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Flipping the switch: how fungal chitin influences the immune response

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Chitin is an essential structural polysaccharide of the fungal cell wall and we recently discovered fungal chitin to trigger an anti-inflammatory immune response in myeloid cells. Activity of human chitinases was essential to release chitin particles with anti-inflammatory properties from \textit{C. albicans}. In addition, fungal chitin induced eosinophilia in vivo, a typical sign for a Th2 driven allergic reaction.

Interestingly, high level expression of the acidic mammalian chitinas I (AMCase I) is associated with allergies and asthma. AMCase I expression, together with the ratio between Arginase I and nitric oxide synthase (iNOS) expression, serves as marker for allergy-associated alternative activated macrophages. Myeloid Arginase I has been shown to down-regulate excessive Th1-induced inflammation and further, enhances wound healing. The production of nitric oxide (NO) on the other hand, is essential for host defence and immune homeostasis, and several pathogens, including fungi, can target these balance in their favour

Here we report for the first time, that fungal chitin increases Arginase I protein levels in human myeloid cells, which can be blocked by the chitinase inhibitor Bisdionin C. We further show that fungal chitin shifts classical activated macrophage towards a more alternative activated phenotype. Treatment of \textit{C. albicans} with the β-1,3-glucan synthesis inhibitor caspofungin increases fungal cell wall chitin and moreover, leads to chitin surface presentation. Caspofungin-treated \textit{C. albicans} cells fail to induce a pro-inflammatory immune response and moreover, are shifting classical macrophages towards an alternative phenotype, as observed for fungal chitin alone.