Phagocytosis of *Aspergillus fumigatus* conidia by murine macrophages involves recognition by the dectin-1 beta-glucan receptor and Toll-like receptor 2.

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Abstract

Aspergillus fumigatus is a fungal pathogen causing severe infections in immunocompromised patients. For clearance of inhaled conidia, an efficient response of the innate immune system is required. Macrophages represent the first line of defence and ingest and kill conidia. C-type lectins represent a family of receptors, which recognize pathogen-specific carbohydrates. One of them is beta1-3 glucan, a major component of the fungal cell wall. Here we provide evidence that beta1-3 glucan plays an important role for the elimination of A. fumigatus conidia. Laminarin, a soluble beta1-3 glucan and antibodies to dectin-1, a well known beta1-3 glucan receptor, significantly inhibited conidial phagocytosis. On resting conidia low amounts of surface accessible beta1-3 glucan were detected, whereas high amounts were found on small spores that appear early during germination and infection as well as on resting conidia of a pksP mutant strain. Swollen conidia also display larger quantities of beta1-3 glucan, although in an irregular spotted pattern. Resting pksP mutant conidia and swollen wild-type conidia are phagocytosed with high efficiency thereby

confirming the relevance of beta1-3 glucans for conidial phagocytosis. Additionally we found that TLR2 and the adaptor protein MyD88 are required for efficient conidial phagocytosis, suggesting a link between the TLR2-mediated recognition of A. fumigatus and the phagocytic response.

Beteiligte Forschungseinheiten

Molekulare und Angewandte Mikrobiologie Axel Brakhage Mehr erfahren

Leibniz-HKI-Autor*innen

Axel A. Brakhage

Details

Identifier

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