Candida albicans induces metabolic reprogramming in human NK cells and responds to perforin with a Zinc depletion response.

Hellwig D, Voigt J, Bouzani M, Löffler J, Albrecht-Eckardt D, Weber M, Brunke S, Martin R, Kurzai O, Hünniger K (2016) *Candida albicans* induces metabolic reprogramming in human NK cells and responds to perforin with a Zinc depletion response. *Front Microbiol* 7, 750.

Details



Abstract

As part of the innate immune system, natural killer (NK) cells are directly involved in the response to fungal infections. Perforin has been identified as the major effector molecule acting against many fungal pathogens. While several studies have shown that perforin mediated fungicidal effects can contribute to fungal clearance, neither the activation of NK cells by fungal pathogens nor the effects of perforin on fungal cells are well-understood. In a dual approach, we have studied the global gene expression pattern of primary and cytokine activated NK cells after co-incubation with Candida albicans and the transcriptomic adaptation of C. albicans to perforin exposure. NK cells responded to the fungal pathogen with an up-regulation of genes involved in immune signaling and release of cytokines. Furthermore, we observed a pronounced increase of genes involved in glycolysis and glycolysis inhibitor 2-deoxy-D-glucose impaired C. albicans induced NK

cell activation. This strongly indicates that metabolic adaptation is a major part of the NK cell response to C. albicans infections. In the fungal pathogen, perforin induced a strong up-regulation of several fungal genes involved in the zinc depletion response, such as PRA1 and ZRT1. These data suggest that fungal zinc homeostasis is linked to the reaction to perforin secreted by NK cells. However, deletion mutants in PRA1 and ZRT1 did not show altered susceptibility to perforin.

Involved units

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Topics

Interactions with immune cells (MPM)

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