Induction of Caspase-11 by aspartyl proteinases of *Candida albicans* and implication in promoting inflammatory response.

Gabrielli E, Pericolini E, Luciano E, Sabbatini S, Roselletti E, Perito S, Kasper L, Hube B, Vecchiarelli A (2015) Induction of Caspase-11 by aspartyl proteinases of *Candida albicans* and implication in promoting inflammatory response. *Infect Immun* 83(5), 1940-1948.

Abstract

We recently demonstrated that the secreted aspartyl proteinases (Saps), Sap2 and Sap6, of Candida albicans have the potential to induce the canonical activation of the NLRP3 inflammasome, leading to the secretion of interleukin-1β (IL-1β) and IL-18 via caspase-1 activation. We also observed that the activation of caspase-1 is partially independent from the NLRP3 activation pathway. In this study, we examined whether Sap2 and Sap6 are also able to activate the noncanonical inflammasome pathway in murine macrophages. Our data show that both Sap2 and Sap6 can activate caspase-11 through type I interferon (IFN) production. Caspase-11 cooperates to activate caspase-1, with a subsequent increase of IL-1β secretion. Endocytosis and internalization of Saps are required for the induction of type I IFN production, which is essential for induction of noncanonical inflammasome activation. Our study indicates a sophisticated interplay between caspase-1 and caspase-11 that connects the canonical and noncanonical pathways of inflammasome activation in response to C. albicans Saps.

Involved units

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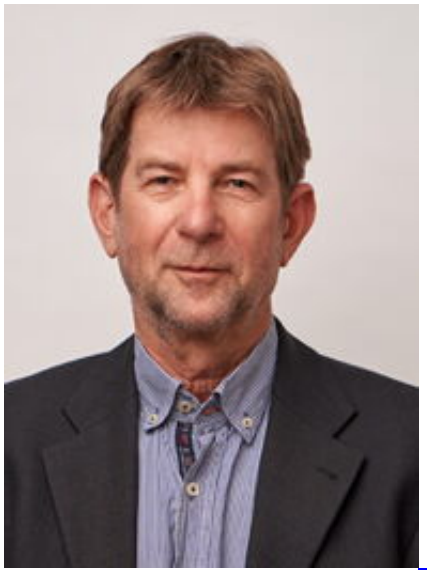

Bernhard Hube

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Lydia Kasper

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Topics

Interactions with immune cells (MPM)

Identifier

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