

Fungal Immunity

Avani R. Modi

M.V.Sc. Scholar, Department of Veterinary Microbiology
College of Veterinary Science & Animal Husbandry, Anand
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Introduction

Several species in the kingdom of fungi are linked to a variety of human and animal diseases. There are three main categories of fungus infections. The first is primary fungus infections that affect the skin or other surfaces and result in diseases like ringworm or thrush. These fungi include the *Microsporum* or *Candida* species. The second class comprises primary infections by dimorphic fungi, such as *Histoplasma capsulatum*, *Blastomyces dermatitidis*, and *Coccidioides immitis*, which primarily cause respiratory infections. The third class includes secondary infections caused by opportunistic fungi in immunocompromised animals, including *Pneumocystis* and the *Mucorales* (*Rhizopus*, *Mucor*, and *Absidia*). Globally, the great majority of mycotic disorders are infectious diseases brought on by different pathogenic skin fungi.

Immunity To Fungi

The body defends itself against primary infections through both innate and adaptive immunological responses. A specific disease that will arise after contact with the common pathogen is Figure 1 | Balancing protection and immunopathology in fungal infections largely determined by the host immune system. It has been made clear by the T HELPER 1 (TH1)/TH2 dichotomy that various effector functions are necessary for the eradication of various fungal diseases. The barrier function of the skin and the mucosal epithelial interfaces of the respiratory, gastrointestinal, and genito-urinary tracts are among the inherent mechanisms of innate defense that are present at locations of ongoing interaction with fungi. The majority of host defense mechanisms, however, are inducible after infection; as a result, their activation necessitates the detection of a set of pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs), by invariant molecular



structures shared by broad groups of pathogens.

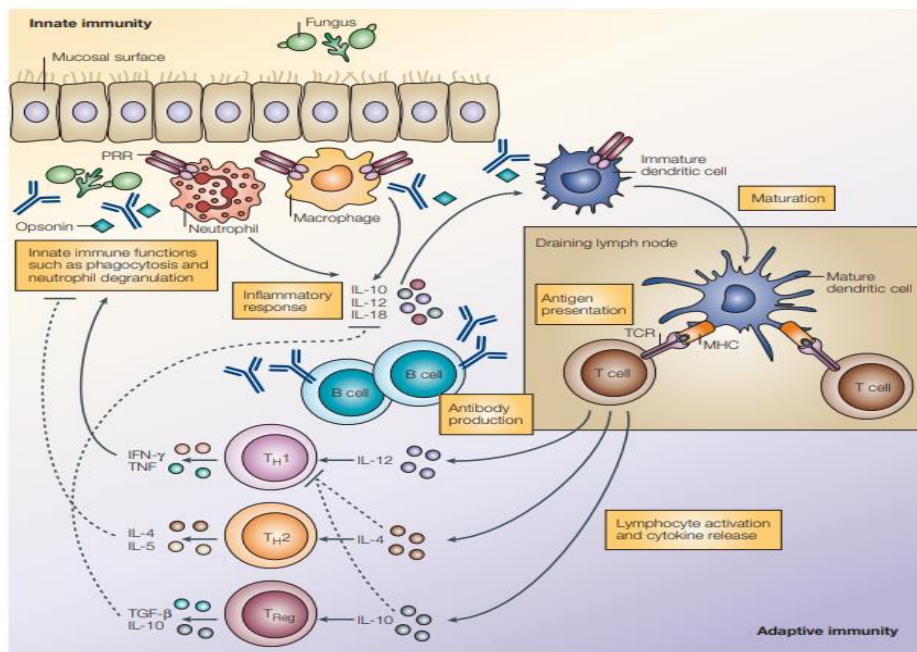


Figure 1 | Balancing protection and immunopathology in fungal infections

Neutrophils are also triggered by the IL-23/IL-17 axis during fungal infections. IL-23 production is triggered by fungus PAMPs via TLR2 or the cell surface lectin dectin-1. Th17 cells are activated by IL-23. These cells subsequently release IL-17, which stimulates neutrophils and endothelial cells and causes an initial inflammatory response.

Antibodies In Immunity to Fungi

Toxin neutralization, opsonization, adhesion prevention, and antibody-dependent cellular cytotoxicity are among the primary roles that antibodies are known to play in fungal infections. Antibodies against fungi can be both protective and non-protective, with a wide range in their proportions and relative composition. Antibodies specific for heat-shock protein 90 are associated with recovery from infections with *Candida albicans* and protection against disseminated disease in patients with AIDS, and they synergize with antifungal chemotherapy. Mice are protected against candidiasis by antibodies specific for a mannan adhesin fraction. Idiotype-specific antibodies, or even just one of their chains, are highly effective in treating experimental infections due to their wide fungicidal action.

