioxidants

Vitamin E	Trap peroxy radicals in cellular membranes.
Vitamin C	Reduce radicals from a variety of sources. Recycling of Vitamin E radicals.
Glutathione	Defense against reactive oxygen species.

Common Toxins Mimicking Disease

- Chemicals capable of damaging the central nervous system (CNS) are abundant in the environment, particularly in occupational settings.
- Various neurotoxins **(substance that alters the structure or functions of the nervous system),** including **heavy metals, organic solvents** and **other chemicals**, have been found to be responsible for these relatively specific neurological conditions.
- **The key thing is, these diseases shared many symptoms with other diseases.**

Common Toxins Mimicking Disease

- In general, the greater the exposure, the more severe the impairment of cerebral function and consciousness.
- Most of these agents gain entry because they are highly lipid soluble and can readily diffuse across membranes.
- The causative agents include organic solvents, which can alter cellular membrane function, and some gases (e.g., gas anesthetics-**substance that induce insensitivity to pain (nitrous oxide and others, are used for induction and maintenance of general anesthesia in the operating room)**, carbon monoxide, hydrogen sulfide, and cyanide), which can diffusely affect brain function.

Common Toxins Mimicking Disease

- **Methyl bromide intoxication**
- Methyl bromide is a highly toxic gas that is used widely as an insecticidal fumigant for dry foodstuffs. **Use to prevent pests (eg. Grapes)**
- It can be toxic to both the CNS and the peripheral nervous system.
- Most neurological manifestations of methyl bromide intoxication occur as a result of inhalation.
- Chronic exposure can cause peripheral polyneuropathy **(weakness,** numbness, and pain from nerve damage, usually in the hands and feet), optic neuropathy and cerebellar dysfunction, sometimes with neuropsychiatric disturbances.
- Typically, occupational history is vital to the diagnosis of bromide intoxication.

Common Toxins Mimicking Disease

- **Organic tin intoxication**
- Organic tins, such as the dimethyl and trimethyl compounds, are widely used as polyvinyl-chloride (**PVC, water service pipe, blood storage bags, etc.)** stabilizers, catalysts and biocides.
- Selective cerebellar dysfunction **(uncoordinating movements, imbalance, speech problems, etc.)** is most prominent upon recovery from coma due to acute severe organic tin intoxication.
- It is easy to diagnose acute organic tin intoxication in patients whose **work history** and circumstances of exposure are known, and whose signs and symptoms are typical and consistent with those reported in the literature.

Common Toxins Mimicking Disease

- **Manganese intoxication (Manganism)**
- Manganism is one of the most typical forms of parkinsonism **(A disorder of the central nervous system that affects movement, often including tremors).**
- Chronic excessive exposure to manganese (Mn) can affect the globus pallidus **(structure in the brain that involved in the regulation of voluntary movement),** resulting in parkinsonian signs and symptoms, sometimes along with psychiatric features called locura manganica or Mn madness.
- Historically, **miners** developed psychosis due to exposure to Mn at levels of up to several hundred milligrams per cubic meter. **Sources: rice, leafy vegetable, coffee, tea, etc.**

Toxicology Screening in Diagnosis-Case of Lead

- Lead introduced into the bloodstream is excreted in urine and bile at a clearance rate of 1 to 3 mL/min, with a half-life of roughly 30 days.
- The remaining lead binds to red blood cells, is distributed throughout the soft tissues of the body, and eventually accumulates in bone.
- The half-life of bone deposited lead ranges from 20 to 30 years.
- Turnover of bone tissue **(the process of Resorption followed by replacement by new bone with little change in shape)** releases lead back into the bloodstream, and such processes as pregnancy, menopause, or lactation (**mammal that produce milk to feed her babies)** may increase

Toxicology Screening in Diagnosis-Case of Lead

- blood lead levels by speeding bone tissue turnover.
- Lead in the body is measured with both blood and bone levels.
- Blood lead levels are more reflective of acute exposure, whereas bone lead levels better reflect cumulative exposure over time.

Toxicology Screening in Diagnosis-Case of Lead

- The presence of lead in the human body causes damage to the nervous system through several mechanisms.
- Direct effects on the nervous system may be classified as either morphological or pharmacological.
- Morphological effects alter the development of the nervous system, particularly from the prenatal period through childhood.

Toxicology Screening in Diagnosis-Case of Lead

- Occupational lead exposure continues to be a source **(street food is another source)** of both acute and chronic exposure, resulting in blood levels of 40 to 120 μg/dL among participants.
- Prenatal exposure presents an additional risk for lead neurotoxicity.
- Maternal exposure to lead and overall maternal body burden of lead are closely associated with lead levels in the fetus, likely because lead appears to cross the placenta freely and because pregnancy increases systemic demand for calcium, resulting in higher bone turnover and consequent lead release into the bloodstream.

Toxicology Screening in Diagnosis-Case of Lead

- Poor nutrition appears to increase risk of toxic effects of lead when exposure is held constant.
- Deficiencies in calcium, iron, and zinc have been specifically identified as risk factors.
- Calcium deficiency appears to increase both retention of lead and the severity of its toxic effects.
- Low intake of dietary iron has similar effects and is perhaps more important because of relatively high risk of iron deficiency in childhood.

Toxicology Screening in Diagnosis-Case of Lead

- Zinc deficiency appears to result in a vicious cycle in that it increases lead absorption, which in turn increases zinc excretion.
- The role of nutrition perhaps accounts for the finding that low socioeconomic status increases risk for persistence of cognitive deficits after prenatal lead exposure.

Toxicology Considerations in Infectious Diseases

- Infectious diseases are among the top five leading causes of death globally. Even before the COVID-19 pandemic, infectious diseases accounted for 6 of the leading 10 causes of death among low-income populations.
- Major infectious diseases in LMICs are lower respiratory infections, tuberculosis (TB), malaria, HIV/AIDS, neonatal infection and diarrhoea.

Toxicology Considerations in Infectious Diseases

- Poverty, inadequate nutrition, lack of access to clean water, low sanitation and poor hygiene practices lead to infection vulnerability in the developing regions.
- Scientists have long known that the environment plays a significant role in the spread of infectious diseases.

Toxicology Considerations in Infectious Diseases

- Emerging findings suggest that environmental toxicants such as airborne particulate matter, pesticides and heavy metals may weaken the immune system.
- In addition, exposure to some pollutants may reduce vaccine effectiveness against communicable diseases.
- Exposure to environmental toxicants also contributes to non-communicable diseases such as endocrine, reproductive, metabolic and neurodegenerative diseases..

Toxicology Considerations in Infectious Diseases

- Environmental toxicants such as air pollutants and suspended particles play essential roles in developing TB.
- **Exposure to environmental pollutants** destroys epithelial cells of the respiratory tracts, so they no longer act as a defence mechanism for TB.
- **Pesticide exposure (for example: malathion and chlorphyrifos are commonly used on all fruits, vegetable, and wheat)** causes varying immune system changes, such as modifications in well-regulated immune responses to allergens **(cause an allergic reaction)**, self-antigens and microbial antigens, increasing the organism's susceptibility to autoimmune and infectious diseases.

Toxicology and Neurological Disorder

- Neurodegeneration leads to the loss of structural and functioning components of neurons over time
- The majority of neurological diseases are mainly related to prenatal and postnatal exposure to industrially produced environmental toxins.
- Some neurotoxic metals, like lead (Pb), aluminium (Al), Mercury (Hg), manganese (Mn), cadmium (Cd), and arsenic (As), and also pesticides and metal based nanoparticles, have been implicated in Parkinson's and Alzheimer's **(A progressive disease that destroys memory and other important mental functions)** disease.

Toxicology and Neurological Disorder

- Sources of Led (Pb) .. Some jewelry, some toys, dust, water, discarded batteries, paint, Lipstick, and cosmetics **(note; creates grip and smoothness for the skin).**
- Heavy metals like Lead, arsenic, mercury, aluminum, zinc, chromium and iron are found in a wide variety of personal care products including lipstick, whitening toothpaste, eyeliner pencil and nail polish. Some metals are intentionally added as ingredients, while others are contaminants.

Toxicology and Neurological Disorder

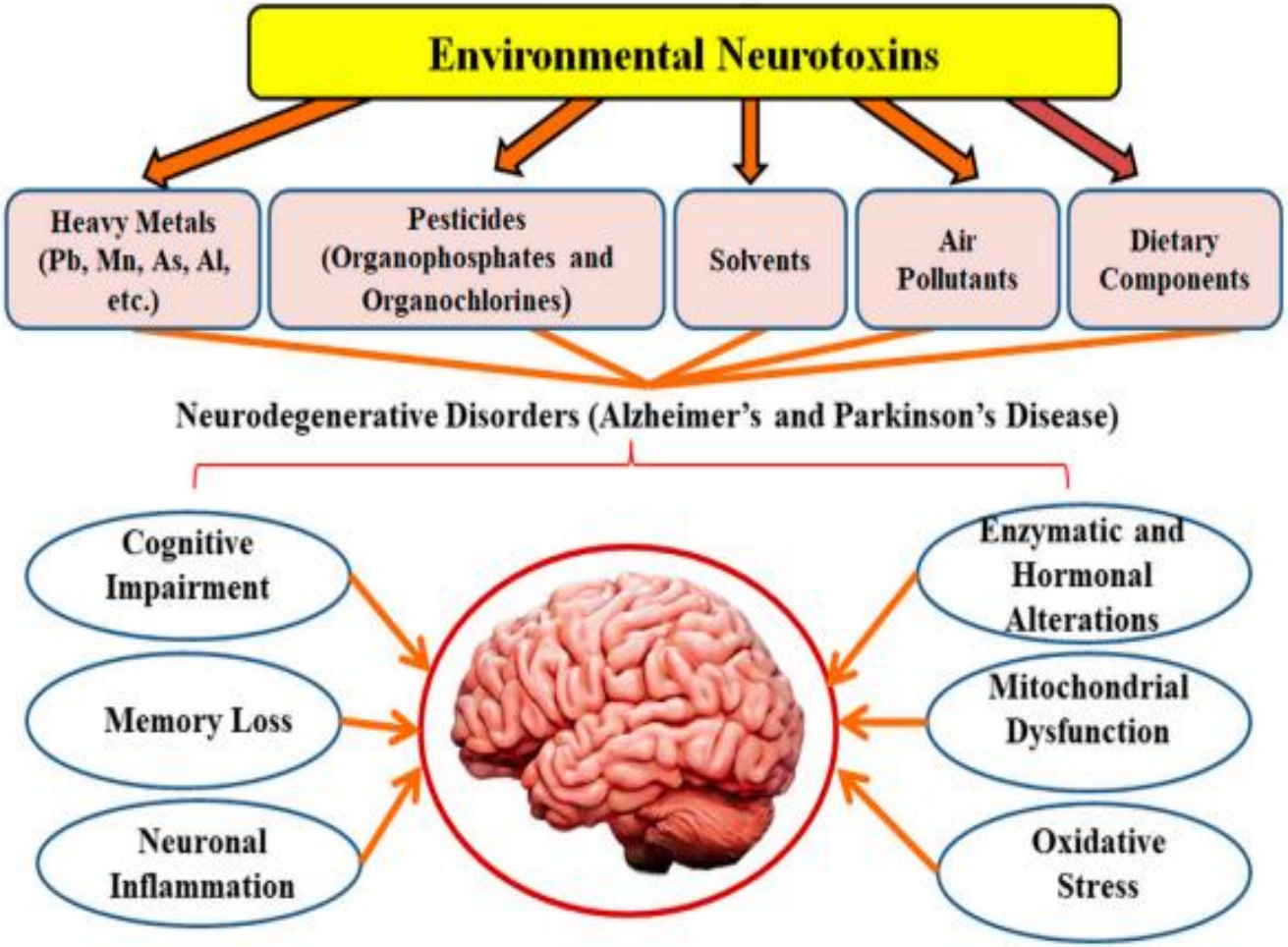


FIGURE 1 | The impact of environmental toxicants on neurodegenerative disorders.

Patient History and Toxic Exposure

- **Taking an Exposure History**
- Because many environmental diseases either manifest as common medical problems or have nonspecific symptoms, an exposure history is vital for correct diagnosis.
- The primary care clinician can play an important role in detecting, treating, and preventing disease due to toxic exposure by taking a thorough exposure history.

Patient History and Toxic Exposure

- Most environmental and occupational diseases either manifest as common medical problems or have nonspecific symptoms.
- It is the etiology **(cause)** that distinguishes a disorder as an environmental illness.
- Unless an exposure history is pursued by the clinician, the etiologic diagnosis may be missed, treatment may be inappropriate, and exposure can continue.

Patient History and Toxic Exposure

- The exposure history form, which can be completed by the clinician or by the patient (to save staff time), will guide the clinician through various aspects of this process.
- The form elicits many important points of an exposure history including job descriptions and categories associated with hazardous substances, physical and biologic agents, and temporal and activity patterns related to environmental and occupational disease.
- The form explores past and current exposures.

Patient History and Toxic Exposure

Organ/System	Exposure Risks
Respiratory	asbestos,* radon,* cigarette smoke, glues
Dermatologic	dioxin,* nickel, arsenic,* mercury,* cement (chromium*), PCBs,* glues, rubber cement
Liver	carbon tetrachloride,* methylene chloride,* vinyl chloride*
Kidney	cadmium,* lead,* mercury,* chlorinated hydrocarbon solvents*
Cardiovascular	carbon monoxide, noise, tobacco smoke, physical stress, carbon disulfide, nitrates,* methylene chloride*
Reproductive	methylmercury,* carbon monoxide, lead,* ethylene oxide
Hematologic	arsenic,* benzene,* nitrates,* radiation
Neuropsychologic	tetrachloroethylene,* mercury,* arsenic,* toluene,* lead,* methanol,* noise, vinyl chloride*

Patient History and Toxic Exposure

- **Toxicants in the Home/Environment**
- The clinician should consider the following sources, which are discussed below, when eliciting information on exposures in the home and environment:
- Indoor air pollution
- Common household products
- Pesticides and lawn **(an area of short grass)** care products
- Lead products and waste
- Recreational hazards
- Water supply
- Soil contamination

Patient History and Toxic Exposure

- **Indoor Air Pollution**
- *Wood Stoves/Gas Ranges*
- **Does the patient have a wood stove?**
- **Is there a smoke smell indoors?**
- **When was the last time the pipe and stove were cleaned?**
- When not properly maintained and vented, wood stoves emit noxious gases including carbon monoxide, oxides of nitrogen, particulates, and hydrocarbons.
- Studies have shown that children living in homes heated with wood stoves have a significant increase in respiratory symptoms compared with children living in homes without wood stoves.

Patient History and Toxic Exposure

- **Common Household Products**
- **Does the patient use any of the following on a regular basis: cleaners for glass, oven, floors, drains, toilets, polishes, air fresheners and disinfectants, glues, solvents, paint strippers, sealants?**
- A 1987 EPA study found approximately 12 common organic pollutants in concentrations 2 to 5 times higher in air inside homes than in outdoor air from use of household products. Product warning labels are often inadequate and pertain to acute exposures only. Long-term or repeated use of some household chemicals, such as chlorinated hydrocarbons, can result in cancer.

Patient History and Toxic Exposure

- Commonly used compounds that can have serious adverse effects are methylene chloride (found in paint strippers and thinners, and adhesive removers), tetrachloroethylene (used in dry cleaning of clothes), and paradichlorobenzene (found in room air fresheners, toilet bowl deodorizers, and moth crystals)

THANK YOU