Phase I oxidation to reactive intermediates, which can bind to cellular macromolecules and induce toxic effects if not effectively conjugated during Phase II reactions. This incomplete detoxification can lead to oxidative stress, DNA damage, and carcinogenic outcomes in aquatic organisms.

In addition to organism-level metabolism, microbial biodegradation plays a crucial role in the environmental transformation of xenobiotics. Microorganisms in aquatic systems can metabolize a wide range of pollutants, including hydrocarbons, pesticides, and pharmaceutical residues, thereby contributing to the natural attenuation of contaminants (Wang and Wang, 2016; Varjani et al., 2020; Singh et al., 2021). The interaction between microbial processes and fish metabolism ultimately determines the persistence, bioavailability, and ecological impact of xenobiotic compounds in aquatic ecosystems.

6 Oxidative Stress and Cellular Damage Induced by Xenobiotics

One of the most significant mechanisms through which xenobiotics exert toxic effects in aquatic organisms is the induction of oxidative stress. Xenobiotic compounds such as heavy metals, pesticides, and pharmaceutical residues can stimulate the excessive production of reactive oxygen species (ROS) including superoxide radicals, hydrogen peroxide, and hydroxyl radicals. These reactive molecules can damage cellular macromolecules such as proteins, lipids, and nucleic acids, ultimately leading to impaired physiological functions in aquatic organisms (Livingstone, 2001; Valavanidis et al., 2006). Fish exposed to xenobiotic pollutants frequently exhibit increased lipid peroxidation, DNA damage, and enzyme inhibition due to oxidative stress. Antioxidant defense systems such as superoxide dismutase, catalase, glutathione peroxidase, and glutathione-S-transferase play essential roles in mitigating oxidative damage. However, prolonged exposure to pollutants can overwhelm these defense mechanisms, resulting in cellular dysfunction and tissue damage (Monteiro et al., 2010; Lushchak, 2011). Recent studies have demonstrated that oxidative stress biomarkers in fish can be used as sensitive indicators of environmental contamination. Monitoring antioxidant enzyme activity and oxidative damage products therefore provides valuable information for assessing the ecological impact of xenobiotic pollutants in aquatic ecosystems (Valavanidis et al., 2006).

7 Endocrine Disruption and Reproductive Toxicity in Aquatic Organisms

Certain xenobiotic pollutants function as endocrine-disrupting chemicals (EDCs) that interfere with hormonal signaling pathways in aquatic organisms. These substances can mimic, block, or alter the synthesis, transport, and metabolism of natural hormones, resulting in disturbances in growth, reproduction, and development. Endocrine-disrupting compounds commonly detected in aquatic environments include industrial chemicals, pesticides, plasticizers, and pharmaceutical residues (Sumpter and Johnson, 2005; Diamanti-Kandarakis et al., 2009).

At the molecular level, many EDCs exert their effects by binding to nuclear hormone receptors such as estrogen receptors (ERs), androgen receptors (ARs), and thyroid hormone receptors, thereby altering transcriptional regulation of target genes involved in reproductive and developmental processes. This receptor-mediated interaction can lead to changes in gene expression, protein synthesis, and endocrine feedback mechanisms, ultimately disrupting physiological homeostasis. In fish populations, exposure to endocrine-disrupting xenobiotics has been associated with abnormalities such as reduced fertility, altered sex ratios, intersex conditions, and impaired reproductive behavior. Synthetic estrogens, for example, have been shown to induce feminization of male fish in contaminated aquatic environments (Jobling et al., 2003).

Recent studies have highlighted the growing significance of emerging endocrine disruptors, including pharmaceutical residues and microplastics, in aquatic ecosystems. Pharmaceutical compounds such as synthetic hormones and antidepressants can interact with endocrine signaling pathways even at low concentrations, while microplastics may act as carriers for adsorbed EDCs or directly interfere with endocrine function. These contaminants have been shown to modulate gene expression related to reproductive development and endocrine regulation, leading to sublethal but ecologically significant effects in fish populations (Sharma et al., 2022; Wilkinson et al., 2022).