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Perspective

# Alterations in Microbiota and Immune Barriers of Fish Exposed to Pollutants

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## DESCRIPTION

Emerging pollutants are synthetic or naturally occurring substances that enter aquatic environments through industrial, agricultural, or domestic activities. Unlike traditional pollutants, these compounds include pharmaceuticals, personal care products, microplastics, and various industrial chemicals that persist at low concentrations but can produce biological effects over time. Teleost fish, as a major component of freshwater and marine ecosystems, are exposed to these contaminants, which can affect multiple organs. Among these, mucosal organs skin, gills, and gut play central roles in nutrient absorption, respiration, and defense against pathogens. Recent studies indicate that emerging pollutants can disrupt the structural and functional integrity of these organs, impacting mucosal microbiota, the physical barrier, and immune defense.

The mucosal surfaces of teleost fish represent primary interfaces with the environment. The skin, a multilayered organ covered by mucus, provides a physical barrier against pathogens while participating in osmoregulation. Gills, with their extensive surface area and thin epithelium, facilitate gas exchange but are highly vulnerable to waterborne contaminants. The gut is involved in digestion and nutrient absorption, as well as immune surveillance. Each mucosal organ contains a resident microbiota that contributes to homeostasis, pathogen resistance, and immunomodulation. Alterations in microbial composition due to pollutants can lead to dysbiosis, which compromises overall health.

Emerging pollutants interact with mucosal microbiota in multiple ways. Pharmaceuticals such as antibiotics, antidepressants, and hormone analogs can selectively inhibit or promote the growth of specific microbial taxa. This results in changes in microbial diversity, richness, and functional capacity. Reduced microbial diversity has been associated with impaired digestion, increased susceptibility to opportunistic infections, and altered metabolic profiles. Heavy metals, including cadmium, lead, and mercury, can also modify microbial communities by promoting metal-tolerant strains, potentially increasing the production of reactive species and toxins. Microplastics serve as carriers for chemical pollutants and microbes, providing surfaces for biofilm formation and facilitating the introduction of pathogens into mucosal environments.

The physical barrier of mucosal organs can be compromised by emerging pollutants. In the skin, pollutants may disrupt mucus secretion, reduce epidermal thickness, or damage keratinocytes, diminishing the first line of defense. In gills, contaminants can induce epithelial hyperplasia, lamellar fusion, or clubbing of filaments, impairing respiration and increasing susceptibility to infection. The gut epithelium may experience villi atrophy, mucous cell reduction, and epithelial cell detachment under chronic pollutant exposure. These structural alterations reduce the effectiveness of the physical barrier, facilitating pathogen entry and systemic dissemination.

The immune barrier in mucosal organs involves innate and adaptive components. Mucus contains antimicrobial peptides, enzymes, and immunoglobulins that neutralize pathogens. Mucosal epithelial cells express pattern recognition receptors, cytokines, and chemokines that orchestrate local immune responses. Exposure to emerging pollutants can modulate these immune mechanisms. For instance, endocrine-disrupting chemicals may suppress immunoglobulin production or alter cytokine expression, weakening pathogen defense. Antibiotic residues can impair leukocyte function, including phagocytosis and reactive oxygen species generation. Heavy metals can cause immunotoxicity through oxidative stress, apoptosis, or interference with signaling pathways. Microplastics may induce chronic low-grade inflammation, leading to immune exhaustion or dysregulation.

Interactions among mucosal microbiota, physical barrier, and immune barrier are complex and interdependent. Dysbiosis induced by pollutants can increase pathogen colonization, which further challenges epithelial integrity and immune responses. Conversely, physical damage to the mucosal surface can allow opportunistic microbes to penetrate deeper tissues, triggering inflammation and systemic effects. Immune suppression can prevent effective microbial control, exacerbating dysbiosis and

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tissue damage. These interconnected effects highlight the importance of considering mucosal organs as integrated systems when assessing pollutant toxicity.

Management of emerging pollutants in aquatic systems is challenging. Conventional water treatment may remove only a fraction of pharmaceuticals or microplastics, leaving residual concentrations capable of affecting aquatic organisms. Strategies for mitigating exposure include improving wastewater treatment, regulating chemical discharge, and monitoring environmental concentrations. At the organismal level, understanding the responses of mucosal organs can inform selection of resilient species or populations, feeding strategies that support mucosal health, and prophylactic measures against infections. Such measures aim to reduce adverse effects while maintaining aquaculture productivity and ecosystem stability.

The ecological consequences of impaired mucosal organs extend beyond individual fish health. Reduced mucosal immunity increases susceptibility to pathogens, which can spread through populations and affect community dynamics. Changes in microbial communities may alter nutrient cycling, biogeochemical processes, and energy flow in aquatic systems. Pollutant-induced alterations in fish health can have cascading effects on fisheries, aquaculture sustainability, and food security. Therefore, assessing the impact of emerging pollutants on mucosal organs is relevant for both environmental management and economic considerations.

In conclusion, emerging pollutants represent a significant challenge for aquatic ecosystems, with particular effects on the mucosal organs of teleost fish. These organs integrate microbial, physical, and immune functions that collectively maintain homeostasis and protect against pathogens. Exposure to pharmaceuticals, heavy metals, microplastics, and industrial chemicals can disrupt mucosal microbiota, compromise physical barriers, and modulate immune responses. The resulting dysbiosis, epithelial damage, and immunotoxicity have implications for individual fish health, population resilience, and ecosystem function. Comprehensive evaluation of mucosal responses, combined with environmental monitoring and management, is necessary to understand and mitigate the impact of emerging pollutants.