Chapter 1
Introduction to Environmental Pollutants and Human Exposure

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The new concept of health, developed in the recent years, considers the person's well-being more heterogeneously. A new model that considers the relationship between human health and the environment has strongly emerged during the last three decades. Our state of well-being is continually threatened by a series of internal and external disturbing factors, which tend to move the body away from a condition of homeostasis. The awareness of the indissoluble link between human health and the environment is increasingly widespread.

Climate change, loss of biodiversity, poor air quality, desertification, deforestation, often irreversible contamination of groundwater and the food chain, and exponential growth of the electromagnetic field (EMF) due to over-the-air communications are the direct consequences of a focused "growth" of globalized economics.

Pollution is a problem that affects organisms, especially the developing ones, such as embryos and children, in consideration of the vulnerability of their status. Prolonged exposure to minimal quantities of pollutants can progressively alter the functioning of cells, tissues, and organs, essentially interfering with deoxyribonucleic acid (DNA) expression. Unfortunately, the absolute limits of toxicity and tolerability of many pollutants are not yet known.

Today, we are detecting a rapid and progressive transformation of the molecular composition of the ecosphere and, in particular, the rapid production and diffusion of atmospheric pollutants (ultra-fine particles, heavy metals, and radiation). The World Health Organization (WHO), indeed, has recognized that environmental factors cause around 24% of diseases worldwide, and more than 33% of diseases in children under the age of 5 years are due to environmental factors [1].

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The pediatric age is much more sensitive than adults to the effect of pollutants. Children under the age of 5 years, who represent only 12% of the population, contract more than 40% of health diseases compared with adults [2]. The role of the environment has been recognized over the years in the pathophysiology of numerous pathologies. The WHO, for example, has defined obesity and diabetes as a real pandemic. Even in Italy, the phenomenon is assuming worrying proportions, particularly in children in primary schools whose obesity rate, which was 7% between 1976 and 1980 and reached 21% in 2015–2017 [3]. Childhood obesity is generally considered a systemic and multifactorial pathology, determined by several causes (excessive intake of food, sedentary lifestyle, and genetic predisposition). However, it is increasingly evident that these factors cannot alone explain the alarming phenomena, such as the recent, dramatic increase in cases and the constant anticipation of the age of onset of related diseases (in particular insulin resistance and type 2 diabetes mellitus) and the insufficient efficacy of individual therapeutic strategies. Recent studies have shown that early exposure to many pollutants can induce obesity and type 2 diabetes [4].

Numerous types of pollutants contribute to air pollution. Transportation, industrial and agricultural activities, energy production, and waste disposal plants emit thousands of tons of pollutants into the atmosphere every day, and they are the leading causes of environmental pollution. The main pollutants studied are ground-level ozone, heavy metals, polyaromatic hydrocarbons, and particulate, which constitute a serious threat to our health [5].

Air pollution affects health in various ways: The subject's health conditions, age, and duration of exposure are the main factors that affect the way through which pollutants influence our health. Air pollutants can have effects on both the respiratory tract and other organs, inducing or contributing to the onset of numerous diseases, including respiratory diseases such as asthma (especially in pediatric age), reduction in the development and functions of the apparatus respiratory tract, arteriosclerosis and cardiovascular diseases, neurodegenerative diseases, tumors, and infertility.

Human beings can be exposed to environmental contaminants through the air, water, food, and soil. Environmental contaminants can be divided into three broad categories: biological agents, chemical agents, and radiation (Fig. 1.1).

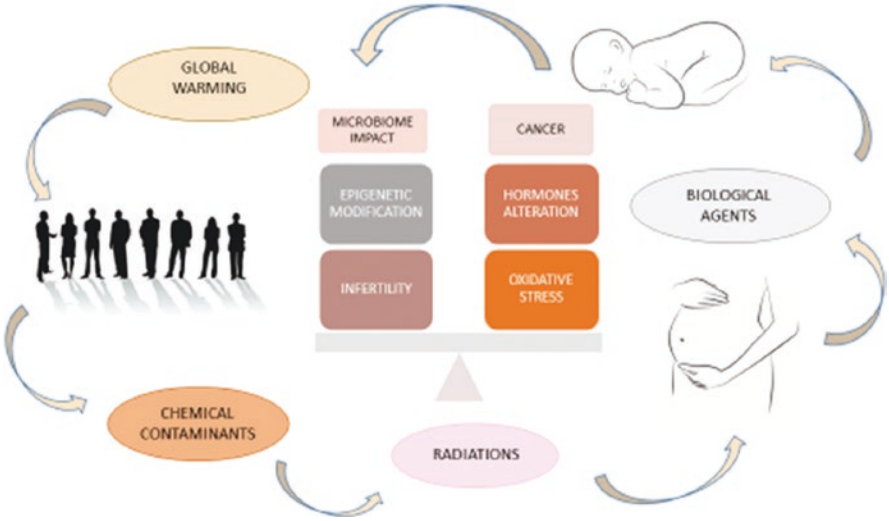

Fig. 1.1 Pollution and interaction with human health

1.1 The Biological Agents

Biological agents are living organisms such as bacteria, viruses, and fungi that are naturally present in the environment and can be responsible for gastrointestinal, allergic, and respiratory diseases. [6].

The biological agent is defined, according to the current legislation (European Directives 90/679/EEC, 93/88/EEC, and 2000/54/EC) [7], as "any microorganism, even if genetically modified, cell culture and human endoparasite, which could cause infections, allergies or poisoning".

The onset of diseases depends on many factors related to the characteristics of the single biological agent, the conditions of the subject exposed, the environmental conditions, and the methods of exposure or contact.

Although there is extensive information about the dangers of chemical and physical agents, the same cannot be said for biological agents [8].

Biological agents are infectious agents that include bacteria, rickettsiae, viruses, yeasts, molds, and single and multicellular parasites.

Each infectious agent species can have subtypes, strains, and variants that differ from the parental pathogenic potential, host specificity, transmissibility, and sensitivity to antimicrobial agents.

According to Italian Legislative Decree 81/08 Title X [9], biological agents are divided into four groups according to the risk of infection:

1. Biological agent of group 1: an agent that is unlikely to cause disease in human subjects.
2. Biological agent of group 2: an agent that can cause disease in human subjects and pose a risk to workers; it is unlikely to spread to the community; effective prophylactic or therapeutic measures are usually available.
3. Biological agent of group 3: an agent that can cause severe diseases in human subjects and constitutes a severe risk to workers; the biological agent can spread throughout the community, but effective prophylactic or therapeutic measures are usually available.
4. Biological agent of group 4: a biological agent that can cause severe diseases in human subjects and constitutes a severe risk to workers and can present a high risk of propagation in the community; there are usually no effective prophylactic or therapeutic measures available.

1.2 Radiations

Radiations are waves or particles of energy to which humans, plants, and animals are exposed. We are exposed to natural and artificial sources of high-energy ionizing radiation and low-energy nonionizing radiation, such as ultraviolet rays and electromagnetic fields.

Ionizing radiations (X-, gamma-, alpha-, beta-, and neutron rays) are electromagnetic waves or corpuscular rays with enough energy to release electrons from atoms as they pass through matter. These modifying atoms, called ions, can induce chemical reactions that cause a biological damage. At the cellular level, radiation can induce damage to DNA molecules [10]. This damage can cause cells to die or be adequately repaired by the cell's protective mechanisms. However, it can also happen that an incorrect repair is made, which still produces a viable cell. Radiation-mediated cancer is assumed to be induced by the latest mechanism.

There are two types of radiation effects on the body. The long-term effects are due to mutations produced at the cellular level. These include the onset of cancer in irradiated people and malformations in their descendants. For these effects, it is not possible to identify a threshold; the same could theoretically occur even with a shallow dose. The risk of cancer development is hypothesized to increase linearly with dose. It should be noted that children are more sensitive than adults to ionizing radiation [11].

On the other hand, the immediate effects are linked to the destruction of a large number of cells by radiation, which leads to the loss of functionality of an organ. These effects, for which there is a threshold dose (a minimum dose with which they occur), occur only at high doses. These effects include destroying the active bone marrow, intestinal mucosa, skin burns, and sterilization. [12].

Exposure to electromagnetic fields (EMFs) represented another source of health risks. For several decades, it has been known that there has been an increase in leukemia among residents near EMF [13]. In 2011, the International Agency for Research on Cancer (IARC), the European cancer research agency, definitively included cell phones and radio frequency (wireless) electromagnetic fields among the "Group 2B" carcinogens, which indicates a "possible" carcinogenic risk on humans. In addition, in this case, it is imperative to reduce the exposure of women during pregnancy and of developing subjects. In this case, too, children represent the most risk category for various reasons:

- The exposure is destined to last for decades.
- The brain is in the process of a functional organization (synapses and circuits).
- The blood–brain barrier is very permeable.
- The part of brain tissue exposed is, in proportion, much more significant than in adults.
- The bone tissue is less thick.
- The tissue has higher water content and higher cerebral concentration and therefore conducts and absorbs more energy.

1.3 Chemical Contaminants

Chemical contaminants include organic and inorganic compounds of natural and human origin. Organic compounds contain carbon, usually combined with hydrogen and other elements such as fluorides, chlorides, bromides, iodides, nitrogen, sulfur, and phosphorus. Examples of organic compounds are pesticides, polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and trihalomethanes (THMs). Inorganic compounds include air pollutants, such as ozone, nitrogen oxides, and sulfur dioxide; metals; lead; and fluorides. Chemicals occur naturally in our environment due to weathering and erosion and are also released by human activities, such as agriculture, industry, power generation, transport, and the use and disposal of consumer products. Exposure to high levels of chemical contaminants can result in a variety of health effects, including allergies; skin and eye irritation; heart, respiratory, reproductive, kidney, or neurological problems; and cancer [14].

In particular, ozone is an odorless and a colorless gas present both in the earth's upper atmosphere (stratosphere) and at the ground level (troposphere). The ozone present at high altitudes constitutes a protective band from solar radiation. In the lower layers of the atmosphere (tropospheric ozone), on the other hand, it behaves as a pollutant, constituting a severe problem for public health. Ground-level ozone is a secondary pollutant produced by the reaction of oxygen with nitrogen dioxide (NO_2) and the contribution of volatile organic compounds in intense solar radiation and high temperatures [15].

Particulate matter is a mixture of solid and liquid particles suspended in the air that reaches its maximum concentration in winter. It includes particles of various

sizes into which dust, earth, materials from roads, pollen, molds, spores, bacteria, viruses, and thousands of chemicals can converge and causes respiratory, cardiovascular, and neurodegenerative diseases. The primary sources of particulate matter are vehicular traffic, industrial activities, and heating systems. The most dangerous particulate fraction is the particulate resulting from the product of thermochemical reactions in foundries, cement factories, steel mills, waste incinerators, diesel engines, and other combustion processes. Thanks to its submicroscopic dimensions, the particulate passes through the alveoli and penetrates the arteries, the brain, and the cell nuclei, opening the way to many chronic degenerative, inflammatory, and cancerous diseases [16].

1.3.1 Endocrine Disruptors

Since the 1990s, an increasingly growing scientific interest has been placed in studying endocrine disruptors (EDs). In 2016, the UN Environment Programme commissioned the International Panel on Chemical Pollution (IPCP) to develop three reports relating to the various EDs and their mechanisms of action [17–19]. In June 2018, the European Food Safety Authority (EFSA) and the European Chemicals Agency (ECHA) published a guide to identifying substances with the characteristics of EDs [20]. There are numerous substances considered probable EDs. Currently, 107 substances have passed the complete evaluation process to be identified as EDs (Table 1.1) as the European Union (EU) regulated under the Plant Protection Products Regulation (PPPR), of the Biocides Products Regulation (BPR) or Registration, Evaluation, Authorisation and Restriction (REACH) (the list of candidates and authorizations). EDs include a wide range of chemicals that can alter the hormonal balance of living organisms, including humans. EDs interact with the standard biochemical signals released by the glands of our body, which are responsible for regulating extremely delicate functions: immune, endocrine, metabolic, reproductive, and neuropsychic. The pathologies induced by frequent exposure to minimal doses of EDs are thyroid and neurodevelopmental disorders, abortion, infertility, genital and reproductive anomalies, endometriosis, obesity and type 2 diabetes, tumors, and immune-mediated diseases [21]. According to the Istituto Superiore Di Sanità, "an ED is an exogenous substance, or a mixture, which alters the functionality of the endocrine system, causing adverse effects on the health of an organism, or of its progeny or (under) population".

EDs act subtly, even at minimal doses, especially in crucial stages of development, such as intrauterine life [22–26] or childhood [27]. Exposure to EDs can also alter gametes and implantation mechanisms [28]. Harmful effects have been found in various female pathologies [29] and neoplasms such as endometrial cancer [30, 31].

The impact of EDs on the environment can be considered for their ubiquitous presence, in some cases, their persistence and their potential effects on living beings.

The primary sources of EDs' environmental risk are behaviors that do not comply with current legislation, industrial processing and disposal processes, and incorrect

Table 1.1 Substances identified as endocrine disruptors at EU level (data latest update 2022/4: https://edlists.org/)

Name and abbreviation	Health effects	Environmental effects
(±)-1,7,7-trimethyl-3-[(4-methylphenyl)methylene]bicyclo[2.2.1] heptan-2-(4-MBC)	X	X
(1R,3E,4S)-1,7,7-trimethyl-3-(4- methylbenzylidene) bicyclo[2.2.1]heptan-2-one (4-MBC)	X	
(1R,3Z,4S)-1,7,7-trimethyl-3-(4- methylbenzylidene) bicyclo[2.2.1]heptan-2-one (4-MBC)	X	
(1R,4S)-1,7,7-trimethyl-3-(4-methylbenzylidene)bicyclo[2.2.1] heptan-2-one (4-MBC)	X	
(1S,3E,4R)-1,7,7-trimethyl-3-(4- methylbenzylidene) bicyclo[2.2.1]heptan-2-one (4-MBC)	X	
(1S,3Z,4R)-1,7,7-trimethyl-3-(4-methylbenzylidene) bicyclo[2.2.1]heptan-2-one (4-MBC)	X	
(3E)-1,7,7-trimethyl-3-(4-methylbenzylidene)bicyclo[2.2.1] heptan-2-one (4-MBC)	X	
1,7,7-trimethyl-3-[(4-methylphenyl)methylene]bicyclo[2.2.1] heptan-2-one; 3-BC		X
14-(nonylphenoxy)-3,6,9,12-tetraoxatetradecan-1-ol		X
17-(4-nonylphenoxy)-3,6,9,12,15-pentaoxaheptadecan-1-ol		X
2-(4-nonylphenoxy)ethanol		X
2-[2-(4-nonylphenoxy)ethoxy]ethanol		X
2-[2-[2-[2-(4-nonylphenoxy)ethoxy]ethoxy]ethoxy]ethanol		X
2-[4-(1,1,3,3-tetramethylbutyl)phenoxy]ethanol		X
2-[4-(3,6-dimethylheptan-3-yl)phenoxy]ethanol		X
2-{2-[4-(2,4,4-trimethylpentan-2-yl)phenoxy]ethoxy}ethanol		X
2-{2-[4-(3,6-dimethylheptan-3-yl)phenoxy]ethoxy}ethanol		X
20-(4-nonylphenoxy)-3,6,9,12,15,18-hexaoxaicosan-1-ol		X
20-[4-(1,1,3,3-tetramethylbutyl) phenoxy]-3,6,9,12,15,18-hexaoxaicosan-1-ol		X
23-(nonylphenoxy)-3,6,9,12,15,18,21-heptaoxatricosan-1-ol		X
26-(4-Nonylphenoxy)-3,6,9,12,15,18,21,24- octaoxahexacosan -1-ol		X
26-(nonylphenoxy)-3,6,9,12,15,18,21,24-octaoxahexacosan-1-ol		X
3,6,9,12-Tetraoxatetradecan-1-ol, 14-(4-nonylphenoxy)-		X
4-(1-ethyl-1-methylhexyl)phenol		X
4-(1-Ethyl-1,3-dimethylpentyl)phenol		X
4-(1-Ethyl-1,4-dimethylpentyl)phenol		X
4-(1,1,3,3-tetramethylbutyl)phenol		X
4-(1,1,5-Trimethylhexyl)phenol		X
4-(2-methylhexan-2-yl)phenol		X
4-(2,2-dimethylpentan-3-yl)phenol		X
4-(2,3-dimethylpentan-2-yl)phenol		X
4-(2,3,3-trimethylbutan-2-yl)phenol		X

(continued)

Table 1.1 (continued)

Name and abbreviation	Health effects	Environmental effects
4-(2,4-dimethylpentan-2-yl)phenol		X
4-(2,4-dimethylpentan-3-yl)phenol		X
4-(3-ethylheptan-2-yl)phenol		X
4-(3-ethylpentan-3-yl)phenol		X
4-(3-ethylpentyl)phenol		X
4-(3-methylhexan-2-yl)phenol		
4-(3-methylhexan-3-yl)phenol		X
4-(3-methylhexyl)phenol		X
4-(3,3-dimethylpentan-2-yl)phenol		X
4-(4-methylhexan-2-yl)phenol		X
4-(4-methylhexyl)phenol		X
4-(4,4-dimethylpentan-2-yl)phenol		X
4-(5-methylhexan-2-yl)phenol		X
4-(5-methylhexan-3-yl)phenol		X
4-(5-methylhexyl)phenol		X
4-(heptan-2-yl)phenol		X
4-(heptan-3-yl)phenol		X
4-(heptan-4-yl)phenol		X
4-heptylphenol		X
4-isododecylphenol	X	X
4-Nonylphenol, branched, ethoxylated		X
4-Nonylphenol, branched, ethoxylated 1–2.5 moles ethoxylated		X
4-Nonylphenol, ethoxylated 1–2.5 moles ethoxylated		X
4-t-Nonylphenol-diethoxylate		X
4-tert-butylphenol		X
4,4′-(1-methylpropylidene)bisphenol: Bisphenol B	X	X
4,4′-isopropylidenediphenol; Bisphenol A	X	X
Benzyl butyl phthalate (BBP)	X	
Bis(2-ethylhexyl) phthalate (DEHP)	X	X
Butyl 4-hydroxybenzoate; Butylparaben	X	
Cholecalciferol	X	X
Dibutyl phthalate (DBP)	X	
Dicyclohexyl phthalate (DCHP)	X	
Diisobutyl phthalate (DIBP)	X	
Formaldehyde, reaction products with branched and linear heptylphenol, carbon disulfide and hydrazine		X
Formaldehyde, reaction products with phenol heptyl derivs. and 1,3,4-thiadiazolidine-2,5-dithione		X
Isononylphenol		X
Isononylphenol, ethoxylated		X
Mancozeb	X	X

Table 1.1 (continued)

Name and abbreviation	Health effects	Environmental effects
Nonylphenol		X
Nonylphenol, branched, ethoxylated		X
Nonylphenol, branched, ethoxylated 1–2.5 moles ethoxylated		X
Nonylphenol, ethoxylated		X
Nonylphenol, ethoxylated (10-EO)		X
Nonylphenol, ethoxylated (15-EO)		X
Nonylphenol, ethoxylated (6,5-EO)		X
Nonylphenol, ethoxylated (8-EO)		X
Nonylphenol, ethoxylated (EO = 10)		X
Nonylphenol, ethoxylated (EO = 4)		X
Nonylphenol, ethoxylated (polymer)		X
Nonylphenolpolyglycolether		X
p-(1-methyloctyl)phenol		X
p-(1,1-dimethylheptyl)phenol		X
p-(1,1-dimethylpropyl)phenol		X
p-isononylphenol		X
p-nonylphenol		X
Phenol, (tetrapropenyl) derivatives	X	X
Phenol, 4-(1-ethyl-1,2-dimethylpropyl)-		X
Phenol, 4-dodecyl, branched	X	X
Phenol, 4-isododecyl-	X	X
Phenol, 4-nonyl-, branched		X
Phenol, 4-nonyl-, phosphite (3:1)		X
Phenol, 4-tert-heptyl-		X
Phenol, dodecyl-, branched	X	X
Phenol, heptyl derivs		X
Phenol, nonyl-, branched		X
Phenol, p-isononyl-, phosphite (3:1)		X
Phenol, p-sec-nonyl-, phosphite		X
Phenol, tetrapropylene-	X	X
Poly (oxy-1,2-ethanediyl), alpha-(nonylphenyl)-omega-hydroxy-, branched		X
Poly(oxy-1,2-ethanediyl), a-(nonylphenyl)-w-hydroxy-		X
Poly(oxy-1,2-ethanediyl),α-[(1,1,3,3-tetramethylbutyl)phenyl]-ω-hydroxy-		X
Polyethylene glycol p-(1,1,3,3-tetramethylbutyl)phenyl ether		X
Tris (4-nonylphenol, branch) phosphorous acid ester		X
Tris(nonylphenyl) phosphite		

disposal of products containing plastics, glues, and paints. EDs characterized by high environmental persistence have a greater accumulation capacity in organisms.

The pollutants that can interfere with the function of sex hormones are of particular importance for all organisms, especially for their effects on the conservation

of species and the maintenance of biodiversity. Transfer from one organism to another occurs through the food chain, increasing concentrations along the food chain. The presence of EDs in the environment is assessed through environmental monitoring using water samples, soil and sediments, and sentinel animals (indicator organisms). By comparing the data obtained, the state of environmental quality and the effects on organisms are determined. The PREVIENI project, for example, is an integrated study on the risk assessment of contaminants (perfluorooctane sulfonate [PFOS] and perfluorooctanoic acid [PFOA], di-2-ethylhexyl phthalate (DEHP) and its active metabolite mono-2-ethylhexyl phthalate (MEHP), and bisphenol A [BPA]) in ecosystems and the human population promoted by the Ministry of the Environment and the Protection of the Territory and the Sea and with the support of the World Wildlife Fund (WWF), Italy. EDs and the related biomarkers measured by the PREVIENI in sentinel organisms (four animal species representative of different habitats: earthworm, barbel, trout, and coot) represent values that can be associated with a state of exhibition background, contributing to the evaluation of reference values ([32]). The second research program of the study is based on the case–control approach for the assessment of fertility. The results show how factors associated with lifestyle and environmental exposure to EDs represent independent risk factors for human reproductive health [33].

Due to their complex nature, exposure to EDs can result in numerous clinical phenotypes: Understanding the mechanisms of action represents a constantly evolving field of research. The heterogeneity of the compounds helps to hinder the identification of a common mechanism of action. Suppose some substances exhibit some similar characteristics, such as molecular weight or the presence of specific highly reactive groups. In such case, no common characteristic can be identified among all the EDs, and therefore, the generalization, or even less the prediction, of the exact mechanism of action is impossible [34]. However, it is possible to divide the entire group of EDs into three broad categories:

- Hormone agonists, whose intake involves, directly or indirectly, a phenomenon of receptor activation (hyperstimulation); phytoestrogens and thyroid-stimulating substances are into this category.
- Hormone antagonists can interact with hormone receptors preventing, directly or indirectly, their physiological activation (inhibition); for example, substances with antiestrogenic and antiandrogenic actions belong to this group.
- -Metabolic modifiers can interfere with the physiological endogenous hormone secretion or other stages of the regular action of hormones, including their transport in the blood, intracellular preprocessing or postprocessing and, therefore, their degradation and elimination. Substances that stimulate hepatic metabolism or can chelate circulating hormones can be included in this category.

Despite significant advances in this field, research in the world of EDs has not so far led to irrefutable scientific evidence. The main criticalities encountered in the study of EDs are remarkably heterogeneous. Many substances have a short half-life, others have very low molecular weights, and others still act through metabolites; therefore, identifying these substances is not simple and requires adequate instrumentation and considerable clinical background. In many cases, the intake of

EDs does not have immediate effects. Exposure before puberty or during intrauterine life can, for example, lead to significant effects on fertility after many years [24, 26, 35]. The foundations of many adult pathologies could be traced back to the exposure to EDs during life in utero. Epigenetic effects, as in the case of the modulation of DNA expression by methylation, can be transmitted to generation, sometimes without giving apparent clinical manifestations in indirectly exposed subjects [36]. Most studies on EDs aim to identify the effects and mechanisms of action of individual substances. However, given the heterogeneous nature of EDs, it is safe to assume that their distribution is ubiquitous and that simultaneous exposure to multiple substances is anything but theoretical. The possible interactions between different substances are largely ignored: The mechanisms of action of several EDs may be additive or even synergistic, leading to more striking manifestations in the face of less exposure to the individual components. In addition, long-term exposure to low doses of EDs often makes it challenging to identify a causal link between the agents and the clinic.

Another problem is related to animal models that are commonly used to evaluate the effects of some substances in vivo; however, it is difficult to provide an adequate estimate of how much the results can be extrapolated to the human being, if only for the temporal dynamics. Moreover, studies aimed at identifying the effects of individual substances are hardly representative of reality. Furthermore, some substances may be inert if studied individually and biologically active following the presence of other agents or within complex biological matrices such as blood. Furthermore, it should be considered that a similar exposure to a mixture of EDs can result in clinical manifestations of a different entity in male or female subjects. Despite all these limitations, animal models still represent the most reliable study method for EDs.

1.4 Global Warming

Man exerts an increasing influence on the Earth's climate and temperature through fossil fuels, deforestation, and cattle breeding.

These activities add vast amounts of greenhouse gases to those naturally present in the atmosphere, fueling the greenhouse effect and global warming.

The leading cause of climate change is the greenhouse effect. Some gases in the Earth's atmosphere act like glass in a greenhouse: They capture the sun's heat, preventing it from returning to space and causing global warming.

Many of these gases occur naturally, but human activity increases the concentrations of some of them in the atmosphere, in particular carbon dioxide (CO_2), methane, nitric oxide, and fluorinated gases.

The CO_2 produced by human activities is the main element of global warming. In 2020, its concentration in the atmosphere was 48% above the preindustrial level (before 1750).

Other greenhouse gases are emitted by human activity in smaller quantities. Nitric oxide, like CO_2, is a long-lived greenhouse gas that accumulates in the atmosphere for decades and even centuries. Methane is a more potent greenhouse gas than CO_2 but has a shorter atmospheric life.

Natural causes, such as changes in solar radiation or volcanic activity, are estimated to have contributed less than 0.1 °C to total warming between 1890 and 2010.

The period 2011–2020 was the hottest decade, with an average global temperature of 1.1 °C above the preindustrial levels in 2019. Human-induced global warming is currently increasing at a rate of 0.2 °C.

A 2 °C increase over the preindustrial temperature is associated with severe impacts on the natural environment and human health and well-being, including a much higher risk of dangerous and catastrophic changes in the global environment [37].

For this reason, the international community has recognized the need to keep warming well below 2 °C and to continue efforts to limit it to 1.5 °C.

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