Functional surface proteomic profiling reveals the host heat-shock protein A8 as a mediator of *Lichtheimia corymbifera* recognition by murine alveolar macrophages.

Hassan MIA, Kruse JM, Krüger T, Dahse HM, Cseresnyés Z, Blango MG, Slevogt H, Hörhold F, Ast V, König R, Figge MT, Kniemeyer O, Brakhage AA, Voigt K (2020) Functional surface proteomic profiling reveals the host heat-shock protein A8 as a mediator of *Lichtheimia corymbifera* recognition by murine alveolar macrophages. *Environ Microbiol* 22(9), 3722-3740.

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Abstract

Mucormycosis is an emergent, fatal fungal infection of humans and warm-blooded animals caused by species of the order Mucorales. Immune cells of the innate immune system serve as the first line of defense against inhaled spores. Alveolar macrophages were challenged with the mucoralean fungus *Lichtheimia corymbifera* and subjected to biotinylation and streptavidin enrichment procedures followed by LC-MS/MS analyses. A total of 28 host proteins enriched for binding to macrophage-*L. corymbifera* interaction. Among those the HSP70-family protein Hspa8 was found to be predominantly responsive to living and heat-killed spores of a virulent and an attenuated strain of *L. corymbifera*. Confocal scanning laser microscopy of infected macrophages

revealed colocalisation of Hspa8 with phagocytosed spores of *L. corymbifera*. The amount of detectable Hspa8 was dependent on the multiplicity of infection. Incubation of alveolar macrophages with an anti-Hspa8 antibody prior to infection reduced their capability to phagocytose spores of *L. corymbifera*. In contrast, anti-Hspa8 antibodies did not abrogate the phagocytosis of *Aspergillus fumigatus* conidia by macrophages. These results suggest an important contribution of the heat-shock family protein Hspa8 in the recognition of spores of the mucoralean fungus *L. corymbifera* by host alveolar macrophages and define a potential immunomodulatory therapeutic target.

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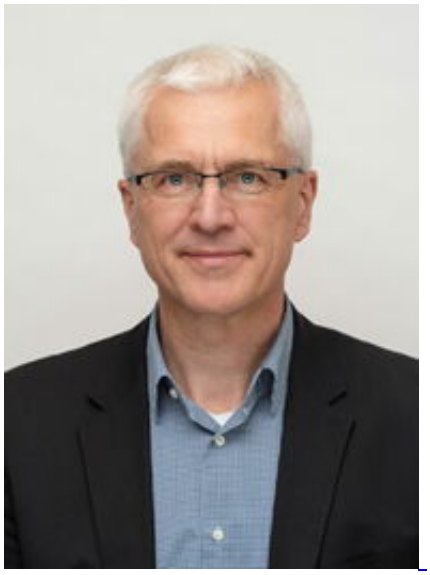

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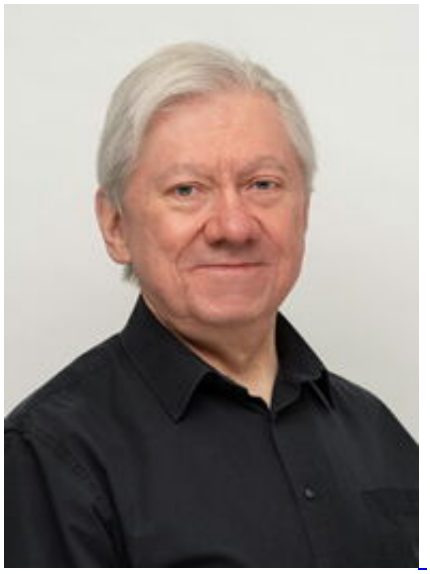

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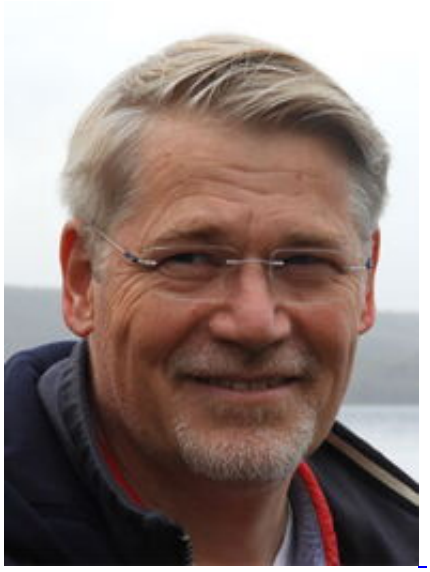

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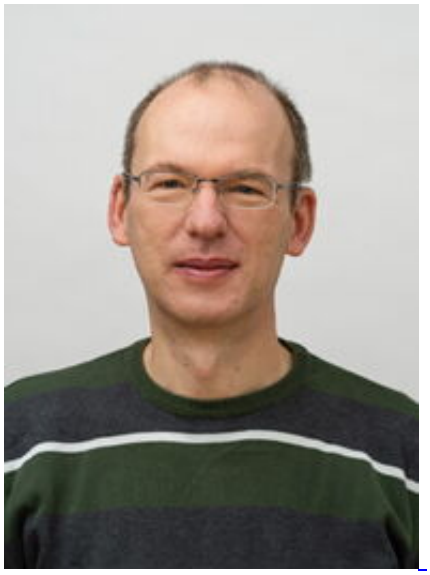

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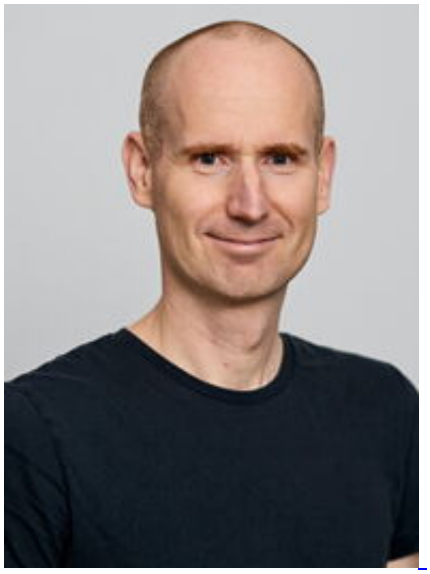

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