1

Marine Ecosystems under Toxic Pressure

1.1. Introduction

In terms of the pressures of anthropogenic origin to which marine ecosystems are subjected, the ideas that spring to mind are intensive fishing, indirect forms of destruction, the destruction of habitats – by fishing equipment during the exploitation of the deep sea, the development of ports – eutrophication, plastic macrowaste, etc. On the subject of toxic pressure, we can also mention the incidents of accidental pollution, of which the explosion of the oil-rig Deepwater Horizon, in the Gulf of Mexico in February 2013, recently gave us a sad example, or even, chemical shipwrecks, such as that of the Ievoli Sun in October 2000.

These events are indeed particularly striking because of the extent of the immediate mortalities that they cause, which are generally short-lived. However, the toxic pressures that marine ecosystems undergo due to the chronic and ubiquitous contamination of the environment by multiple contaminants are less well known and understood by the general public.

The first research on metallic trace elements in marine environments dates from the 1970s, and belongs mostly to the studies

---

Chapter written by Véronique LOIZEAU and Marie-Hélène TUSSEAU-VUILLEMIN.

of geochemists, searching to understand the global cycle of the elements of the Earth's crust, including some toxic metals, such as mercury, lead and cadmium and other essential metals, such as zinc and copper (see the reference work *Tracers in the Sea* by Broeclker and Peng [BRO 82]). Nevertheless, the goal, above all, is then to arrive at a precise knowledge of the quantity of these metals in seawater, which contains so little of them that handling them carries high chances of contamination... These metals are not, therefore, being studied as toxic pollutants. The emergence of ecotoxicology (the term was coined for the first time by René Truhaut in 1969) will very gradually lead to an understanding at least of the local effects in the sea, when accidents occur, or indeed in the vicinity of pollutant refuse. However, it was only with the application of the 1996 law on water that treatment plants emptying waste into the sea saw themselves obliged to control their emissions. Until then, it was thought that the oceans' power to dilute waste was sufficient protection. Finally, more recently, developments in analytical environmental and organic chemistry allowed the detection, in all environments, of organic xenobiotic[1] substances with toxic effects. Environmental chemists and ecologists became aware of the fact that the ocean, the environment where terrestrial life originated (see Chapter 1 of [MON 14c] also from the Seas and Ocean set of books), but which mankind does not inhabit, had nevertheless absorbed manmade chemical emissions and that marine ecosystems were living with this chronic pressure.

Since then, the major challenge facing scientists has been to understand how marine ecosystems behave under toxic pressure, what evolutions and adaptations this pressure causes, and at what cost (metabolic, phenotypic and genetic). In effect, other major pressures are being exerted, among them climate change and acidification of the oceans, etc. (see [MON 14a, b and c] also from the Seas and Oceans set). And the future of the entire biosphere is directly linked to the oceans' capacity to sustain significant primary production, trapping atmospheric carbon dioxide, which is the basis whole of the

---

[1] A substance present in a living organism, which is nevertheless foreign to it.

trophic oceanic chain, which feeds not only the sea birds... but also people.

First, these are the details of the ocean environment that are affected *vis-à-vis* the toxic pressure. Then, the biological responses will be described at the level of individuals exposed to toxic pressure, independent of each another ("direct effects"). Finally, the focus turns to the group of effects known as "indirect effects", that is to say, those that affect the relationships between the individuals of which an ecosystem is composed. Little is yet known about these indirect effects, but initial observations have tended to show that they are the primary impact; understanding the behavior of these systems under toxic pressure has to be taken into account.

1.2. Details of the marine environment

All aquatic environments are subject to pollution of anthropogenic origin, and all the associated ecosystems are subject to the toxic stress that results. The ocean, because of its dimensions – it is the most vast of the biosphere's ecosystems (1.4 billion km^3) whose depth reaches, on average 3,800 m – and because of its distance from the continents appears relatively protected in comparison to rivers and lakes. Rivers and lakes are often very directly impacted by human use: runoff from agricultural land or soil that has been made impermeable, sources of diverse phytosanitary products, hydrocarbons, dioxins, metals, etc.; they are the recipients of more or less well-treated collection networks, sources of molecules from pharmaceutical synthesis, cosmetics, detergents, products from eroded materials, etc.; outlets, finally, of the widespread contamination of our environment by extremely varied products (see also Chapters 2 and 3). Locally, the impacts of these contaminations can be very pronounced (for example [DED 09], chemosphere), even if they are difficult to prove, because of the mobility of flowing water [FEC 14] and the physico-chemical variability of these environments: diurnal variations in pH and temperature, seasonal variations in organic matter and in shade from forest cover, regional variations in the concentration of eroded minerals, etc., all are modulating factors in the bioavailability of the contaminants [TUS 07].

Assessment of the contamination of the marine environment – which is vast, chemically and thermically stable, and relatively homogeneous in the oceanic areas – and of the consequences for the associated ecosystems is, therefore, fundamentally different. In fact, it is important to distinguish coastal environments from open oceanic environments, situated beyond the continental plateau. The risks of contamination in the coastal zone, to which estuaries and laguna can be added, are fairly similar to those of continental environments, down to a few specific details.

1.2.1. *The coastal zone*

This interface between the continent and oceans is home to specific ecosystems where important transfers of matter, energy and genes occur. The marshes, the seasonal nature of rivers' hydrology and the pre-eminence of primary production confer on the coastal zone a physico-chemical instability analogous to that of continental waters. The biodiversity housed by coastal regions is adapted to the strong variability in the characteristics of these transition environments, but its resilience has been broken down by anthropic impacts, leading to an increased vulnerability to pollution and global change, even more critical in the case of islands and lagoons. The recipient of nutritive salts eroded or washed from continents, the coastal and littoral zone, which is not very deep, provides numerous services to ecosystems (support and regulation especially) via primary production, the recycling of major elements, the metabolization of contaminants or their export into sediments and hydrological regulation. Costanza *et al*. [COS 97] estimate that a third of the global benefits and services to ecosystems are formed there. Because of this, but also due to the access to waterways that they provide, as well as the attraction that they exercise for our contemporaries, coastal areas concentrate 60% of the world's population – which is becoming increasingly urban and concentrated in megacities – at least 100 km from the coasts.

Coastal ecosystems, rich and vulnerable by nature, are therefore subject not only to pressures provoked by global changes, including climate change, but also to pressures due to this very strong concentration of continental activities as well as maritime activities.

The species exploited (fishing, conchiliculture (the farming of shellfish), the farming of sea vegetation, etc.) are also subject to these pressures, which could explain certain recurrent weaknesses in the immune system (F. Akcha, personal communication). Moreover, exposure to pollutants can lead to a contamination of the biomass, rendering it unfit for human consumption [LEB 06].

Once they have passed through the filters of lagoons, deltas and estuaries, in which the levels of salinity trigger a significant precipitation of matter, trapping certain components (cadmium in the Gironde or in the Bay of the Seine, for example, among the most well-known instances [SHI 13]), a proportion of the collection of micropollutants issuing from the drainage basin are found in the coastal zone (hydrocarbons, pesticides, metals, persistent organic products, medication, cosmetics, etc.). For example, the supplies of hydrocarbons to the marine environment account for 80% of the telluric supplies, accidental pollution, therefore, only represents a small fraction, of which the impact is mainly local and often significant for macrofauna. At the end of the 1980s, the development of chromatography in liquid form for environmental research, coupled with mass spectrometry at high resolution, enabled hydrophilic molecules of pharmaceutical, cosmetic or hygienic origin to be gradually detected, at weak concentrations that qualify as emergent. Pioneering studies have enabled the identification of a number of these substances active in rivers, lakes and aquifers (for example, in the United States [KOL 02]). In France, the first studies on coastal waters and estuaries were only carried out quite recently [CAS 06]. Antibiotic and anti-inflammatory products, fungicides, antidepressants, analgesics and anticancer medications have been identified, of which it is still difficult to evaluate the real impact on fauna and aquatic flora. More specifically, the use of the coast leads locally to strong concentrations of cosmetic sun protection products, oils and perfumes, as well as products used to protect the hulls of boats from biological fouling (copper, tributyltin (TBT) and its replacements). For example, at only 20 $ng.L^{-1}$, TBT considerably disrupts the growing metabolism of mussels. At 2 $ng.L^{-1}$, TBT, an endocrine disruptor, is capable of modifying the sex of certain marine gastropods (masculinization of the females by the effect called imposex) [ABI 12]. Nanoparticles of titanium oxide, used in a lot of sun protection products, are part of the emerging concerns. Nevertheless, in their review on the subject, [KLA 09] do not report any more observations *in situ*. Research is currently concentrated on the evaluation *in vitro* of the potential effects of these nanoparticles on coastal organisms [CAN 10]. Finally, the coastal zone permits active exchanges between the column of water and the sediments, which unfortunately often constitute a significant reservoir of persistent contaminants. Much like continental lakes, the toxic threat associated with them thus lasts decades, being reactivated following the reshaping of the sediment or modifications of its "redox status".

1.2.2. *The open ocean*

The risks from contamination of the open ocean by toxic substances are different. There is less biodiversity there, which results from the low habitat fragmentation of the environments, and from their relative homogeneity. Nevertheless, marine ecosystems are of the greatest importance for mankind: for their supply of protein biomass (15% of the total supply) and for their value to local communities: the cradle of life on Earth, still largely unexplored, is believed by some to be outside the reach of anthropogenic pressures [GOU 12, p. 18]. They are home to wild species of great longevity, at the top of the trophic chain.

One way of taking account of the immensity of the oceans and their inertia consists of evaluating the residence time of elements in their different compartments. This amount, homogeneous for a time, is obtained by dividing the volume of a reservoir by the fluxes that cross it, under the hypothesis of stationarity. The residence time of water in the global ocean is in the order of 3,000 years [DE 09]. For the Mediterranean, it is, for example, a hundred years. This means that an easily biodegradable contaminant, carried from the continent, such as glyphosate (in the order of a month in water, INERIS, 2010), will not be found again in a significant concentration in the whole of the

Mediterranean basin. However, persistent organic contaminants, such as PolyChlorinated Biphenyls (PCB), can easily be dispersed. In effect, the duration of the half-life in water (suggested by [MAC 92] to be two years for tri- and tetra-chlorides, and six years for penta- to hepta-chlorides) does not include transfers in the food chain, one of the most efficient methods of storage and transport for hydrophobic substances. Chlordecone, a chlorine insecticide used to combat the banana-tree weevil in the Antilles, is one of the contaminants for which it is still difficult to suggest a typical biodegradation time. In fact, the risks for the open ocean, where ecotoxicology is concerned, are on the one hand those of persistent contaminants on the scale of oceanic fluxes (a decade and more), and on the other hand of substances whose planetary cycle is in part controlled by specific marine processes. Mercury can be counted among the latter, of which the atmospheric supplies through snow, then the arrival at the ice interface of sea and seawater appears to be primordial [COS 11, DAS 14], but whose planetary cycle remains to be elucidated.

These compounds are generally hydrophobic – this is what makes them difficult to biodegrade in an aquatic environment – and lipophilic, and they, therefore, spread in the trophic chain in spite of the fairly weak concentrations that cause them to be diluted in the ocean. Their enduring presence, associated with this particular mode of transfer, leads large sea predators to become contaminated, whether these are, for example, tuna [KRA 03], mammals or even birds [DIE 13]. The risks are, therefore, both the chronic toxicity of these substances for large organisms and the ecosystems to which they belong – which for a long time were thought to be unable to carry pollution of human origin – as well as the fitness of the human foodstuffs that are taken from them. This concern is even more important when a population's food supply is mainly taken from marine sources, as is the case, for example, for the Inuit [DAL 13] and Polynesians [DEW 08].

More than 37 million chemical substances are currently listed in the world, for the most part substances resulting from biosynthesis. Around 100,000 chemical substances are produced, imported and used on the European market, and 5,000 of them (5%) are considered to be dangerous for mankind and the environment. Sources of contamination by metals are multiple and include mining activity, the steel industry, transport, the use of different types of batteries and the painting and dying industries, as well as the use of phosphorous fertilizer (cadmium). Taking account of the diversity of the molecules, the study of organic contaminants represents a very important undertaking. Very schematically, it is possible to distinguish four main substance groups:

– hydrocarbons, of which aromatic polycyclic hydrocarbons (APHs) are the most worrying for aquatic environments;

– pesticides, with some 900 types in current use and a usage rate of 80,000 tons applied each year;

– biocides, which refers to substances used in a non-phytopharmaceutic context, such as additives included in anti-fouling paints for use at sea, which cause non-negligible contamination by different organometallic (TBT) or organic (diuron or atrazine such as Irgarol 1057) or active metallic substances (copper);

– other organic synthetic substances that represent a large number of substances (chlorinated solvents, PCB, flame retardants, phthalates, detergents, colorants, etc.). The selection criteria for chemical contaminants judged to be a priority for the environment are based on three properties: persistence (P) defining persistent substances in the environment (for example, persistent organic pollutants (POPs) such as DichloroDiphenylTrichloroethane (DDT)), bioaccumulation (B) defining their capacity to accumulate in organisms and toxicity (T). These three properties define a group of substances that are called PBT substances. To this group should be added substances that have carcinogenic and mutagenic properties and effects on the reproductive system that are called CMR substances. Endocrine disruptors are also associated with this group.

Box 1.1. *The chemical universe and the environment*

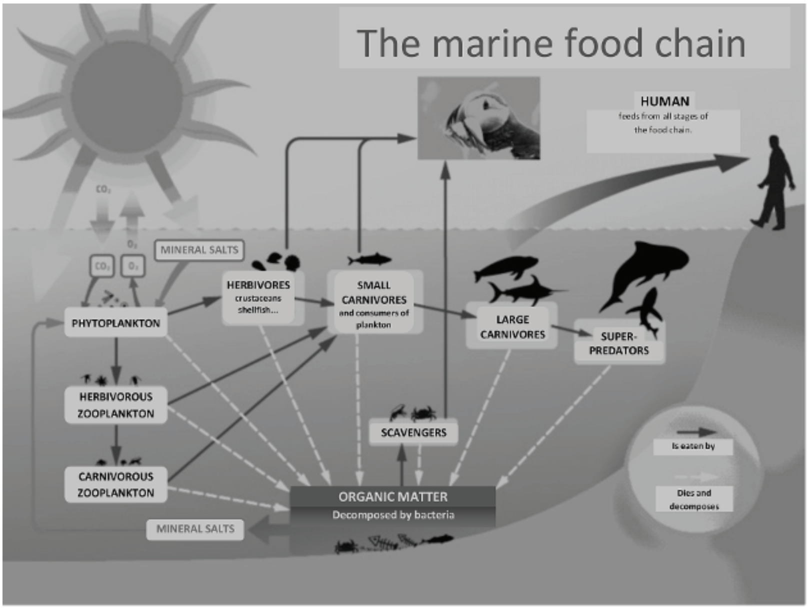

Figure 1.1. *Example of the marine trophic chain*

1.3. What is the biological response of organisms to contaminants?

The absorption of a contaminant by a living organism triggers a disruption in its metabolism. This disruption leads to a biological response, which results either in the cell returning to its non-disturbed state or in a manifestation of toxic effects. An organism's biological response to a toxin can be seen as the result of the interaction between the intrinsic properties of the substance and those of the organism exposed to it. It depends on the chemical properties of the contaminant (structure, activity and mode of action) and the physiological, biological and ecological properties that characterize the organism at the moment in its life when it is exposed to this contaminant. It is also variable within a single species and a single age group, depending on individuals. For example, many of us are exposed to the flu virus each winter, and only some will actually become ill. Its general physiological state has, in effect, an impact on the reaction of an organism in the face of a stressor. Each species, and each individual within a species, therefore shows a specific response to each chemical product. Moreover, the environment in which this species evolves and its connection with other parts of the ecosystem will also condition its response. Finally, the direct impact of contaminants on a species and/or a subsection of it can generate indirect effects on the entirety of the ecosystem, as we will see later (Figure 1.2).

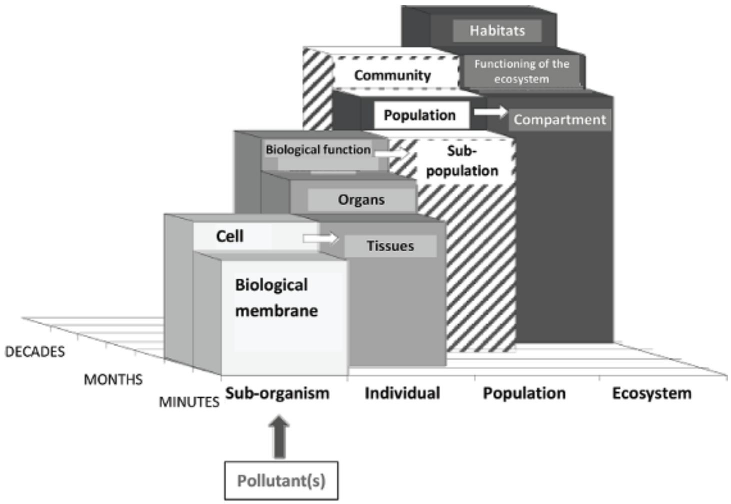

Figure 1.2. *Adaptation of the schematic representation of the field of ecotoxicology studies depending on the level of complexity of the lifeform and the time, from [MUN 95] and [ADA 00]*

1.3.1. *At cellular level*

The manifestation of a disturbance in the functioning of the cell is the result of interaction between the cellular biomolecules and contaminants. This interaction is very specific: its occurrence and intensity depend on the organism's physiology at the moment it is exposed and on the toxic product's mode of action.

1.3.1.1. *General remarks on the modes of action*

The contaminant's mode of action is determined by its chemical structure [ESC 02, PAK 00, TRE 04]. Pollutants have been classified into four categories, on the basis of their structure/activity ratios (SARs) [HER 89]. The "inert" products (class I) and "slightly reactive" products (class II) have a narcotic action: they react in a non-selective manner with biological membranes, thus modifying their structure and functioning. Their effect depends mainly on their hydrophobia [VAN 92]. The "reactive" compounds (class III) and the compounds "with a specific mode of action" (class IV) react selectively or not at all with the cellular biomolecules. Once in the cell, they are generally hydroxylated and eventually combined with other molecules in order to be eliminated. However, the combinations are sometimes more toxic than the initial substance.

For a family of contaminants with a given mode of action, the cell's response depends on the presence and the abundance of the product's targets and on its metabolic capacities, as well as on its ability to repair damage [ESC 02]. These physiological properties characterize species. For example, the active elimination of the contaminant by cells calls upon different metabolic paths whose biomolecules are unequally distributed between different species [BAZ 97, CAL 83, IBR 98] or within the same species. Thus, a given cell's membership of a taxonomic group determines its response to a given product.

1.3.1.2. *The cellular response: the means of identifying exposure to contaminants before the event*

The impact of pollutants on a subcellular level can lead to the inhibition and/or triggering of diverse proteins and enzymes implicated in the metabolism and the excretion of xenobiotics. These detoxification mechanisms allow organisms to maintain themselves in the fact of exposure to pollutants. Modulations of biotransformation enzymes have, therefore, been the subject of a very large number of investigations over the last 30 years, notably among fish [AND 92, GOK 98, WHY 00]. Much effort has, in particular, been devoted to the identification of biomarkers of detoxification, that is to say proteins, or indeed enzymes, whose activity levels reveal the starting of processes within the cell. This work focuses on the measurement of cytochrome protein levels P450 (phases 1A and 3A) [MUR 97, WEB 02], the measurement of ethoxyresorufin-O-deethylase activity (EROD) [GOK 98, TEL 04, WHY 00] or the enzymes from the glutathione-S-transferase family [GEO 94, KIM 10, VAR 89]. As an example, Figure 1.3 shows a correlation between levels of contamination and EROD activity [BUR 94, GAL 91]. These molecular biomarkers are not, however, specific to the contaminants that trigger their activation, and their responses are potentially affected by biotic or abiotic factors.

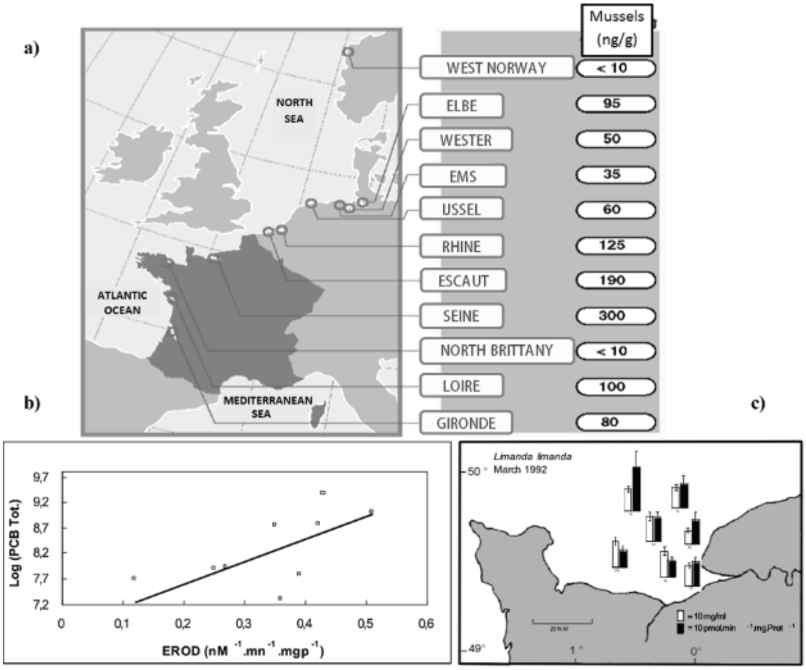

Figure 1.3. *a) Contamination in mussels (ng.g^{-1}) on European coasts by PCB from measurements in RNO 1991; b) correlation between EROD activity (nMol produced by enzymatic activity (resorufin), per minute and per mg of liver protein) and level of contamination in PCB (ng.g^{-1}) in samples of plaice [GAL 91] and c) EROD activity (by the same units as (b)) in samples of flounder in the Bay of the Seine [BUR 94]*

1.3.2. *On an individual level*

Disturbance in cellular physiology generally manifests itself by the effects on the survival, growth, reproduction and indeed the behavior of individuals. It is also at an individual level that contamination occurs. This does not depend only on the chemical form of the contaminants, from which it has wrongly been thought possible to define “the” bioavailability [GOU 13], but above all on the specific details of an individual’s life history.

1.3.2.1. *How specific and individual variability influences contamination*

The biological and ecological characteristics of individuals are implicated at all stages of the contamination process and the biological response: exposure, absorption, elimination and eventual compensation for the product’s effects. In the first place, the duration of contact between the organism and contaminated environment depends on the number and duration of the developmental stages undergone during the lifecycle, as well as the presence of defense mechanisms over the course of this cycle [SPR 05]. The habitat and feeding method determine the organism’s behavior and influence its level of exposure [KOI 92].

The biological and ecological characteristics of species are moreover involved in the kinetics of the organism’s contamination [ESC 02]. In effect, the speed at which a contaminant is absorbed depends on the intensity of the exchanges between the organism and its environment. This absorption speed can be described using food assimilation rates [CAN 02] and the exchange surface between the organism and environment. This exchange surface is generally represented by the ratio between the surface of the body and its volume (S/V) [ESC 02]. In this ratio, assessment of the body surface takes account of the toxin’s different absorption routes: the integument, the digestive tract and the respiratory surfaces [WEI 04]. For two organisms of similar size, the higher the S/V ratio, the more rapid the kinetics of the toxin’s absorption [KOI 92]. In practice, it is, therefore, mainly the mode of feeding and the respiratory system (gills or integument) that is involved in the organism's contamination kinetics.

1.3.2.2. *How specific variability and individual influence the depuration rate*

The biological and ecological characteristics of species and individuals are also involved in their capacity to eliminate or store the contaminant in a non-dangerous form. Passive elimination of the compound implies its excretion or accumulation in inert compartments of the organism [GRO 99]. Furthermore, tissues with a high lipid content offer a significant storage volume of hydrophobic contaminants. Differences in the presence and volume of these compartments generate a strong interspecies variability in biological response to toxins and, in particular, to organic products [ESC 02]. These differences are linked to the relative size of species: in effect, it is generally the largest organisms that possess the most lipidic reserves [CAN 02, ESC 02].

Thus, it is mainly the characteristics linked to use of the habitat and food, as much as the characteristics linked to the pattern of the organisms' life history, which determines their response to contaminants at an individual level.

1.3.2.3. *Some types of toxic effects*

Certain pollutants, characterized as "endocrine disruptors" (PE), act on organisms' hormonal equilibrium. Endocrine disruptors are exogenous substances that trigger effects harmful to the health of an organism or its descendents, following changes to endocrine function. The action mechanisms of PEs are multiple, since they can act on all the stages of endocrine regulation, from the synthesis of hormones to activity at the level of the target tissues. Among aquatic organisms, exposure to PEs has been associated with harmful effects on the reproduction (Figure 1.4; [CRA 08, DAO 11, MEN 08, MIL 05]) of individuals and populations. Certain pollutants have also been identified as neurotoxic, leading to effects on the neural functions of fish, and they can, therefore, potentially affect the species's behavior and learning [PEA 13].

Diverse chemical agents have the capacity to interact with the DNA molecule and to modify its nature [CAJ 03, HEB 96]. Types of damage to DNA are generally separated into two categories: genetic lesions (functional lesions) caused by mutagenous agents and chromosomic lesions (structural lesions) caused by clastogenic agents [EVE 94, LIV 00, MAR 10].

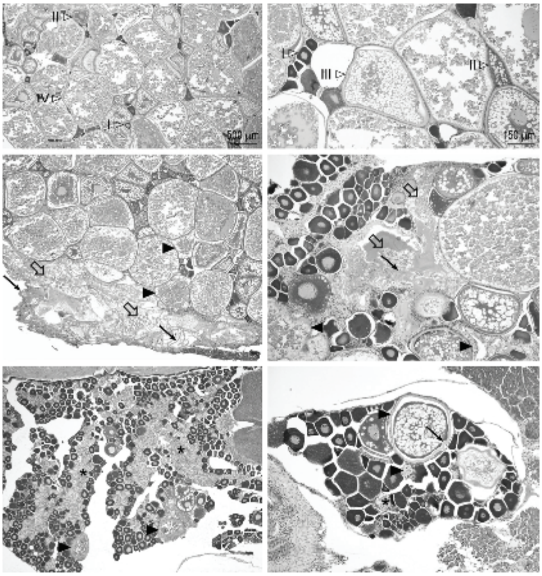

Figure 1.4. *Histological structure of the ovaries of female zebra fish (taken from [DAO 11])*

COMMENTARY ON FIGURE 1.4.– The different stages of maturity of ovarian follicles (I–IV) can be identified in the ovary of a control fish. The fish ovary exposed to PCBs (at levels in the order of those encountered in the Loire estuary; photo, center) shows a number of atretic (arrow) follicles. The number of follicles at stage III (vitellogenous) and stage IV (mature ovocite) is slightly diminished in comparison to those of the control. The ovaries of fish exposed to elevated concentrations of PCB (Seine estuary; photo below) show an almost total absence of follicular stages III and IV, which makes reproduction impossible.

1.3.3. *On the level of the population*

The decrease in rates of survival, growth and reproduction, as well as the modification of individual behavior – as far as it is due to them – leads to a modification in the population's dynamic, which can go as far as the disappearance of the species in a polluted habitat [CAS 01]. For a single disruption at an individual level, the effect on the population is lesser or greater according to the pattern of the species' life history [SPR 05].

In practice, the pattern of the species' life history is a group of data that includes: the species' biodemographic parameters in a given environment (lifespan, number of developmental stages, number of offspring, fecundity, etc.) and the ratios between these parameters (ratio between lifespan and age at which an organism first reproduces, ratio between rates of growth and fecundity, etc.). Each pattern of the species' life history corresponds to a coadaptation of the species' biological and ecological characteristics depending on the biotic and abiotic factors of its habitat. These characteristics can be classified into two categories. Those that are linked to the developmental cycle of the organism influence the probability of survival above all. Those that are linked to reproduction influence fecundity above all.

Thus, the number of developmental stages and the population's vulnerability in terms of the probability of survival at these stages are major determinants of a population's resistance to disruptions [SPR 05]. In effect, among certain organisms, there are critical periods or stages of development during which the organism's probability of survival is weaker than at other stages [KOE 91]. These stages are particularly vulnerable to instances of pollution. For example, this is the case during periods of larval molts among crustaceans [KOI 92] or the metamorphosis between the larval and juvenile stages of fish (for example, flat fish, which pass from a pelagic larval life stage to a benthic stage during their juvenile and adult phases).

The characteristics influencing organisms' reproduction, along with their lifespan, strongly influence a population's response to contaminants [CAL 97, IND 99]. In effect, the number of descendants produced per year in a population depends, according to [SPR 05]:

– on the age of sexual maturity compared to the lifespan and the number of potential reproductive opportunities;

– on the number of reproductions per year;

– on the organisms' fecundity (the more energy the parent invests in the egg, the less productive it is, but the higher the chance of survival of the egg, and then the juvenile);

– on the existence of parental care of the eggs and/or the young.

In a non-polluted environment, there is no "good" or "bad" strategy in terms of a population's persistence [SPR 05]. However, certain strategies are more "efficient" than others in terms of persistence when pollution is added to the normal level of disruption in the environment [KAM 96]. For example, in the case of a short and temporary disruption lasting for the duration of the reproductive season, species that can reproduce several times in the course of the season will probably be less affected than those that only reproduce once [CAL 97].

In conclusion, a cell's response to a contaminant results from the interaction between its physiology, function and activity, and the mode of action of the contaminant to which it is exposed. The biological response of an individual or a population results from the interaction between the disruptions caused by the toxin at a cellular level and the organism's biological and ecological characteristics. These characteristics are mainly determined by the properties of the habitat and the organism's life history strategy.

Nevertheless, specific populations are only one element of their communities and ecosystems. It is becoming much clearer that it is at this level of lifeforms' organization, when exchanges of matter, energy and genes occur, that modes of evolution, adaptation and resilience are made permanently, notably in response to toxic pressures. Figure 1.5, taken from [ADA 05a], shows the different levels at which environmental stress produces direct and indirect effects on the ecosystem's structure.

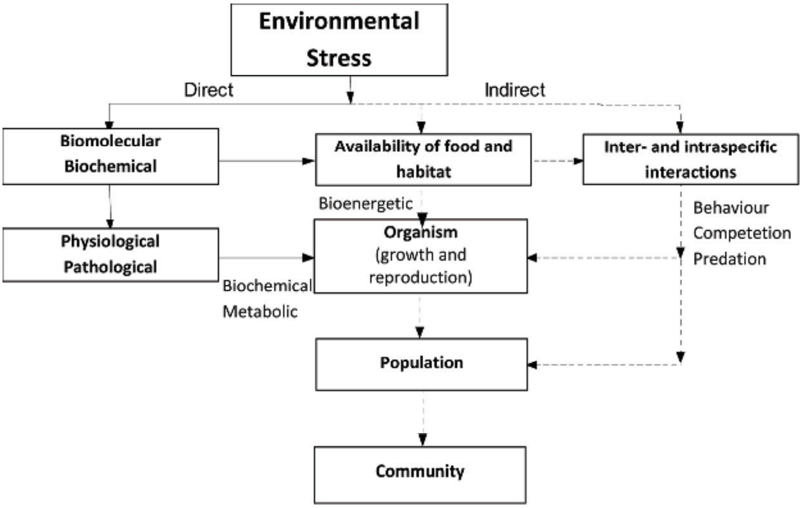

Figure 1.5. *Direct and indirect effects of environmental stress on biological systems (taken from [ADA 05a])*

The direct effects affect organisms by acting on the biochemical and metabolic processes; indirect effects disrupt the biota[2] via the availability of food and habitat, and via intra- and inter-species interactions.

1.4. Consequences of toxic pressure on ecosystems

When pollutants are ejected into aquatic ecosystems, direct (toxic) effects on biota are possible. These direct effects of toxic substances tend to reduce the number of organisms (by an increase in mortality and/or a decrease in fertility). The biota of a given habitat often show a large range of tolerance to specific toxic substances (for example, insecticides and herbicides target specific organisms in a community), the toxin can have lethal effects on certain species, but have no observable effect on others. Pollutants can, however, have effects on tolerant species through a number of ecological mechanisms. These

---

[2] A group of living organisms present in a particular habitat or more generally in a given region.

effects are called indirect (or secondary) effects of the contaminants [PAL 08]. Toxicity tests in laboratories based on a single species do not permit indirect effects to be detected. To assess the impact of pollutants on the scale of a population, a community or an ecosystem, studies carried out in a microcosm or *in situ* are necessary [CAI 83, CLE 94].

The effect of contaminants on one section of an ecosystem (predators and/or prey) can lead to a cascade of indirect effects on species *a priori* resistant at other trophic levels. These effects on sensitive species can, therefore, modify concurrential interactions in resistant members, both producers and consumers, of the community. Furthermore, toxic substances can act directly on “key species” of an ecosystem. These species, including, for example, “engineer species” or “keystone species”[3] have an influence on other biological compartments via modifications in the environment [BRU 01]. Thus, mechanisms linked to the population and/or community after exposure to contaminants are potentially complex and very varied. The indirect effects of toxic products can then lead to an increase (reduced concurrence) or a decrease in the abundance (reduced availability of the “preferred food”).

1.4.1. *Interspecies relationships*

At relatively high concentrations, the contaminants are fatal and cause indirect effects on the “density control” relationships between species [FLE 03, DE 89]. The contaminants, present at non-lethal concentrations, can also have an incidence on a broad range of individual biological traits (changes in the neurotransmitters, hormones, immune response and reproduction) and behavior (hunting for food, capacity to swim, detection of predators, learning and social interactions) [WEI 01]. These modifications of course affect the individual, but can also have indirect effects on the community in which the individuals are integrated.

---

[3] Of which the disappearance leads to a modification, indeed to a brutal degradation of the ecosystem.

1.4.1.1. *Predator–prey interactions (top-down)*

The "top-down" effects are triggered when a predator is more sensitive to a contaminant than its prey (Figure 1.3). This sensitivity, without consequently directly causing the predator's death, can modify the ingestion and/or predation rates [GRE 97, WAL 00, WEI 01, WID 91] and thus causes an increase in the abundance of the prey (Figure 1.6). "Top-down" effects have thus been used as evidence in marine benthic systems after contamination by metals, fungicides and hydrocarbons [BEL 00, CAR 97, LAY 85, JAK 96, VAN 00]. These different contaminations have all led to trophic cascades. In addition to reducing pressure from predation, the contaminants can also benefit producers via the release of nutrients from the decomposition of deceased animals [KNA 05].

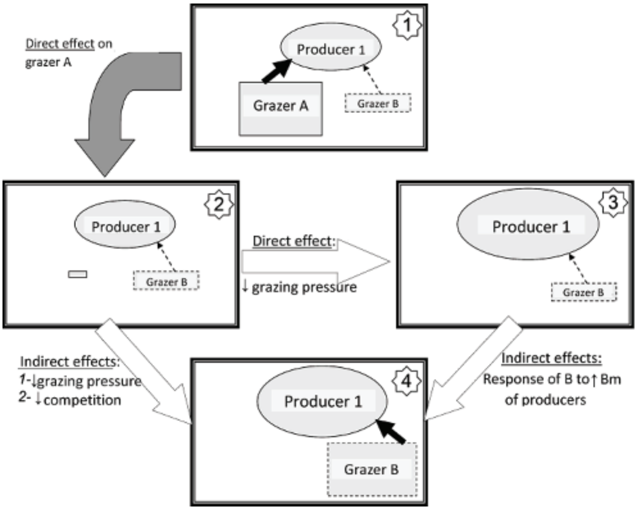

Figure 1.6. *Illustration of indirect "top-down" effects of contaminants (modified from [FLE 03])*

COMMENTARY ON FIGURE 1.6.– Box 1 represents a community of primary producers and two species of grazers (A = dominant; B = rare) before modification by a contaminant. Box 2 shows the result of a possible direct effect (rapid and selective) of the contaminant on the dominant grazers (A). The primary producers benefit from the reduction in pressure from grazing due to the death of (A). Boxes 3 and 4 illustrate two indirect effects able to lead to an increase in the abundance of grazers (B) (box 4). First of all, the “rare” grazers can benefit from the death of the common grazers and thus increase their rate of feeding on the primary producers through a decrease in the concurrence, which leads to an increase in the population. Box 4 shows that the increase in the abundance of primary producers generates a functional response for rare grazers, who become dominant.

The structure of the community has evolved from 1 to 4 following exposure to a contaminant.

1.4.1.2. *Interaction between resources and consumers: “bottom-up”*

Contaminants can have indirect effects at the base of the food chain (vegetables, detritic and/or bacterial biomasses, primary producers) by modifying, for example, the organic substrate of detritivores or by affecting the liberation rates of nutritive elements for primary producers. Hydrocarbons from petrol represent an important source of organic matter that can lead to a stimulation in productivity or the bacterial biomass. This stimulation can be sufficiently important to feed a reaction of the “bottom-up” type on bacteriophagic species and/or detritivores [PET 96]. In another domain, Podemski and Culp [POD 01] found evidence for a stimulation in the growth of diatoms subjected to effluent coming from a paper-whitening factory. This phenomenon generated an important proliferation of grazers feeding on the diatoms.

Herbicides can have direct effects on microalgae and generate indirect effects on communities of zooplankton via variations in competiveness between species. Through cascade effects, these are the species at higher trophic levels, such as salmon, which are more susceptible to being affected [BRO 10].

Fleeger *et al.* [FLE 03] list 56 cases of indirect effects from pesticides on the competition between species or predation on the biota and conclude that, at least in aquatic systems, pesticides exert a strong selection pressure on invertebrates.

Few studies on aquatic systems have examined the effects of contaminants on the cycle of nutritive elements or their bioavailability. Studies carried out on land ecosystems suggest, however, that the contaminants can increase or on the contrary decrease the liberation of nutritive elements at such a level that the abundance of the vegetation and/or the bacterial population can be affected [BOG 96, SAL 97, SAL 01].

1.4.1.3. *The alteration of behavior*

The behavior is defined as the group of coordinated internal responses (action or inaction) of living organisms (individuals or groups) to internal and/or external stimulae, excluding responses linked to development [LEV 09]. A behavior is, therefore, an interaction of living beings with each other or with their environment (Figure 1.7). It can appear to change or be inhibited following a change in physiological state, a change in the environment or due to a new social interaction. It is a very early and sensitive indicator of physiological disturbances. An individual response, alteration in behavior very often leads to indirect effects linked to predation.

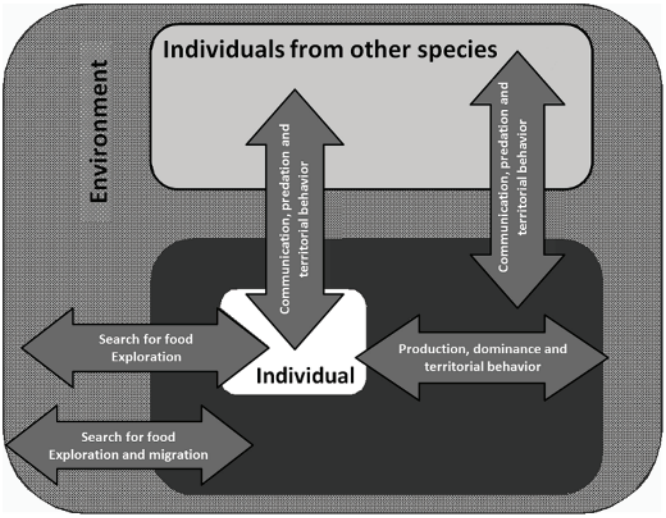

Figure 1.7. *Different behavioral relationships between an individual with the other individuals of its species, other species or its environment (taken from [PEA 12])*

The behavior of living organisms is susceptible to being affected by the presence of toxic substances in the environment [WEI 01]. Disturbances in organisms' locomotive activities can lead to deficiencies in their capacity to flee when faced with predators and an increase in the time required to search for prey as well as modifications in migratory activities indirectly affecting their reproduction [TRI 97, WEI 02, WEI 04]. Several studies have also indicated that pollutants can decrease the alimentary activity of fish. Finally, the presence of undesirable molecules can lead to modifications in behavior linked to reproduction (display, choice of partner and parental care). All these behavioral changes result from modifications in the biochemical and physiological processes on an individual level and can have significant effects on the equilibrium of populations and communities. In effect, the contaminants can cause behavioral or defensive responses, which can modify biological interactions or even intensify the effect of a contaminant.

Three general types of pollutant influence on the behavior have been observed:

– reduced rates of predation: although numerous studies demonstrate that ingestion or rates of predation by various animals can be diminished by contaminants, very few identify a behavioral mechanism associated with the hunt for food. Smith and Weis [SMI 97], however, have stated that exposure to mercury has been shown to correlate with a decrease in the predation intensity of the killifish (*Fundulus heteroclitus*); Temara *et al.* [TEM 99] have described changes in the predatory behavior of a starfish exposed to soluble fractions of crude oil;

– an increased sensitivity to predation: exposure to contaminants can inhibit a prey's specific behavior used to avoid a predator. This effect can lead to an increase in rates of encounters between predators and prey. As an example, Hinkle-Conn *et al.* [HIN 98] have found evidence of a decrease in several species of invertebrates' capacity to dig holes in contaminated sediments, increasing their exposure to predators. Many other examples of increased sensitivity to predation, triggered by a variety of contaminants, are identified in several studies in fresh and marine waters, involving both vertebrates and invertebrates at once [CLE 99, CLE 09, DOD 95, HAM 95, KIF 96, KRU 94, LEF 99, PRE 99, TAY 95, WEI 95, ZHO 99]. The increase in the intensity of predation can lead to an increase in the rates of trophic transfer from contaminants to contaminated prey to larger predators;

– a reduced sensitivity to predation: the changes caused by the contaminants on the behavior of prey that would decrease the probability of encounters between prey and predators, thus reducing the rates of predation, have received relatively little attention. However, Taylor *et al.* [TAY 95] have found evidence of a decrease in a cladoceran's capacity to swim in the presence of lindane. This disruption has reduced its vulnerability to predation by the hydra (*Hydra oligactis*).

1.4.1.4. *Interspecies competition within a same trophic behavior*

One of the clearest cases in which contaminants can affect interspecies competition can be found in the examples of differential sensitivity in several species within a single trophic group. For example, several studies have demonstrated that different groups of zooplankton, which are in competition for phytoplankton, have different sensitivities to insecticide (typically, cladocerans are more sensitive than copepodes). Thus, when moderate concentrations of insecticides are applied to communities containing cladocerans and copepodes, a spectacular drop in the more sensitive cladocerans is observed while the number of copepodes increases [HAV 93, HAV 95, VAN 02, REL 05]. Several authors have also examined the effects of insecticides on communities of macroinvertebrates composed of several competing species. Thus, populations of gastropods and oligochetes increase following exposure to a mixture of lindane and chlorpyrifos to the detriment of crustaceans more sensitive to these insecticides [CUP 02]. Differential sensitivity to herbicides has also been documented among producers. In a green-macrophyte algae system, the application of linuron leads to a drop in the abundance of macrophytes, but an increase in the abundance of green algae. Although herbicide has direct toxicity for green algae, the nutrients released by the decomposition of macrophytes also benefited algae [SLI 05].

1.4.2. *Contamination and impact on genetic diversity*

The main genetic impacts reported in the context of contamination studies on populations include:

– increases in mutation rates;

– changes in the whole genome of genetic diversity due to demographic bottlenecks;

– changes in alleles or genotypic frequencies caused by selection triggered by contaminants;

– changes in the dispersion methods of gene fluxes that modify the genetic relationship between populations [BIC 11].

Evidence of the direct effects of pollution on genomes has been found in land organisms and in particular in the case of contamination by radionuclides [DUB 96] and aromatic polycyclic compounds [SOM 02, YAU 00].

The indirect effects of pollutants on genomes can result from a massive reduction in the size of the population resulting from mortality or even the selection of alleles or genotypes associated with "tolerance" in the contaminated zones and elimination of the majority of "sensitive" genotypes.

Only a few studies have found evidence for the existence of genetic erosion due to a massive reduction in population size in the contaminated sites. For example, Demarais *et al.* [DEM 93] concluded that the modifications in the genetic structure of freshwater fish populations (Gila de seminuda), observed after acute accidental exposure to rotenone (insecticide), were probably due to a genetic bottleneck caused by an elevated mortality rate. Murdoch and Hebert [MUR 94] put forward the same hypothesis to explain the mitochondrial genetic diversity observed in populations of cat-fish (*Ameiurus nebulosus*) taken from contaminated sites. These selective pressures can lead to changes in the distribution frequencies of alleles and genotypes in exposed populations [HEI 97, MAR 03, MUL 02, VIR 03]. Analyses of persistent fish populations surviving in chronically contaminated estuaries for more than 60 years in the United States and Europe, compared to populations living in untouched estuaries, have furnished new information on the identification of candidate genes potentially implicated in the response of the fish to chemical agents [HAH 04, HAH 05, MAR 10]. Thus, Hemmer-Hansen *et al.* [HEM 07] have demonstrated the flounder's excellent capacity to adapt (*Platichthys flesus*) to contrasting environments in estuaries. They have underlined the genetic base of these species' adaptation to the particular environment of the Baltic Sea compared to the North Sea (salinity gradient, chemical stress, hypoxia, etc.). Later on, different studies carried out on populations of flounder in estuaries confirmed these results [LAR 02, MAR 03, MAR 04]. The authors found a greater capacity to maintain the integrity of DNA among individuals carrying the allele PGM-85 in different estuaries contaminated along the Atlantic Coast of France (the estuaries of the Loire, Seine and Vilaine), compared to an uncontaminated control estuary (Ster); the hypothesis of selective pressure acting on the locus PGM has been formulated. Associations (genotype–phenotype) have been observed for several aquatic species exposed to diverse contaminants [GIL 99, KAM 00 VAN 00, WEI 02]. Correlations between individual heterozygosis and fitness components (such as survival, stability of development, growth rate, fecundity and metabolism) have been observed among different aquatic species exposed to contamination. [BEN 92, GIL 99, HAR 04, KOP 92, MAR 03, MAR 04] have observed that flounder displaying the greatest genetic variability (the most heterozygotic) were also the most capable of maintaining DNA integrity in the contaminated estuaries, confirming the conclusions of several studies carried out on natural fish populations exposed to complex mixtures of contaminants [LAR 01, LAR 02]. These correlations can in part be explained by a slower base metabolism among the heterozygotic individuals, thus leading to a smaller energy need for vital functions and finally to an improved capacity to adapt to environmental stress [DEP 96, HAU 03]. Numerous studies suggest that contamination can constitute a strong selective pressure, susceptible to leading to adaptive changes in the natural populations.

1.4.3. *Host–parasite interactions*

Parasites represent one of the most important natural biotic factors of environmental stress on natural populations. They can modify the physiology and metabolism of animals, and in consequence can influence characteristics of the lifecycle such as survival, growth and fecundity. Parasite infection can modify intra- and inter-species competition for resources, by rendering the infected individuals less competitive [REL 06].

Other stress factors, such as chemical products in the form of heavy metals or organic compounds, can exacerbate the harmful effects of infection. Conversely, a greater vulnerability of parasites to contaminants can lead to a decrease in the rate of infection [BOO 05]. Eira *et al*. [EIR 09] indicated that infestations of cestodes in eels (*Anguilla anguilla*) can modify the metabolic/storing processes of metals in the host's tissues and thus reduce the bodily charge in chrome and nickel.

Numerous chemical compounds have a tendency to alter the immune function of organisms, which is an animal's main system of protection against infections [GAL 01, GAL 03]. Two types of effect can be seen: first, the impact of pathogenic agents on animals present in the polluted zones and second, the interactions between the pesticides and biological combating agents [HOL 10]. Thus, environmental contaminants and infectious diseases are considered to be the main factors contributing to the global decline in amphibians [FOR 06, KIE 02]. Eder *et al*. [EDE 07] have found evidence of a significant infestation by the hematopoietic necrosis virus that infects salmon (*Oncorhynchus tshawytscha*) following exposure to pesticides. Several examples of evidence have also been found in fish (contamination by PCB [DUF 00] or sea-dwelling mammals). The strongest correlations between parasites and chemical contaminants have been discovered among vertebrates. However, Heinonen *et al.* [HEI 00] have shown differences in the toxicokinetic of benzo(a)pyrene between infected and healthy clams (*Pisidium amnicum*).

To protect cultures against threats in the most efficient way, conventional pesticides can be used jointly with biological combating agents. Koppenhoffer *et al.* [KOP 00] have revealed synergic interactions of imidacloprid (insecticide) applied jointly with entomopathogenic nematodes (*Steinernema glaseri* and *Heterorhabditis bacteriophora*) against white grubs (*Clidemia hirta*, *Clintonia borealis* and *Pieris japonica*). The main factor responsible for this synergic interaction between the pesticide and nematodes seems to be the general disruption of the nervous system due to the imidacloprid.

1.4.4. *Resilience and resistance*

Resistance is defined here as the capacity of a community to maintain conditions of equilibrium and to maintain its functions following exposure to contaminants. However, resilience is defined as a community’s capacity to return to its initial state or to a state that enables the maintenance of its functions after exposure to toxic compounds. A better comprehension of the ecological factors that determine a system’s resistance and resilience will enable observers to improve their capacity to predict how communities react to and recover from exposure to xenobiotics [CLE 02, PAL 08].

When an environmental condition – such as the atmospheric temperature and the rate at which oxygen dissolves – changes gradually over the course of time (Figure 1.8(a)), we might expect that ecosystems themselves respond gradually (Figure 1.8(b)). However, this is not always the case. Under the effect of growing pressure, certain ecosystems reach points of no return, at which they undergo sudden and unexpected transformations called “catastrophic transitions” (Figure 1.8(c)) [SCH 01].

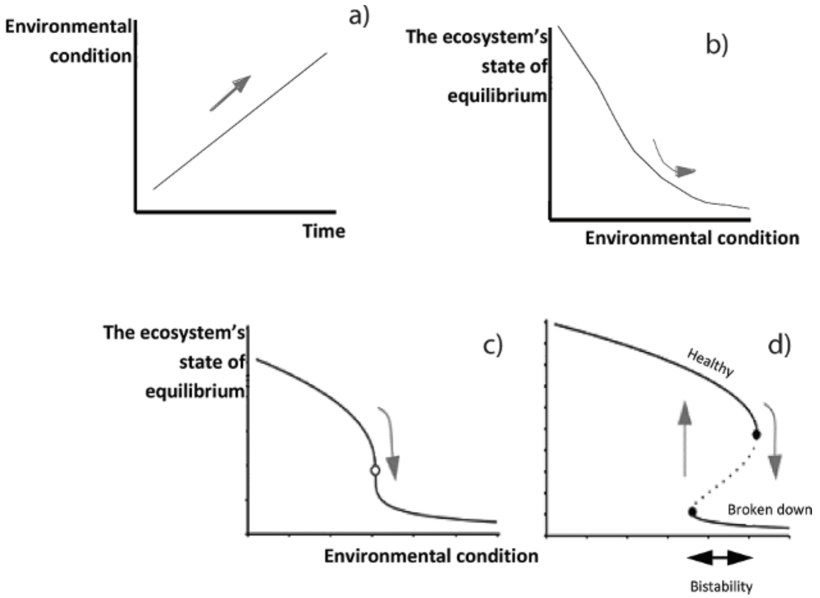

Figure 1.8. *Types of ecosystem response to a change in environmental condition (taken from [SCH 01])*

COMMENTARY ON FIGURE 1.8.– (a) Environmental condition varying gradually over time (for example, temperature, supply of nutrients or contaminants). (b)–(d): three types of ecosystem responses to these changes. (b) Continuous, gradual transition: the state of the ecosystem varies gradually in response to the change in the environmental condition. (c) Continuous, abrupt transition: the ecosystem's response becomes abrupt, and therefore less predictable but remains reversible. (d) Discontinuous transition (or catastrophic transition): the state of the system varies little until a threshold value for the environmental condition is reached. The ecosystem then swings to another state and then another mode of functioning.

Because of the ecosystems' diversity, the resilience of a community should be assessed while taking account of other stress factors such as climate change and/or invasive species [ADA 05a]. Thus, Kaufman [KAU 82] has reported that the communities associated with environments with an elevated level of stress were more tolerant than those from stable environments. The hypothesis put forward is that communities in disrupted natural habitats are preadapted to disturbances and effectively more resistant to anthropogenic stress factors [KIF 96]. However, it has been established that the effects of multiple disturbances are not necessarily cumulative [PAI 98]; consequently, the superposition of disturbances in an ecosystem already subject to chemical stress can trigger unexpected reactions from an ecological point of view [BEL 04, SCH 01].

Resistance to contaminants has been identified in numerous taxa, from microorganisms to vertebrates and vegetation [AMI 11, FEC 14] and results from two processes: (1) a physiological acclimatizing by individuals and/or (2) evolutionary adaptation in a population where the contaminants can be considered as selective factors. However, manifestations of resistance (adaptive responses) in populations exposed to complex mixes of pollutants, in fresh water or seawater, are still rare in the literature.

Individuals living in contaminated systems can produce descendants (generation F1 and F2) who display a resistance to pollutants similar to that observed for their parents; thus, this tolerance can be considered as a genetic adaptation of the population [BEL 01, JOH 11]. By using this approach, [KLE 01] have studied the Cyprinodon fish's potential resistance to chemical stress in experimental conditions. They underlined a decrease in the heritability of resistance to chemical stress with the increasing number of pollutants in the mixture. First, artificial selection carried out in a laboratory on another fish (*Heterandria formosa*) showed a rapid response to selection following exposure to cadmium; after a single generation, a proportion of two selected lines of descent out of three displayed an increase in resistance to cadmium [XIE 04]. Several studies have highlighted the fact that populations of flounder (*P. flesus*) in three chronically polluted estuary systems in the United States are resistant to aromatic hydrocarbons in their environment, compared to fish in the region of habitats little impacted by HAP. Resistance in the first generation, and sometimes in the embryos of second generation, suggests that differential survival can be due to genetic adaptation rather than to physiological acclimatization [BUR 07, VAN 08].

The energy an organism allots to developing its resistance to stress increases its probability of survival, but can have repercussions for the energy necessary for essential functions such as growth and reproduction. These energetic compromises between maintenance (survival) and production (measured, for example, by the rate of development and fertility) are found in populations subjected to chemical stress [MOU 11]. In their summary on the cost of tolerance to toxins, measured by experimental approaches, Van den Brink [VAN 00] have underlined the cost of tolerance to metals in plants, of invertebrates and fish, which show modifications in the traits of their life history. Arthropods' resistant to insecticides show a cost that is characterized by a loss of physiological performance in terms of fecundity, rates of development and fertility [ROU 87].

Several studies, carried out in laboratories, based on the successive generations of fish selected for their resistance to contamination, have typified the cost of resistance, showing:

– a reduction in the size of the offspring, a weaker fecundity and increase in the age of first maturity, for fish exposed to cadmium compared to control fish [XIE 04];

– a greater vulnerability in fish exposed to contaminated estuary sediments compared to control fish faced with other stress factors such as ultraviolet (UV) radiation and hypoxia [MEY 03];

– in the case of flounder (*P. flesus*), resistant genotypes, characterized by their significant capacity to maintain the integrity of their DNA in polluted estuaries, have shown a reduced fecundity and a weaker condition index [MAR 04].

Different molecular mechanisms for resisting toxins, therefore, generate energy costs that can affect essential physiological functions [TAY 96]. However, the majority of studies carried out in this domain were performed with experimental measurement in laboratory, and a major question remains: what is the validity of costs estimated in a laboratory, in a natural environment?

A system's "recuperation" time (resilience) depends on the capacity of species to recolonize the system (generation time) [BRO 00] and the half-life of the contaminant, which can vary from a few days to several years. The resilience can occur due to an increase in the abundance of species still in the system or by the migration of species coming from the exterior of the system. For example, studies in lotic systems have shown that the diversity of macroinvertebrates can fluctuate following the application of insecticides, because individuals can colonize sites upstream that are not affected by the insecticide [WAL 96]. In the same way, insects can recolonize lentic systems after exposure to pesticides, which contributes to the system's resilience [WAN 96]. This suggests that the geographic distribution of species and the structuration of metapopulations can play an important role in the recuperation of ecosystems.

The contaminants can have long-term effects on the ecosystems; Woin [WOI 98] has demonstrated that two years after the application of fenvalerate insecticide (a pyrethrinoid, organochlorinated insecticide), a community of invertebrates in a pond was significantly different in the diversity of the species and in abundance compared to control sites. In the same way, the application of herbicides can lead to a reduction in the biomass of producers, which persists because of recuperation periods specific to the species [SPA 97]. Overall, these studies show that the resilience of ecosystems to exposure to pesticides will depend on a certain number of parameters (beyond lethal and sublethal effects on each member of the community) such as the type of habitat, rates of migration, the dynamics of extinction and recolonization, the specific sensitivity of the species and the species' specific rates of recuperation.

The resilience *stricto sensu* of an ecosystem, following an improvement in the quality of the water and/or sediment, can be retarded by the installation of tolerant species that check recolonization by sensitive species, such as has been reported in polluted lakes [FRO 06]. The ecosystem has, therefore, evolved in a lasting manner, eventually returning to its initial functions, supported by other species.

Theoretical and empirical studies suggest that certain communities show abrupt and nonlinear changes in their structure or functioning in response to disturbances (Figure 1.8(d)) [CON 83, EST 95, MAY 77]. The concept of an ecological threshold has a direct relationship with the concepts of resistance and resilience. The threshold for resistance represents the concentrations of the contaminant that will lead to sudden modifications of the community's structure. The threshold of resilience represents "the point in time" where recuperation is initiated and finally completed after the elimination of the stress factor. This notion of a threshold is illustrated in several studies carried out on different systems such as lakes, coral reefs and the pelagic system [GRO 06, SCH 01].

1.5. Indirect effects and multiple stress factors

Generally speaking, marine ecosystems are simultaneously exposed to a combination of stress factors (contaminants, temperature, eutrophization, anoxia, etc.). Each stress factor can have an impact on an individual or the community, and their combination may or may not produce other additive effects [CAS 98]. Few studies have been carried out on the effects of multiple stress factors on biological communities [BRE 99], and even fewer have examined the consequences in terms of indirect effects [CUP 02]. Resistance to different types of contaminant is not necessarily correlated, which renders the impact of concomitant stresses unpredictable at the level of the community. For example, several studies [BRO 95, CUP 95, VAN 95] suggest that insecticides increase the negative effects of eutrophization by a "top-down" effect where the biomass of periphyton is no longer controlled by grazers vulnerable to insecticide.

One of the challenges of ecotoxicology – ecology under toxic stress – is to understand the toxic chemical pressure, amid the complex mix of multiple disruptions to natural environments. In this context, the importance of global climate change (GCC) and its potential interaction with contaminants in the environment has recently received increasing attention [CLE 09, NOY 09, SCH 07, VAN 08]. Projections from the intergovernmental group of experts on the climate *Groupe d'experts Intergouvernemental sur l'Evolution du*

Climate (GIEC), on the subject of changes for the end of this century, concern a wide range of environmental conditions: raised temperature, changes in precipitation regimes, an increase in the acidity of the oceans and a reduction in ice covering the seas [INT 07] (see also the [MON 14a, b and c] also from the Seas and Oceans set). These projections include significant uncertainties in regional variations but agree on an increase in the frequency of extreme meteorological phenomena such as heat waves, droughts and storms. The transfer, the arrival and then the exposure to contaminants will also be impacted by GCC [VER 08], although there is already great uncertainty associated with the effects of GCC on future concentrations of contaminants in the environment [NOY 09].

1.5.1. ***Impact on the future of contaminants***

It is at the level of the highest latitudes that the increase in temperature is the most pronounced. Reports from IPCC indicate that the average of temperatures in the Arctic has increased by almost twice more than the global average in the last hundred years [IPC 07]. POPs are, mostly, semi-volatile compounds that can be found thousands of kilometers from the place where they were emitted. In effect, they are transported, in the form of gas and/or adsorbed to aerosols, by global atmospheric circulation into higher latitudes. As the gradients in temperature between high and low latitudes will be less pronounced, the presence of persistent organic pollutants in high latitudes could diminish [BEY 03, BRA 05, BRE 04, WAN 96]. Moreover, this increase in temperature will also have an impact on the degradation processes of POP, which are more significant at higher temperatures. It is, however, important to note that this degradation can lead to metabolites that are more toxic than the initial compounds (see [DAL 07] on PCB and the dioxins in the Venice laguna). Organic carbon cycles in land and aquatic systems will also be modified by climate change, which will have a direct impact on the bioavailability of a large number of contaminants [MAC 03, MAG 97, SCH 97]. This phenomenon has been found in the boreal lakes of north-west Ontario following a long period of warming and drought associated with forest fires [SCH 97]. Moreover, the warming will “release” more easily the contaminants “trapped” in the permafrost, rendering them “available” for exchanges with air and/or running water [MAC 05]. This availability will also augment the degradation processes [BEY 03, BRU 98, MA 04, SCH 05].

In addition to numerous abiotic factors that can influence the behavior of contaminants, modifications in the migratory habits of several species due to climate change could be an important modulating factor for the transport of POP [BLA 07]. In effect, migratory species, such as fish, birds and sea mammals, can be exposed to contaminants in an impacted geographic zone (coastal or estuary zone) and by migrating, transport these contaminants in substantial quantities to other sites that are not directly impacted. This "biotic" transport of contaminants can have an order of size similar to atmospheric and oceanic transport [BUR 08]. In effect, Blais *et al.* [BLA 07] have shown that birds from the Arctic and Antarctica act as vectors for persistent contaminants from oceans to land systems *via* their guano. Thus, on the Canadian coast under the cliffs where northern fulmars nest (*Fulmarus glacialis*), concentrations in HexaChloroBenzene (HCB), DDT and mercury are 10–60 times higher than concentrations of contaminants in sediments not impacted by nesting. Similar results have been observed for Antarctica [BLA 07]. Thus, if climate change modifies the migrations of several species, the local and global transport of POPs will also be modified with impacts on other ecosystems [BUR 08, WRO 05].

Finally, the adaptation of societies to climate change (CC) also risks affecting the presence of contaminants in different regions due to modifications in agricultural practices, the more significant use of pesticides due to the proliferation of pests [KAT 11] and an increase in the exploitation of resources from polar regions [DE 11].

1.5.2. *Effects of contaminants and climate change on different organization levels of life forms*

Physiological mechanisms implicated in the combined effects of toxic substances and climatic stresses can be interpreted from two different angles, as proposed by [HOO 12] (Figure 1.9): (1) the increased sensitivity to toxins caused by the climate, where exposure to a stress factor linked to the climate renders an organism more sensitive to exposure to toxic substances (Figure 1.9, arrow 3), and (2) exposure to a contaminant renders an organism more vulnerable to climate changes (Figure 1.9, arrow 4).

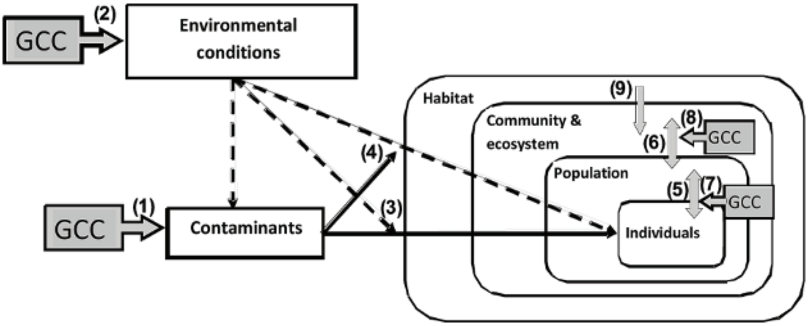

Figure 1.9. *Combined effects of the impact of global climate change (GCC) and contaminants on different levels of biological organization (taken from [MOE 13])*

COMMENTARY ON FIGURE 1.9.– The term "GCC" represents the climatic factors, such as the temperature and precipitation. The environmental conditions represent other abiotic factors (for example, hydrological regimes, UV radiation and concentrations of nutrients). GCC can affect the transfer and exposure of toxic products directly (arrow 1) or by means of environmental conditions (arrow 2) [GOU 13]. Individuals can be affected by GCC due to exposure to toxic substances and/or other environmental conditions; interactions between these factors can lead to a sensitivity to toxins caused by the climate (arrow 3) or a sensitivity to climate change caused by the contaminant (arrow 4) [HOO 12]. The combined effects of the contaminants and GCC on the individuals can spread to higher levels of biological organization (arrows 5 and 6). These transfer processes in turn can be affected by GCC, directly or indirectly (arrows 7 and 8). Finally, the properties habitats can influence the responses of populations and communities to the combined effects of contaminants and GCC (arrow 9).

Interactions between different environmental stresses can be synergic, antagonistic or cumulative. They vary depending on the trophic level (herbivore and predator) and the level of response (individual, population and community) [CRA 08]. It is, therefore, difficult to predict the impact that the effect of the climate, combined with chemical stress observed at an individual level, will have on other levels of biological organization [DE 11, PAI 98].

Many authors have found evidence that climate change has affected the phenology of organisms and the geographical distribution of species, as well as the composition and dynamic of communities [LOV 05, PEN 01, ROO 03, WAL 01].

The toxico-kinetic accumulation and the toxicity of POPs and pesticides in ecosystems are susceptible to increasing in response to the increase in temperatures and salinity [CAP 06, GAU 00, HEU 01, MOO 03, SCH 07, WAN 01, WAR 04]. A study carried out by Maruya *et al.* [MAR 05] on an estuary-dwelling fish (*F. heteroclitus*) has thus found evidence that rates of elimination of several toxaphene congeners are higher at 25°C than in water at 15°C. Similar results have been observed among perch on elimination rates of different PCB congeners over a seasonal cycle [PAT 07a] and on rainbow trout when following hydroxylation rates for PCB [BUC 07].

In parallel with these processes, the increase in temperature can also alter key physiological functions, aggravating the harmful effects of contaminants [BRO 02, BRO 04]. Although the exact mechanisms that underlie this relationship are not fully understood, several studies indicate that the temperature causes changes in the metabolism and notably in the activation of enzymatic systems implicated in the detoxification processes [BUC 07, LYD 99]. Thus, Capkin *et al.* [CAP 06] have shown that mortality rates in rainbow trout (*Oncorhynchus mykiss*) exposed to the insecticide endosulfan were higher at 16°C than at 13°C. Monserrat and Bianchini [MON 95] suggest a similar explanation for the increase in the toxicity of methyl parathion for crabs (*Chasmagnathus granulata*) after exposure to temperatures of 12–30°C. Conversely, DDT is generally considered to be more toxic at low temperatures, which could be due to an increase in the modulation sodium channels, which would lead to vulnerability in the nervous system [NAR 00].

Species living at the limits of their homeostatic or physiological tolerance will then be the most vulnerable to the double stress caused by climate change and exposure to contaminants [GOR 03, HEA 94, PAT 07]. In effect, the capacity of species and populations to tolerate raised temperatures can be altered by toxic products. This double exposure acts as a “co-stressor”: the toxic substances affecting the physiological functions can diminish the capacity of organisms to maintain homeostasy [BRO 04]. Ectoderms, like fish, are particularly vulnerable to these temperature–contaminant interactions. The capacity of brook trout (*Salvelinus fontinalis*) and Atlantic salmon (*Salmo salar*) to acclimatize to an increase in temperature is weakened by sublethal doses of DDT [AND 69].

Moreover, modifications in habitat and trophic networks caused by climate change can affect the toxicity of contaminants by modifying the means of exposure and the sensitivity of certain populations, in particular those that are already in a stress situation [BRE 04, BRO 02, GAS 03, GIL 03, MAC 05].

Finally, these interactions between climate change and exposure to contaminants will also be dependent on the life stage subject to this exposure. In effect, the sensitive stages of life induce responses that, in their turn, modify the physiological processes. This process is particularly marked in contamination by endocrine disruptors [BRI 08].

1.6. Conclusion

Marine ecotoxicology is a very recent avenue of research. Until the 1990s, the dilution capacity of the oceans appeared to be so great that a “drop of PCB in the sea” would not have any consequences... Indeed, it is no longer possible to ignore the harmful impact of xenobiotic contaminants. First, it has been proved that they are present in the confines of the ocean, in the environment as much as in the fauna to which it is home. Second, the chronic toxicity that they cause constitutes a still unvalidated but probable hypothesis for explaining certain collapses in marine species that are not otherwise understood: such as stocks of farmed species not being replaced after the cessation of farming, non-farmed species in decline and immune deficiencies in cultivated species... Finally, the contamination of sea produce consumed by mankind poses proven health problems and triggers numerous halts in fishing activity (eels, shad, sardines, etc.).

Even more than for aquatic continental ecosystems, the object of marine ecotoxicology is necessarily ecology under chronic toxic stress. In effect, the contaminants involved are transported over tens of thousands of kilometers, in a relatively little-fragmented environment. They can survive over decades and infect the whole of the trophic chain. These scales of time and biological organization immediately pose questions at the level of ecology and indirect effects.

However, knowledge of them remains limited, since relatively few experiments have been devised specifically to test it. Changes in behavior, physiology, trophic interactions and/or competition between species can produce changes in populations and the composition of the community that intensify or mask the direct toxic effects. “Trophic cascades” seem to be a shared type of indirect effect, but the setup of most of the experiments does not permit the univocal distinction between trophic interactions and interactions involving competition; these two types of response are indirect effects, but result from very different ecological processes.

More work is also necessary to understand the multiple stress factors and know how, directly and indirectly, they influence the structure and behavior of aquatic communities. Similarly, complementary research should be carried out to develop ecosystem models that describe and predict direct and indirect effects of contaminants on a large variety of aquatic habitats. The aim of these approaches should be to understand the ecological implications of environmental stress factors, and, finally, to aid the development of management strategies for preserving and restoring the integrity of natural habitats. To understand the direct toxic effects of the contaminants is, and will continue to be, an important part of this process. However, communities and ecosystems are much more than the sum of their components, and the challenge to come is to understand the sometimes subtle but important integration of the indirect influence of contaminants in a realistic approach to exposure.

Experiments in micro- and meso-cosmes constitute necessary tools for studying the combined effects of different environmental stresses; although there is disagreement and debate concerning the modeling methods for the analysis and interpretation of these data [LIE 11, VAN 12]. Long-term ecotoxicological experiments that integrate the combination of climate evolution and environmental variations on a credible scale would permit more reliable predictions of the impact of toxic products in the context of climate change. Three approaches seem particularly promising:

– studying the potential of species to adapt, with the help of genetic analyses (variability and correlations);

– to carry out experiments (in a controlled setting) on microevolution;

– to compare populations tolerant to climate changes with populations resistant to toxic substances [SCH 11].

Marine ecosystems have been living under toxic pressure, probably for decades, but the level of this pressure has increased considerably with the rapid expansion of synthetic chemistry and the production of manufactured goods from the middle of the 20th Century. If marine life has developed with ingenious mechanisms enabling it to regulate, indeed to use, metals that are a constituent of the Earth’s crust, it is not necessarily the same for the new, synthetic organic molecules, which are endlessly renewed, with which it is faced today. The formidable distances over which these substances are transported, physically or biologically, separate the origins and impacts of these pollutions in space and time. Everything should be done to reduce contamination on such a vast scale in environments that have a heritage value and are rich in practical uses.

But, we also have a lot to learn about the dynamics of these marine ecosystems that have developed strategies of maintenance, indeed of development. Life is fundamentally fluid, “unpredictable and uncertain [...]. We should credit it with the ability to surprise us" advises Tassin [TAS 14]. To study ecological mechanisms developed in the face of toxic pressure without let up can also give us keys to understanding the living world that may help the human species itself to live with global changes, hand-in-hand with the trajectory of the biosphere of which it is part.

1.7. Bibliography

[ABI 12] ABIDLI S., MACHADO SANTOS M., LAHBIB Y., *et al.*, "Tributyltin (TBT) effects on *Hexaplex trunculus* and *Bolinus brandaris* (Gastropoda:Muricidae): imposex induction and sex hormone levels insights", *Ecological Indicators*, vol. 13, pp. 13–21, 2012.

[ADA 00] ADAMS M., GREELEY M.S., "Ecotoxicological indicators of water quality: using multi-response indicators to assess the health of aquatic ecosystems", *Water, Air, & Soil Pollution*, vol. 123, pp. 103–115, 2000.

[ADA 05a] ADAMS M., "Assessing cause and effect of multiple stressors on marine systems", *Marine Pollution Bulletin*, vol. 51, pp. 649–657, 2005.

[ADA 05b] ADAMS M., RYON M.G., SMITH J.G., "Recovery in diversity of fish and invertebrate communities following remediation of a polluted stream: investigating causal relationships", *Hydrobiologia*, vol. 542, pp. 77–93, 2005.

[AMI 11] AMIARD-TRIQUET C., "Pollution tolerance: from fundamental biological mechanisms to ecological consequences", in AMIARD-TRIQUET C., RAINBOW P.S., ROMÉO M. (eds.), *Tolerance to Environmental Contaminants*, CRC Press, Boca Raton, pp. 1–24, 2011.

[AND 69] ANDERSON J.M., PETERSON M.R., "DDT – sublethal effects on brook trout nervous system", *Science*, vol. 164, no. 3878, pp. 440–441, 1969.

[AND 92] ANDERSSON T., FÖRLIN L., "Regulation of the cytochrome P450 enzyme system in fish", *Aquatic Toxicology*, vol. 24, pp. 1–20, 1992.

[BAZ 97] BAZZANTI M., CHIAVARINI S., CREMISINI C., *et al.*, "Distribution of PCB congeners in aquatic ecosystems: a case study", *Environment International*, vol. 23, pp. 799–813, 1997.

[BEL 00] BELANGER S.E., GUCKERT J.B., BOWLING J.W., *et al.*, "Responses of aquatic communities to 25-6 alcohol ethoxylate in model stream ecosystems", *Aquatic Toxicology*, vol. 48, pp. 135–150, 2000.

[BEL 01] BELFIORE N.M., ANDERSON S.L., "Effects of contaminants on genetic patterns in aquatic organisms: a review", *Mutation Research*, vol. 489, pp. 97–122, 2001.

[BEL 04] BELLWOOD D.R., HUGHES T.P., FOLKE C., *et al.*, "Confronting the coral reef crisis", *Nature*, vol. 429, pp. 827–833, 2004.

[BEN 92] BENTON M.J., GUTTMAN S.I., "Allozyme genotype and differential resistance to mercury pollution in the caddisfly, *Nectopsyche albida*: II. Mutilocus genotypes", *Canadian Journal of Fisheries and Aquatic Sciences.*, vol. 49, pp. 147–149, 1992.

[BEY 03] BEYER A., WANIA F., GOUIN T., *et al.*, "Temperature dependence of the characteristic travel distance", *Environmental Science & Technology*, vol. 37, pp. 766–771, 2003.

[BIC 11] BICKHAM J.W., "The four cornerstones of evolutionary toxicology", *Ecotoxicology*, vol. 20, pp. 497–502, 2011.

[BLA 01] BLACK W.C., BAER C.F, ANTOLIN M.F, *et al.*, Population genomics: genome-wide sampling of insect populations", *Annual Review of Entomology*, vol. 46, pp. 441–469, 2001.

[BLA 07] BLAIS J.M., MACDONALD R.W., MACKEY D., *et al.*, "Biologically mediated transport of contaminants to aquatic systems", *Environmental Science & Technology*, vol. 41, no. 4, pp. 1075–1084, 2007.

[BOG 96] BOGOMOLOV D.M., CHEN S.K., PARMELEE R.W., *et al.*, "An ecosystem approach to soil toxicity testing: a study of copper contamination in laboratory soil microorganisms", *Applied Soil Ecology*, vol. 4, pp. 95–105, 1996.

[BOO 05] BOONSTRA R., MANZON R.G., MIHOK S., *et al.*, "Hormetic effects of gamma radiation on the stress axis of natural populations of meadow voles (Microtus pennsylvanicus)", *Environmental Toxicology and Chemistry*, vol. 24, pp. 334–343, 2005.

[BRA 05] BRAUNE B.M., OUTRIDGE P.M., FISK A.T., *et al.*, "Persistent organic pollutants and mercury in marine biota of the Canadian Arctic: an overview of spatial and temporal trends", *Science of the Total Environment*, vol. 351, pp. 4–56, 2005.

[BRE 99] BREITBURG D.L., SANDERS J.G., GILMOUR C.C., *et al.*, "Variability in responses to nutrients and trace elements, and transmission of stressor effects through an estuarine food web", *Limnology and Oceanography*, vol. 44, pp. 837–863, 1999.

[BRE 04] BREIVIK K., ALCOCK R., LI Y.F., *et al.*, "Primary sources of selected POPs: regional and global scale emission inventories", *Environmental Pollution*, vol. 128, nos. 1–2, pp. 3–16, 2004.

[BRI 08] BRIAN J.V., HARRIS C.A., RUNNALLS T.J., *et al.*, "Evidence of temperature-dependent effects on the estrogenic response of fish: implications with regard to climate change", *Science of the Total Environment*, vol. 397, pp. 72–81, 2008.

[BRO 82] BROECLKER W., PENG T., *Tracers in the Sea*, Eldigio, Palisades, p. 691, 1982.

[BRO 95] BROCK T.C.M., ROIJACKERS R.M.M., ROLLON R., *et al.*, "Effects of nutrient loading and insecticide application on the ecology of *Elodea*-dominated freshwater microcosms II. Responses of macrophytes, periphyton and macroinvertebrate grazers", *Archiv fur Hydrobiologie*, vol. 134, pp. 53–74, 1995.

[BRO 00] BROCK T., LAHR J., VAN DEN BRINK P.J., *Ecological Risks of Pesticides in Freshwater Ecosystems. Part 1: Herbicides*, Alterra Green World Research, Alterra, Wageningen, Netherlands, 2000.

[BRO 02a] BROOK R.K., RICHARDSON E.S., "Observations of polar bear predatory behaviour toward caribou", *Arctic*, vol. 55, no. 2, pp. 193–196, 2002.

[BRO 02b] BROOMHALL S., "The effects of endosulfan and variable water temperature on survivorship and subsequent vulnerability to predation in *Litoria citropa* tadpoles", *Aquatic Toxicology*, vol. 61, nos. 3–4, pp. 243–250, 2002.

[BRO 04] BROOMHALL S.D., "Egg temperature modifies predator avoidance and the effects of the insecticide endosulfan on tadpoles of an Australian frog", *Journal of Applied Ecology*, vol. 41, no. 1, pp. 105–113, 2004.

[BRO 10] BRODMAN R., NEWMAN W.D., LAURIE K., *et al.*, "Interaction of an aquatic herbicide and predatory Salamander density on wetland communities", *Journal of Herpetology*, vol. 44, pp. 69–82, 2010.

[BRU 98] BRUBAKER W.W., HITES R.A., "OH reaction kinetics of polycyclic aromatic hydrocarbons and polychlorinated dibenzo-p-dioxins and dibenzofurans", *Journal of Physical Chemistry A*, vol. 102, no. 6, pp. 915–921, 1998.

[BRU 01] BRUNO J.F., BERTNESS M.D., "Habitat modification and facilitation in benthic marine communities", in BERTNESS M.D., GAINES S.D., HAY M.E. (eds.), *Marine Community Ecology*, Sinauer Associates, Sunderland, pp. 201–218, 2001.

[BUC 07] BUCKMAN A.H., BROWN S.B., SMALL J., *et al.*, "Role of temperature and enzyme induction in the biotransformation of polychlorinated biphenyls and bioformation of hydroxylated polychlorinated biphenyls by rainbow trout (Oncorhynchus mykiss)", *Environmental Science and Technology*, vol. 41, pp. 3856–3863, 2007.

[BUR 94] BURGEOT T., BOCQUENE G., PINGRAY G., *et al.*, "Monitoring biological effects of contamination in marine fish along French coast by measurement of ethoxyresorufin-o-deethylase activity", *Ecotoxicology and Environmental Safety*, vol. 29, pp. 131–147, 1994.

[BUR 07] BURNETT K.G., BAIN LISA J., BALDWIN WILLIAM S., *et al.*, "*Fundulus* as the premier teleost model in environmental biology: opportunities for new insights using genomics", *Comparative Biochemistry and Physiology*, vol. 2D, pp. 257–86, 2007.

[BUR 08] BUREK K.A., GULLAND F.M.D., OHARA T.M., "Effects of climate change on Arctic marine mammal health", *Ecological Applications*, vol. 18, no. 2, pp. S126–S134, 2008.

[CAI 83] CAIRNS J., "Are single species toxicity tests alone adequate for estimating environmental hazard?", *Hydrobiologia*, vol. 100, pp. 47–57, 1983.

[CAJ 03] CAJARAVILLE M.P., HAUSER L., CARVALHO G., *et al.*, "Genetic damage and the molecular/cellular response to pollution", in LAWRENCE A.J., HEMINGWAY K.L. (eds.), *Effects of Pollution on Fish, Molecular Effects and Population Responses*, Blackwell Sciences, Oxford, pp. 14–82, 2003.

[CAL 83] CALABRESE E., *Principles of Animals Extrapolations*, John Wiley & Sons, New York, NY, 1983.

[CAL 97] CALOW P., SIBLY R., FORBES V., "Risk assessment on the basis of simplified life history scenarios", *Envionmental Toxicology and Chemistry*, vol. 16, pp. 1983–1989, 1997.

[CAN 02] CANIVET V., GIBERT J., “Sensitivity of epigeanand hypogean freshwater macroinvertebrates to complex mixture. Part I: laboratory experiments”, *Chemosphere*, vol. 46, pp. 999–1009, 2002.

[CAN 10] CANESI L., FABBRI R., GALLO G., *et al.*, “Biomarkers in Mytilus galloprovincialis exposed to suspensions of selected nanoparticles (nano carbon black, C60 fullerene, nano-TiO2, nano-SiO2)”, *Aquatic Toxicology*, vol. 100, no. 2, pp. 168–177, 2010.

[CAP 06] CAPKIN E., ALTINOK I., KARAHAN S., “Water quality and fish size affect toxicity of endosulfan, an organochlorine pesticide, to rainbow trout”, *Chemosphere*, vol. 64, no. 10, pp. 1793–800, 2006.

[CAR 97] CARMAN K.R., FLEEGER J.W., POMARICO S., “Response of a benthic food web to hydrocarbon contamination”, *Limnology and Oceanography*, vol. 42, pp. 561–571, 1997.

[CAS 98] CASSEE F.R., GROTEN J.P., VAN BLADEREN P.J., *et al.*, “Toxicological evaluation and risk assessment of chemical mixtures”, *Critical Reviews in Toxicology*, vol. 28, pp. 73–101, 1998.

[CAS 01] CASWELL H., *Matrix Population Models: Construction, Analysis and Interpretation*, 2nd ed., Sinauer Associates, Sunderland, 2001.

[CAS 06] CASELLAS C., “Risques écologiques et sanitaires des résidus de médicaments dans les eaux”, *Colloque Hydrosciences*, Montpellier, France, 16–17 June 2005, proceedings published in *Environnement, Risques & Sant*é (ERS), vol. 5, no. 4, p. 225, 2006.

[CLE 94] CLEMENTS W.H., KIFFNEY P.M., “Assessing contaminant effects at higher levels of biological organization”, *Environmental Toxicology and Chemistry*, vol. 13, pp. 357–359, 1994.

[CLE 99] CLEMENTS W.H., “Metal tolerance and predator-prey interactions in benthic macroinvertebrate stream communities”, *Ecological Applications*, vol. 9, pp. 1073–1084, 1999.

[CLE 02] CLEMENTS W.H., NEWMAN M.C., *Community Ecotoxicology*, John Wiley & Sons, 2002.

[CLE 09] CLEMENTS W.H., ROHR J.R., “Community responses to contaminants: using basic ecological principles to predict ecotoxicological effects”, *Environmental Toxicology and Chemistry*, vol. 28, pp. 1789–1800, 2009.

[CON 83] CONNELL J.H., SOUSA W.P., "On the evidence needed to judge ecological stability or persistence", *The American Naturalist*, vol. 121, pp. 789–824, 1983.

[COS 97] COSTANZA R., DARGE R., DEGROOT R., *et al.*, "The value of the world's ecosystem services and natural capita", *Nature*, vol. 387, p. 253, 1997.

[COS 11] COSSA D., HEIMBURGER L.E., LANNUZEL D., *et al.*, "Mercury in the Southern ocean", *Geochimica et Cosmochimica Acta*, vol. 75, no. 14, pp. 4037–4052, 2011.

[CRA 08a] CRAIN D.A., JANSSEN S.J., EDWARDS T.M., *et al.*, "Female reproductive disorders: the roles of endocrine-disrupting compounds and developmental timing", *Fertility and Sterility*, vol. 90, pp. 911–940, 2008.

[CRA 08b] CRAIN C.M, KROEKER K., HALPERN B.S., "Interactive and cumulative effects of multiple human stressors in marine systems", *Ecology Letters*, vol. 11, pp. 1304–1315, 2008.

[CUP 95] CUPPEN J.G.M., GYLSTRA R., VAN BEUSEKOM S., *et al.*, "Effects of nutrient loading and insecticide application on the ecology of Elodea-dominated freshwater microcosms III. Responses of macroinvertebrate detritivores, breakdown of plant litter, and final conclusions", *Archiv fur Hydrobiologie*, vol. 134, pp. 157–177, 1995.

[CUP 02] CUPPEN J.G.M., CRUM S.J.H., VAN DEN HEUVEL H., *et al.*, "Effects of a mixture of two insecticides in freshwater microcosms: I. Fate of chlorpyrifos and lindane and responses of macroinvertebrates", *Ecotoxicology*, vol. 11, pp. 165–180, 2002.

[DAL 07] DALLA VALLE M., CODATO E., MARCOMINI A., "Climate change influence on POPs distribution and fate: a case study", *Chemosphere*, vol. 67, no. 7, pp. 1287–1295, 2007.

[DAL 13] DALLAIRE R., DEWAILLY E., AYOTTE P., *et al.*, "Exposure to organochlorines and mercury through fish and marine mammal consumption: associations with growth and duration of gestation among Inuit newborns", *Environment International*, vol. 54, pp. 85–91, 2013.

[DAO 11] DAOUK T., LARCHER T., ROUPSARD F., *et al.*, "Long-term food-exposure of zebrafish to PCB mixtures mimicking some environmental situations induces ovary pathology and impairs reproduction ability", *Aquatic Toxicology*, vol. 105, pp. 270–278, 2011.

[DAS 14] DASTOOR A.P., DURNFORD D.A., "Arctic ocean: is it a sink or a source of atmospheric mercury?", *Environmental Science & Technology*, vol. 48, no. 3, pp. 1707–1717, 2014.

[DE 89] DE NOYELLES F. JR., KETTLE W.D., FROMM C.H., *et al.*, "Use of experimental ponds to assess the effects of a pesticide on the aquatic environment", *Miscellaneous Publications Entomological Society of America*, vol. 75, pp. 41–56, 1989.

[DE 09] DE MARSILY G., *L'eau, un trésor en partage*, Dunod, Paris, 2009.

[DE 11] DE HOOP L., SCHIPPER A.M., LEUVEN R.S.E.W., *et al.*, "Sensitivity of polar and temperate marine organisms to oil components", *Environmental Science & Technology*, vol. 45, pp. 9017–9023, 2011.

[DED 09] DEDOURGE-GEFFARD O., PALAIS F., BIAGIANTI-RISBOURG S., *et al.*, "Effects of metals on feeding rate and digestive enzymes in Gammarus fossarum: an *in situ* experiment", *Chemosphere*, vol. 77, no. 11, pp. 1569–1576, 2009.

[DEM 93] DEMARAIS B.D., DOWLING T.E., MONCKLEY W.L., "Post-perturbation genetic changes in populations of endangered Virgin River chubs", *Conservation Biology*, vol. 7, pp. 334–41, 1993.

[DEP 96] DEPLEDGE M.H., "Genetic ecotoxicology: an overview", *Journal of Experimental Marine Biology and Ecology*, vol. 200, pp. 57–66, 1996.

[DEW 08] DEWAILLY E., SUHAS E., MOU Y., *et al.*, "High fish consumption in French Polynesia and prenatal exposure to metals and nutrients", *Asia Pacific Journal of Clinical Nutrition*, vol. 17, no. 3, pp. 461–470, 2008.

[DIE 13] DIETZ R., SONNE C., BASU N., *et al.*, "What are the toxicological effects of mercury in Arctic biota?", *Science of the Total Environment*, vol. 443, pp. 775–790, 2013.

[DOD 95] DODSON S.I., HANAZATO T., GORSKI P.R., "Behavioral responses of *Daphnia pulex* exposed to carbaryl and *Chaoborus* Kairomone", *Environmental Toxicology and Chemistry*, vol. 14, pp. 43–50, 1995.

[DUB 96] DUBROVA Y.E., NESTEROV V.N., KROUCHINSKY N.G., *et al.*, "Human minisatellite mutation rate after the Chernobyl accident", *Nature*, vol. 380, pp. 683–686, 1996.

[DUF 00] DUFFY J.E., HAY M.E., "Strong impacts of grazing amphipods on the organization of a benthic community", *Ecological Monographs*, vol. 70, pp. 237–263, 2000.

[EDE 07] EDER K.J., KÖHLER H.R., WERNER I., "Pesticide and pathogen: heat shock protein expression and acetylcholinesterase inhibition in juvenile Chinook salmon in response to multiple stressors", *Environmental Toxicology and Chemistry*, vol. 26, pp. 1233–1242, 2007.

[EIR 09] EIRA C., TORRES J., MIQUEL J., *et al.*, "Trace element concentrations in *Proteocephalus macrocephalus* (Cestoda) and *Anguillicola crassus* (Nematoda) in comparison to their fish host, *Anguilla anguilla* in Ria de Aveiro, Portugal", *Science of the Total Environment*, vol. 407, pp. 991–998, 2009.

[ESC 02] ESCHER B., HERMENS J., "Modes of action in ecotoxicologie: their role in body burdens, species sensitivity, QSARs, and mixture effects", *Environmental Sciences and Technology*, vol. 36, pp. 4201–4217, 2002.

[EST 95] ESTES J.A., DUGGINS D.O., "Sea otters and kelp forests in Alaska – generality and variation in a community ecological paradigm", *Ecological Monographs*, vol. 65, pp. 75–100, 1995.

[EVE 94] EVERAARTS J.M., SLEIDERINK H.M., DEN BESTEN P.J., *et al.*, "Molecular responses as indicators of marine pollution: DNA damage and enzyme induction in *Limanda limanda* and *Asterias rubens*", *Environmental Health Perspectives*, vol. 102, Suppl. 12, pp. 37–43, 1994.

[FEC 14] FECHNER L., GOURLAY-FRANCÉ C., TUSSEAU-VUILLEMIN M.H., "Linking community tolerance and structure with low metallic contamination: a field study on 13 biofilms sampled across the Seine river basin", *Water Research*, vol. 51, pp. 152–162, 2014.

[FLE 03] FLEEGER J.W., CARMAN K.R., NISBET R.M., "Indirect effects of contaminants in aquatic ecosystems", *Science of the Total Environment*, vol. 317, pp. 207–233, 2003.

[FOR 06] FORSON D., STORFER A., "Effects of atrazine and iridovirus infection on survival and lifehistory traits of the long-toed salamander (*Ambystoma macrodactylum*)", *Environmental Toxicology and Chemistry*, vol. 25, pp. 168–173, 2006.

[FRO 06] FROST T.M., FISCHER J.M., KLUG J.L., *et al.*, "Trajectories of zooplankton recovery in the Little Rock Lake whole-lake acidification experiment", *Ecological Applications*, vol. 16, pp. 353–367, 2006.

[GAL 91] GALGANI F., BOCQUENE G., LUCON M., *et al.*, "EROD measurements in Fis from the Northwest Part of France", *Marine Pollution Bulletin*, vol. 22, pp. 494–500, 1991.

[GAL 01] GALLOWAY T.S., DEPLEDGE M.H., "Immunotoxicity in invertebrates: measurement and ecotoxicological relevance", *Ecotoxicology*, vol. 10, pp. 5–23, 2001.

[GAL 03] GALLOWAY T., HANDY R., "Immunotoxicity of organophosphorous pesticides", *Ecotoxicology*, vol. 12, pp. 345–363, 2003.

[GAS 03] GASTON A.J., WOO K., HIPFNER J.M., "Trends in forage fish populations in northern Hudson Bay since 1981, as determined from the diet of nestling thick-billed murres *Uria lomvia*", *Arctic*, vol. 56, no. 3, pp. 227–233, 2003.

[GAU 00] GAUNT P., BARKER S.A., "Matrix solid phase dispersion extraction of triazines from catfish tissues; examination of the effects of temperature and dissolved oxygen on the toxicity of atrazine", *International Journal of Environment and Pollution*, vol. 13, nos. 1–6, pp. 284–312, 2000.

[GEO 94] GEORGE S.G., "Enzymology and molecular biology of phase II xenobiotic-conjugating enzymes in fish", in MALINS D.C., OSTRANDER G.K. (eds.), *Aquatic Toxicology Molecular, Biochemical, and Cellular Perspectives*, Lewis, Boca Raton, pp. 37–85, 1994.

[GIL 99] GILLEPSIE R.B., GUTTMAN S.I., "Chemical-induced changes in the genetic structure of populations: effects on allozymes", in FORBES V.E. (ed.), *Genetics and Ecotoxicology*, Taylor and Francis, New York, pp. 55–77, 1999.

[GIL 03] GILBERTSON M.K., HAFFNER G.D., DROUILLARD K.G., *et al.*, "Immunosuppression in the northern leopard frog (*Rana pipiens*) induced by pesticide exposure", *Environmental Toxicology and Chemistry*, vol. 22, no. 1, pp. 101–110, 2003.

[GOI 13] GOIN T., ARMITAGE J., COUSINS I., *et al.*, "Influence of global climate change on chemical fate and bioaccumulation: the role of multimedia models", *Environmental Toxicology and Chemistry*, vol. 32, no. 1, pp. 20–31, 2013.

[GOK 98] GØKSOYR A., HUSOY A.M., "Immunochemical approaches to studies of CYP1A localization and induction by xenobiotics in fish", in BRAUNBECK T., HINTON D.E., STREIT B. (eds.), *Fish Ecotoxicology*, Birkhäuser Verlag, Berlin, pp. 165–202, 1998.

[GOR 03] GORDON C.J., "Role of environmental stress in the physiological response to chemical toxicants", *Environmental Research*, vol. 92, no. 1, pp. 1–7, 2003.

[GOU 12] GOULLETQUER P., GROS P., WEBER J., *et al.*, *Biodiversité en environnement marin*, Quae, Versailles, 2012.

[GOU 13] GOURLAY-FRANCÉ C., TUSSEAU-VUILLEMIN M., “Bioavailability of contaminants”, in FÉRARD J., BLAISE C. (eds.), *Encyclopedia of Aquatic Ecotoxicology*, Springer-Verlag, Berlin, Heidelberg, pp. 181–191, 2013.

[GRE 97] GREGG J.C., FLEEGER J.W., CARMAN K.R., “Effects of suspended, diesel-contaminated sediment on feeding rate in the darter goby, *Gobionellus boleosoma* (Teleostei: Gobiidae)”, *Marine Pollution Bulletin*, vol. 34, pp. 269–275, 1997.

[GRO 06] GROFFMAN P., BARON J., BLETT T., *et al.*, “Ecological thresholds: the key to successful environmental management or an important concept with no practical application?”, *Ecosystems*, vol. 9, pp. 1–13, 2006.

[GRO 99] GROENENDIJK D., KRAAK M., ADMIRAAL W., “Efficient shedding of accumulated metals during metamorphosis in metal-adapted populations of the midge Chironomus riparius”, *Environmental Toxicology and Chemistry*, vol. 18, pp. 1225–1231, 1999.

[HAH 04] HAHN M.E., KARCHNER S.I., FRANKS D.G., *et al.*, “Aryl hydrocarbon receptor polymorphisms and dioxin resistance in Atlantic killifish (*Fundulus heteroclitus*)”, *Pharmacogenetics*, vol. 14, pp. 131–43, 2004.

[HAH 05] HAHN M.E., FRANKS D.G., KARCHNER S.I., *et al.*, Mechanism of PCB- and dioxin-resistance in fish in the Hudson River estuary: role of receptor polymorphisms, Final Report, Hudson River Foundation Grant, New York, available at www.hudsonriver.org/ls/, p. 215, 2005.

[HAM 95] HAM L., QUINN R., PASCOE D., “Effects of cadmium on the predator–prey interaction between the turbellarian *Dendrocoelum lacteum* (Muller, 1774) and the isopod crustacean *Asellus aquaticus* (L)”, *Archives of Environmental Contamination and Toxicology*, vol. 29, pp. 358–365, 1995.

[HAR 04] HARPER-ARABIE R.M., WIRTH E.F., FULTON M.H., *et al.*, “Protective effects of allozyme genotype during chemical exposure in the grass shrimp *Palaemonetes pugio*”, *Aquatic Toxicology*, vol. 70, pp. 41–54, 2004.

[HAU 03] HAUSER L., ROCAP G., LARIMER F.W., *et al.*, "Molecular/cellular processes and the population genetics of a species", in LAWRENCE A.J., HEMINGWAY K.L. (eds.), *Effects of Pollution on Fish, Molecular Effects and Population Responses*, Blackwell Science, Oxford, pp. 256–288, 2003.

[HAV 93] HAVENS K.E., HANAZATO T., " ooplankton community responses to chemical stressors – a comparison of results from acidification and pesticides contamination research", *Environmental Pollution*, vol. 82, no. 3, pp. 277–288, 1993.

[HAV 95] HAVENS K.E., "Insecticide (Carbaryl, 1-Napthyl-N-Methylcarbamate) effects on a freshwater plankton community: zooplankton size, biomass, and algal abundance", *Water Air & Soil Pollution*, vol. 84, pp. 1–10, 1995.

[HEA 94] HEATH S., BENNETT W.A., KENNEDY J., *et al.*, "Heat and cold tolerance of the fathead minnow, *Pimephales promelas*, exposed to the synthetic pyrethroid cyfluthrin", *Canadian Journal of Fisheries and Aquatic Sciences*, vol. 51, no. 2, pp. 437–440, 1994.

[HEB 96] HEBERT P.D., LUIKER M.M., "Genetic effects of contaminant exposure – towards an assessment of impacts on animal populations", *Science of the Total Environment*, vol. 191, pp. 23–58, 1996.

[HEI 97] HEITHAUS M.R., LAUSHMAN R.H., "Genetic variation and conservation of stream fishes: influence of ecology, life history, and water quality", *Canadian Journal of Fisheries and Aquatic Sciences*, vol. 54, pp. 1822–1836, 1997.

[HEI 00] HEINONEN J., KUKKONEN J.V.K., HOLOPAINEN I.J., "Toxicokinetics of 2, 4, 5-trichlorophenol and benzo(a)pyrene in the clam Pisidium amnicum: effects of seasonal temperatures and trematode parasites", *Archives of Environmental Contamination and Toxicology*, vol. 39, pp. 352–359, 2000.

[HEM 07] HEMMER-HANSEN J., NIELSEN E.E., FRYDENBERG J., *et al.*, "Adaptive divergence in a high gene flow environment: Hsc70 variation in the European flounder (*Platichthys flesus* L.)", *Heredity*, vol. 99, pp. 592–600, 2007.

[HER 89] HERMENS J., "Quantitative structure-activity relationships for environmental pollutants", *Handbook of Environmental Chemistry*, Springer-Verlag, Berlin, pp. 111–162, 1989.

[HEU 01] HEUGENS E.H.W., HENDRIKS A.J., DEKKER T., *et al.*, "A review of the effects of multiple stressors on aquatic organisms and analysis of uncertainty factors for use in risk assessment", *Critical Reviews in Toxicology*, vol. 31, no. 3, pp. 247–284, 2001.

[HIN 98] HINKLE-CONN C., FLEEGER J.W., GREGG J.C., *et al.*, "The effect of sediment-bound polycyclic aromatic hydrocarbons on feeding behavior in juvenile spot (*Leiostomus xanthurus* Lace´pe`de: Pisces)", *Journal of Experimental Marine Biology and Ecology*, vol. 227, pp. 113–132, 1998.

[HOL 10] HOLMSTRUP M., BINDESBOL A.M., OOSTINGH G., *et al.*, "Interactions between effects of environmental chemicals and naturals stressors: a review", *Science of the Total Environment*, vol. 408, pp. 3746–3762, 2010.

[HOO 12] HOOPER M.J., ANKLEY G.T., CRISTOL D.A., *et al.*, "Interactions between chemical and climate stressors: a role for mechanistic toxicology in assessing climate change risks", *Environmental Toxicology and Chemistry*, vol. 32, pp. 32–48, 2012.

[IBR 98] IBRAHIM H., KHEIR R., HELMI S., *et al.*, "Effects of organophosphorus, carbamate, pyrethroid and organochlorine pesticides, and a heavy metal on survival and cholinesterase activity of Chironomus riparius Meigen", *Bulletin of Environmental Contamination and Toxicology*, vol. 60, pp. 448–455, 1998.

[IND 99] INDEHERBERG M., VAN STRAALEN N., SCHOCKAERT E., "Combinig life-history and toxicokinetic parameters to interpret differences in sensitivity to cadmium between populations of Polycelis tenuis (Plathelminthes)", *Ecotoxicology and Environmental Safety*, vol. 44, nos. 1–11, pp. 1–11, 1999.

[INT 07] INTERGOVERNMENTAL PANEL ON CLIMATE CHANGE, "Climate change 2007: the physical science basis", *Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*, Cambridge University Press, Cambridge, MA, 2007.

[IPC 07] INTERGOVERNMENTAL PANEL ON CLIMATE CHANGE, Climate Change: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, Cambridge University Press, Cambridge, UK, 2007.

[JAK 96] JAK R.G., MAAS J.L., SCHOLTEN M.C.T., "Evaluation of laboratory derived toxic effect concentrations of a mixture of metals by testing fresh water plankton communities in enclosures", *Water Resource*, vol. 30, pp. 1215–1227, 1996.

[JOH 11] JOHNSTON E., "Tolerance to contaminants: evidence from chronically exposed populations of aquatic organisms", in AMIARD-TRIQUET C., RAINBOW P.S., ROMÉO M. (eds.), *Tolerance to Environmental Contaminants*, CRC Press, Boca Raton, pp. 25–48, 2011.

[KAM 96] KAMMENGA J., BUSSHERS M., VAN STRAALEN N., *et al.*, "Stress-induced fitness reduction is not determined by the most sensitive life-cycle trait", *Functional Ecology*, vol. 10, pp. 106–111, 1996.

[KAM 00] KAMMENGA J., ARTS M.J., DOROSZUK A., "Multi-generation effects at the population level: fitness maximisation and optimal allocation in a nematode", in KAMMENGA J., LASKOWSKI J. (eds.), *Demography in Ecotoxicology*, pp. 163–77, 2000.

[KAT 11] KATTWINKEL M., KÜHNE J.V., FOIT K., *et al.*, "Climate change, agricultural insecticide exposure, and risk for freshwater communities", *Ecological Applications*, vol. 21, pp. 2068–2081, 2011.

[KAU 82] KAUFMAN L.H., "Stream aufwuchs accumulation: disturbance frequency and stress resistance and resilience", *Oecologia*, vol. 52, pp. 57–63, 1982.

[KIE 02] KIESECKER J.M., "Synergism between trematode infection and pesticide exposure: a link to amphibian limb deformities in nature?", *Proceedings of the National Academy of Sciences*, vol. 99, pp. 9900-9904, 2002.

[KIF 96] KIFFNEY P.M., CLEMENTS W.H., "Size-dependent response of macro invertebrates to metals in experimental streams", *Environmental Toxicology and Chemistry*, vol. 15, pp. 1352–1356, 1996.

[KIM 10] KIM K.H., DAHMS H.U.D., RHEE J.S., *et al.*, "Expression profiles of seven glutathione S-transferase (GST) genes in cadmium-exposed river pufferfish (*Takifugu obscurus*)", *Comparative Biochemistry and Physiology, Part C*, vol. 151, pp. 99–106, 2010.

[KLA 09] KLAINE S.J., "Considerations for research on the environmental fate and effects of nanoparticles", *Environmental Toxicology and Chemistry*, vol. 28, no. 9, pp. 1787–1788, 2009.

[KLE 01] KLERKS P., MOREAU C., “Heritability of resistance to individual contaminants and to contaminant mixtures in the sheepshead minnow (*Cyprinodon variegatus*)”, *Environ. Toxicol. Chem.*, vol. 20, pp. 1746–51, 2001.

[KNA 05] KNAPP C.W., CAQUET T., HANSON M.L., *et al.*, “Response of water column microbial communities to sudden exposure to deltamethrin in aquatic mesocosms”, *FEMS Microbiology Ecology*, vol. 54, pp. 157–165, 2005.

[KOE 91] KOEMAN J., “From comparative physiology to toxicological risk assessment”, *Comparative Biochemistry and Physiology*, vol. 100, pp. 7–10, 1991.

[KOI 92] KOIVISTO S., KETOLA M., WALLS M., “Comparison of five cladoceran species in short and long term copper exposure”, *Hydrobiologia*, vol. 248, pp. 125–136, 1992.

[KOL 02] KOLPIN D.W., BARBASH J.E., GILLIOM RJ., *et al.*, Pharmaceuticals, hormones and other organic wastewater contaminants in US streams, 1999-2000: a national reconnaissance”, *Environmental Science and Technology*, vol. 36, p. 1202, 2002.

[KOP 92] KOPP R.L., GUTTMAN S.I., WISSING T.E., “Genetic indicators of environmental stress in central mudminnows (*Umbra limi*) populations exposed to acid deposition in the Adirondack Mountains”, *Environmental Toxicology and Chemistry*, vol. 11, pp. 665–676, 1992.

[KOP 00] KOPPENHÖFFER A.M., GREWAL P.S., KAYA H.K., “Synergism of imidacloprid and entomopathogenic nematodes against white grubs: the mechanism”, *Entomologia Experimentalis et Applicata*, vol. 94, pp. 283–293, 2000.

[KRA 03] KRAEPIEL A.M.L., KELLER, K., CHIN, H.B., *et al.*, “Sources and variations of mercury in tuna”, *Environmental Science & Technology*, vol. 37, no. 24, pp. 5551–5558, 2003.

[KRU 94] KRUZYNSKI G.M., BIRTWELL I.K., “A predation bioassay to quantify the ecological significance of sublethal responses of juvenile chinook salmon (*Onchorynchus tshawytscha*) to the antisapstain fungicide TCMTB”, *Canadian Journal of Fisheries and Aquatic Science*, vol. 51, pp. 1780–1790, 1994.

[LAR 01] LARNO V., LAROCHE J., LAUNEY S., *et al.*, "Responses of chub (*Leuciscus cephalus*) populations to chemical stress, assessed by genetic markers, DNA damage and cytochrome P450 1A induction", *Ecotoxicology*, vol. 10, pp. 145–158, 2001.

[LAR 02] LAROCHE J., QUINIOU L., JUHEL G., *et al.*, "Genetic and physiologic responses of flounder (*Platichthys flesus*) populations to chemical contamination in estuaries", *Environmental Toxicology and Chemistry*, vol. 21, pp. 2705–2712, 2002.

[LAY 85] LAY J.P., SCHAUERTE W., PEICHL L., *et al.*, "Influence of benzene on the phytoplankton and on *Daphnia pulex* in compartments of an experimental pond", *Ecotoxicology* and *Environmental Safety*, vol. 10, pp. 218–227, 1985.

[LEB 06] LEBLANC J.C., CALIPSO study, ANSES, 2006.

[LEF 99] LEFCORT H., THOMSON S.M., COWLES E.E., *et al.*, "Ramifications of predator avoidance: predator and heavy metal-mediated competition between tadpoles and snails", *Ecological Applications*, vol. 9, pp. 1477–1489, 1999.

[LEV 09] LEVITIS D.A., LIDICKER JR. W.Z., FREUND G., "Behavioural biologists do not agree on what constitutes behaviour", *Animal Behaviour*, vol. 78, no. 1, pp. 103–110, 2009.

[LIE 11] LIESS M., BEKETOV M., "Traits and stress: keys to identify community effects of low levels of toxicants in test systems", *Ecotoxicology*, vol. 20, pp. 1328–1340, 2011.

[LIV 00] LIVINGSTONE D.R., CHIPMAN J.K., LOWE D.M., *et al.*, "Development of biomarkers to detect the effects of organic pollution on aquatic invertebrates: recent molecular, genotoxic, cellular and immunological studies on the common mussel (*Mytilus edulis* L.) and other mytilids", *International Journal of Environment and Pollution*, vol. 13, pp. 56–91, 2000.

[LOV 05] LOVEJOY T.E., HANNAH L., *Climate Change and Biodiversity*, Yale University Press, New Haven, 2005.

[LYD 99] LYDY M.J., BELDEN J.B., TERNES M.A., "Effects of temperature on the toxicity of M-parathion, chlorpyrifos, and pentachlorobenzene to Chironomus tentans", *Archives of Environmental Contamination and Toxicology*, vol. 37, no. 4, pp. 542–547, 1999.

[MA 04] MA J., HUNG H., BLANCHARD P., "How do climate fluctuations affect persistent organic pollutant distribution in North America? Evidence from a decade of air monitoring", *Environmental Science & Technology*, vol. 38, no. 9, pp. 2538–2543, 2004.

[MAC 92] MACKAY D., "Correlation of bioconcentration factors", *Environmental Science and Technology*, vol. 16, pp. 274–278, 1992.

[MAC 03] MACDONALD R.W., MACKAY D., LI Y.F., *et al.*, "How will global climate change affect risks from long-range transport of persistent organic pollutants?", Human *and Ecological* Risk Assessment, vol. 9, no. 3, pp. 643–660, 2003.

[MAC 05] MACDONALD R.W., HARNER T., FYFE J., "Recent climate change in the Arctic and its impact on contaminant pathways and interpretation of temporal trend data", *Science of the Total Environment*, vol. 342, nos. 1–3, pp. 5–86, 2005.

[MAG 97] MAGNUSON J.J., WEBSTER K.E., ASSEL R.A., *et al.*, "Potential effects of climate changes on aquatic systems: Laurentian Great Lakes and Precambrian Shield region", *Hydrological Processes*, vol. 11, pp. 825–871, 1997.

[MAR 03] MARCHAND J., TANGUY A., LAROCHE J., *et al.*, "Responses of European flounder *Platichthys flesus* populations to contamination in different estuaries along the Atlantic coast of France", *Marine Ecology Progress Series*, vol. 260, pp. 273–284, 2003.

[MAR 04] MARCHAND J., QUINIOU L., RISO R., *et al.*, "Physiological cost of tolerance to toxicants in the European flounder *Platichthys flesus*, along the French Atlantic Coast", *Aquatic Toxicology*, vol. 70, pp. 327–343, 2004.

[MAR 05] MARUYA K.A., SMALLING K.L., VETTER W., "Temperature and congener structure affect the enantioselectivity of toxaphene elimination by fish", *Environmental Science and Technology*, vol. 39, no. 11, pp. 3999–4004, 2005.

[MAR 10] MARCHAND J., EVRARD E., GUINAND B., *et al.*, "Genetic polymorphism and its potential relation to environmental stress in five populations of the European flounder *Platichthys flesus*, along the French Atlantic coast", *Marine Environmental Research*, vol. 70, pp. 201–209, 2010.

[MAY 77] MAY R.M., "Thresholds and breakpoints in ecosystems with a multiplicity of stable states", *Nature*, vol. 269, pp. 471–477, 1977.

[MEN 08] MENDOLA P., MESSER L.C., RAPPAZZO K., "Science linking environmental contaminant exposures with fertility and reproductive health impacts in the adult female", *Fertility and Sterility*, vol. 89, pp. e81–e94, 2008.

[MEY 03] MEYER J.N., WASSENBERG D.M., KARCHNER S.I., *et al.*, "Antioxidant defenses in killifish *(Fundulus heteroclitus)* exposed to contaminated sediments and model prooxidants: short term and heritable responses", *Aquatic Toxicology*, vol. 65, pp. 377–395, 2003.

[MIL 05] MILLS L.J., CHICHESTER C., "Review of evidence: are endocrine-disrupting chemicals in the aquatic environment impacting fish populations?", *Science of the Total Environment*, vol. 343, pp. 1–34, 2005.

[MOE 13] MOE S., DE SCHAMPHELAERE K., CLEMENTS W., *et al.*, "Combined and interactive effects of global climate change and toxicants on populations and communities", *Environmental Toxicology and Chemistry*, vol. 32, no. 1, pp. 49–61, 2013.

[MON 95] MONSERRAT J., BIANCHINI A., "Effects of temperature and salinity on the toxicity of a commercial formulation of methyl parathion to Chasmagnathus granulata (Decapoda, Grapsidae)", *Brazilian Journal of Medical and Biological Research*, vol. 28, no. 1, pp. 74–78, 1995.

[MON 14a] MONACO A., PROUZET P., *Ocean in the Earth System*, ISTE, London and John Wiley & Sons, New York, 2014.

[MON 14b] MONACO A., PROUZET P., *Land-Sea Interactions*, ISTE, London and John Wiley & Sons, New York, 2014.

[MON 14c] MONACO A., PROUZET P., *Ecosystem Sustainability and Global Change*, ISTE, London and John Wiley & Sons, New York, 2014.

[MOO 03] MOORE A., SCOTT A.P., LOWER N., *et al.*, "The effects of 4-nonylphenol and atrazine on Atlantic salmon (*Salmo salar* L) smolts", *Aquaculture*, vol. 222, nos. 1–4, pp. 253–263, 2003.

[MOU 11] MOUNEYRAC C., LEUNG P.T.Y., LEUNG K.M.Y., "Cost of tolerance", in AMIARD-TRIQUET C., RAINBOW P.S., ROMÉO M. (eds.), *Tolerance to Environmental Contaminants*, CRC Press, Boca Raton, pp. 265–297, 2011.

[MUL 02] MULVEY M., NEWMAN M.C., VOGELBEIN W., *et al.*, "Genetic structure of *Fundulus heteroclitus* from Polycyclic Aromatic Hydrocarbon (PAH)-contaminated and neighboring sites in the Elizabeth and York Rivers", *Aquatic Toxicology*, vol. 61, pp. 195–209, 2002.

[MUN 95] MUNKITTRICK K.R., MCCARTY L.S., "An integrated approach to aquatic ecosystem health: topdown, bottom-up or middle-out?", *Journal of Aquatic Ecosystem Stress and Recovery (Formerly Journal of Aquatic Ecosystem Health)*, vol. 4, pp. 77–90, 1995.

[MUR 94] MURDOCH M.H., HEBERT P.D.N., "Mitochondrial DNA diversity of brown bullhead from contaminated and relatively pristine sites in the great lakes", *Environmental Toxicology and Chemistry*, vol. 13, pp. 1281–1289, 1994.

[MUR 97] MURPHY D.L., GOOCH J.W., "EROD and CYP1A protein in channel catfish (*Ictalurus punctatus*) from an urban estuary relative to that in benzo[a]pyrene-exposed hatchery specimens", *Environmental Pollution*, vol. 95, no. 2, pp. 235–239, 1997.

[NAR 00] NARAHASHI T., "Neuroreceptors and ion channels as the basis for drug action: past, present, and future", *Journal of Pharmacology and Experimental Therapeutics*, vol. 294, no. 1, pp. 1–26, 2000.

[NOY 09] NOYES P.D., MCELWEE M.K., MILLER H.D., *et al.*, "The toxicology of climate change: environmental contaminants in a warming world", *Environmental International*, vol. 35, pp. 971–986, 2009.

[PAI 98] PAINE R.T., TEGNER M.J., JOHNSON E.A., "Compounded perturbations yield ecological surprises", *Ecosystems*, vol. 1, pp. 535–545, 1998.

[PAK 00] PAKERTON T., KONKEL W., "Application of quantitative structure-activity relationships for assessing the aquatic toxicology of phthalate esters", *Ecotoxicology and Environmental Safety*, vol. 45, pp. 61–78, 2000.

[PAL 08] PALUMBI S., MCLEOD K., GRUNBAUM D., "Ecosystems in action: lessons from marine ecology about recovery, resistance, and reversibility", *Bioscience*, vol. 58, no. 1, pp. 33–42, 2008.

[PAT 07a] PATERSON G., DROUILLARD K.G., HAFFNER G.D., "PCB elimination by yellow perch (Perca flavescens) during an annual temperature cycle", *Environmental Science and Technology*, vol. 41, pp. 824–829, 2007.

[PAT 07b] PATRA R.W., CHAPMAN J.C., LIM R.P., *et al.*, "The effects of three organic chemicals on the upper thermal tolerances of four freshwater fishes", *Environmental Toxicology and Chemistry*, vol. 26, no. 7, pp. 1454–1459, 2007.

[PEA 12] PÉAN S., Effets des polluants organiques persistants sur le comportement des poissons, PhD Thesis, University of La Rochelle, 2012.

[PEA 13] PÉAN S., DAOUK T., VIGNET C., *et al.*, "Long-term dietary-exposure to non-coplanar PCBs induces behavioral disruptions in adult zebrafish and their offspring", *Neurotoxicology and Teratology*, vol. 39, pp. 45–56, 2013.

[PEN 01] PENUELAS J., FILELLA L., "Responses to a warming world", *Science*, vol. 294, pp. 793–795, 2001.

[PET 96] PETERSON C.H., KENNICUTT M.C., GREEN R.H., *et al.*, "Ecological consequences of environmental perturbations associated with offshore hydrocarbon production: a perspective on long-term exposures in the Gulf of Mexico", *Canadian Journal of Fisheries and Aquatic Science*, vol. 53, pp. 2637–2654, 1996.

[POD 01] PODEMSKI C.L., CULP J.M., "Toxicant interactions with food algae: a missing link between laboratory and field effects?", *Environmental Toxicology and Chemistry*, vol. 16, pp. 31–42, 2001.

[PRE 99] PRESTON B.L., CECCHINE G., SNELL T.W., "Effects of pentachlorophenol on predator avoidance behavior of the rotifer *Brachionus calyciflorus*", *Aquatic Toxicology*, vol. 44, pp. 201–212, 1999.

[REL 05] RELYEA R.A., "The impact of insecticides and herbicides on the biodiversity and productivity of aquatic communities", *Ecological Applications*, vol. 15, pp. 618–627, 2005.

[REL 06] RELYEA R.A., HOVERMAN J., "Assessing the ecology in ecotoxicology: a review and synthesis in freshwater systems", *Ecology Letters*, vol. 9, pp. 1157–1171, 2006.

[ROO 03] ROOT T.L., PRICE J.T., HALL K.R., *et al.*, "Fingerprints of global warming on wild animals and plants", *Nature*, vol. 421, no. 6918, pp. 57–60, 2003.

[ROU 87] ROUSH R.T., MCKENZIE J.A., “Ecological genetics of insecticide and acaricide resistance”, *Annual Review of Entomology*, vol. 32, pp. 361–380, 1987.

[SAL 97] SALMINEN J., SETALA H., HAIMI J., “Regulation of decomposer community structure and decomposition processes in herbicide stressed humus soil”, *Applied Soil Ecology*, vol. 6, pp. 265–274, 1997.

[SAL 01] SALMINEN J., ANH B.T., VAN GESTEL C.A.M., “Indirect effects of zinc on soil microbes via a keystone enchytaeid species”, *Environmental Toxicology and Chemistry*, vol. 20, pp. 1167–1174, 2001.

[SCH 97] SCHINDLER D.W., CURTIS P.J., BAYLEY S.E., *et al.*, “Climate-induced changes in the dissolved organic carbon budgets of boreal lakes”, *Biogeochemistry*, vol. 36, pp. 9–28, 1997.

[SCH 01] SCHEFFER M., CARPENTER S., FOLEY J.A., *et al.,* “Catastrophic shifts in ecosystems”, *Nature*, vol. 413, pp. 591–596, 2001.

[SCH 05] SCHEYER A., GRAEFF C., MORVILLE S., *et al.*, “Analysis of some organochlorine pesticides in an urban atmosphere (Strasbourg, east of France)”, *Chemosphere*, vol. 58, no. 11, pp. 1517–1524, 2005.

[SCH 07] SCHIEDEK D., SUNDELIN B., READMAN J.W., *et al.*, “Interactions between climate change and contaminants”, *Marine Pollution Bulletin*, vol. 54, pp. 1845–1856, 2007.

[SCH 11] SCHAMPHELAERE K.A.C., GLAHOLT S., ASSELMAN J., *et al.*, “Will genetic adaptation of natural populations to chemical pollution result in lower or higher tolerance to future climate change?”, *Integrated Environmental Assessment and Management*, vol. 7, pp. 141–143, 2011.

[SHI 13] SHIEL A., WEIS D., COSSA D., *et al.*, “Determining provenance of marine metal pollution in French bivalves using Cd, Zn and Pb isotopes”, *Geochimica et Cosmochimica Acta*, vol. 121, no. 15, pp. 155–167, 2013.

[SLI 05] SLIJKERMAN D.M.E., MOREIRA-SANTOS M., JAK R.G., “Functional and structural impact of linuron on a freshwater community of primary producers: the use of immobilized algae”, *Environmental Toxicology and Chemistry*, vol. 24, suppl. vol. I, no. 10, pp. 2477–2485, 2005.

[SMI 97] SMITH G.M., WEIS J.S., “Predator-prey relationships in mummichogs (*Fundulus heteroclitus* (L)): effects of living in a polluted environment”, *Journal of Experimental Marine Biology and Ecology*, vol. 209, pp. 75–87, 1997.

[SOM 02] SOMERS C.M., YAUK C.L., WHITE P.A., *et al.*, "Air pollution induces heritable DNA mutations", *Proceedings of the National Academy of Sciences*, vol. 99, pp. 15904–15907, 2002.

[SPA 97] SPAWN R.L., HOAGLAND K.D., SIEGFRIED B.D., "Effects of alachlor on an algal community from a midwestern agricultural stream", *Environmental Toxicology and Chemistry*, vol. 16, pp. 785–793, 1997.

[SPR 05] SPROMBERG J., BIRGE J., "Modeling the effects of chronic toxicity on fish populations: the influence of life history strategies", *Environmental Toxicology and Chemistry*, vol. 24, pp. 1532–1540, 2005.

[TAS 14] TASSIN J., *La grande invasion*, Odile Jacob, Paris, 2014.

[TAY 95] TAYLOR E.J., MORRISON J.E., BLOCKWELL S.J., *et al.*, "Effects of lindane on the predator-prey interaction between *Hydra oligactis* Pallas and *Daphnia magna* Strauss", *Archives of Environmental Contamination Toxicology*, vol. 29, pp. 291–296, 1995.

[TAY 96] TAYLOR M., FEYEREISEN R., "Molecular biology and evolution of resistance to toxicants", *Molecular Biology and Evolution*, vol. 13, pp. 719–734, 1996.

[TEL 04] TELES M., GRAVATO C., PACHECO M., *et al.*, "Juvenile sea bass biotransformation, genotoxic and endocrine responses to β-naphthoflavone, 4-nonylphenol and 17 β-estradiol individual and combined exposures", *Chemosphere*, vol. 57, pp. 147–158, 2004.

[TEM 99] TEMARA A., GULEC I., HOLDWAY D.A., "Oil-induced disruption of foraging behaviour of the asteroid keystone predator, *Coscinasterias muricata* (Echinodermata)", *Marine Biology*, vol. 133, pp. 501–507, 1999.

[TRE 04] TREMOLADA P., FINIZIO A., VILLA S., *et al.*, "Quantitative inter-specific chemical activity relationships of pesticides in the aquatic environment", *Aquatic Toxicology*, vol. 67, pp. 87–103, 2004.

[TRI 97] TRIEBSKORN R., KÖHLER H.R., HONNEN W., *et al.*, "Induction of heat shock proteins, changes in liver ultrastructure, and alterations of fish behavior: are these biomarkers related and are they useful to reflect the state of pollution in the field?", *Journal of Aquatic Ecosystem Stress and Recovery* (Formerly Journal of Aquatic Ecosystem Health), vol. 6, no. 1, pp. 57–73, 1997.

[TUS 07] TUSSEAU-VUILLEMIN M.H., GOURLAY C., LORGEOUX C., *et al.*, "Dissolved and bioavailable contaminants in the Seine river basin", *Science of the Total Environment*, vol. 375, nos. 1–3, pp. 244–256, 2007.

[VAR 89] VARANASI U., *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*, CRC Press, Boca Raton, 1989.

[VAN 92] VAN LEEUWEN C., VAN DER ZANDT T., "Application of QSARs, extrapolation and equilibrium partitioning in aquatic effects assessment. I. Narcotic Industrial pollutants", *Environmental Toxicology and Chemistry*, vol. 11, pp. 267–282, 1992.

[VAN 95] VAN DONK E., PRINS H., VOOGD H.M., *et al.*, "Effects of nutrient loading and insecticide application on the ecology of *Elodea*-dominated freshwater microcosms I. Responses of plankton and zooplanktivorous insects", *Archiv für Hydrobiologie*, vol. 133, pp. 417–439, 1995.

[VAN 00] VAN DEN BRINK P.J., HATTINK J., BRANSEN F., *et al.*, "Impact of the fungicide carbendazim in freshwater microcosms. II. Zooplankton primary producers and final conclusions", *Aquatic Toxicology*, vol. 48, pp. 251–264, 2000.

[VAN 02] VAN DEN BRINK P.J., ROELSMA J., VAN NES E.H., *et al.*, "PERPEST model, a case-based reasoning approach to predict ecological risks of pesticides", *Environmental Toxicology and Chemistry*, vol. 21, pp. 2500–2506, 2002.

[VAN 08] VAN DEN BRINK P.J., "Ecological risk assessment: from book-keeping to chemical stress ecology", *Environmental Science and Technology*, vol. 42, pp. 8999–9004, 2008.

[VAN 12] VAN DEN BRINK P.J., TER BRAAK C.J.F., "Response to ''Traits and stress: keys to identify community effects of low levels of toxicants in test systems'' by Liess and Beketov (2011)", *Ecotoxicology*, vol. 21, pp. 297–299, 2012.

[VER 08] VERDONCK F.A.M.,VAN SPRANG P.A., VANROLLEGHEM P.A., "An intelligent data collection tool for chemical safety/risk assessment", *Chemosphere*, vol. 70, pp. 1818–1826, 2008.

[VIR 03] VIRGILIO M., BARONCINI N., TROMBINI C., *et al.*, "Relationships between sediments and tissue contamination and allozymic patterns in *Hediste diversicolor* (Polychaeta Nereididae) in the Pialassa lagoons (north Adriatic Sea)", *Oceanologica Acta*, vol. 26, pp. 85–92, 2003.

[WAL 96] WALLACE J.B., GRUBAUGH J.W., WHILES M.R., *et al.* "Biotic indices and stream ecosystem processes: results from an experimental study", *Ecological Applications*, vol. 6, pp. 140–151, 1996.

[WAL 00] WALLACE W.G., HOEXUM BROUWER T.M., BROUWER M., *et al.*, "Alterations in prey capture and induction of metallothioneins in grass shrimp fed cadmium-contaminated prey", *Environmental Toxicology and Chemistry*, vol. 19, pp. 962–971, 2000.

[WAN 96] WANIA F., MACKAY D., "Tracking the distribution of persistent organic pollutants", 1996", *Environmental Science and Technology*, vol. 30, no. 9, pp. A390–A396, 1996.

[WAN 01] WANG J., GRISLE S., SCHLENK D., "Effects of salinity on aldicarb toxicity in juvenile rainbow trout (*Oncorhynchus mykiss*) and striped bass (Morone saxatilis x chrysops)", *Toxicology Science*, vol. 64, no. 2, pp. 200–207, 2001.

[WAR 04] WARING C.P., MOORE A., "The effect of atrazine on Atlantic salmon (Salmo salar) smolts in freshwater and after sea water transfer", *Aquatic Toxicology*, vol. 66, no. 1, pp. 93–104, 2004.

[WEB 02] WEBER L.P., DIAMOND S.L., BANDIERA S.M., *et al.*, "Expression of HSP70 and CYP1A protein in ovary and liver of juvenile rainbow trout exposed to b-naphthoflavone", *Comparative Biochemistry and Physiology, Part C*, vol. 131, pp. 387–394, 2002.

[WEI 04] WEINER J., DE LORENZO M., FULTON M., "Relationship between uptake capacity and differential toxicity of the herbicide atrazine in selected microalgal species", *Aquatic Toxicology*, vol. 68, pp. 121–128, 2004.

[WEI 95] WEIS J.S., WEIS P., "Swimming performance and predator avoidance by mummichog (*Fundulus heteroclitus*) larvae after embryonic or larval exposure to methylmercury", *Canadian Journal of Fisheries and Aquatic Science*, vol. 52, pp. 2168–2173, 1995.

[WEI 01] WEIS J.S., SAMSON J., ZHOU T., *et al.*, "Prey capture ability of mummichogs (*Fundulus heteroclitus*) as a behavioural biomarker for contaminants in estuarine systems", *Canadian Journal of Fisheries and Aquatic Science*, vol. 58, pp. 1442–1452, 2001.

[WEI 02] WEIS J.S., "Tolerance to environmental contaminants in the mummichog, *Fundulus heteroclitus*", *Human and Ecological Risk Assessment*, vol. 8, pp. 933–953, 2002.

[WID 91] WIDDOWS J., DONKIN P., "Role of physiological energetics in ecotoxicology", *Comparative Biochemistry and Physiology, Comparative Pharmacology Toxic Endocrinology*, vol. 100, pp. 69–75, 1991.

[WHY 00] WHYTE J.J., JUNG R.E., SCHMITT C.J., *et al.*, "Ethoxyresorufin-O-deethylase (EROD) activity in fish as a biomarker of chemical exposure", *Critical Review in Toxicology*, vol. 30, pp. 347–570, 2000.

[WOI 98] WOIN P., "Short- and long-term effects of the pyrethroid insecticide fenvalerate on an invertebrate pond community", *Ecotoxicology and Environmental Safety*, vol. 41, pp. 137–156, 1998.

[WRO 05] WRONA F.J., PROWSE T.D., REIST J.D., *et al.*, "Freshwater ecosystems", *Arctic Climate Impact Assessment 2005*, Chapter 8, Cambridge University Press, New York, pp. 147–169, 2005. Available at: www.acia.uaf.edu/pages/scientific.html.

[XIE 04] XIE L., KLERKS P., "Fitness cost of resistance to cadmium in the least killifish (*Heterandria formosa*)", *Environmental Toxicology and Chemistry*, vol. 23, pp. 1499–1503, 2004.

[YAU 00] YAUK C.L., FOX G.A., MCCARRY B.E., *et al.*, "Induced minisatellite germline mutations in herring gulls (*Larus argenteus)* living near steel mills", *Mutation Research*, vol. 452, pp. 211–218, 2000.

[ZHO 99] ZHOU T., WEIS J.S., "Predator avoidance in mummichog larvae from a polluted habitat", *Journal of Fish Biology*, vol. 54, pp. 44–57 vol. 147, pp. 373–399, 2000, 1999.