CHAPTER 1

Sources and Toxicity of Pollutants

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Abstract: Modern living standards depend largely on the production and usage of thousands of chemicals, many of which are toxic and synthetically produced. These substances are discharged into the air, soil, water bodies and the sea through a variety of ways, becoming pollutants of our environment. The investigation of their fate and impacts they have on ecosystems is called ecotoxicology, a multidisciplinary science which intends to evaluate the nature of the discharge, the transformation and distribution of toxicants in the environment, exposure, lethality and sublethal effects on organisms, population responses, and changes in community structure and ecosystem function. The sources and mode of action of some of the most common groups of toxicants are described in this chapter, leaving their fate and effects in organisms and ecosystems for the subsequent chapters.

INTRODUCTION

We are living in the Chemical Era. Indeed, the most distinctive characteristic of our modern society is the production and use of an enormous amount of chemical products. Currently, some 70,000 chemicals are utilised worldwide, while the rate of introduction of new substances can be estimated between 200 and 1000 each year [1]. Our civilization depends to a large extent on the search for new materials that are employed to develop technology, medicines, textiles and construction materials of all kinds. What would happen to us if such production were to stop suddenly?

Throughout history, civilizations have relied on the use of natural materials to manufacture tools, clothing and furnishings, while poisons and medicinal plants must have been known to the first humans. The development of agriculture during the Neolithic (11,000 BP) brought with it the manufacturing of textiles as well as dyes and paints made from minerals, plant and animal products, some of which are quite toxic. With the discovery of metals during the Bronze and Iron Ages (ca. 5000 and 3000 BP, respectively) came mining and consequently pollution by toxic metals. Alchemy started in Persia about 2500 BP, and since then each civilization in Asia looked to develop new substances, mainly for medicinal purposes, using the diverse array of natural products available to them. In Europe, alchemy laid the foundations of toxicology and modern chemistry in the 16th and 17th centuries respectively, which would result in the discovery and manufacturing of hundreds of entirely new substances. During the industrial revolution of the 19th century, mining and chemical companies were created specifically to exploit natural resources and create new products. The discovery of large deposits of crude oil in Baku (Azerbaijan) and North America in the 1850s [2], together with the realisation that petroleum could be used as fuel for combustion engines, boosted the mechanised and oil-dependent society we still live in. As a result of these activities, pollution on a large scale began at that time and still continues despite amelioration efforts by governments and industries in most developed countries.

The technological race that started in the 20th century, particularly since the end of World War II, included chemicals as an essential part of modern development. For instance, communications and transport have benefited enormously from the use of new metals and alloys to make transistors, batteries and more durable metallic products. Synthetic organic compounds, mostly derived from petroleum, underwent a revolution of their own: polychlorinated biphenyls (PCBs), used as insulating fluids in the electrical industry since their introduction in 1929; chlorofluorocarbons (CFCs) used in refrigeration and air conditioning systems; plastics to serve a wide range of uses, from building materials to household items and toys; pesticides to control insect and rodent pests, weeds and plant diseases; and the immense array of chemicals used to make paints, cleaning products, cosmetics and pharmaceuticals. Many of these new substances are toxic, have become environmental pollutants in air, water and soil, and created unforseen problems related to their waste and disposal.

Although the discovery of toxic substances dates from ancient times, their systematic study or toxicology began during the European Renaissance with Paracelsus (1493-1541), a medical doctor and alchemist who sought to understand the effects of toxicants and drugs used in medicine. However, it wasn't until the effects of new pollutants

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from the industrial revolution started to take a toll on ecosystems that people realised the dangers they posed to our environment and our own health. In Japan, a country which experienced the fastest transformation from a feudal to an industrial society, Tanaka Shozo (1841-1913) appealed to the Meiji Emperor in protest against fish kills due to careless discharges from the Ashio copper mine north of Tokyo [3]. It might have been the first time that a local politician tried to protect the environment and the lives of his community by demanding regulation of indiscriminate exploitation of resources. Japan would suffer dearly the consequences of such a rush for industrial development, with Minamata and '*itai-itai*' being added to the infamous list of modern diseases caused by pollutants [4]. These unintended problems prompted a rethink of treating the natural environment as a receptacle for untreated waste, and yet many other industrialised societies would have to endure a large human toll from *smog* before taking any action to regulate the burning of fossil fuels in their cities [5]. In this climate, the publication of *Silent Spring* in 1962 [6] brought to the attention of ordinary people in the street what scientists were still trying to comprehend: the negative effects that pesticides can pose to the environment. Such a book would mark the start of the environmental movement in America and the rest of the world.

The investigation of the ecological impacts that toxic pollutants have on ecosystems constitutes a new science, emerged in the 1970s, called ecotoxicology [7]. Its approach is multidisciplinary, combining the knowledge from chemistry, toxicology and ecology to reach an understanding of the complex interactions of toxicants in the environment. In a broader sense, ecotoxicology has the role of assessing, monitoring and predicting the fate of foreign substances in the environment [8], with the ultimate end of helping the regulatory authorities establish limits that protect human health and nature.

This first chapter aims at providing the reader with a glimpse of the different kinds of pollutants currently in existence, where they come from and how they exert their toxic effects on organisms. Subsequent chapters will examine the ways these chemicals move between air, soil, rivers and oceans, with special attention given to the overall impacts on specific communities and types of ecosystems.

TYPES AND SOURCES OF TOXICANTS

Toxic chemicals that pollute the environment can be called ecotoxicants [1]. They can be natural or man-made substances, but a common characteristic to all of them is that they can exert a deleterious effect on living organisms at relatively small doses, measured in milligrams or micrograms per litre or per kilogram [9]. An important aspect to consider with ecotoxicants is whether they are available to organisms (see risk assessment below). Indeed, pollutants are discharged into the air and water or disposed of in or on the ground, where they may be absorbed by plants or taken up by animals, which may in turn be affected by their toxic activity. By contrast, the vast majority of naturally occurring toxicants (e.g. plant poisons) are stored in tissues that are only available to animals if eaten. In the case of crude oil, natural deposits are many metres underground and out of reach…except to humans! For this reason, biological toxins very rarely become pollutants – these originate mostly from human activities of our modern society (Table **1**). The following is a brief description of the most common types.

Toxins of Biological Origin

Although our knowledge is still limited, the variety of plant and animal poisons is staggering [10], with many of them being utilised as medical drugs or in the production of pesticides – the toxins of the soil microbe *Bacillus thuringiensis*, for instance, are used for pest control, either directly or through transgenic plants [11]. Most natural toxins produced by organisms are used as defence tools in mechanisms that evolved over millions of years, but some animals produce toxic venoms to capture and kill their prey [12]. In any case, very few of these toxins are ecotoxicants: botulin produced by the soil bacterium *Clostridium botulinum*, mycotoxins produced by some species of fungi, cyanotoxins and microcystins produced by certain blue-green algae [13], and saxitoxins and brevetoxins produced by several species of dinoflagellates (e.g. *Alexandrium* sp.) are the most notorious [14], as they can cause fish deaths through algal-blooms and serious intoxication or health problems in humans.

Waste Products

Natural ecosystems recycle the elements through a variety of pathways which end up in mineralization, thus ensuring that all organic wastes in soil, water and sediments are eliminated as soon as possible. Raw sewerage is processed by naturally occurring micro-organisms in waters provided its volume is within the capability of aquatic ecosystems, but

large cities discharge excessive volumes of refuse into rivers, lakes and coasts, which if insufficiently treated can lead to eutrophication of the waters and foster toxic algal blooms [15]. Moreover, stormwater runoff and waste discharges from cities often contain a variety of toxic chemicals, including metals, petroleum hydrocarbons, pharmaceuticals, pesticides [16,17], phenols, steroids and many others which have endocrine disrupting activity [18].

Table 1: Sources of toxic pollutants and their mode of action.

Types of toxicant	Chemical groups	Common sources	Mode of action
Natural	Polycyclic aromatic hydrocarbons (PAH)	Bushfires, fuel emissions	Carcinogens (DNA adduct formation)
	Biological toxins	Micro-organisms (bacteria, fungi, dinoflagellates, blue-green algae)	Gastrointestinal, narcotic, neurotoxic
	Inorganic (SO_2, CO, CO_2, NO_2 SH_2, NH_3, etc)	Volcanic eruptions, coal and vehicle emissions, fertilisers	Blocking of biochemical pathways
	Metallic (As, Cd, Cr, Cu, Hg, Pb, Sn, Sb, Zn)	Mining, smelters, metallurgic and transport industries, electronics, batteries, paints, herbicides	Several modes of action: neurotoxic, blocking respiration and biosynthesis
	Phenolic compounds	Plant wastes, disinfectants	Antioxidants, cell inhibitors, EDC
Artificial	Anilines*	Dyes (textiles, paints), rubber and pharmaceutical industries	Oxidation, dehydrogenation, *etc.*
	Antibiotics*, sulfonylureas and sulfonamides	Biocides, herbicides, pharmaceuticals	Amino acids and/or protein inhibitors
	Benzoylureas	Insecticides	Chitin synthesis inhibitors
	Carboxamides, phthalimides, pyrroles, strobilurins	Biocides	Respiration inhibitors
	Chlorofluorocarbons (CFC)	Coolants, propellants, fire-suppressants, solvents	Greenhouse gasses; ozone depletion
	DDT and synthetic pyrethroids*	Insecticides	Neurotoxic - alter Na^+ channels
	Dibenzodioxins (PCDD), dibenzofurans (PCDF)	Pesticide industry, waste-burning emissions	Disruption of Ah receptor; teratogenic
	Imidazoles, morpholines, triazoles	Fungicides	Ergosterol biosynthesis inhibitors
	Narcotics*	Pharmaceutical industries	Neurotoxic
	Neonicotinoids	Insecticides	Neurotoxic - nicotinic receptor inhibitors
	Nonylphenols, nonylphenol ethoxylates, steroids	Detergent and pharmaceutical industries	Endocrine disrupters (EDC)
	Organochlorines	Insecticides and fungicides	Neurotoxic - GABA receptors; respiration
	Organometallic* compounds	Biocides, antifoulants	Chelating, oxidants, respiration inhibitors
	Organophosphorous and carbamate pesticides	Insecticides, herbicides	Neurotoxic - acetyl-cholinesterase inhibitors
	Perfluorinated compounds (PFOS, etc)	Semiconductors, oil/water-proof materials	Unknown

	Phenoxy and pyridine herbicides	Herbicides	Control/stop plant growth
	Phthalates	Plasticizers, surfactants, pharmaceutical industry	Endocrine disrupters (EDC)
	Polychlorinated biphenyls (PCB)	Electrical insulators, lubricants, paints	Disruption of Ah receptor; EDC
	Polybrominated diphenyl ethers (PBDE)	Fire retardants	Unknown
	Solvents	Chemical industry, paints, cleaning products	Anesthetic or narcotic action
	Synthetic coumarins* and indandiones	Rodenticides	Anticoagulants
	Thiocarbamate, chloroacetamides, dinitroanilines and benzimidazoles	Herbicides and fungicides	Germination inhibitors - microtubules, lipid, fatty acid or cellulose biosynthesis
	Triazines and urea derivatives	Herbicides	Photosynthesis inhibitors

* Some compounds occur naturally as well

Metals and other Elements

All elements, whether they are toxic or not, occur naturally in soils, air, oceans and sediments, usually at very low, non-toxic levels. Some of them are called micronutrients, since they are essential for the synthesis of certain biomolecules: iron, cobalt, chromium, copper, iodine, manganese, selenium, zinc and molybdenum. However, human activities such as mining, manufacturing and transport have produced abnormally elevated levels of many toxic elements, thus causing detrimental effects in the environment [19]. For example, urban soils throughout the world contain high concentrations of lead due to the intense use of leaded-fuel in motor vehicles for many decades. Mine tailings contain very high concentrations of residual metals and constitute a high risk to the surrounding environments, with their accidental release causing enormous damage to aquatic ecosystems and associated fishing industries [20]. Smelters and factories that process large amounts of metals may also be sources of metal pollution through smoke stacks and discharges into waterbodies.

Synthetic and Natural Organic Toxicants

Although the majority of organic toxicants that contaminate the environment are man-made, some can be produced by natural events. Bushfires, for instance, are major sources of air and water contamination by polycyclic aromatic hydrocarbons (PAHs), which are present in nature at very low concentrations – background or baseline levels [21]; however, their environmental levels have increased in recent years due to the large increase of fossil fuels usage worldwide. Crude oil, composed mainly of a cocktail of aliphatic hydrocarbons of biological origin, has been stored safely underground for millions of years [2]; the petroleum spillages that result from the accidental break up of oil-tankers or oil-field shafts, even if they are not very toxic, may have other temporary impacts on the ecosystems exposed [22].

In addition to these natural ecotoxicants there is an immense range of synthetic compounds, man-made for a variety of purposes: pesticides and fertilizers used in agriculture, industrial chemicals such as PCBs and CFCs, chemical reagents, solvents, plasticizers, dyes, surfactants, detergents, pharmaceutical drugs and explosives used in warfare and industrial activity. All these chemicals can be released into the environment through manufacturing, usage, accidental spillage or inefficient disposal. For example, chlorodibenzodioxins (PCDD) and chlorodibenzofurans (PCDF) are by-products formed in the manufacturing of some chlorinated pesticides and also from incineration of chlorophenolic wastes [23].

Inorganic Toxicants

Volcanic eruptions discharge enormous volumes of sulphur dioxide, carbon monoxide and other inorganic poisons into the air (Table **1**), where in addition to their inherent toxicity are subsequently transformed into acids by reacting either with atmospheric water vapour or in direct contact with water [24]. Even carbon dioxide can be toxic at concentrations higher than 2%. Once again, the levels of these toxicants in the environment have increased due to

human activities such as fertiliser usage or burning of coal and petroleum fuels. On a different matter, they can affect the global weather patterns, with CO_2 and NO_2 contributing to the warming of the atmosphere while SO_2 forms aerosols which have the opposite effect [25].

MODE OF ACTION OF TOXICANTS

Toxicants are chemicals that have toxic effects on organisms. Such effects occur when the toxicant, after being taken up through the roots or leaves in plants, or through the skin, digestive or respiratory systems in animals, is transferred by the circulatory fluids to the site of action within the plant or animal body. Some of the original compound, or its biotransformed active products, may reach the site of action, while the remainder may be excreted and metabolised, usually to non-toxic products, or stored in lipid tissues [26].

For most chemicals, the site of toxic action is at the cellular level, and their activity translates into one or more physiological effects which are manifested as several toxicity symptoms in individual organisms. For example, mercurial compounds block the degradation pathway of catecholamines in neuronal cells, so the resulting excess of epinephrine (adrenaline) in the blood stream causes profuse sweating and hypersalivation, whereas excess of dopamine in the brain induces tachycardia and hypertension. But organisms are rarely affected in isolation; since toxic pollutants are spread over certain areas, they usually affect a number of organisms at the same time – effects at the population level can translate into reduction of numbers (mortality) or decreased reproduction success in certain species but not in others. In turn, these changes typically result in altered communities of animals and/or plants, which impact the ecosystem functionality (e.g. biomass productivity, nutrient and predator-prey dynamics) to a lesser or greater degree [27]. Effects at different levels of organization are not always observed: while all ecotoxicants have effects at the individual level, or at most at the population level in acute exposures, impacts on communities and ecosystems depend mainly on the persistence and/or bioaccumulation of the chemicals concerned. However, short pulses can also have a large, long-lasting impact when the recovery of the communities affected is slow or when the ecosystem has been pushed to an alternative stable state.

Inorganic and Elementary Compounds

Inorganic toxicants usually disable the functionality of essential biomolecules; for example, CO binds to haemoglobin and prevents it from transporting oxygen in the blood. Even if some are essential micronutrients, toxic metals (As, Cd, Cr, Cu, Hg, Ni, Pb, Sb, Sn, Zn) tend to form covalent bonds with organic molecules and effectively disable their funcionality:

- Arsenic and antimony compete with phosphorus in several phosphorylation processes, disrupting ATP production; some organoarsenic compounds are neurotoxic [12].
- Cadmium replaces zinc, magnesium and calcium in some metabolic systems; if inhaled through smoking it can induce cancer [28].
- Hexavalent chromium (Cr^{6+}) is mutagenic, and nickel is carcinogenic.
- Copper ions (Cu^{2+}) from copper salts (e.g. $CuSO_4$) accumulate in certain cells such as algae and fungi spores and prevent their germination; in fish, copper ions disrupt the sodium regulation [29], and in mammals can also produce cirrhosis (liver damage), but the mechanism of toxicity is poorly understood.
- Mercury binds to sulfide groups in proteins, but organomercurial forms are much more toxic because they penetrate the tissues and reach the nervous system [12].
- Lead inhibits several enzymes involved in the synthesis of haemoglobin; it also interferes with calcium ions during nerve conduction.
- Zinc competes with copper in the uptake and synthesis of biomolecules; its free ion is a corrosive acid with broad-spectrum biocidal activity. However, organotin compounds inhibit the oxidative phosphorylation and ATP production in mitochondria [30]; tributyltin (TBT) also inhibits the P450 mono-oxygenase detoxification system in fish and marine invertebrates and causes imposex in gastropods [31].

Organic Compounds

Within the enormous variety of organic toxicants, the following are some of the most common mechanisms of toxicity:

a. *Anticoagulants*. Inhibit the regeneration of vitamin K, leading to dysfunctional hepatocytes that prevent blood clotting and, in certain conditions, can cause haemorrhaging. Exclusively used as rodenticides, but they also affect birds [32] and possibly other vertebrates.

b. *Auxin-type disrupters*. Phenoxy and pyridine herbicides (e.g. 2,4-D and triclopyr respectively) stop plant growth by mimicking plant hormones (auxins).

c. *Chemicals destructive of tissues*. Reactive substances such as chemical reagents (acids, formalin, strong bases), anilines and some herbicides (e.g. paraquat and diquat), cause either oxidation or reduction of tissues and consequently their destruction.

d. *Endocrine disrupting chemicals (EDCs)*. Some toxic chemicals mimic the role of hormones that control specific physiological processes in animals. For instance, PCBs disrupt the aryl hydrocarbon (Ah) receptor in the cytosol, triggering the induction of the detoxification complex P450; in some cases, the metabolites thus produced compete with thyroxine, reduce the level of retinol in blood and lead to vitamin A deficiency [12]. Certain phthalates, organochlorines, phenolic and organometallic compounds, and other toxicants described above act in similar ways [33]. Human hormones (synthetic and natural) can be discharged into sewage and cause endocrine disruption in other organisms.

e. *Inhibitors of biosynthetic processes*. Usually selective toxins for some taxa, as they target specific metabolic processes. Antibiotics inhibit the synthesis of specific proteins in bacteria; sulfonamides (e.g. flusulfamide) inhibit specific protein or amino acid synthesis in fungi; sulfonylurea herbicides (e.g. chlorsulfuron) act the same way in plant cells. Glyphosate also inhibits the biosynthesis of essential aromatic amino acids (histidine, phenylalanine, tryptophan, tyrosine), which cannot be produced by animals. Benzoylurea insecticides (e.g. lufenuron) inhibit the production of chitin in arthropods, effectively impeding moulting and stopping development in those organisms.

f. *Germination inhibitors*. Broad-spectrum herbicides and fungicides disrupt cell division. Thiocarbamates (e.g. thiobencarb), chloroacetamides (e.g. metolachlor) and dinitroanilines (e.g. trifluralin) inhibit the synthesis of certain proteins, lipids or fatty acids required for plant germination, while dichlobenil inhibits the production of cellulose. Triazoles (e.g. difenoconazole), imidazoles (e.g. imazethapyr) and morpholines (e.g. fenpropidin) inhibit the synthesis of ergosterol, an essential component of the membranes of fungi; the fungicide benomyl inhibits the synthesis of microtubules, and consequently stop cell division.

g. *Mutagenic, carcinogenic and teratogenic*. Aromatic hydrocarbons (e.g. benzene) and PAHs (e.g. anthracene) form adducts with the DNA, thus causing mutations in the genome that lead to cancers and malformations. In contrast, the aliphatic hydrocarbons (e.g. butane) are not as toxic and can be readily metabolised by bacteria [34]. Dioxins and related compounds act like PCBs but are more toxic and also have teratogenic effects [12].

h. *Neurotoxic*. Most insecticides are included here. Organochlorines (e.g. lindane) inhibit the GABA receptors in neuronal cells, whereas DDT, pyrethrum and synthetic pyrethroids (e.g. cypermethrin) act upon the axonal sodium channels that are voltage dependent [35], altering the nervous impulse and causing convulsions and paralysis. Organophosphorous (e.g. chlorpyrifos) and carbamate (e.g. carbaryl) insecticides inhibit the acetyl-cholinesterase receptor at the neuronal synapses, while nicotine and synthetic neonicotinoid insecticides (e.g. imidacloprid) inhibit the nicotinic receptor, as a result of which the transmission of the nervous impulse is blocked, thus causing hyper-excitability [36]. Narcotic drugs (e.g. morphine) activate the opioid receptors in the brain and spinal cord, causing analgesic and sedative effects.

i. *Photosynthesis inhibitors*. The photosystem II electron transport process, carried out in the chloroplasts within plant cells and algae, can be inhibited by many herbicides: triazines (e.g. atrazine), urea-derivatives (e.g. diuron), bromoxynil, *etc.*

j. *Respiratory inhibitors*. Most biocides are included in this category. The complex III (cytochrome C — oxidoreductase or cytochrome BC_1 complex) involved in electron-transfer mechanisms to produce ATP in the mitochondria and bacteria can be inhibited by some organometallic compounds (e.g. TBT), strobilurin fungicides and other toxicant groups (Table **1**).

BASIC TOXICOLOGICAL PRINCIPLES

Paracelsus established that "All things are poison and nothing (is) without poison; only the dose permits something not to be poisonous" [37]. In toxicology, the degree of exposure or dose is as important as the nature of a chemical. Common salt (NaCl), for instance, can be toxic to most aquatic organisms at concentrations above 6%. For a typical toxicant, the response is represented by a sigmoid curve which indicates increasing toxic effects at increasing doses, usually on a logarithmic scale (Fig. **1**). For essential elements (*i.e.* copper), the relationship is parabolic: at lower than normal doses for growth and development, the organisms may die of nutrient deficiency; as the levels increase within a short range there are not harmful effects, whereas higher doses produce the normal toxic response. In aquatic environments the dose is replaced by the concentration of the toxicant in water, since the internal dose (which causes the effect) is a function of the uptake rate by the organism and the external concentration [38]. The toxicity of a compound is usually evaluated as the dose required to cause a 50 percent effect in an organism (EC50), which in the case of mortality is the median lethal dose or concentration: LD50 or LC50 respectively. The no-observed effect level (NOEL) and lowest-observed effect level (LOEL) are also used, but they are less reliable measures and sometimes difficult to obtain.

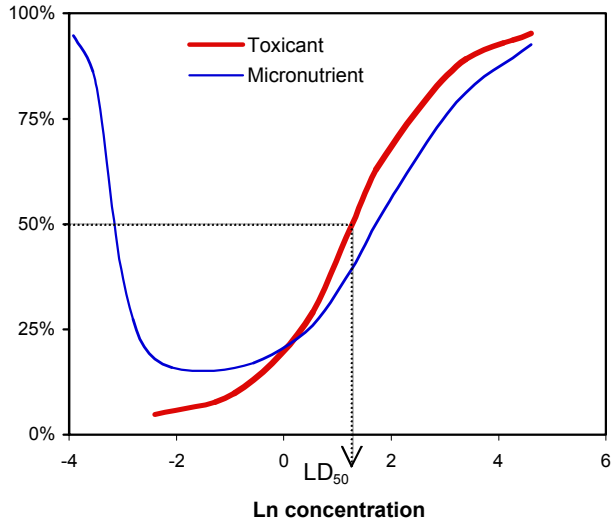

Figure 1: Dose-effect relationships.

In addition to dose, time is an important factor to be considered in toxicology. Indeed, the same effect can be produced by a large dose applied to an organism at once than by repeated small doses applied over a certain period of time (chronic exposure), as long as the toxicant is not degraded (Fig. **2**). The exposure time is in fact related to the actual internal dose, and both are linked by a linear relationship in the logarithmic scale [39]. Obviously this relationship is more important where a continuous input occurs or in the case of persistent ecotoxicants, which can linger in the environment for months or years, and can be accumulated in the tissues of the organisms affected. For example, many chlorinated organic toxicants (PCBs, PCDDs, DDT and other pesticides) are recalcitrant and non-polar compounds with a tendency to be stored in fatty tissues because they are lipophilic; the remobilization of these toxins from the body fat during periods of starvation or exertion, such as long migration, can take them to the sites of action (e.g. neuronal system) and produce a toxic effect [40].

Finally, sublethal effects – those effects caused by doses smaller than the LD50 or acute NOEL – are commonly found after chronic exposure of organisms to low levels of ecotoxicants. These effects are usually unrelated to the specific mode of action of the chemicals, and therefore are unpredictable. For example, some organochlorine insecticides which are neurotoxic at relatively high doses (mg/kg body weight), can also produce endocrine disruption when present at very low doses (µg/kg body weight) [41]. In other instances, such as DDT, the metabolite DDE causes the thinning of egg-shells and consequently can lower the hatching success of many bird species [42].

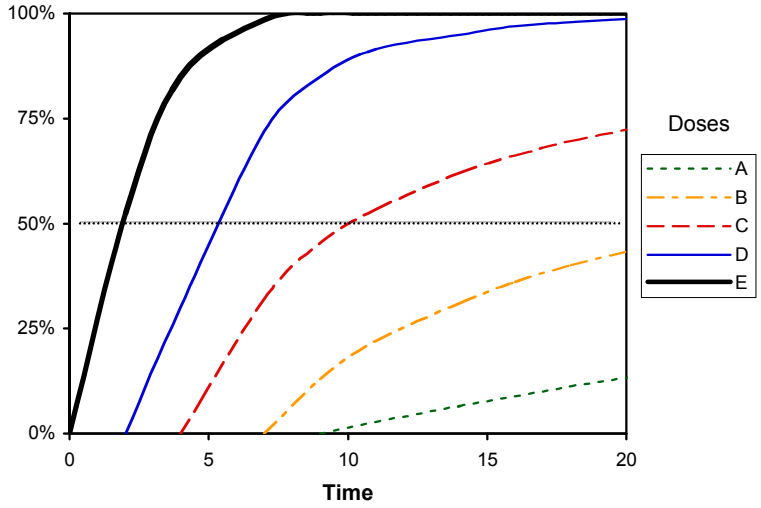

Figure 2: Time-to-effect relationships.

ECOLOGICAL RISK ASSESSMENT

The hazardous substances that pollute our environment (Table **1**) can cause detrimental effects only when organisms are exposed to them at sufficient, toxic doses. Ecological risk assessments aim at determining the extent of harm caused to the environment by the release of toxic chemicals. Such assessments consider the exposure of organisms and inherent toxicity of the substances as the two main components, and usually indicate the impacts in terms of probabilities [43]. Most risk assessments refer to individual toxicants discharged over specific areas either in one event (e.g. accidental spills) or repeatedly (e.g. continuous industrial effluents, pulsed pesticide applications), and consequently tend to be site-specific.

The exposure of organisms to toxicants depends on many factors: distribution and concentration of the chemicals in water, air, soil and sediment; uptake through the roots and/or leaves in plants or through contact, ingestion and inhalation in animals; persistence of the residues in the environment and bioaccumulation in tissues; proximity to the source of emissions and probability of being affected. The toxicity component is based on experimental laboratory data, which derive the LD(C)50 or NOEL (acute or chronic) of the chemical for certain species of organisms such as algae, *Daphnia*, worms, insects, fish, birds and mammals. Typically, endpoints include mortality as well as sublethal effects. A chemical's hazard indicates the danger it may pose to organisms; risk implies the probability of being affected. The simplest way to assess the risk is by comparing the amounts of chemical present in water, air, soil and sediment with the toxic endpoints to each organism [44]:

$$HQ = \frac{\text{Predicted Environmental Concentration (PEC)}}{\text{Toxicity endpoint (LD(C)50 or NOEL)}}$$

If the hazard quotient (HQ) between the two numbers is greater than 1 the organisms would be at high risk, meaning that more than 50% of the individuals of a given species may die or experience sublethal effects. Obviously, the PEC can be replaced by actual measured concentrations. Based on years of experience and numerous field trials – mainly with insecticides – it is concluded that for an ecotoxicant to be considered 'safe' to organisms, the HQ should be smaller than 0.1 [45]. Since the toxicity endpoints are fixed for each chemical and species the main variable of the quotient is the environmental concentration – this forms the basis for establishing contaminant guidelines for air, soil/sediment and water quality, to ensure that levels of pollutants are below harmful thresholds. In any case, such quotients only indicate potential hazards to certain species, while additional information on the chemical's persistence, bioaccumulation factors and probability of actual exposure to a community of species, under normal and worst-case scenarios, are needed for a more comprehensive risk assessment to ecosystems [46].

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