Available online at
http://currentagriculturetrends.vitalbiotech.org/

ISSN (E): 2583 – 1933

Curr. Agri.Tren.:e- Newsletter, (2026) 5(2), 23-27

Current Agriculture Trends: e-Newsletter

Article ID: 561

Impact of Gut Mycobiome Alteration on Disease Susceptibility in Cultured Fish: The Forgotten Kingdom of Aquaculture Health

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Open Access

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Article History
Received: 3. 2.2026
Revised: 7. 2.2026
Accepted: 12. 2.2026

INTRODUCTION

For over two decades, the global aquaculture industry has operated under a "bacterio-centric" worldview. As we evolved from small-scale farming to the primary source of the world's seafood, our understanding of fish health focused almost exclusively on pathogenic bacteria (*Vibrio*, *Aeromonas*, *Streptococcus*) and beneficial probiotics (*Lactobacillus*, *Bacillus*). When fish pathologists spoke of the "microbiome," they effectively meant the "bacteriome." We sequenced 16S rRNA genes exhaustively, treating the gut as if it were a bacteria-only club (Li *et al*., 2015). This was a critical oversight. Recent advances in Next-Generation Sequencing (NGS), specifically targeting the Internal Transcribed Spacer (ITS) regions, have revealed that the fish gut is a multi-kingdom ecosystem where fungi the mycobiome engage in a complex biological dialogue with bacteria and the host immune system (Siriyappagouder et al., 2018). While fungi are less abundant than bacteria in the gut, their cell volume is roughly 100-fold greater, and their metabolic and immunologic impact is profound. They are not merely transients; they are key tenants. This article aims to correct the historical bacterial bias by exploring how the alteration of this fungal community directly impacts the susceptibility of cultured fish to disease. We will demonstrate that a healthy mycobiome is a gatekeeper of immunity, while a dysbiotic mycobiome is a silent precursor to the bacterial outbreaks that plague the industry (Lv et al., 2025).

2. The Fungal Frontier: What Lives in a Fish Gut?

To understand how things go wrong (dysbiosis), we must first understand what "right" (eubiosis) looks like. Unlike the human gut, which is heavily colonized by *Candida* and *Saccharomyces*, the fish gut mycobiome is heavily influenced by the aquatic environment and diet, making it far more transient and dynamic.

2.1 The Core Mycobiome

Research indicates that the teleost gut is dominated by two primary phyla: Ascomycota and Basidiomycota. In a landmark study on zebrafish (*Danio rerio*), Siriyappagouder *et al*. (2018) identified fungal taxa belonging to more than 15 classes.

- **Ascomycota:**

Often the most abundant phylum, comprising genera such as *Saccharomyces*, *Debaryomyces*, and *Penicillium*. These are crucial for enzymatic breakdown of complex carbohydrates.

- **Basidiomycota:**

Includes yeasts like *Rhodotorula* and *Cryptococcus*. *Rhodotorula* species, specifically, are emerging as significant carotenoid producing commensals in healthy salmonids.

2.2 Wild vs. Captive Disparity

A critical finding in mycobiome research is the stark difference between wild and cultured fish. Wild fish tend to harbour a diverse mycobiome often dominated by *Dothideomycetes*, reflecting a natural, varied diet. In contrast, laboratory and farmed fish often show a mycobiome dominated by *Saccharomycetes* and other saprotrophic guilds (Siriyappagouder et al., 2018). This loss of diversity in captivity an unintended consequence of standardized feeds and controlled environments may be the first step toward increased disease susceptibility, a concept known as the "hatchery effect."

3. Drivers of Dysbiosis

Dysbiosis is the breakdown of the microbial community structure. In the context of the mycobiome, this often manifests as a reduction in diversity or the blooming of opportunistic "pathobionts" (commensals that turn nasty under stress). Three primary drivers are responsible for fungal dysbiosis in modern aquaculture.

3.1 The Shift to Plant-Based Feeds (The Mycotoxin Vector)

This is the single biggest driver. To make aquaculture sustainable, the industry has aggressively replaced finite fish meal (FM) with plant proteins (PP) like soybean meal, corn gluten, and wheat. While economically necessary, this introduces distinct challenges:

- **Substrate Change:** Plant ingredients introduce fibres (cellulose, lignin, non-starch polysaccharides) that select for different fungal decomposers compared to marine proteins.
- **Mycotoxins:** Perhaps the most insidious threat. Feeds contaminated with mycotoxins (e.g., Aflatoxin B1, Ochratoxin A) do not just damage the liver; they radically alter the gut environment. Recent work by Mamdouh and Zahran (2024) highlighted that Aflatoxin B1 exposure increases the abundance of pathogenic bacterial genera like *Aeromonas* and *Pseudomonas* while suppressing beneficial microbes. The mycotoxin acts as a chemical selection pressure, favouring robust, often pathogenic, microbes over delicate commensals.

3.2 Antibiotic Usage: The "Empty Niche"

We treat bacterial infections with antibiotics, often ignoring the "collateral damage" to the fungal kingdom. Antibiotics create an "empty niche" phenomenon. By decimating the bacterial population, we remove the competition that keeps fungal populations in check. This can lead to:

- **Fungal Blooms:** Rapid overgrowth of yeasts (e.g., *Candida* spp.), which can physically damage the gut epithelium and trigger excessive inflammation.
- **Secondary Invasion:** The destabilized community becomes vulnerable to colonization by secondary pathogens once the antibiotic treatment ceases.

3.3 Environmental Stressors

Fish are poikilotherms; their metabolism and their microbiome match their water temperature. Thermal stress has been shown to reduce the alpha-diversity of the gut microbiota. According to the Diversity

Resistance Hypothesis proposed by Li et al. (2013), higher diversity creates a barrier against invasion. When stress lowers this diversity, the "colonization resistance" of the gut collapses, leaving the door open for pathogens like *Vibrio* or *Flavobacterium*.

4. Mechanisms: How Fungal Shifts Lead to Disease

4.1 The Immune Connection: Dectin-1 and β-Glucans

Fungal cell walls contain β-1,3-glucans and mannans. These are potent Pathogen-Associated Molecular Patterns (PAMPs). The fish innate immune system recognizes these via Pattern Recognition Receptors (PRRs), specifically C-type lectin receptors like Dectin-1 and Toll-like receptors (TLRs).

- **The "Goldilocks" Zone:** In a healthy mycobiome, low-level stimulation by commensal yeasts keeps the fish's immune system "primed" (Dawood et al., 2019). The macrophages are alert but not aggressive.
- **The Dysbiotic State:** If the mycobiome shifts towards overgrowth (fungal bloom), the massive load of antigens can trigger chronic inflammation (high levels of TNF-α and IL-1β). This diverts energy away from growth and specific disease resistance, causing "immune exhaustion." Conversely, if the fungal community is decimated, the immune system loses that baseline priming, rendering the fish sluggish in responding to bacterial invasion.

4.2 The Bacterial-Fungal Axis

Bacteria and fungi in the gut are not ignoring each other; they are in a constant state of warfare and trade.

- Synergy: Some bacteria assist fungi in breaking down complex carbohydrates.
- Antagonism: Commensal bacteria secrete short-chain fatty acids (SCFAs) like butyrate that inhibit fungal hyphae formation. When we disrupt the mycobiome, we disrupt this axis. He et al. (2017) demonstrated that dysbiosis is frequently associated with disease because the breakdown of this cross-kingdom network destroys the mucosal barrier.

5. Case Studies in Aquaculture

5.1 The Probiotic Success: *Saccharomyces cerevisiae*

The most compelling evidence for the mycobiome's impact comes from probiotic studies. Research by Dawood et al. (2019) and others has consistently shown that supplementing feed with *S. cerevisiae* (baker's yeast) enhances disease resistance.

- Observation: Fish fed yeast-supplemented diets show significantly higher survival rates when challenged with *Aeromonas hydrophila*.
- Mechanism: The yeast enhances the height of intestinal villi (increasing absorption) and upregulates the expression of immune genes (Lysozyme, complement system) (Dawood et al. 2019). This proves that *adding* a specific fungus can reverse disease susceptibility.

5.2 The Mycotoxin Failure: Aflatoxin-Induced Susceptibility

Conversely, when fungal metabolites (mycotoxins) disrupt the gut, susceptibility skyrockets. In studies involving Nile Tilapia, exposure to contaminated feed resulted in a "Leaky Gut" syndrome. The tight junctions between intestinal cells loosened, allowing bacteria to translocate from the gut into the bloodstream (septicaemia). This was not caused by the bacteria primarily, but by the fungal toxins disrupting the gut integrity first (Mamdouh and Zahran, 2024).

5.3 The Fungal Pathogen: *Saprolegnia*

While often considered a water mold (Oomycete), *Saprolegnia* infections are heavily influenced by the host's overall health and slime coat integrity. Dysbiosis in the gut can lead to systemic stress that compromises the skin mucosal barrier, making the fish more susceptible to external fungal infections like

Saprolegniasis. This highlights the "Gut-Skin Axis" a dysbiotic gut leads to a vulnerable skin surface.

6. Therapeutic Implications

If we accept that gut fungi control disease susceptibility, our management strategies must change. We must move beyond simple antibiotic treatments.

1. **Mycobiotics:** Beyond standard bacterial probiotics, we need "Mycobiotics" consortia of beneficial fungi tailored to specific fish species. Strains of *Debaryomyces hansenii* are showing promise as marine probiotics due to their polyamine production, which aids gut healing (Angulo *et al*., 2020).
2. **Next-Gen Prebiotics:** We need feed ingredients specifically designed to nourish beneficial fungi (mycobiome-targeted fibers). Mannan-oligosaccharides (MOS) are effectively "fungal food" that blocks pathogen adhesion (Torrecillas et al., 2014).
3. **Fungystats in Feed:** We must be more rigorous about mycotoxin binders (clay minerals, yeast cell walls) in plant-based feeds to prevent chemical dysbiosis (Khatoon et al., 2025).
4. **Integrative Monitoring:** Routine health checks should not just count bacteria. They should use ITS sequencing to monitor the fungal diversity index. A drop in fungal diversity should be treated as an early warning system for an impending disease outbreak (Lv et al., 2025).

CONCLUSION

As we look toward the next 25 years of aquaculture, we must abandon the simplified "bacteria vs. host" view of disease. The fish gut is a theatre of war and cooperation between kingdoms. The mycobiome, though often hidden in the data, acts as a critical gatekeeper of health. Dysbiosis in the fungal community is a silent precursor to many of the bacterial outbreaks we fight daily. By understanding the composition of the healthy mycobiome and the drivers that alter it, we can move from reactive treatments (antibiotics) to proactive resilience (ecological engineering). The fungal kingdom is no longer forgotten; it is the new frontier of disease control.

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