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Bone Destruction by Talaromyces Marneffei: Proteomic Insights.

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Introduction

Talaromyces marneffei (TM), formerly known as *Penicillium marneffei*, is a thermally dimorphic fungus endemic to Southeast Asia and southern China. It is a significant opportunistic pathogen, particularly in immunocompromised individuals, such as those living with HIV/AIDS. While TM typically causes systemic infections involving the lungs, skin, and reticuloendothelial system, recent studies have uncovered its potential to cause **bone destruction**, a rare but serious manifestation. Proteomic analysis has emerged as a powerful tool to unravel the molecular mechanisms behind TM-induced bone pathology, offering new diagnostic and therapeutic avenues [1, 2].

TM exhibits thermal dimorphism, growing as a mold at 25°C and converting to a yeast-like form at 37°C—the temperature of the human body. This switch is crucial for its pathogenicity. Once inside the host, TM can disseminate through the bloodstream and invade various tissues. Bone involvement is uncommon but increasingly recognized, especially in patients with advanced immunosuppression. The mechanisms by which TM damages bone tissue remained poorly understood until recent proteomic studies began to shed light on the molecular interactions at play [3, 4].

A landmark study published in *Medical Mycology* used Data-Independent Acquisition (DIA) proteomics to analyze bone tissue samples from patients infected with TM. The researchers compared samples from infected individuals with non-infectious controls, identifying 509 differentially expressed proteins (DEPs) out of 5,930 quantifiable proteins. Histopathological analysis revealed extensive bone destruction, inflammatory infiltration, and altered hematopoietic cell populations in infected specimens [5, 6].

Among the DEPs, COMMD1 was significantly downregulated, while IL-17 was upregulated. COMMD1 is known to regulate NF-κB signaling and maintain immune homeostasis, while IL-17 is a pro-inflammatory cytokine implicated in bone resorption and autoimmune diseases. These findings suggest that TM-induced bone destruction may be driven by dysregulated inflammatory pathways, particularly those involving IL-17-mediated osteoclast activation [7, 8].

The identification of COMMD1 and IL-17 as key players in TM-related bone damage has important clinical implications. First, these proteins could serve as biomarkers for early diagnosis of bone involvement in talaromycosis. Second, targeting IL-17 signaling may offer a novel therapeutic strategy to mitigate bone loss. Given the challenges in diagnosing fungal osteomyelitis—often requiring invasive biopsies and advanced imaging—proteomic biomarkers could revolutionize detection and monitoring. A case report published in *BMC Infectious Diseases* described a patient with HIV co-infected with TM and *Salmonella*, presenting with osteoarticular destruction. Metagenomics sequencing confirmed TM infection, and treatment with amphotericin B halted bone degradation. This case underscores the importance of early recognition and aggressive

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antifungal therapy in preventing irreversible skeletal damage [9, 10].

Conclusion

Proteomic and transcriptomic studies have revealed several virulence factors that may contribute to TM's ability to invade and damage bone tissue. A comprehensive review in *Toxins* identified Mp1p, a surface protein that binds host lipids and facilitates immune evasion. Other virulence factors include polyketide synthases involved in pigment production and oxidative stress resistance, as well as microRNA-like RNAs that regulate fungal gene expression during infection. Comparative proteomics between TM's yeast and hyphal phases showed differential expression of adhesion molecules and metabolic enzymes, suggesting that phase-specific proteins may mediate tissue tropism and immune modulation. These insights highlight the complexity of TM's pathogenic arsenal and the need for multi-omics approaches to fully understand its behavior in host tissues.TM is endemic in tropical regions, particularly in Southeast Asia, where environmental exposure to fungal spores is common. The fungus is found in soil and decaying organic matter, and infection often occurs through inhalation. With increasing travel and migration, cases are now being reported outside endemic areas, making global awareness and surveillance essential.

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