

Complement plays a central role in *Candida albicans*-induced cytokine production by human PBMCs.

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Abstract

In experimental studies, the role of complement in antifungal host defense has been attributed to its opsonizing capability. In this study, we report that in humans an activated complement system mainly augments *Candida albicans*-induced host proinflammatory cytokine production via C5a-C5aR signaling, while phagocytosis and intracellular killing of *Candida* are not influenced. By blocking the C5a-C5aR signaling pathway, either with anti-C5a antagonist antibodies or with the C5aR antagonist W-54001, *C. albicans*-induced IL-6 and IL-1 β levels were significantly reduced. Recombinant C5a augmented cytokine production. In addition, using serum from patients with various complement deficiencies, we demonstrated a crucial role of C5, but not C6 or the membrane attack complex, in *C. albicans*-induced IL-6 and IL-1 β production in monocytes. These findings reveal a central role of anaphylatoxin C5a in augmenting host proinflammatory cytokine production upon contact with *C. albicans*, and define the role of the complement system in anti-*Candida* host defense in humans.

Beteiligte Forschungseinheiten

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