

The Interaction of Human Pathogenic Fungi With C-Type Lectin Receptors.

Goyal S, Castrillón-Betancur JC, Klaile E, Slevogt H (2018) The Interaction of Human Pathogenic Fungi With C-Type Lectin Receptors. *Front Immunol* 9, 1261. [PubMed](#)

ILRS Authors

[Juan Camilo Castrillón Betancur](#)

Projects

The impact of the interaction of the receptors of the Dectin-1 gene cluster in fungal and bacterial infections

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Abstract

Fungi, usually present as commensals, are a major cause of opportunistic infections in immunocompromised patients. Such infections, if not diagnosed or treated properly, can prove fatal. However, in most cases healthy individuals are able to avert the fungal attacks by mounting proper antifungal immune responses. Among the pattern recognition receptors (PRRs), C-type lectin receptors (CLRs) are the major players in antifungal immunity. CLRs can recognize carbohydrate ligands, such as β -glucans and mannans, which are mainly found on fungal cell surfaces. They induce proinflammatory immune reactions, including phagocytosis, oxidative burst, cytokine, and chemokine production from innate effector cells, as well as activation of adaptive immunity via Th17 responses. CLRs such as Dectin-1, Dectin-2, Mincle, mannose receptor (MR), and DC-SIGN can recognize many disease-causing fungi and also collaborate with each other as well as other PRRs in mounting a fungi-specific immune response. Mutations in these receptors affect the host response and have been linked to a higher risk in contracting fungal infections. This review focuses on how CLRs on various immune cells orchestrate the antifungal response and on the contribution of single nucleotide polymorphisms in these receptors toward the risk of developing such infections.

Identifier

doi: 10.3389/fimmu.2018.01261 PMID: 29915598