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Abbreviations list

ADI	Acceptable Daily Intake
ALARA	As Low As Reasonably Achievable
ANSES	French Agency for Food, Environmental and Occupational Health & Safety
AOI	Area of Interest
ATMO	Approved Air Quality Monitoring Associations Network
AZF	Nitric Acid Fertilization Plant
BE	Biomonitoring Equivalent
BMD	Benchmark Dose
BMDL	Benchmark Dose Lower Confidence Limit
BPA	Bisphenol A

CFCs	Chlorofluorocarbons
CI	Inhaled Concentration
CO	Carbon Monoxide
COPD	Chronic Obstructive Pulmonary Disease
DASRI	Healthcare Waste with Infectious Risks
DCE	Water Framework Directive
DED	Daily Exposure Dose
DHHS	Department of Health and Human Services
EASE	Estimation and Assessment of Substance Exposures
EEA	European Environment Agency
EPA	Environmental Protection Agency
ERS	Health Risk Assessment
EU	European Union
HQ	Hazard Quotient
HRA	Health Risk Assessment
ICDDR,B	International Centre for Diarrhoeal Disease Research, Bangladesh
ICPE	Classified Installations for Environmental Protection
ICRP	International Commission on Radiological Protection
IER	Individual Excess Risk
IUR	Inhalation Unit Risk
ISO	International Organization for Standardization
LC₅₀	Lethal Concentration 50
LCPE	Canadian Environmental Protection Act
LD₅₀	Lethal Dose 50
LEMA	Water and Aquatic Environments Law
LOAEL	Lowest Observed Adverse Effect Level

MF	Modifying Factor
MRL	Minimal Risk Level
NO₂	Nitrogen Dioxide
NOAEL	No Observed Adverse Effect Level
NO_x	Nitrogen Oxides
NRC	National Research Council
ONU (UN)	United Nations
PAHs	Polycyclic Aromatic Hydrocarbons
PET scan	Positron Emission Tomography scan
PM_{2.5}	Fine Particulate Matter ($\leq 2.5 \mu\text{m}$)
PM₁₀	Particulate Matter ($\leq 10 \mu\text{m}$)
PNUE (UNEP)	United Nations Environment Programme
POP / POPs	Persistent Organic Pollutants
PSDs	Passive Sampling Devices
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfC	Reference Concentration
RfD	Reference Dose
SAR	Structure–Activity Relationship
SF	Slope Factor
SIA	Acute Radiation Syndrome
SPF	French Public Health Agency
TRV	Toxicological Reference Value
UE (EU)	European Union
UF	Uncertainty Factor
UN	United Nations
UNEP	United Nations Environment Programme

VOCs

Volatile Organic Compounds

WHO

World Health Organization

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Our surroundings continuously interact with us, whether through the air we inhale, the water we consume, the food we eat, or the surfaces we come into contact with. Often, we remain unaware of these exchanges. However, they are underpinned by a sophisticated web of chemical, physical, and biological elements that can affect our well-being in both subtle and considerable manners. In an era characterized by swift industrial growth and environmental transformation, grasping the nature of these interactions is becoming ever more crucial.

This course, Environmental and health risks, invites students to take a step back and look more closely at what “risk” really means in everyday life. It is not only about identifying hazards, but also about understanding how and why exposure occurs, and what happens once a substance enters the human body. Why do some exposures lead to disease while others do not? Why are certain populations more affected than others? These are some of the questions that guide this course.

Throughout the semester, students will explore how pollutants originate, how they move through environmental compartments such as air, water, and soil, and how they eventually reach humans. The course gradually introduces the tools used by scientists to study these processes, including the key steps of health risk assessment. Rather than treating these steps as abstract concepts, the course aims to show how they are applied in real situations to evaluate potential dangers and support decision-making.

At the same time, attention is given to the broader context in which these risks exist. Environmental issues are rarely isolated; they are connected to societal choices, technological development, and regulatory frameworks. Understanding how risks are monitored, assessed, and managed helps students see the link between scientific knowledge and public health action.

By the end of this course, students are expected not only to have acquired essential knowledge, but also to develop a more critical and reflective perspective on environmental health issues. In doing so, they will be better prepared to face the challenges of a world where the boundaries between environment and health are increasingly intertwined.

1. General Concepts

1.1.Introduction

Risk assessment in environmental and human health has become a central field within public health sciences and toxicology. Although its structured development is relatively recent on the scientific scale, it draws upon contributions from multiple disciplines, including epidemiology, toxicology, engineering, and statistics.

Each of these disciplines has provided specific approaches that contribute to a better understanding, quantification, and management of risks associated with environmental exposures. However, most published studies tend to focus on particular aspects of risk assessment, without consistently offering an integrated view of the different stages of the process.

In this context, it is essential to adopt a comprehensive approach that connects sources of hazards, exposure pathways, and health effects, in order to better understand the challenges associated with environmental risks.

1.2 What is risk?

Risk is a fundamental concept in toxicology and environmental health. It is generally defined as: **“The probability of occurrence of an adverse health effect resulting from exposure to a hazard, taking into account the severity of that effect”**.

Thus, risk is based on two essential components :

1. The probability that an adverse event will occur;
2. The severity of the consequences associated with that event.

In the absence of either component, the concept of risk loses its meaning.

For example: a hazard without exposure does not generate risk, whereas exposure without harmful effect does not constitute a health risk.

In the field of environmental health, risk is therefore closely linked to the interaction between a hazard, defined as an intrinsic property of a substance or situation, and the level of population exposure.

Risk is generally expressed as a function of hazard and exposure:

$$\text{Risk} = f(\text{hazard, exposure})$$

where the function f integrates dose–response relationships as well as biological factors influencing the occurrence of adverse effects (**risk factors**).

1.3 Mode of exposure

1.3.1 Definition of exposure

Exposure is defined as the amount (or concentration) of a chemical substance reaching a target organism, system, or (sub-)population at a specified frequency and for a defined duration, as well as the timing and frequency of its interaction with individuals or other organisms at risk.

According to the International Programme on Chemical Safety ([WHO, 2001](#)), exposure is defined as the “**contact of an organism with a chemical or physical agent, quantified as the amount of the chemical available at the exchange boundaries of the organism and accessible for absorption.**”

The term “**agent**” refers to a chemical, biological, or physical entity in contact with a target. The “**target**” refers to any physical, biological, or ecological entity exposed to an agent.

In the case of human exposure, contact occurs with the external surfaces of the body (the target), such as the skin, as well as through openings such as the mouth, nostrils, and lesions. When an agent crosses an external exposure surface without necessarily passing through an absorption barrier (for example, via ingestion or inhalation), this is referred to as intake. The resulting dose is termed the intake dose (also referred to in the literature as administered dose or potential dose).



Damage caused by chemical substances occurs only if these substances reach sensitive sites within an individual or another living organism at a sufficiently high concentration and for a sufficient duration; this is referred to as the **absorbed dose**.

For highly toxic substances, the tolerable level of exposure may be close to zero. When determining what constitutes a tolerable exposure, it is essential to have data linking exposure to the occurrence of toxicity or adverse effects.

The terms “exposure” and “dose” are closely related and often confused:

- **Dose** refers to the amount of an agent that enters a target over a given period after crossing a contact barrier.
- **Exposure** does not necessarily result in a dose; however, there can be no dose without a corresponding exposure.

1.3.2. What is an adverse effect?

An adverse effect is defined as an abnormal, undesirable, or harmful change occurring following exposure to a potentially toxic substance. The ultimate adverse effect is death; however, less severe effects may include alterations in food consumption, changes in body and organ weights, observable pathological changes, or simply modifications in enzyme levels.

1.3.3 What is the mode of exposure?

The mode of exposure refers to the route by which a toxic substance comes into contact with an organism.

The main routes of exposure are the skin (topical or percutaneous exposure), the lungs (inhalation), and the gastrointestinal tract (ingestion).

In general, for a given concentration of a substance and an identical duration of exposure, inhalation is likely to cause more damage than ingestion, which in turn is more harmful than topical exposure. Eye exposure may also lead to serious consequences and must be carefully considered.

1.3.3.1 Dermal (cutaneous or percutaneous) exposure

Chemical substances can penetrate through the skin, particularly lipophilic compounds. It is often overlooked that these substances are capable of crossing healthy and intact skin, which should be emphasized. Substances known to be absorbed through the skin include aniline, hydrogen cyanide, certain steroid hormones, organic mercury compounds, nitrobenzene, organophosphorus compounds, and phenol. Some substances, such as phenol or methylmercury chloride, may be lethal if absorbed over a relatively small area of skin (a few square centimeters).

When protective clothing is worn, it should be noted that dermal absorption of a chemical that has penetrated beneath such clothing may be even more rapid than on unprotected skin, as the substance cannot evaporate and remains in prolonged contact with the skin.

Examples of potential scenarios of percutaneous exposure include:

- **Showering** : dermal absorption of contaminants present in domestic water;
- **Swimming** : potential exposure to contaminants in surface waters through dermal absorption;
- **Direct contact with soil** : potential exposure to contaminants present in outdoor soils.

1.3.3.2 Inhalation (via the lungs)

Contaminated air may contain vapors, gases, or toxic particles that are inhaled directly into the respiratory system, representing a particularly rapid route of absorption into the bloodstream.

Gases and vapors are readily inhaled, whereas the inhalation of particles depends on their size and shape. The smaller the particle, the deeper it can penetrate into the respiratory tract. Dust particles with an effective aerodynamic diameter between 0.5 and 7 μm (respirable fraction) may persist in the alveoli and respiratory bronchioles after deposition.

Possible exposure scenarios include:

- **Indoor air** : potential exposure to contaminants (volatile compounds and fugitive dust) present in indoor ambient air;
- **Outdoor air** : exposure to contaminants present in outdoor ambient air.

1.3.3.3. Ingestion (via the gastrointestinal tract)

Absorption may occur through the consumption of contaminated food or water. It is important to note that exposure may be continuous or repeated at regular intervals.

A chemical substance may accumulate if its rate of absorption exceeds its rate of excretion. This is particularly likely for substances characterized by high lipid solubility and strong chemical stability. Such substances are often found among persistent organic pollutants (POPs), such as certain organochlorine pesticides.

Possible exposure scenarios include:

- **Drinking water** : ingestion of contaminants present in water used for drinking or cooking;

- **Swimming**: accidental ingestion of contaminants present in surface waters;
- **Accidental soil ingestion**: ingestion of contaminants contained in dust and soils;
- **Consumption of crops**: exposure to contaminated foods (e.g., fruits and vegetables grown in home gardens using contaminated soil, groundwater, or irrigation water);
- **Consumption of dairy products and meat**: exposure to contaminated food products (e.g., livestock contaminated through drinking water, feed derived from contaminated crops, or exposure to polluted air and soils);
- **Seafood consumption**: exposure to contaminated products (e.g., fish and shellfish from polluted waters or exposed to contaminated sediments, leading to bioaccumulation of toxic substances in edible tissues).

1.4. Sources and levels of exposure

1.4.1. Key elements of exposure

Exposure is defined as the interaction between a chemical substance and a receptor. This interaction is the key element required for a toxic effect to occur.

For this interaction to take place, the chemical must reach the receptor. It must be released from a source, pass through one or more environmental media, and, in many cases, be absorbed by the organism or target system in order to interact with the receptor before a hazard can result in an adverse effect. This concept is illustrated in **Figure 1.1**.

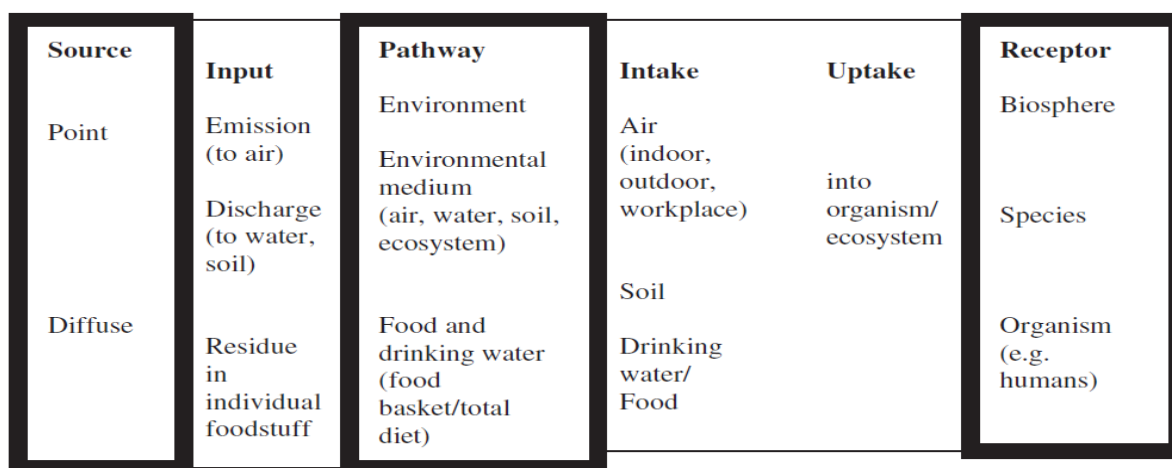


Figure 1.1. Transfer of a chemical from the source to the receptor (idealized model) (Duffus and Worth, 2006).

Although this concept may appear relatively simple, the complexity of these systems becomes evident in **Figure 1.2**, where the target organism is the human being. Similarly complex pathways can be developed for other targets.

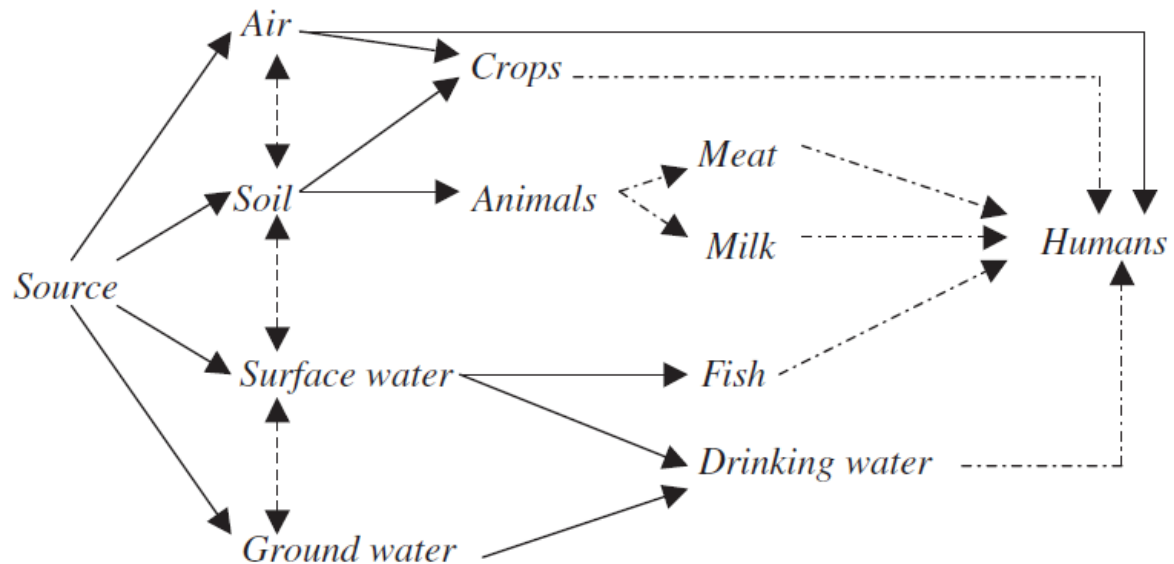


Figure 1.2. Schematic representation of human exposure pathways via the environment (Duffus and Worth, 2006). Environmental pathways: transfer between environmental compartments; uptake through the food chain.

1.4.2 Exposure pathways

Pollutants enter the environment through two main types of sources: **point sources** and **non-point (diffuse) sources**.

1.4.2.1. Point sources

Are fixed entry points of contaminants ; they are localized and measurable, and therefore easier to control. Point sources may include:

- Industrial, municipal, or wastewater treatment plant discharges (effluents);
- Chemical or oil spills;
- Leachates from landfills and disposal sites;
- Industrial stacks and other stationary atmospheric emissions;
- Industrial wastewater;
- Domestic wastewater.

1.4.2.2. Non-point (diffuse) sources

Involve widespread inputs without a single identifiable point of entry. They do not originate from fixed discharge points and are therefore more difficult to control. Examples of diffuse sources include:

- Agricultural runoff (pesticides, fertilizers);
- Vehicle emissions (mobile sources);
- Atmospheric deposition;
- Desorption or leaching from large contaminated areas (polluted sediments, mining residues);
- Groundwater infiltration.

1.4.3 Sources of human exposure

Potential sources of human exposure are illustrated in [Figure 1.3](#).

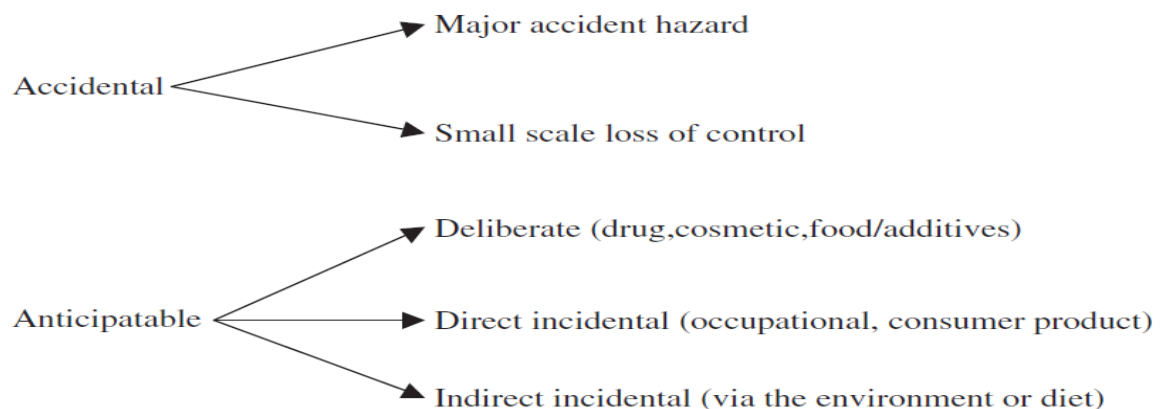


Figure 1.3. Potential exposure scenarios to be considered in human health risk assessment (Duffus and Worth, 2006).

1.4.3.1 Dietary exposure

Exposure to chemicals through diet is a key component of human exposure. Certain chemicals are:

- **Intentionally applied to crops and animals** and may leave residues on or in food. This is typically the case for pesticide residues found on fruits, vegetables, and cereals;
- **Derived from environmental contamination (air, soil)**, such as bacteria (e.g., *Salmonella*, *Listeria*) and toxins (e.g., aflatoxins), or from food packaging materials such as bisphenol A and phthalates;

- **Food additives**, including colorants, preservatives, and sweeteners;
- **Heavy metals**, such as mercury in fish, cadmium in cereals, and lead in water.

In addition to dietary exposure, humans may also be exposed through consumer products such as cosmetics and personal care products containing parabens and phthalates, household products such as detergents, disinfectants, and solvents, as well as building materials such as asbestos, formaldehyde, and lead-based paints. All these substances are considered potential contaminants.

1.4.3.2. Occupational exposure

Workers handling chemical products are mainly exposed through inhalation (dusts, fumes) and dermal contact with chemicals (asbestos, silica, solvents). They may also be exposed to noise (a nuisance factor) following prolonged exposure to high sound levels.

Occupational exposure assessments are based on environmental or biological measurements (blood, urine, exhaled air).

1.4.3.3. Environmental sources of exposure

a) Air pollution

Given that the primary route of exposure to air pollutants is respiratory, toxicologists classify them as:

- Gases
- Particulate matter

Two types of atmospheric pollutants are distinguished:

- **Primary pollutants** : directly released into the atmosphere as a result of natural processes or anthropogenic activities.
- **Secondary pollutants** : formed when primary pollutants undergo chemical reactions with other atmospheric components.

The main gaseous pollutants include:

- Carbon oxides (carbon monoxide and carbon dioxide)
- Sulfur oxides
- Nitrogen oxides
- Ozone

- Volatile hydrocarbons (benzene, methane, and a specific class of halogenated molecules known as CFCs)

The main particulate pollutants include:

- Dust
- Pollen
- Heavy metals

Most of these pollutants originate from combustion processes, i.e., the burning of organic materials.

In more developed countries, fossil fuels such as oil, gas, and coal are burned to produce energy and heat, whereas in less developed countries, wood and other agricultural residues are used as fuel.

In addition, the burning of forests and grasslands also contributes to the release of pollutants into the atmosphere.

b) Water pollution

Water, regardless of its source, is vulnerable to pollution. Water pollution is a complex issue due to the large number of sources and types of aquatic pollutants. Moreover, these pollutants may interact with each other or with water itself, leading to chemical transformations.

Thus, the toxicity of pollutants in aquatic environments depends on several factors (e.g., water temperature, hardness, and pH) and can be difficult to predict. Nevertheless, the protection of water resources is essential for both ecological and human health.

Water pollutants are generally classified into several major categories:

- **Organic substances** : include dead or decomposing plant and animal matter, as well as organic wastes such as petroleum products, solvents, pesticides, and polymers.
- **Inorganic substances** : include metals, nitrates, and phosphates.
- **Biological agents** : include viruses, bacteria, protozoa, and other disease-causing parasites.
- **Suspended matter** : insoluble solid particles such as soil and rock materials.
- **Radioactive substances and heat** : each constitutes a separate category.

Water pollutants may originate from industrial, municipal, or agricultural sources.

- **Industrial sources** : generally point sources, meaning pollutants are discharged at well-defined locations (e.g., an effluent pipe).
- **Municipal sources** : some, such as wastewater treatment plant discharges, are also point sources.
- **Agricultural sources** : mainly diffuse sources, where pollutants are released over a wide area rather than a single point. A typical example is runoff containing fertilizers and pesticides from cultivated land.

Urban stormwater drainage systems are another example of diffuse pollution. Runoff waters transport pollutants associated with precipitation, often more mobile than agricultural chemicals, which may represent an even higher risk.

Most water pollution originates from diffuse sources, which are unfortunately the most difficult to control.

1.4.4. Exposure levels

The exposure level refers to the amount of an agent to which an individual is exposed. It is generally expressed as the concentration in the environment (air, water, food) or as the dose absorbed by the organism. This level depends on several factors, including proximity to the source, duration and frequency of exposure, as well as environmental conditions.

Several factors influence exposure levels. The dose reaching the target organ may increase due to :

- High pollutant concentrations
- Prolonged exposure duration
- Behavioral factors (level of physical activity, time spent in different environments)

Risk analysts refer to these latter elements as risk factors. A risk factor must generally meet two conditions :

- Exposure to the risk factor precedes the onset of the adverse effect.
- The risk factor and the adverse effect are consistently associated, meaning that the adverse effect is generally not observed in the absence of this factor.

Examples of different exposure levels :

- Outdoor concentrations of particulate matter are generally higher outdoors than indoors, except for ultrafine particles, where the difference is minimal.

- Nitrogen dioxide is often more concentrated indoors, particularly when gas is used for heating or cooking.
- Individuals working or playing outdoors are more exposed to particulate matter, whereas those staying indoors are more exposed to nitrogen dioxide.

Physical exercise also influences the dose by increasing:

- The volume of air inhaled per minute
- Mouth breathing, thereby reducing the efficiency of the nasal filter

However, rapid breathing decreases the deposition of ultrafine particles due to insufficient time for sedimentation and diffusion.

Exposure may also be aggravated by :

- Smoking
- Occupational exposure
- Co-exposure to other pollutants or viral infections

Thus, two individuals exposed to the same source may present different exposure levels depending on their lifestyle habits, occupational activity, or environmental conditions. Taking into account both sources and exposure levels is essential for an accurate assessment of health risks.

1.5. Biological effects of exposure

Adverse effects on human health are often difficult to identify and assess. Even when an adverse effect is confirmed, it remains challenging to determine the environmental factors responsible.

1.5.1. What is a biological effect of exposure?

An effect is any biochemical, cellular, or physiological alteration resulting from exposure to a toxic agent.

It refers to the immediate or delayed biological response of the organism following exposure to a toxic substance. This may include irritation, inflammation, oxidative stress, genetic mutation, and others. These effects may be reversible or irreversible.

1.5.2. Types of biological effects

The categories of biological effects of exposure commonly encountered in public health risk assessment are identified and compared below:

1.5.2.1. Acute effect vs. chronic effect (immediate vs. delayed effect)

An acute effect involves the sudden onset of symptoms that last for a short period, generally less than 24 hours (e.g., intoxication, irritation, allergies, burns), whereas a chronic effect leads to long-lasting and persistent symptoms (e.g., oxidative stress, inflammation, genetic mutations, endocrine disruption).

In general, the cellular damage responsible for acute toxicity symptoms is reversible, whereas chronic toxicity often leads to permanent effects due to irreversible cellular alterations. Indeed, when cellular destruction and the resulting loss of function are severe, organism death may occur.

1.5.2.2. Local effect vs. systemic effect

A local effect occurs when symptoms related to exposure to a toxic substance are confined to the initial site of exposure. This is the case for exposure to respiratory irritants such as sulfur dioxide, or skin contact with corrosive substances such as strong acids and bases causing chemical burns.

A systemic effect occurs when adverse effects appear at sites distant from the initial exposure point, such as the nephrotoxic effect of cadmium resulting from the ingestion of contaminated food or inhalation of particles containing this metal.

In this case, toxicity occurs after absorption and distribution of the toxicant from its entry point to a distant target site (target organ). Toxic substances are often absorbed at a specific location and then transported through the body via the blood or lymphatic system to other target tissues.

In general, it is easier to attribute local toxicity to a direct contact between the biological system and the toxic agent compared with systemic toxicity.

1.5.3. General nature of the health effects of exposure to chemical substances in humans

Exposure to certain chemicals present in the human environment can lead to various health effects. The following are the main categories of health effects ([Table 1.1](#)) that may be expected following exposure to chemical substances commonly encountered in modern societies:

Table 1.1. Some typical health effects resulting from chemical exposures: a listing for selected toxic chemicals in the human environments ([Asante-Duah, 2002](#)).

Chemical	Typical health effects/symptoms and toxic manifestations/responses
Arsenic and compounds	Acute hepatocellular injury, anemia, angiosarcoma, cirrhosis, developmental disabilities, embryotoxicity, heart disease, hyperpigmentation, peripheral neuropathies
Antimony	Heart disease
Asbestos	Asbestosis (scarring of lung tissue)/fibrosis (lung and respiratory tract)/lung cancer, mesothelioma, emphysema, irritations, pneumonia/pneumoconioses
Benzene	Aplastic anemia, CNS depression, embryotoxicity, leukemia and lymphoma, skin irritant
Beryllium	Granuloma (lungs and respiratory tract)
Cadmium	Developmental disabilities, kidney damage, neoplasia (lung and respiratory tract), neonatal death/fetal death, pulmonary edema
Carbon tetrachloride	Narcosis, hepatitis, renal damage, liver tumors
Chromium and compounds	Asthma, cholestasis (of liver), neoplasia (lung and respiratory tract), skin irritant
Copper	Gastrointestinal irritant, liver damage
Cyanide	Asthma, asphyxiation, hypersensitivity, pneumonitis, skin irritant
Dichlorodiphenyl trichloroethane (DDT)	Ataxic gait, convulsions, human infertility/reproductive effects, kidney damage, neurotoxicity, peripheral neuropathies, tremors
Dieldrin	Convulsions, kidney damage, tremors
Dimethylfumarate (DMFu)	Dermatological symptoms/effects (contact dermatitis)— <i>viz.</i> , skin irritation and skin sensitization, cutaneous allergic reactions; possible respiratory allergic symptoms or diseases
Dioxins and furans (PCDDs/PCDFs)	Hepatitis, neoplasia, spontaneous abortion/fetal death; bioaccumulative
Formaldehyde	Allergic reactions; gastrointestinal upsets; tissue irritation
Lead and compounds	Anemia, bone marrow suppression, CNS symptoms, convulsions, embryotoxicity, neoplasia, neuropathies, kidney damage, seizures; biomagnifies in food chain
Lindane	Convulsions, coma and death, disorientation, headache, nausea and vomiting, neurotoxicity, paresthesias
Lithium	Gastroenteritis, hyperpyrexia, nephrogenic diabetes, Parkinson's disease
Manganese	Bronchitis, cirrhosis (liver), influenza (metal-fume fever), pneumonia, neurotoxicity
Mercury and compounds	Ataxic gait, contact allergen, CNS symptoms; developmental disabilities, neurasthenia, kidney and liver damage, Minamata disease; biomagnification of methyl mercury
Methylene chloride	Anesthesia, respiratory distress, death
Naphthalene	Anemia
Nickel and compounds	Asthma, CNS effects, gastrointestinal effects, headache, neoplasia (lung and respiratory tract)
Nitrate	Methemoglobinemia (in infants)

Chemical	Typical health effects/symptoms and toxic manifestations/responses
Organo-chlorine pesticides	Hepatic necrosis, hypertrophy of endoplasmic reticulum, mild fatty metamorphosis
Pentachlorophenol (PCP)	Malignant hyperthermia
Phenol	Asthma, skin irritant
Polychlorinated biphenyls (PCBs)	Embryotoxicity/infertility/fetal death, dermatoses/chloracne, hepatic necrosis, hepatitis, immune suppression, endocrine effects, neurologic effects, cardiovascular effects, musculoskeletal issues, gastrointestinal systems effects
Silver	Blindness, skin lesions, pneumoconiosis
Toluene	Acute renal failure, ataxic gait, neurotoxicity/CNS depression, memory impairment
Trichloroethylene (TCE)	CNS depression, deafness, liver damage, paralysis, respiratory and cardiac arrest, visual effects
Vinyl chloride	Leukemia and lymphoma, neoplasia, spontaneous abortion/fetal death, tumors, death
Xylene	CNS depression, memory impairment
Zinc	Corneal ulceration, esophagus damage, pulmonary edema

1.6. Health consequences of exposure

Exposure to toxic substances can have profound and diverse consequences on human health. These consequences are not limited to immediate biological responses but also extend to chronic diseases, developmental disorders, and teratogenic effects. The extent of these consequences depends on several factors, including the nature of the toxicant, the duration and intensity of exposure, and individual susceptibility.

1.6.1. Specific diseases and health disorders related to toxic substances

Certain chemical exposures are directly associated with well-defined diseases. These pathologies may affect different body systems depending on the type of toxicant involved.

1.6.1.1. Impact on the nervous system

Heavy metals, particularly mercury and lead, are well known for their neurotoxic effects. In children, chronic exposure to lead can result in reduced intelligence quotient (IQ), attention disorders, and cognitive developmental delays. Mercury, especially in its methylated form (methylmercury), accumulates in the brain and may cause tremors, loss of motor coordination, and in severe cases, Minamata disease, observed in Japan in populations exposed through the consumption of contaminated fish.

1.6.1.2. Respiratory toxicity and pulmonary diseases

Fine particulate matter and polycyclic aromatic hydrocarbons (PAHs), present in urban and industrial pollution, are strongly associated with chronic respiratory diseases such as chronic obstructive pulmonary disease (COPD) and asthma. Prolonged exposure to asbestos is another example of pulmonary toxicity, leading to the development of mesothelioma, a rare and aggressive cancer affecting the pleura.

1.6.1.3. Cardiovascular risks

Certain toxic substances increase the risk of cardiovascular diseases. Air pollutants such as nitrogen dioxide (NO₂) and volatile organic compounds (VOCs) promote systemic inflammation and atherosclerosis, thereby increasing the risk of stroke and myocardial infarction.

1.6.2. Endocrine disruption and reproductive alterations

1.6.2.1. Endocrine disruptors and hormonal imbalance

Endocrine disruptors (bisphenol A, phthalates, dioxins) interfere with the body's natural hormones. These substances may mimic or block hormone action, leading to metabolic disorders, obesity, type 2 diabetes, and thyroid dysfunction.

1.6.2.2. Effects on fertility and pregnancy

Exposure to organochlorine pesticides or heavy metals can reduce fertility by impairing spermatogenesis in men and disrupting the ovarian cycle in women. Moreover, in utero exposure to certain toxicants (e.g., lead, dioxins) is associated with miscarriages, preterm births, and congenital abnormalities.

1.6.3. Impact on quality of life and overall health

1.6.3.1. Reduction in life expectancy

Prolonged exposure to toxic substances has a direct impact on longevity. Exposure to air pollutants reduces life expectancy by several years in highly polluted areas.

1.6.3.2. Psychological and cognitive effects

Toxic substances not only cause physical diseases but also psychological and cognitive disorders. For example, organic solvents (e.g., toluene, benzene) are associated with mood disorders, chronic fatigue, and reduced concentration abilities.

1.6.4. Transgenerational effects and risks for future generations

Some toxic substances can induce epigenetic modifications that are transmissible to subsequent generations. Studies have shown that exposure of pregnant women to endocrine disruptors may affect fetal neurological and immune development, increasing the risk of chronic diseases in adulthood.

1.7. The exposure–dose–effect continuum

The exposure -dose- effect continuum represents the pathway of an agent from its source to the occurrence of an effect. The agent may be transported and transformed in the environment through air, water, soil, dust, or food. Individuals may come into contact with this agent via inhalation, ingestion, or dermal and ocular contact.

An individual's physiology, behavior, and activity patterns, as well as the concentration of the agent, determine the magnitude, frequency, and duration of exposure. Once the agent crosses the absorption barrier (i.e., the skin, lungs, eyes, gastrointestinal tract, or placenta), exposure becomes an absorbed dose.

Interactions of the chemical substance or its metabolites with a target tissue may then lead to adverse health effects. The text under the different stages of the diagram indicates the specific information required to characterize each phase of the exposure–dose–effect continuum ([Figure 1.4](#)).

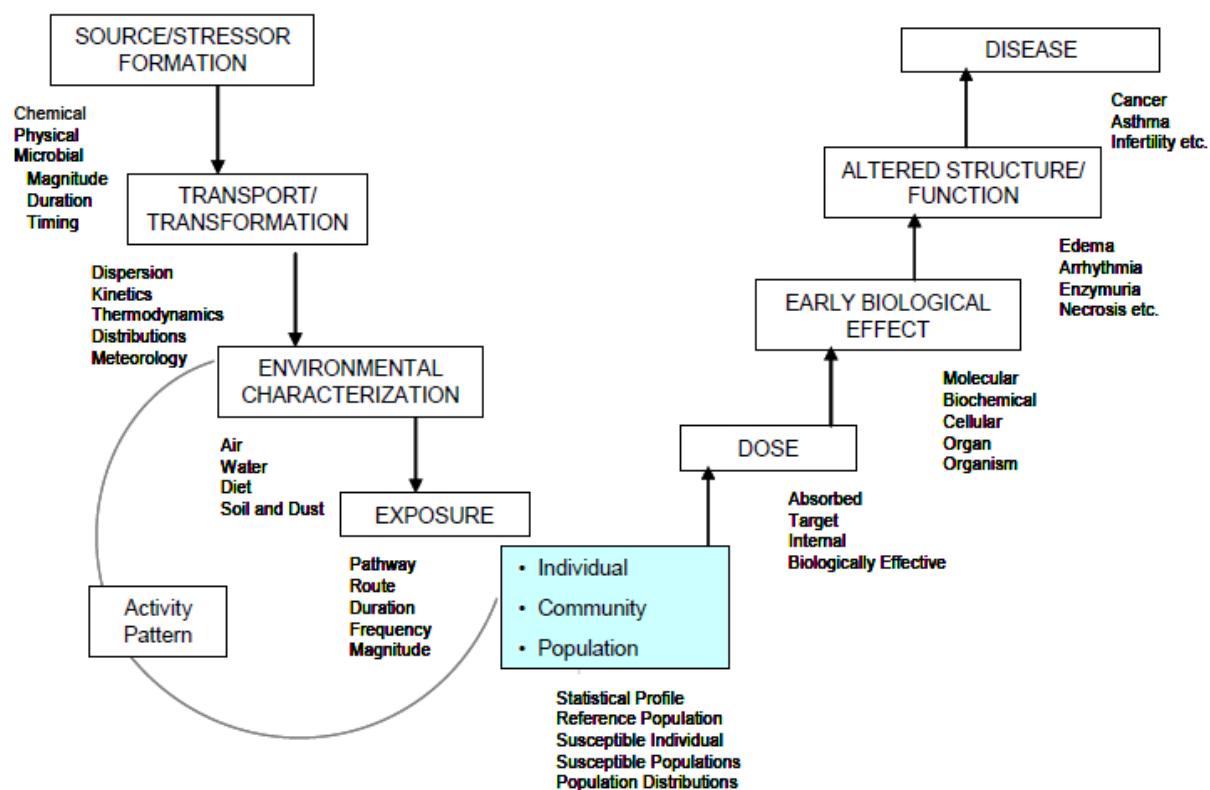


Figure 1.4. Exposure–Dose–Effect Continuum (Adapted from U.S. EPA, 2003c; WHO, 2006; Ott, 2007).

2. Environmental risks

2.1. Health and environment: assessment of health risks related to environmental situations and environmental degradation

2.1.1. General definitions

- **Environment**

The environment encompasses all external physical, chemical, biological, and social factors that surround and influence living organisms and human populations.

- **Health**

According to the World Health Organization ([WHO, 1948](#)), health is defined as a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity.

- **Environmental health**

Environmental health is a multidisciplinary field that examines the interactions between humans and their environment. It focuses on identifying, assessing, and controlling environmental factors that may adversely affect human health and quality of life, both for present and future generations.

There are numerous concerns related to environmental health, as illustrated in [Figure 2.1](#).

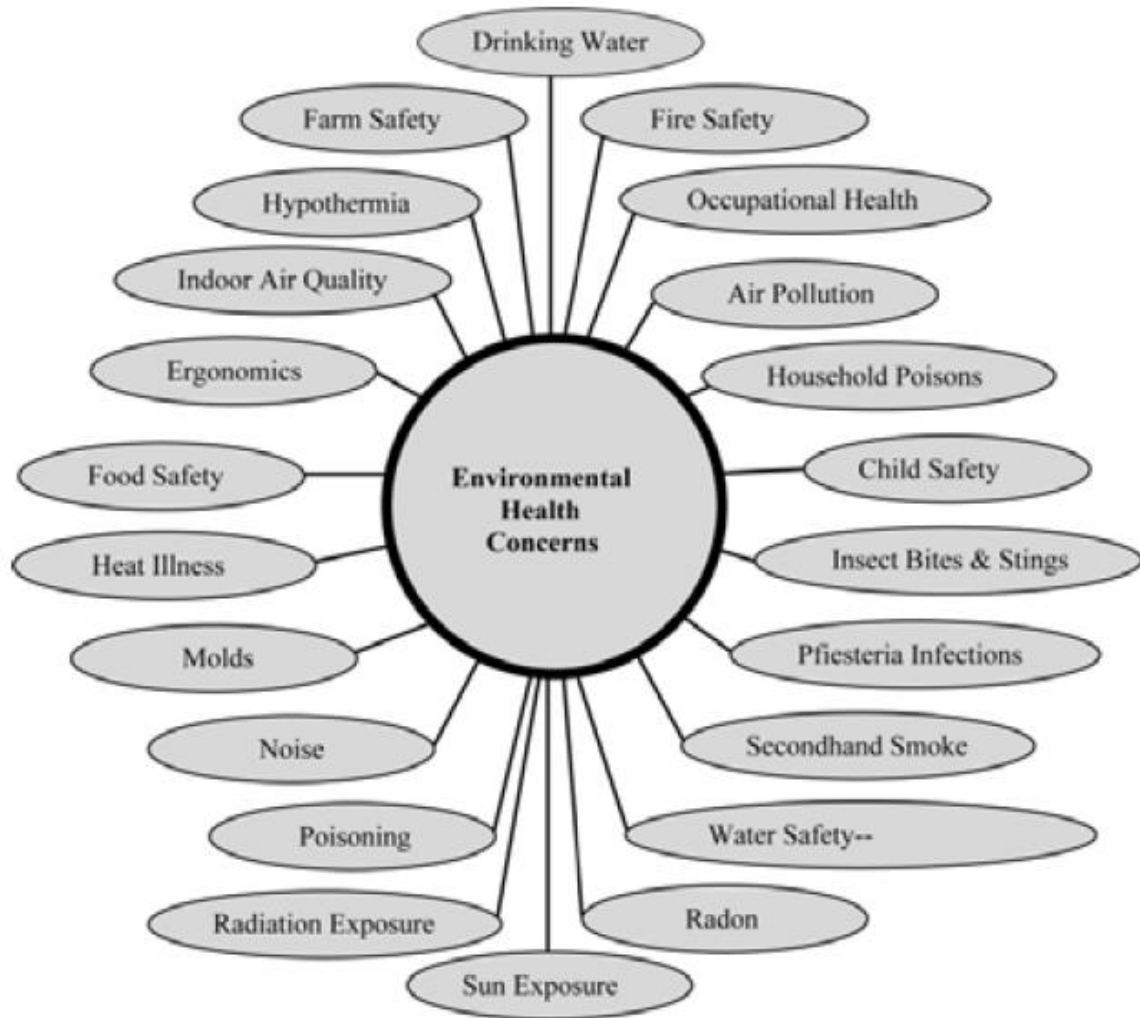


Figure 2.1. Environmental health concerns (Spellman and Stoudt, 2013).

2.1.2. Environmental health risk assessment

Environmental health risk assessment is a systematic process used to identify, analyze, and quantify the potential adverse effects of environmental hazards on human health.

It aims to organize scientific data in order to :

- Identify hazardous situations,
- Estimate the likelihood and severity of adverse effects,
- Support decision-making and risk management strategies.

In modern societies, populations are continuously exposed to two main categories of risks:

- Risks related to chemical substances from consumer products,

- Risks related to environmental contaminants present in air, water, and soil.

Risk assessment is therefore a fundamental tool in public health, providing a scientific basis for prioritizing interventions, developing regulations, and protecting exposed populations.

2.1.3. Examples of environmental degradation situations

Environmental degradation is associated with various situations that may significantly impact ecosystems and human health. The most common include:

- **Industrial pollution** : release of heavy metals, pesticides, hydrocarbons, and microplastics (e.g., Minamata mercury contamination).
- **Air pollution** : increased respiratory diseases due to particulate matter and toxic gases.
- **Water contamination** : pollution of groundwater by nitrates, pharmaceuticals, and hydrocarbons.
- **Deforestation and climate change** : biodiversity loss and increased frequency of extreme weather events.
- **Waste pollution** : accumulation of plastic and hazardous waste affecting terrestrial and marine ecosystems.
- **Agricultural contamination** : use of pesticides leading to soil and food contamination.

2.1.4. Case study: Dublin air pollution control

In 1990, the city of Dublin implemented a ban on the sale of bituminous coal, which was identified as a major source of urban air pollution, particularly black smoke and sulfur dioxide.

Following this intervention, air quality and public health indicators were closely monitored. The results showed a substantial improvement in environmental conditions, including a significant reduction in concentrations of particulate matter and sulfur dioxide.

More importantly, this policy led to measurable health benefits. A marked decrease in respiratory and cardiovascular mortality was observed, with studies reporting reductions of approximately:

- **5–6% in total mortality,**
- **10–15% in respiratory mortality,**
- **And a notable decline in cardiovascular-related deaths.**

This case study clearly demonstrates the direct relationship between environmental exposure and health outcomes. It also highlights the effectiveness of regulatory interventions in reducing population exposure and improving public health.

2.1.5. Use of risk analysis in environmental assessment

The integration of risk analysis into environmental impact assessment is based on three main contributions :

1. **Prioritization of impacts according to probability and severity :**
Risk analysis enables the comparison of rare but severe events with more frequent but less severe impacts, supporting objective risk prioritization.
2. **Incorporation of long-term uncertainties :** It allows the integration of uncertainties related to delayed or poorly characterized environmental and health effects, particularly over long time horizons.
3. **Shift toward risk-based regulatory frameworks :** Modern regulatory systems, particularly in the United States and several international contexts, are increasingly adopting risk-based approaches rather than purely consequence-based standards.

2.1.6. Steps in environmental risk assessment

Environmental risk assessment is structured into four interrelated steps ([Figure 2.2](#)) :

a) Release (source) assessment

This step identifies and quantifies potential sources that may release hazardous agents into the environment, including:

- Type, quantity, frequency, and probability of releases ;
- Variability under normal and accidental conditions.

b) Exposure assessment

This step characterizes the contact between populations or ecosystems and hazardous agents, including:

- Intensity, duration, and frequency of exposure;
- Exposure pathways (inhalation, ingestion, dermal contact);
- Characteristics of exposed populations or systems;
- Factors influencing contaminant transport and accessibility.

c) Effects (consequence) assessment

This stage establishes the relationship between exposure and resulting health and environmental effects:

- Human health outcomes (mortality, morbidity, injury);
- Ecological impacts (ecosystem degradation, biodiversity loss).

d) Risk characterization

This step integrates the previous components to produce an overall risk estimate:

- Quantitative estimation of impacts on populations and the environment;
- Uncertainty analysis (confidence intervals, probability distributions);
- Synthesis of risk scenarios over time.

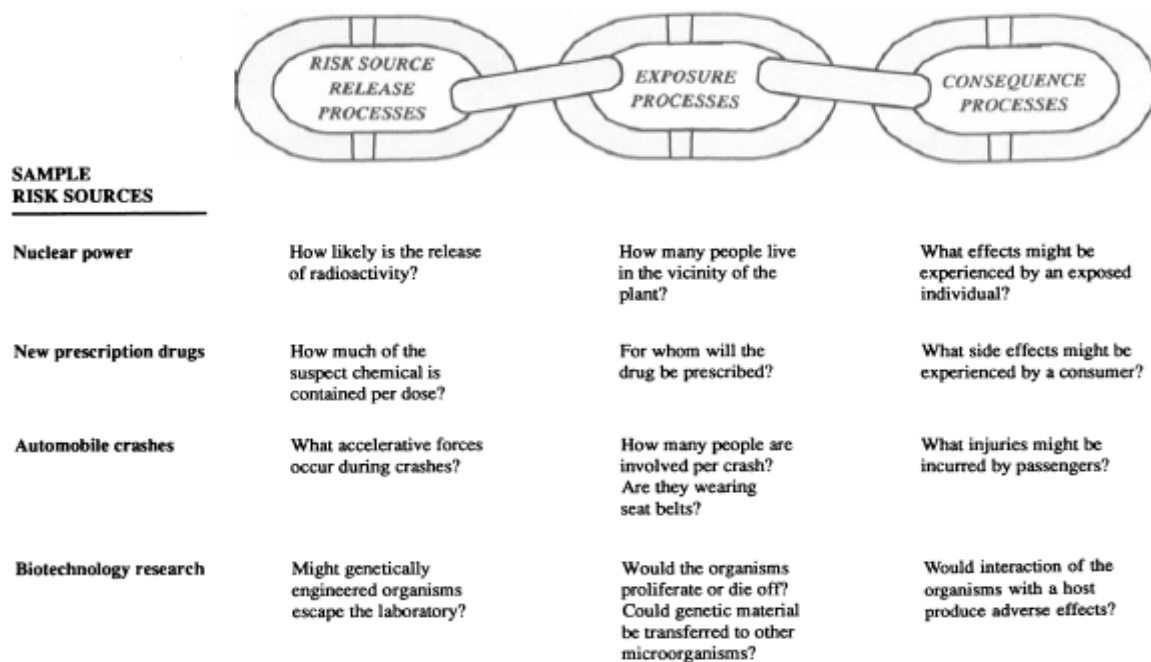


Figure 2.2. The risk assessment chain and key questions in environmental risk analysis (Covello and Merkhofer, 1993).

2.1.7. Application example: nuclear power plant risk assessment

Risk assessment is widely used to estimate the probability and consequences of potential accidents in nuclear power plants. It involves identifying accident scenarios, analyzing technical failures, modeling radioactive releases, assessing exposure pathways, and quantifying health and environmental consequences.

This approach supports comparison between scenarios, evaluation of safety levels, and informed decision-making in risk management (**Figure 2.3**).

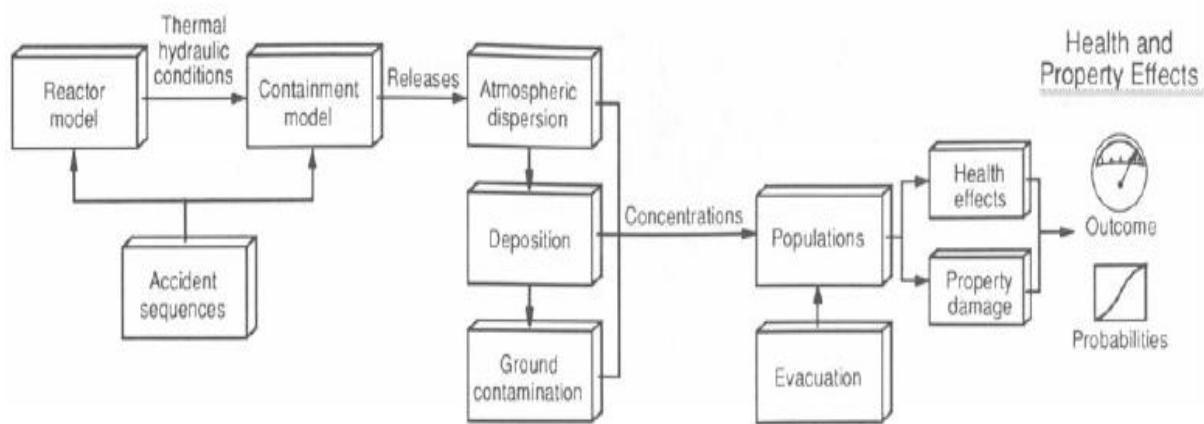


Figure 2.3. Main components of a risk assessment model for a nuclear power plant (**Covello and Merkhofer, 1993**).

2.2. Environmental monitoring : sampling strategies for environmental compartments (air, water, soil) and regulatory frameworks

2.2.1. Types of environmental monitoring

Environmental monitoring of environmental compartments is generally conducted through two main approaches:

- **Ambient environmental monitoring**

This approach involves measuring air or water quality downstream of a pollution source (e.g., in the direction of prevailing winds or water flow).

It is primarily used to assess the overall state of the environment.

- **Emission point monitoring ("End-of-Pipe")**

This method measures the levels of effluents discharged from outlets such as industrial stacks or drainage systems.

Its main objective is to verify compliance with regulatory standards and legislative requirements.

2.2.2 Sampling strategies

The primary objective in designing a sampling plan is to obtain a sample that accurately reflects the composition of the matrix under investigation, in accordance with the study objectives—in other words, a **representative sample**.

Sampling strategies are generally classified into three main categories:

2.2.2.1. Random sampling

From a statistical standpoint, random sampling represents the only truly rigorous approach. In a random sampling design, sampling locations within the Area of Interest (AOI) are determined using methods that eliminate selection bias.

For example, a random number generator may be used to define latitude and longitude coordinates within the boundaries of the study area ([Figure 2.4](#)).

(a) Random sampling scheme

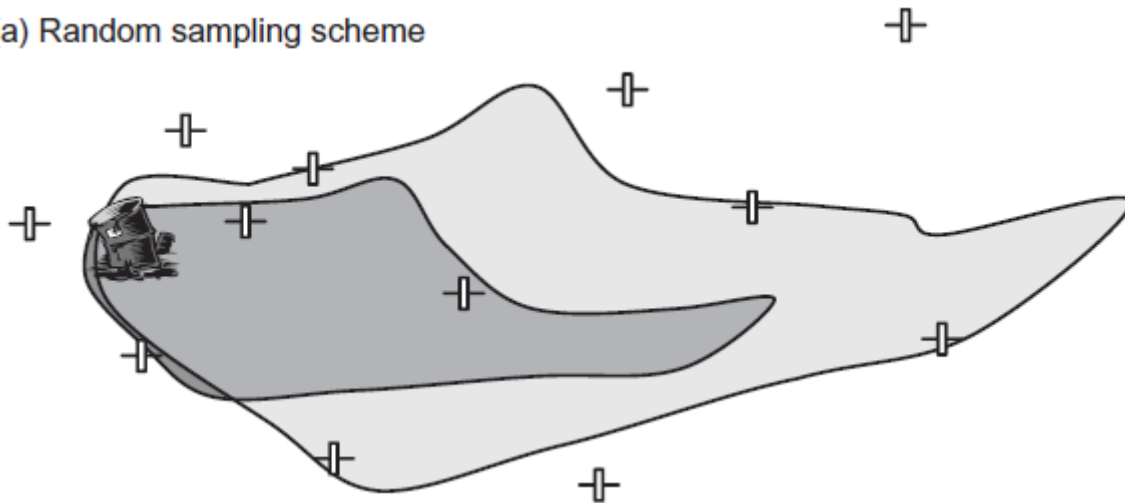


Figure 2.4. Diagram of random sampling ([Hodgson, 2011](#)).

This type of sampling design is particularly useful for establishing baseline conditions at a given site. When properly designed, with a sufficient number of samples, random sampling provides strong statistical power, thereby increasing confidence in conclusions regarding the overall state of the site.

However, this approach presents several limitations.

- First, achieving high statistical precision often requires a large number of samples, which may impose budgetary constraints due to the high costs associated with environmental sampling and analysis.

- Second, in heterogeneous areas, some sampling points may fall in inaccessible or inappropriate locations (e.g., on infrastructure).
- Finally, this method may result in uneven sample distribution, leading to undersampled areas (“gaps”) and oversampled zones.

2.2.2.2 Systematic sampling

In this approach, the area of interest is divided into a grid, and one sample is collected from each cell of the grid (**Figure 2.5**). This type of sampling design is useful when uniform coverage of the study area is required.

(b) Systematic sampling scheme

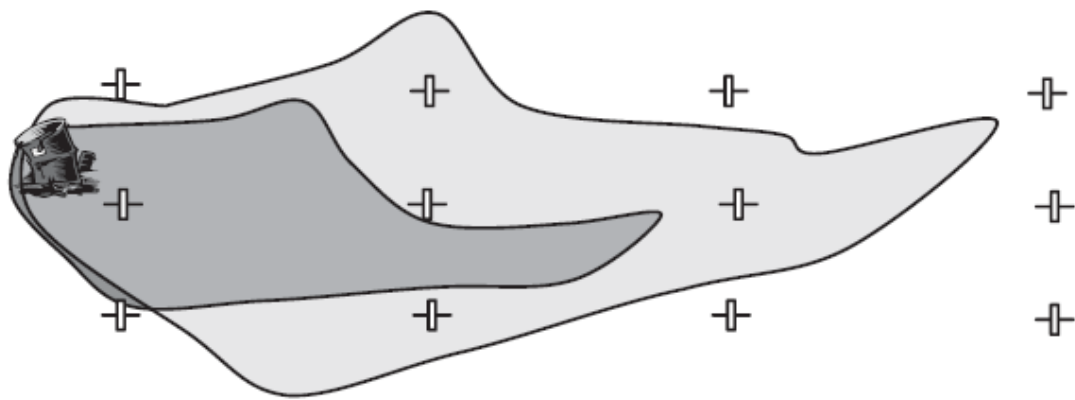


Figure 2.5. Diagram of systematic sampling (**Hodgson, 2011**).

Advantages :

- Helps avoid the “gaps” commonly observed in random sampling.
- Provides uniform coverage of the study site.

Disadvantages :

- Reduced ability to perform certain statistical analyses, as sampling locations are not independent and randomly distributed.

2.2.2.3. Judgmental sampling

In many cases of environmental contamination, the source of pollution is already known. The primary objective is therefore to determine the nature and extent of contamination (i.e., to identify the contaminants and assess their spatial distribution). For this reason, this approach is one of the most commonly used in environmental toxicology and exposure assessment.

In this type of design, sampling is structured to maximize the number of samples collected near the contamination source. Sampling points are then progressively distributed away from the source along transects, with increasing distances between samples as the distance from the source increases ([Figure 2.6](#)).

(c) Judgmental sampling scheme

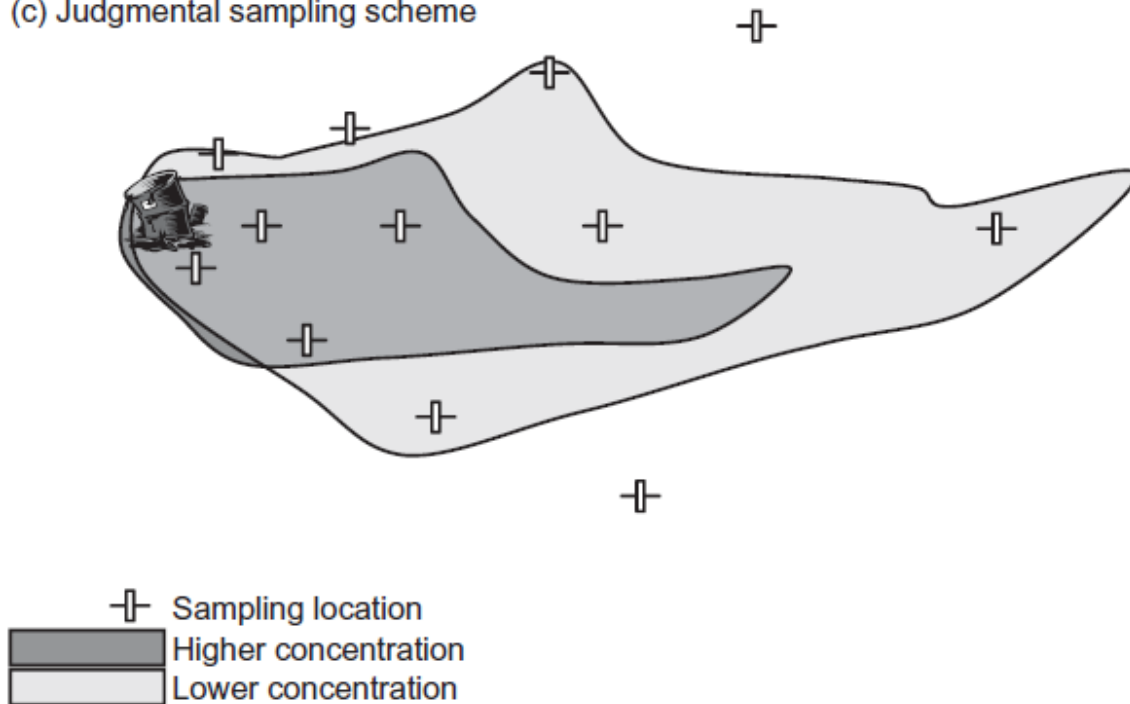


Figure 2.6. Diagram of judgmental sampling ([Hodgson, 2011](#)).

Advantages :

- Provides detailed information on localized contamination.
- Well suited for environmental impact studies and risk assessments.

Disadvantages :

- May overlook secondary sources of contamination that were not initially identified, as sampling is focused on the primary source.

The advantages and disadvantages of each sampling strategy are summarized in [Table 2.1](#):

Table 2.1. Different sampling strategies (Hodgson, 2011).

Sampling Type	Advantages	Disadvantages	Typical Use
Random	Statistical objectivity; no selection bias.	High cost; some areas may be inaccessible for sampling.	Establishing general baseline conditions.
Systematic	Good spatial coverage; avoids “gaps.”	Limits certain statistical analyses.	Large-scale pollution studies.
Judgmental	Focuses on known contamination; efficient for targeted investigations.	May overlook secondary contamination sources.	Environmental impact studies; industrial pollution assessment.

2.2.3. Sampling of different environmental matrices

The main environmental matrices include water, air, solids (soils, sediments), biota, and vegetation. Except for air, these matrices exhibit complex compositions requiring specific strategies to ensure sample representativeness.

2.2.3.1. Water

Water is one of the most frequently analyzed matrices. Representativeness depends on the target contaminant, pollution sources (agricultural, industrial, municipal, or accidental), and environmental factors such as flow rate, temperature, thermal stratification, salinity, and sediment composition.

Sampling may be performed using grab sampling or specialized devices for depth collection. Groundwater is collected through wells or piezometers, requiring careful planning.

Temporal variability, particularly seasonal fluctuations, has led to increased use of passive sampling devices (PSDs). These accumulate contaminants over time, enabling estimation of average concentrations, detection of trace compounds, and assessment of bioavailability of dissolved contaminants.

2.2.3.2. Air

Atmospheric pollutants originate from anthropogenic sources (combustion, industrial processes, waste disposal) and natural sources (wildfires, dust, volcanic emissions). Their impact depends on their ability to remain airborne and be inhaled.

They include liquid aerosols (mists, fogs) and solid particles (dust, fumes, soot). Air samplers use filters, adsorbents, pumps, and flow meters. Portable devices are used to assess occupational exposure.

Sampling methods include glass fiber filters, membrane filters, liquid impingers, and polyurethane foam (PUF), increasingly preferred due to analytical advantages.

2.2.3.3. Soil

Pollutants interact with soil components (organic and inorganic), liquid and gaseous phases, and living organisms. Depending on their properties, they may remain localized, migrate, or be taken up by plants.

Soil sampling requires a rigorous statistical approach considering site characteristics and spatial variability. Sampling areas are divided into homogeneous zones, and the number of samples depends on the desired precision.

Tools include manual or mechanical corers, rotary devices, and large-diameter cylinders for depth-specific sampling.

2.2.3.4. Biota

Environmental monitoring often includes the study of living organisms (biota), particularly in aquatic environments. Three main sampling approaches are commonly employed:

1. **Non-lethal sampling** : Collection of hair or feathers for the analysis of mercury and other heavy metals.
2. **Collection of wild organisms** : The *Mussel Watch Program* (NOAA) involves the sampling of mussels and sediments at more than 300 coastal sites across the United States since 1986, for the analysis of over 100 pollutants.
 - **Advantage:** Direct assessment of contamination levels in organisms within their natural environment.
3. **Bioassays using caged organisms:**
 - “Clean” fish or mussels are placed in the study environment to accumulate site-specific contaminants.
 - **Advantage:** Reduction of biases related to spatial variability.

- **Limitation** : Survival rates may be low due to predation.

2.3. Legal framework for environmental monitoring

The monitoring of environmental compartments (air, water, and soil) is governed by a structured legal framework aimed at ensuring environmental quality and preventing risks to human health and ecosystems. This framework includes **national** and **international regulations, technical standards**, as well as **environmental directives** and **conventions**.

2.3.1. Objectives

The enforcement of environmental laws is based on an environmental management approach. Its objective is to remediate environmental damage rather than merely sanction specific regulatory violations.

All areas of law may be mobilized to prevent threats or damage to the environment, as well as to human health and safety.

Example : Fiscal laws, such as tax incentives, can be used as tools for environmental regulation.

Regulations impose :

- Strict thresholds for pollutants in water, air, and soil.
- Monitoring obligations for governments and industries.
- Transparency of environmental data to the public.

Table 2.2 summarizes the regulations and monitoring obligations for environmental media (water, air, soil).

Table 2.2. Regulations and monitoring requirements for environmental media (water, air, soil) (EEA, 2021; WHO, 2021).

Monitored Medium	Main Regulation	Obligations	Responsible Actors
Water	Water Framework Directive (2000/60/EC)	Monitoring of surface and groundwater	Water agencies, public laboratories
Air	Air Quality Directive (2008/50/EC)	Mandatory measurement of atmospheric pollutants	AASQA, EPA, accredited laboratories
Soil	Environmental Liability Directive (2004/35/EC)	Assessment and monitoring of contaminated sites	Consulting firms, local authorities

2.3.2. Types of regulation

a) **International regulations** : A set of rules and principles adopted at the global or regional level by international organizations (UN, EU, WHO, etc.). These often require transposition into national law to be implemented.

Examples : Paris Agreement (2015) : climate change mitigation ; Stockholm Convention (2001) : restriction of persistent organic pollutants; Water Framework Directive (2000/60/EC, EU).

b) **National regulations** : A body of laws, regulations, and decrees adopted by a state to manage and protect the environment at the national level. These laws are legally binding within the respective country.

Examples : France : Water and Aquatic Environments Law (LEMA, 2006); USA – Clean Water Act (1972); Canada : Canadian Environmental Protection Act (CEPA, 1999); Algeria : Law No. 05-12 (2005) on water resource protection and regulation of withdrawals.

c) **Technical standards** : Frameworks established by standardization bodies to harmonize environmental measurement, sampling, and analytical methods. They are not always legally binding but often serve as a basis for setting regulatory thresholds.

Examples : ISO 14001 : environmental management systems; ISO 10381 : soil sampling protocols; ISO 5667-11 : groundwater sampling.

d) **Environmental directives** : Legislative instruments adopted by supranational organizations (e.g., the European Union) that establish objectives to be achieved by Member States, which must transpose them into national law for implementation.

Example : Water Framework Directive (2000/60/EC) : establishes objectives for achieving good ecological status of surface and groundwater.

e) **Environmental conventions** : International treaties signed between states under the auspices of organizations such as the UN or WHO. They define principles and commitments to address global environmental issues.

Examples: Stockholm Convention (2001) : control of persistent organic pollutants (POPs); Paris Agreement (2015) : global commitment to reduce greenhouse gas emissions.

2.3.3. In Algeria

In Algeria, environmental regulation is based on a set of laws, decrees, and regulations governing natural resource management, environmental protection, and the prevention of pollution and nuisances.

This framework is inspired by international standards and climate commitments. However, challenges remain regarding the effective enforcement of laws and the strengthening of field-level monitoring and control.

2.4. Major natural risks

Major natural risks are phenomena of geological, climatic, or hydrological origin that have significant impacts on human populations and ecosystems.

Floods, droughts, and extreme storms have caused millions of deaths, affected many more people, and resulted in billions of dollars in economic losses. On average, natural disasters caused approximately 123,000 deaths per year worldwide between 1972 and 1996, with Africa experiencing the highest disaster-related mortality rate.

Table 2.3 presents the main types of natural risks along with representative case studies:

Table 2.3. Main Types of Natural Risks (Bilham et al., 2010; Lipman & Mullineaux, 1981; Hulme, 2001)

Risk Type	Definition	Examples
Earthquakes	Sudden release of energy in the Earth's crust due to tectonic fault rupture, generating seismic waves.	• Tōhoku earthquake, Japan (2011) – Magnitude 9.1, triggering a tsunami and the Fukushima nuclear disaster. • Turkey–Syria earthquakes (2023) – Magnitude 7.8, causing massive destruction and casualties.
Volcanic eruptions	Emission of magma, gases, and ash from a volcano, potentially causing destruction and climate effects.	• Mount St. Helens eruption, USA (1980) – Major explosion destroying ~600 km² of forest and causing 57 deaths.
Hurricanes and cyclones	Intense tropical storms with winds exceeding 120 km/h, leading to flooding and large-scale damage.	• Hurricanes Helene and Milton, USA (2024) – Over \$50 billion in damages.

Risk Type	Definition	Examples
Floods	Temporary submersion of land due to heavy rainfall, river overflow, or tsunamis; may increase waterborne diseases (e.g., cryptosporidiosis, giardiasis).	• Bangkok floods, Thailand (2011) – Affected 13.6 million people, ~\$46 billion in losses.
Droughts	Prolonged period of insufficient rainfall leading to water scarcity and agricultural crises.	• Sahel drought, Africa (1970–1980) – Severe famine, over 100,000 deaths.
Tsunamis	Series of large ocean waves caused by underwater earthquakes, volcanic eruptions, or landslides.	• Indian Ocean tsunami (2004) – Magnitude 9.1, waves up to 30 m, over 230,000 deaths. • Tōhoku tsunami, Japan (2011) – ~18,500 deaths and major economic losses.
Landslides	Rapid movement of soil and rocks down slopes, often triggered by heavy rainfall or earthquakes.	• Mocoa landslide, Colombia (2017) – Over 300 deaths following intense rainfall.
Wildfires (megafires)	Large-scale forest fires (>400 hectares), often linked to drought, rising temperatures, and ecological imbalances.	• Australian bushfires (2019–2020) – ~100,000 km ² burned, 33 deaths, over 3,000 homes destroyed.

2.5. Technological risks

Technological risks are associated with human activities involving technologies that may lead to major accidents. They include industrial, nuclear, chemical, biological incidents, and accidents related to the transport of hazardous materials. These events often have severe consequences for human health and the environment.

2.5.1. Nuclear accidents

- **Chernobyl (Ukraine, 1986)** : Reactor explosion releasing a radioactive cloud that spread across several European countries, exposing hundreds of thousands of people and leading to increased cancer cases.
- **Fukushima (Japan, 2011)** : Earthquake and tsunami caused reactor meltdowns, resulting in major radioactive contamination and large-scale population evacuation.
-

2.5.2. Chemical accidents

- **Seveso (Italy, 1976)** : Release of dioxin (TCDD) following an industrial explosion, causing poisoning, skin disorders, and liver damage.
- **Bhopal (India, 1984)** : Massive leak of toxic gas (methyl isocyanate) from a pesticide plant, resulting in thousands of deaths and long-term health effects in exposed populations.

2.5.3. Accidents in the transport of hazardous materials

- **AZF (France, 2001)** : Explosion of ammonium nitrate storage in Toulouse, causing numerous casualties and injuries.
- **Lac-Mégantic (Canada, 2013)** : Train derailment carrying crude oil leading to a major explosion, destruction of the town center, and significant loss of life.

2.6. Water related risks

Water is an essential resource but can also act as a vector for toxic substances and pathogens, posing significant risks to human health and the environment. These risks can be classified into microbiological, chemical, and environmental categories.

2.6.1. Microbiological risks : waterborne diseases

Water contaminated with pathogenic microorganisms is responsible for numerous diseases worldwide. Major agents include bacteria (*Escherichia coli*, *Vibrio cholerae*), viruses (*rotavirus*, *norovirus*, *hepatitis A*), and protozoa (*Giardia lamblia*, *Cryptosporidium*).

Examples :

- **Cholera in Bangladesh** : Regular outbreaks with tens of thousands of cases annually, highlighting the impact of unsafe water.
- **Cryptosporidium outbreak (USA, 1993)** : Contamination of drinking water in Milwaukee affected over 400,000 people, revealing limitations in filtration and disinfection systems.

2.6.2. Chemical risks : Contamination by toxic pollutants

Drinking water may contain chemical contaminants from industrial, agricultural, or treatment sources, posing long-term health risks.

Examples :

- **Heavy metals - Lead contamination in Flint (USA):** Water supply changes caused lead leaching from pipes, exposing large populations to a neurotoxic metal.
- **Nitrates and cyanobacteria:** Agricultural runoff promotes algal blooms producing toxic compounds, affecting water quality in regions such as France and China.

2.6.3. Effects of disinfection by-products

Water treatment processes, particularly chlorination, can generate toxic by-products such as trihalomethanes (THMs) and haloacetic acids. These compounds are suspected carcinogens and may affect the reproductive system.

Epidemiological studies have reported associations between THM exposure in drinking water and an increased risk of bladder cancer, especially at higher concentrations.

2.6.4. Environmental risks : Overexploitation and saline intrusion

Excessive groundwater extraction can deplete freshwater resources and degrade water quality.

Examples :

- **Saltwater intrusion (Jakarta, Indonesia):** Overexploitation of aquifers has led to seawater intrusion, reducing drinking water availability and contributing to land subsidence.
- **Aral Sea desiccation:** Intensive irrigation caused severe ecological degradation, exposing populations to contaminated dust and increasing respiratory and renal diseases.

2.7. Air- related risks

Air pollution is a major environmental determinant of chronic diseases and premature mortality worldwide. According to the World Health Organization (WHO), it accounts for approximately 7 million premature deaths annually. The main pollutants include:

- **Particulate matter (PM_{2.5} and PM₁₀):** Penetrate deep into the respiratory tract and bloodstream, contributing to cardiovascular, respiratory, and neurodegenerative diseases.

- **Nitrogen oxides (NO_x) and sulfur dioxide (SO₂):** Irritant gases that induce pulmonary inflammation and aggravate conditions such as asthma and chronic obstructive pulmonary disease (COPD).
- **Volatile organic compounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs):** Emitted from combustion processes, they are carcinogenic and can impair liver and kidney function.
- **Case Studies**
 - **London Smog (1952) :** Severe coal-related air pollution caused over 12,000 deaths, leading to the UK Clean Air Act (1956).
 - **Delhi (India) :** Chronic exposure to extreme PM_{2.5} levels (up to 500 µg/m³) is associated with increased respiratory diseases, reduced fertility, and higher cancer incidence.
 - **Wildfires (California, Amazon) :** Release of CO, PM_{2.5}, and benzene exposes populations to acute respiratory effects and long-term cancer risks.

2.8. Waste related risks

Improper waste management, particularly of **healthcare waste (HCW)** and **radioactive waste**, poses serious risks to human health and the environment. These wastes may contain infectious agents, toxic chemicals, or radioactive substances. The WHO estimates that about 15% of healthcare waste is hazardous.

2.8.1. Healthcare Waste (HCW)

- **Biological risks:** Transmission of pathogens (HIV, hepatitis B/C, resistant bacteria) through injuries or aerosols.
- **Chemical risks:** Exposure to cytotoxic drugs, anesthetic gases, and heavy metals (e.g., mercury, lead).
- **Environmental risks:** Soil and groundwater contamination due to improper disposal or incineration.

Case study: In India (2018), ~40% of healthcare facilities failed to comply with HCW regulations, increasing infection risks (e.g., hepatitis C transmission via discarded needles).

2.8.2. Radioactive waste

- **External irradiation** : Ionizing radiation causing cancers, DNA damage, and burns.
- **Internal contamination** : Inhalation/ingestion of radionuclides (e.g., Cs-137, Pu-239) leading to chronic toxicity.
- **Storage challenges** : Long-term isolation required; leakage may cause persistent environmental contamination.

Case study: Goiânia accident (Brazil, 1987): Cs-137 exposure led to 249 contaminations and 4 deaths, highlighting risks of poor waste management.

2.9. Risks Related to radioelements and radiation protection

Radioelements (natural or artificial) are widely used in medical, industrial, and energy sectors. Their emission of ionizing radiation (α , β , γ , X) can cause harmful biological effects depending on dose, duration, and tissue sensitivity.

2.9.1. Biological effects of radiation

Radiation induces **direct DNA damage** or **indirect oxidative stress via free radicals**.

- **Acute exposure:** May cause Acute Radiation Syndrome (ARS), affecting hematopoietic, gastrointestinal, and neurovascular systems.
- **Chronic exposure:** Associated with genetic mutations, cancers, and cardiovascular diseases.

Case studies:

- **Chernobyl (1986)** : Increased thyroid cancers due to I-131 exposure.
- **Fukushima (2011)** : Raised concerns about long-term low-dose exposure.
- **Medical exposure** : Growing use of CT and PET scans requires dose optimization.

2.9.2. Principles of radiation protection

According to the International Commission on Radiological Protection (ICRP), three key principles apply:

- **Justification** : Use radiation only when benefits outweigh risks.
- **Optimization (ALARA)** : Minimize exposure through time, distance, and shielding.
- **Dose limitation** : Regulatory limits (20 mSv/year for workers; 1 mSv/year for the public).

3. Health risks

3.1. Health risk assessment: Definition

Health risk assessment in public health begins with determining whether a chemical substance possesses toxic or hazardous properties. This initial step aims to identify the inherent hazard associated with the substance. Once the potential hazard has been established, the focus shifts to evaluating both the likelihood and the magnitude of human exposure.

The risk associated with chemical exposure depends on two fundamental components : the dose received by individuals and the intrinsic toxicity of the substance. This relationship can be formally expressed as follows:

$$\text{Risk associated with chemical exposure} = [\text{Dose of the chemical}] \times [\text{Intrinsic toxicity}]$$

Thus, risk is not solely determined by the presence of a hazardous substance, but rather by the extent to which individuals are exposed to it and the severity of its toxic effects. Even highly toxic substances may pose limited risk if exposure is negligible, whereas low-toxicity substances can become significant concerns under conditions of high or prolonged exposure.

An accurate characterization of exposure is therefore essential and involves:

- The identification of contamination sources,
- The determination of concentrations in contact with individuals,
- And the identification of exposure pathways (inhalation, ingestion, and dermal contact).

3.2. Factors influencing exposure

Risk assessment takes into account several key parameters that influence the nature and magnitude of human exposure to chemical substances.

First, the **identity and concentration of chemical substances** must be clearly established. This involves identifying contaminants of concern and determining their levels in relevant environmental media. The chemical nature of a substance, combined with its concentration, plays a critical role in defining its potential impact on human health.

Second, the **duration and frequency of exposure** are essential determinants of risk. **Acute exposure** (≤ 14 days) may result in immediate or short-term health effects, whereas **chronic exposure** (≥ 365 days) is more likely to be associated with long-term or cumulative health outcomes. The temporal pattern of exposure therefore significantly influences the type and severity of adverse effects.

Third, the **sources and pathways of exposure** must be identified. Humans may be exposed to contaminants through multiple environmental compartments, including water, air, soil, and food. These pathways determine how contaminants enter the body, whether through inhalation, ingestion, or dermal contact.

Finally, the **characteristics of exposed individuals** must be considered. Factors such as age, health status, lifestyle, and prior exposure history can significantly influence individual susceptibility. Vulnerable populations, including children, the elderly, and individuals with pre-existing health conditions, may experience greater health impacts under similar exposure conditions.

Whenever possible, the assessment should rely on **empirical measurements**, such as biological tissue analysis and environmental monitoring data. However, in situations where complete data are not available, **exposure models** may be used to estimate exposure levels. In such cases, the inherent uncertainty associated with these models must be explicitly acknowledged and taken into account in the interpretation of results.

3.3. Risk assessment process

Risk assessment is based on **four main steps**, which together provide a structured framework for evaluating potential health impacts associated with environmental exposures.

3.3.1. Hazard identification

Hazard identification and evaluation involve a **qualitative assessment** of the presence and magnitude of the danger that an agent may pose to potential receptors. This step aims to determine whether a substance is capable of causing adverse health effects under specific conditions of exposure.

Data on the toxicity of chemical substances are derived from four main types of studies:

1. **Structure–activity relationships (SAR)**, which predict toxicity based on chemical structure,
2. ***In vitro* or short-term studies**, providing preliminary information on biological effects,
3. ***In vivo* bioassays in animals**, which allow the assessment of toxic effects in whole organisms,
4. **Human epidemiological studies**, which provide evidence based on observed health outcomes in exposed populations.

This process involves systematic data collection and analysis and may include:

- The identification of sources of chemical exposure,
- The compilation of a comprehensive inventory of chemical agents present at a given site that may affect target receptors,
- The selection of specific chemicals of potential concern (i.e., those to be included in the risk assessment), based on their hazardous properties such as persistence, bioaccumulation potential, toxicity, and environmental fate and behavior,
- The compilation of summary statistics for key constituents selected for detailed analysis.

It is important to emphasize that, in identifying chemicals of concern, the objective is generally to capture those substances that account for the majority (approximately 95%) of the risks associated with the exposures under consideration.

3.3.2. Dose–Response assessment

Dose response assessment (also referred to as effects assessment) aims to estimate the relationship between the dose or level of exposure to a substance and the incidence as well as the severity of the observed adverse effects.

This step considers:

- The types of adverse effects associated with chemical exposures,
- The relationship between the magnitude of exposure and these negative effects,
- And the uncertainties inherent in the available data.

The objective of dose–response assessment is to describe the **quantitative relationship** between the amount of a substance administered or absorbed and the extent of the resulting toxic damage or disease. This relationship is fundamental for predicting health outcomes under different exposure scenarios.

The estimation of dose–response relationships is based on available scientific studies, including **epidemiological data, toxicological studies**, and, when such data are limited, *in vitro* or *in silico* approaches.

It should be noted that a single substance may exhibit multiple dose–response curves, depending on the type of toxic effect and the conditions of exposure. Different endpoints (e.g., carcinogenic, neurological, or reproductive effects) may therefore require separate analyses.

In the context of chemical exposure assessment, this step typically includes:

- A **toxicological analysis**, which involves compiling toxicological profiles for substances of potential concern,
- And/or a **dose–response evaluation**, used to quantify toxicological information and characterize the relationship between the administered or received dose and the incidence of adverse effects in exposed populations.

Based on these quantitative relationships, it is possible to derive **toxicity reference values**, which are then used to estimate the likelihood and severity of adverse health effects in populations exposed to different levels of contaminants.

3.3.2.1. Toxicological Reference Values (TRVs)

Toxicological Reference Values (TRVs) are indicators used, through comparison with exposure levels, to characterize potential health risks in humans, particularly for chemical substances. A TRV represents an estimate of the quantity (expressed in mg/kg/day) or concentration (expressed in mg/m³) to which a population can be exposed without observable adverse effects.

Commonly used TRVs include:

- **ADI (Acceptable Daily Intake)**, also referred to as **DJA (Dose Journalière Admissible)**,
- **RfD (Reference Dose)** and **RfC (Reference Concentration)**,
- **MRL (Minimal Risk Level)**.

In addition to TRVs based on external exposure (i.e., dose or concentration at the point of contact), it is also possible to derive **internal TRVs**, known as **Biomonitoring Equivalents (BE)**, which are based on measured concentrations in biological media.

In dose–response analysis, different approaches are used depending on whether the effects exhibit a **threshold (deterministic effects)** or **no threshold (probabilistic or stochastic effects)**.

a) Threshold Approaches

For deterministic effects, the severity of the effect is proportional to the dose.

At the **individual level**, there exists a threshold above which the body's defense mechanisms are overwhelmed, leading to the occurrence of adverse effects. The intensity of these effects increases with increasing dose.

At the **population level**, once the threshold affecting the most sensitive individuals is exceeded, the proportion of the population likely to develop adverse effects increases with the dose.

Approaches used to characterize threshold-based dose–response relationships include the identification of:

- The **NOAEL (No Observed Adverse Effect Level)**,

- And the **LOAEL (Lowest Observed Adverse Effect Level)**.

In a typical dose–response curve (see [Figure 3.1](#)), tested doses may be represented by points such as E, F, G, H, and I. Statistical significance of observed effects at certain doses (e.g., G, H, and I) is often indicated by an asterisk (*).

The **threshold (T)** corresponds to the dose below which no additional increase in response is observed. The **NOAEL** (e.g., point F) is defined as the highest tested dose that does not produce a statistically significant adverse effect. In this example, the NOAEL is approximately **2 mg/kg body weight**.

The **LOAEL** (e.g., point G), approximately **2.3 mg/kg body weight**, corresponds to the lowest tested dose at which a statistically significant adverse effect is observed.

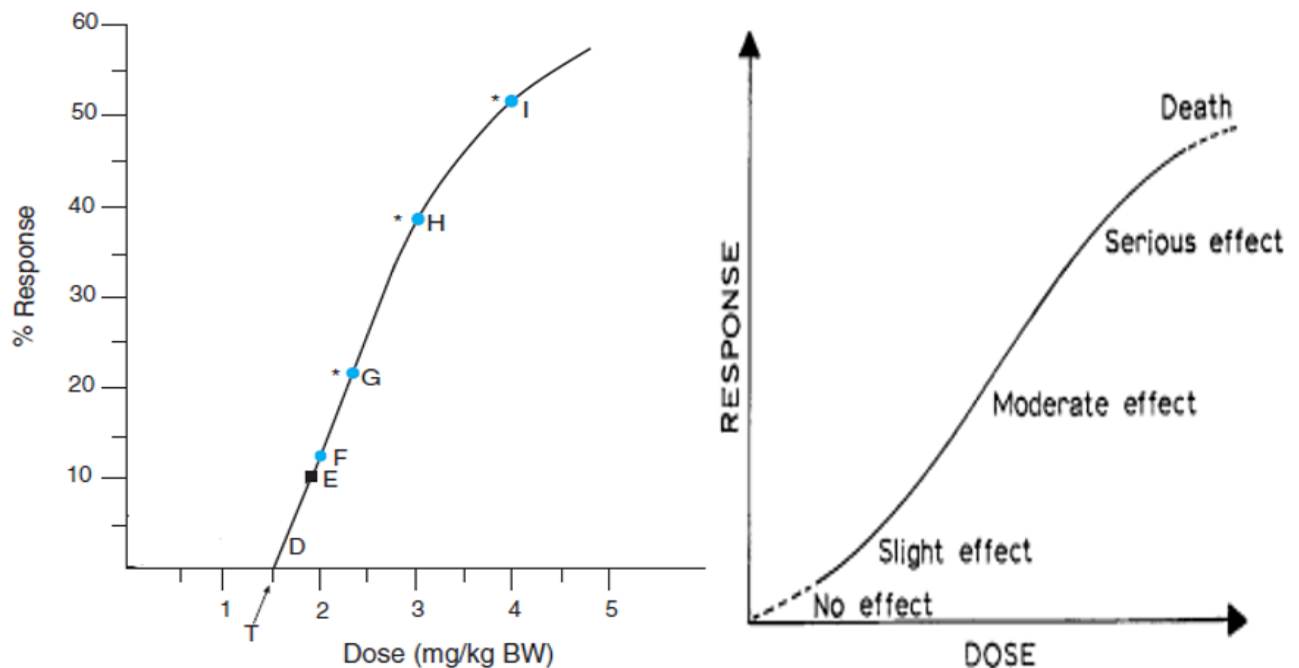


Figure 3.1. Dose - response curve for a threshold effect ([Casarett, 2008](#)).

The NOAEL should not be interpreted as being risk-free, as several studies have shown that NOAEL-derived responses for continuous effects correspond, on average, to a risk of approximately 5%, while NOAELs based on quantal effects may be associated with risks exceeding 10%.

Approaches used to characterize dose- response relationships include the identification of the following critical doses:

- **LD₅₀** (dose producing 50% lethality),
- **LC₅₀** (concentration producing 50% lethality),
- **Benchmark Dose (BMD_{10%})** (dose producing a 10% response),
- As well as **NOAEL** values (**Figure 3.2**).

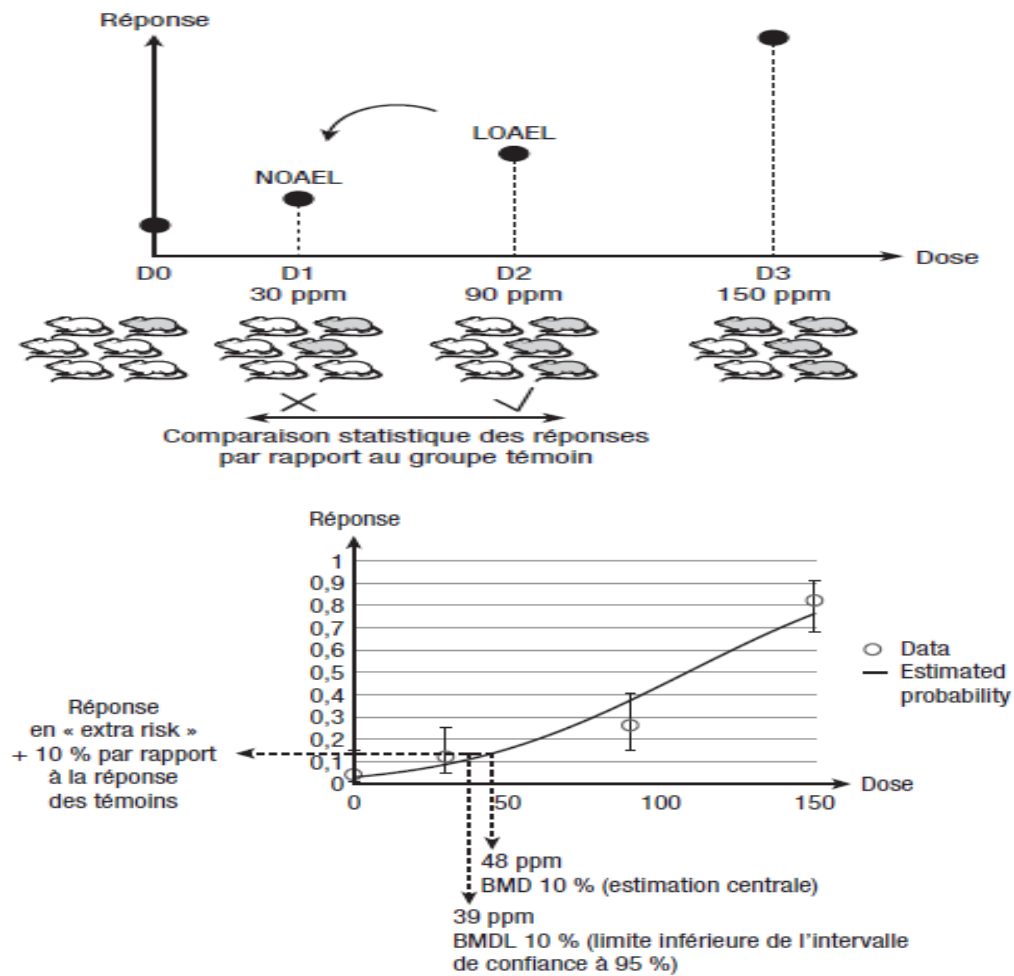


Figure 3.2. Illustration of the calculation of critical doses based on animal data (**Glorennec et al., 2023**).

When the assumption of a toxicity threshold is adopted, the effect occurs only if a certain dose is reached and exceeds the body's detoxification capacity.

RfD and **ADI** values are generally calculated from **NOAEL** values by dividing them by uncertainty factors (**UF**) and/or modifying factors (**MF**) (**See TD N° 4**).

$$RfD = NOAEL / (UF \times MF)$$

$$ADI = NOAEL / UF$$

In principle, division by uncertainty factors accounts for variability:

- Interspecies (animal to human),
- And intraspecies (human variability), with default values of 10 for each.
-
- An additional uncertainty factor is used to account for experimental limitations—for example, to extrapolate from short-term exposure studies to conditions more relevant for chronic exposure, or to account for an insufficient number of animals or other experimental constraints ([Figure 3.3](#)).

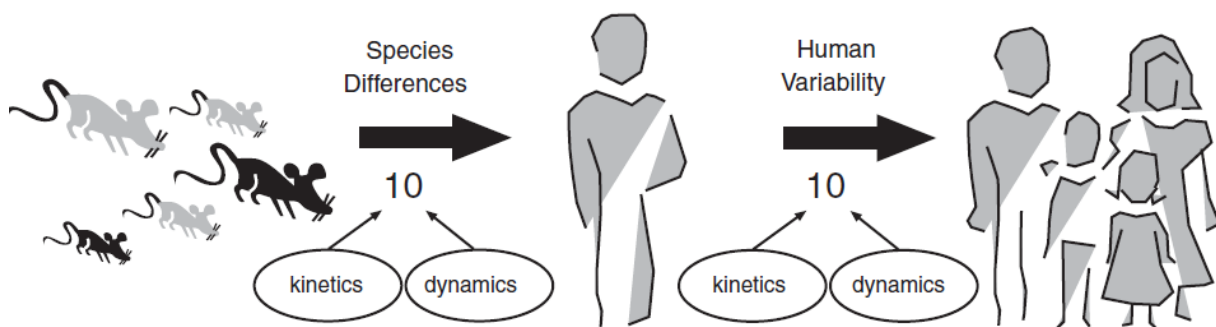


Figure 3.3. Facteurs d’incertitudes et de modification ([Casarett, 2008](#)).

b) Non-threshold approaches

For non-threshold (probabilistic) effects, the assumption is made that no threshold exists. Within the population, it is the probability of occurrence of the effect, rather than its severity, that is proportional to the dose. This is the case for carcinogenic, mutagenic, or genotoxic compounds.

In other words, even the smallest exposure, however minimal, may produce an effect, with a proportionality coefficient between the dose and the probability of occurrence of the effect. The dose–response relationship therefore consists in determining this proportionality coefficient, referred to as the “excess unit risk” (sometimes called “unit risk”).

In this type of effect, the toxicity reference value (TRV) is defined as the theoretical additional probability, compared to an unexposed individual, that a person will develop a disease (generally cancer) if exposed daily over a lifetime to a unit dose or concentration of the substance. In other words, it corresponds to the slope of the line representing the dose–response relationship in the low-dose region (**Figure 3.4**).

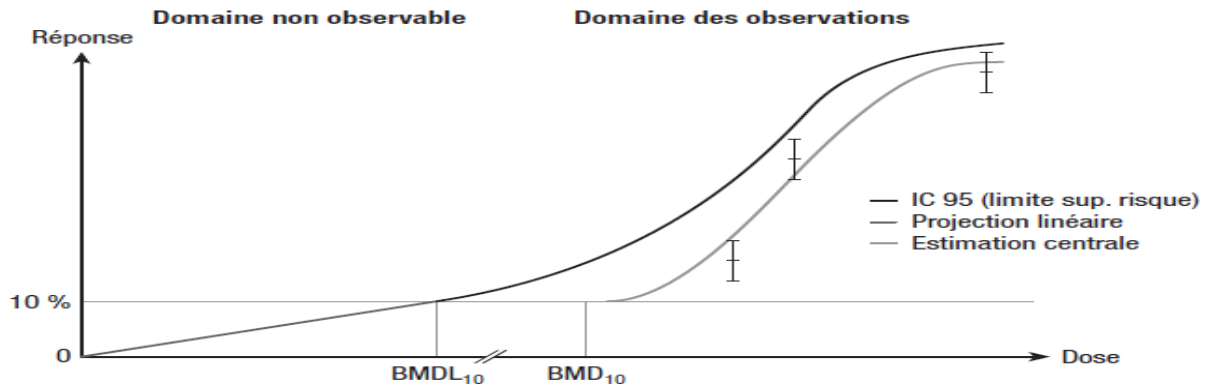


Figure 3.4. Illustration de la projection linéaire représentant l’excès de risque unitaire ou la pente de la droite (*Slope Factor*) (**Glorennec, et al., 2023**).

The slope factor is generally expressed as an excess unit risk (EUR) and is sometimes referred to as the Slope Factor (SF) or Inhalation Unit Risk (IUR). Its unit is the inverse of dose: $(\text{mg/kg})^{-1}$ for the oral route and $(\text{mg/m}^3)^{-1}$ for the inhalation route.

The toxicity reference value (TRV) is calculated using the following equation:

$$\text{ERU} = \frac{0.10}{\text{BMDL}_{10}}$$

3.3.3. Exposure Assessment

Exposure assessment is conducted to estimate the magnitude of actual and/or potential exposures of receptors to chemical substances present in the human environment. This process takes into account:

- The frequency and duration of exposure,
- The nature and size of potentially at-risk populations (risk groups),
- As well as the pathways and routes through which these groups may be exposed.

Indeed, several physical and chemical characteristics of the substances involved help identify the main features of exposure.

These properties also provide essential information for determining distribution, absorption, metabolism, retention time, excretion, bioaccumulation, and half-life of the substances, as well as their transformation into new chemical compounds.

In general, exposure assessment consists of describing the **nature** and **size of the population** exposed to a substance, as well as the **intensity** and **duration** of this exposure. Exposure assessment may concern past, current, or future anticipated exposures.

- **How is exposure expressed?**

Exposure can be expressed as external dose (average ingested dose, average inhaled concentration) or internal dose (a concept reflecting the amount present in the body). It is generally expressed as follows:

- **Daily Exposure Dose (DED)** in mg/kg·day for environmental media *i* (e.g., garden soil at home) or food item *i* (e.g., lettuce grown at home), calculated using the following equation:

$$DJE = \frac{\sum i (C_i \cdot Q_i)}{P}$$

- Where:
- C_i : concentration in medium *i* (mg/kg),
- Q_i : average quantity of medium *i* ingested over the considered period (kg/day),
- P : body weight (kg).
- **Average inhaled concentration (CI)** ($\mu\text{g}/\text{m}^3$), calculated as follows:

$$CI = \frac{\sum (C_j \cdot T_j)}{24}$$

Where:

C_j : pollutant concentration in location *j*,

T_j : time (hours) spent in location *j*.

3.3.3.1. Types of Exposure

- Dietary Exposure

Dietary exposure assessment is based on consumption and residue data. These data include typical dietary patterns (regional, national, or individual models). The assessment may target the average consumer or critical groups (high consumers), using high-exposure scenarios (maximum values) or best estimates (median values).

Depending on data availability, the estimation may rely on realistic scenarios (e.g., agricultural pesticides, based on field trial data) or pessimistic scenarios (e.g., food additives and contaminants, based on theoretical maximum consumption).

- Occupational Exposure

Workers handling chemical products are primarily exposed through inhalation and dermal contact. Occupational exposure assessments are based on environmental or biological measurements (blood, urine, exhaled air).

In the absence of monitoring data, estimation models are used, such as the EASE (Estimation and Assessment of Substance Exposures) model, integrated into the EUSES system.

Modeling of Major Industrial Accident Risks

Exposure related to industrial accidents relies on specialized models designed to predict the impact of a single, short-term chemical release. These models take into account:

- The source of the release (frequency and quantity of emissions),
- Atmospheric dispersion, depending on meteorological conditions,
- characteristics of accidental events, assessed through analysis of technical failures and probabilities of occurrence.

Environmental Exposure

Environmental exposure assessment follows a two-step process :

- **Screening stage** : to determine whether exposure is possible and significant,
- **Quantitative assessment** : if exposure is identified, a detailed risk analysis is performed.

In the absence of experimental data, conservative assumptions are often applied. It may be necessary to calculate bioconcentration factors to estimate the accumulation of contaminants in the food chain (fish, crops, meat, dairy products).

For chemicals applied to food products, withdrawal periods may be calculated to determine the required time before consumption.

$$\text{IER}_{\text{inhalation}} = C \times \text{EUR}_{\text{inhalation}}$$

$$\text{IER}_{\text{ingestion}} = C \times \text{EUR}_{\text{ingestion}}$$

Comprehensive Environmental Exposure Assessment

A comprehensive environmental exposure assessment includes four steps:

- Identification of target compartments (air, surface and groundwater, soil, sediments, biota),
- Estimation of emissions or releases and environmental behavior,
- Estimation of environmental concentrations.

The assessment may be local, regional, or continental. A local assessment generally concerns point sources, whereas regional or continental assessments apply to diffuse sources.

Identification of target compartments covers the entire life cycle of the chemical: manufacturing, formulation, industrial, professional and domestic use, as well as disposal or recycling. Releases into air, soil, and wastewater are estimated using emission scenarios and physicochemical properties of the substance. Wastewater treatment is a key factor in managing releases.

Chemical substances may be transported within the same compartment (intra-media transport) or transferred between different media (inter-media transport). The main transport mechanisms are :

- **Advection** : transport of a substance with the flow of the medium (e.g., rain infiltration carrying contaminants from soil to groundwater),
- **Dispersion** : movement of a contaminant due to concentration gradients (e.g., diffusion of a pollutant from surface water to sediments).

Degradation processes (biodegradation, hydrolysis, photodegradation) influence exposure by reducing chemical concentrations, particularly in wastewater treatment plants.

Another key factor is the bioconcentration factor. The accumulation of chemicals in organisms may lead to secondary contamination in food chains, particularly affecting top predators.

The final step consists in estimating the concentration of the chemical in receiving media (soil, air, surface water, and groundwater).

The conceptual exposure model is illustrated in [Figure 3.5](#).

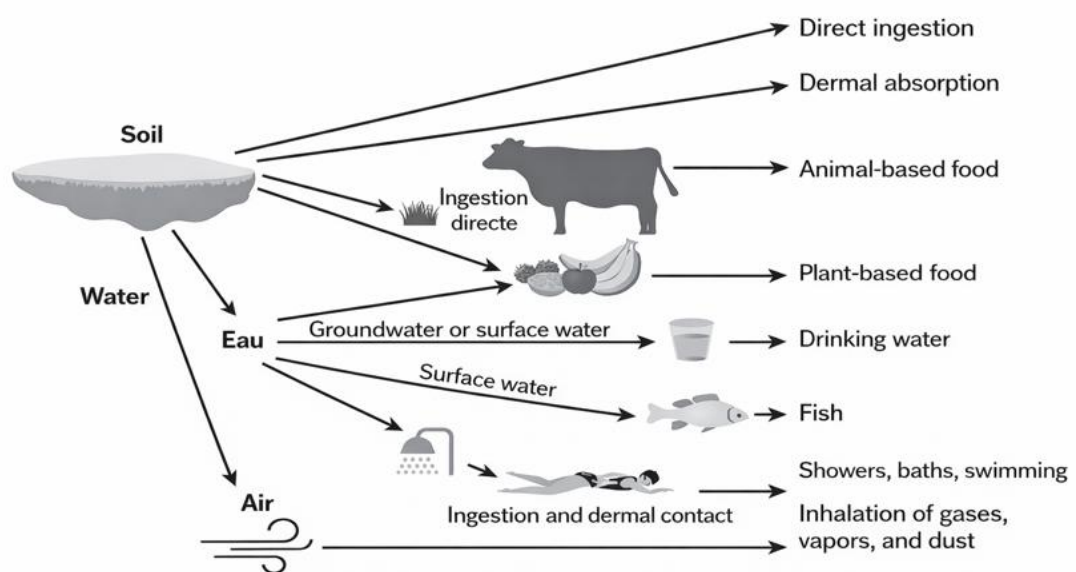


Figure 3.5. Example of a conceptual exposure diagram ([Glorennec et al., 2023](#)).

3.3.4. Risk Characterization

In general, risk characterization summarizes and integrates the results of exposure and toxicity assessments in order to define risk levels qualitatively and/or quantitatively.

More broadly, risk characterization involves integrating data and analyses from the first three steps of the risk assessment process (namely: hazard identification, dose–response assessment, and exposure assessment) to determine the likelihood that humans will experience one or more forms of toxicity related to a substance.

For threshold effects (deterministic), the ratio- commonly referred to as the **hazard quotient (HQ)** (also called hazard ratio (HR), danger quotient (DQ), or risk index (RI))- is calculated by comparing the average inhaled concentration or the average ingested dose to the toxicity reference value (TRV):

$$\text{HQ}_{\text{inhalation}} = \text{CI} / \text{TRV}_{\text{inhalation}} \quad \rightarrow \text{Less than 1: acceptable risk}$$

$$\text{HQ}_{\text{ingestion}} = \text{DED} / \text{TRV}_{\text{ingestion}} \quad \rightarrow \text{Greater than 1: unacceptable risk}$$

Indeed, if the ratio exceeds 1, this indicates that exposure is higher than the TRV.

For non-threshold (probabilistic) effects, the assessment is quantitative. The average inhaled concentration or ingested dose is multiplied by the excess unit risk (EUR), for inhalation or ingestion, to estimate the probability of occurrence of the effect for an average individual, referred to as the **individual excess risk (IER)**:

$$\text{IER}_{\text{inhalation}} = \text{C} \times \text{EUR}_{\text{inhalation}}$$

$$\text{IER}_{\text{ingestion}} = \text{C} \times \text{EUR}_{\text{ingestion}}$$

Where IER is the individual excess risk and EUR is the excess unit risk.

For carcinogenic effects, TRVs are conventionally provided for a 70-year exposure duration. Therefore, exposure concentrations and doses should be expressed as averages over this period.

In addition, results can be expressed at the population level. For non-threshold effects, the proportion of the population exceeding the TRV can be estimated. For probabilistic effects, the number of expected excess cases can be estimated by multiplying the average individual excess risk by the size of the exposed population. This makes it possible, for example, to estimate how many excess cancer cases will theoretically be observed over 70 years in a given city due to the presence of an atmospheric contaminant.

These risk indicators are expressed per hazardous agent, since the assessment is carried out on a substance-by-substance basis. However, in real situations, populations are exposed to several agents, and the effects of these simultaneous exposures are generally not known.

4. Risk analysis and management

4.1. Health risk assessment: Environmental example

Health Risk Assessment (HRA) is a systematic scientific approach aimed at estimating the probability that exposure to an environmental hazard will lead to adverse effects on human health.

In the environmental field, this approach makes it possible to analyze the potential impact of contaminants present in air, water, soil, or food on exposed populations. It is now considered an essential tool for public health, prevention, and regulatory decision-making.

According to the World Health Organization (WHO), a significant proportion of the global burden of disease is attributable to environmental factors such as air pollution, toxic chemical substances, radiation, and water contamination.

In real environmental contexts, this approach is applied to situations such as urban air pollution, drinking water contamination, or pesticide exposure. For example, in a large city with heavy road traffic, the pollutants studied include PM_{2.5}, NO₂, CO, and ozone.

The observed health effects include:

- Worsening of asthma,
- Chronic bronchitis,
- Cardiovascular diseases,
- Premature mortality.

The WHO reports that air pollution is one of the major environmental contributors to global mortality.

4.1.1. Importance for public health

Health risk assessment makes it possible to :

- Establish environmental standards,
- Regulate industrial emissions,
- Protect sensitive populations,
- Guide public policies,
- Prevent health crises.

It also serves to communicate risks scientifically to decision-makers and the public.

4.1.2. Limitations of the method

Despite its scientific robustness, Health Risk Assessment (HRA) has certain limitations :

- Uncertainties in exposure data,
- Individual biological variability,
- Combined effects of multiple pollutants that are difficult to model,
- Lack of toxicological data for certain emerging substances.

Health risk assessment applied to the environment is an essential method for understanding how environmental contaminants affect human health. Through a structured analysis of hazards, exposure, dose–response relationships, and overall risk, this approach effectively supports prevention, regulation, and population protection.

In a context of increasing urbanization, industrial pollution, and climate change, its role is becoming central in building safer and more sustainable environments.

4.2. Health and environmental risk management

4.2.1. Definition

Environmental health risk management refers to all strategies implemented to prevent, reduce, control, or eliminate the negative impacts of environmental factors on human health (**Figure 4.1**). Following the risk assessment stage, this approach aims to translate scientific findings into concrete public health protection actions.

It is part of a preventive public health framework, particularly important in the face of exposures related to air pollution, chemical contaminants, hazardous waste, biological agents, and environmental disasters.

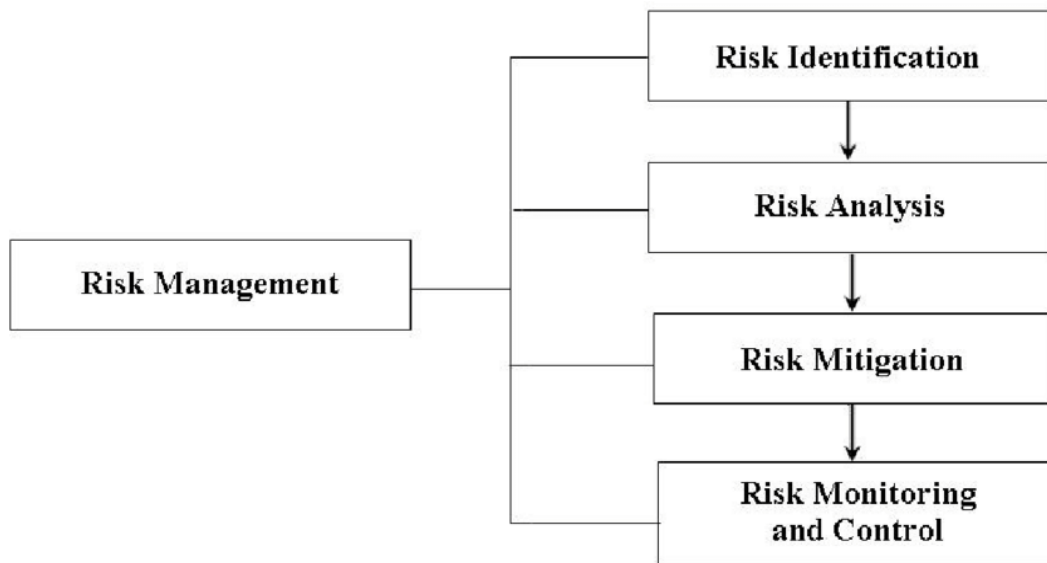


Figure 4.1. Fundamental Concept of Risk Management (Ghazali et Kabir, 2009).

4.2.2. Fundamental principles of risk management

- **Source prevention** : The first strategy consists of acting directly at the origin of the hazard in order to prevent the emission or dispersion of pollutants.

Examples:

- Reduction of industrial emissions,
- Control of chemical discharges,
- Limitation of pesticide use,
- Wastewater treatment,
- Safe management of biomedical waste.

This approach is the most effective, as it reduces the risk even before human exposure occurs.

- **Control of exposure pathways** : When hazard elimination is not possible, it is necessary to limit the contact between the contaminant and the population.

Possible measures:

- Air filtration,
- Drinking water treatment,
- Containment of contaminated soils,
- Personal protective equipment,
- Establishment of safety zones around polluted sites.

These measures reduce inhalation, ingestion, or dermal contact with hazardous substances.

- **Environmental monitoring** : Monitoring enables the tracking of the evolution of contaminants in different environmental media.

Examples of monitoring :

- Air quality (PM_{2.5}, NO₂),
- Drinking water quality,
- Soil contamination,
- Environmental noise,
- Radiation levels.

Regular monitoring facilitates the early detection of threshold exceedances with potential health impacts.

Regulation and standards : Public authorities regulate risk management through laws, standards, and limit values.

- **Key regulatory frameworks include:**
- WHO air quality guidelines,
- European environmental directives,
- ICPE regulations (classified installations for environmental protection),
- ISO 14001 for environmental management systems.

Risk communication : Properly informing the population is essential to promote protective behaviors.

Examples:

- Pollution alerts,
- Health advisories,
- Information campaigns,
- Public participation initiatives.

Risk communication enhances community resilience in the face of environmental threats.

4.2.3. Case study : Management of health-risk waste

Medical, chemical, and industrial wastes require specific management procedures. The essential actions include: Waste segregation, safe storage, controlled transportation, specialized treatment and regulated disposal. Poor management increases infectious and toxic risks.

4.2.4. Limitations and challenges

Despite significant progress, several obstacles remain:

- High costs of control technologies,
- Lack of monitoring in certain regions,
- Poorly understood emerging pollutants,
- Institutional coordination difficulties,
- Scientific uncertainties.

Environmental health risk management constitutes a crucial step following hazard assessment. It translates scientific analysis into concrete interventions aimed at protecting human health.

By combining prevention, monitoring, regulation, and communication, this approach enables a long-term reduction of pollution impacts on populations. In a global context marked by industrialization, urbanization, and climate change, it represents a fundamental pillar of modern public health.

4.3. Standard setting and the role of national and international institutions

The establishment of health and environmental standards is a fundamental pillar of public health protection. These standards define acceptable exposure thresholds to physical, chemical, or biological agents present in the environment in order to limit harmful effects on human health and ecosystems.

Their development is based on scientific data derived from toxicology, epidemiology, health risk assessment, and the precautionary principle. These standards are then used as references to regulate industrial activities, monitor environmental quality (air, water, soil), and guide public policies.

This mission involves both national institutions (health agencies, ministries, public laboratories) and international organizations (WHO, UN, European Union, EPA, etc.), which collaborate to harmonize health safety criteria.

4.3.1. Principles of health standard setting

4.3.1.1. Scientific basis of standards

Regulatory thresholds are established based on several elements:

- Toxicological studies,

- Dose–response relationships,
- Chronic or acute exposure levels,
- Vulnerable populations,
- Safety margins.

For example, for an air pollutant such as NO₂ or PM_{2.5}, experts determine the maximum concentration compatible with the protection of human health.

According to the WHO, these guideline values are based on available scientific evidence regarding respiratory, cardiovascular, and neurological effects.

4.3.1.2. Precautionary principle

When scientific uncertainty exists but a plausible risk is identified, authorities may adopt protective standards even in the absence of absolute certainty.

This principle is particularly applied to :

- Endocrine disruptors,
- Nanomaterials,
- Emerging pollutants,
- Suspected carcinogenic substances.

4.3.2. Role of international institutions

International organizations play a central role in developing recommendations based on scientific data and in promoting the harmonization of practices between countries. These references then serve as a basis for national policies, thereby ensuring global coherence in protecting human health from environmental risks. **Table 4.1** summarizes the main international institutions as well as their respective roles in this process.

Table 4.1. Main international institutions and their roles in the establishment of health and environmental standards (WHO, 2021; UN, 2020; EU, 2022; EEA, 2021; ISO, 2015; ICRP, 2007).

International Body	Field of Intervention	Main Roles in Setting Health and Environmental Standards
World Health Organization (WHO)	Global public health	- Develops guideline values for air, water, and noise quality. - Defines exposure limits to toxic substances. - Issues international health

International Body	Field of Intervention	Main Roles in Setting Health and Environmental Standards
		recommendations. - Supports countries in health risk assessment and management.
United Nations (UN)	Global environmental governance	- Coordinates international environmental agreements. - Promotes cooperation among states. - Oversees global conventions on hazardous pollutants and climate.
United Nations Environment Programme (UNEP)	Environmental protection	- Implements global environmental policies. - Monitors the state of the global environment. - Supports conventions on hazardous chemicals.
European Union (EU)	Regional regulation	- Establishes directives on air and water quality. - Regulates hazardous waste. - Governs the use of chemical substances (REACH). - Harmonizes standards among member states.
European Environment Agency (EEA)	Environmental monitoring	- Collects pollution data. - Assesses the state of the environment in Europe. - Provides scientific reports to support decision-making.
International Commission on Radiological Protection	Radiological protection	- Defines exposure limits to ionizing radiation. - Establishes principles of radiological protection.
International Organization for Standardization	Technical standardization	- Develops environmental management standards. - Frames industrial best practices. - Example: ISO 14001 for environmental management.
Stockholm Convention	Persistent organic pollutants	- Regulates the reduction or elimination of POPs. - Protects human health from toxic bioaccumulative substances.

4.3.3. Role of national authorities

National authorities play a key role in implementing health and environmental standards established at the international level ([Table 4.2](#)). They act as the operational interface between global recommendations and national contexts, adapting them to local socio-economic, environmental, and health conditions.

Their core functions include the development of scientific data, risk assessment, regulation, environmental monitoring, and crisis management.

Ministries in charge of health, environment, and labor translate scientific recommendations into regulatory frameworks (laws, decrees, and official orders). They establish national exposure

limits, regulate industrial activities, and implement national action plans, such as air quality management plans and hazardous substance control strategies.

Environmental monitoring agencies ensure continuous surveillance of air, water, and soil quality. This monitoring enables the detection of regulatory exceedances, supports early warning systems, and informs the public. For instance, networks such as Atmo France provide real-time data on air pollutant concentrations.

National authorities are also essential in managing environment-related health crises, including acute pollution events, drinking water contamination, and industrial accidents. They coordinate emergency responses, implement protective measures, and ensure effective risk communication.

Finally, they contribute to international cooperation by sharing data, participating in research programs, and implementing international conventions, thereby ensuring consistency in environmental health risk management.

Table 4.2. Main national authorities and their roles in the management of health and environmental standards ([ANSES, 2022](#); [Ministry of Health, 2021](#); [Santé publique France, 2022](#); [Atmo France, 2023](#); [Ministry for Ecological Transition, 2022](#)).

National Authority	Field of Intervention	Main Roles
Ministry of Health	Public health	- Development of national health policies - Definition of health safety thresholds - Management of health crises
Ministry of the Environment	Environmental protection	- Regulation of pollution - Implementation of environmental policies - Protection of natural resources
ANSES (France)	Scientific expertise	- Health risk assessment - Independent scientific opinions - Support for public decision-making
Santé publique France	Epidemiological surveillance	- Monitoring of health impacts - Analysis of health data - Prevention and health alert systems
Atmo France (regional networks)	Air quality	- Monitoring of atmospheric pollutants - Public information - Alerts in case of threshold exceedance
Water Agencies	Water resource management	- Monitoring of water quality - Protection of aquatic environments - Sustainable water management

National Authority	Field of Intervention	Main Roles
Classified Installations Inspection (ICPE)	Industrial control	- Monitoring of industrial sites - Prevention of technological risks - Enforcement of regulations
National Public Laboratories	Research and analysis	- Production of scientific data - Toxicological and environmental analyses - Technical support to authorities

4.3.4. Interaction between national and international levels

The effectiveness of environmental health policies largely depends on coordination between national and international authorities. This interaction ensures consistency of standards, harmonization of practices, and coordinated responses to global challenges such as transboundary pollution, climate change, and the circulation of toxic substances.

International organizations, such as the World Health Organization and the United Nations Environment Programme, provide a scientific and normative framework based on the best available knowledge. These recommendations serve as references for countries, which adapt them according to their local constraints, technical capacities, and public health priorities.

For example, WHO air quality guidelines (WHO, 2021) are widely used as a basis for developing national standards. However, countries may adopt stricter or more flexible thresholds depending on their economic and industrial context, allowing realistic implementation while maintaining a common health protection objective.

International cooperation also involves participation in multilateral agreements such as the Stockholm Convention and the Paris Agreement. These legal instruments promote collective commitment to reducing environmental risks at the global scale.

In addition, the exchange of scientific and technical data between countries strengthens risk monitoring and anticipation capacities. International research networks and health surveillance systems enable early detection of emerging threats and coordinated responses.

However, this coordination may face limitations due to economic disparities, regulatory differences, and unequal institutional capacities across countries. Developing countries, in particular, may encounter financial and technical constraints in implementing certain standards.

Despite these challenges, collaboration between national and international levels remains essential to address complex environmental health issues. It facilitates knowledge sharing, harmonizes practices, and enhances population protection worldwide.

Environmental and health risk analysis constitutes a fundamental component of modern public health and environmental protection. This course has progressively established a comprehensive framework, beginning with the fundamental concepts of risk, exposure, and hazard (Chapter 1), followed by an exploration of environmental risks and their sources in real-world contexts (Chapter 2).

Building on these foundations, Chapter 3 introduced the scientific methodology of Health Risk Assessment (HRA), providing a structured and quantitative approach based on hazard identification, dose–response relationships, exposure assessment, and risk characterization. This methodological framework enables a rigorous evaluation of potential health impacts associated with environmental contaminants and supports evidence-based decision-making.

Finally, Chapter 4 extended this analysis toward risk management and governance, highlighting the translation of scientific knowledge into practical actions, regulatory frameworks, and public health policies. It emphasized the importance of prevention strategies, environmental monitoring, regulatory standards, and effective risk communication. The role of national and international institutions was also examined, underlining the necessity of coordinated efforts to address global environmental challenges.

Overall, this course demonstrates that environmental health risk is not only a scientific issue but also a societal and regulatory challenge requiring multidisciplinary approaches. The integration of scientific assessment, policy implementation, and international cooperation is essential to ensure sustainable development and the protection of human health.

In a context marked by increasing environmental pressures, emerging pollutants, and climate change, strengthening risk assessment and management capacities remains a priority. The concepts and tools presented in this course provide a solid foundation for students to understand, analyze, and contribute to the management of environmental health risks in both academic and professional contexts.

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