Human anti-fungal Th17 immunity and pathology rely on cross-reactivity against *Candida albicans*.

Bacher P, Hohnstein T, Beerbaum E, Röcker M, Blango MG, Kaufmann S, Röhmel J, Eschenhagen P, Grehn C, Seidel K, Rickerts V, Lozza L, Stervbo U, Nienen M, Babel N, Milleck J, Assenmacher M, Cornely OA, Ziegler M, Wisplinghoff H, Heine G, Worm M, Siegmund B, Maul J, Creutz P, Tabeling C, Ruwwe-Glösenkamp C, Sander LE, Knosalla C, Brunke S, Hube B, Kniemeyer O, Brakhage AA, Schwarz C, Scheffold A (2019) Human anti-fungal Th17 immunity and pathology rely on cross-reactivity against *Candida albicans*. *Cell* 176(6), 1340-1355.

Details

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Abstract

Th17 cells provide protection at barrier tissues but may also contribute to immune pathology. The relevance and induction mechanisms of pathologic Th17 responses in humans are poorly understood. Here, we identify the mucocutaneous pathobiont Candida albicans as the major direct inducer of human anti-fungal Th17 cells. Th17 cells directed against other fungi are induced by cross-reactivity to C. albicans. Intestinal inflammation expands total C. albicans and cross-reactive Th17 cells. Strikingly, Th17 cells cross-reactive to the airborne fungus Aspergillus fumigatus are selectively activated and expanded in patients with airway inflammation, especially during acute

allergic bronchopulmonary aspergillosis. This indicates a direct link between protective intestinal Th17 responses against C. albicans and lung inflammation caused by airborne fungi. We identify heterologous immunity to a single, ubiquitous member of the microbiota as a central mechanism for systemic induction of human anti-fungal Th17 responses and as a potential risk factor for pulmonary inflammatory diseases.

Beteiligte Forschungseinheiten

Mikrobielle Pathogenitätsmechanismen Bernhard Hube Mehr erfahren

Molekulare und Angewandte Mikrobiologie Axel Brakhage Mehr erfahren

Leibniz-HKI-Autor*innen



Matthew Blango

Details



Axel A. Brakhage

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Sascha Brunke

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Marie Goldmann

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Bernhard Hube

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Olaf Kniemeyer

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Themenfelder

Interaktion mit Immunzellen (MPM)

Pathomechanismen an der Epithelbarriere

Evolution & Anpassung bei Pathogenen

Awards

Recommended by Faculty 1000 as exceptional

Identifier

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