

Unaltered fungal burden and ethality in human CEACAM1-transgenic mice during *Candida albicans* dissemination and systemic infection.

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Abstract

Carcinoembryonic antigen-related cell adhesion molecule 1 (CEACAM1, CD66a) is a receptor for *Candida albicans*. It is crucial for the immune response of intestinal epithelial cells to this opportunistic pathogen. Moreover, CEACAM1 is of importance for the mucosal colonization by different bacterial pathogens. We therefore studied the influence of the human CEACAM1 receptor in human CEACAM1-transgenic mice on the *C. albicans* colonization and infection utilizing a colonization/dissemination and a systemic infection mouse model. Our results showed no alterations in the host response between the transgenic mice and the wild-type littermates to the *C. albicans* infections. Both mouse strains showed comparable *C. albicans* colonization and

mycobiota, similar fungal burdens in various organs, and a similar survival in the systemic infection model. Interestingly, some of the mice treated with anti-bacterial antibiotics (to prepare them for *C. albicans* colonization via oral infection) also showed a strong reduction in endogenous fungi instead of the normally observed increase in fungal numbers. This was independent of the expression of human CEACAM1. In the systemic infection model, the human CEACAM1 expression was differentially regulated in the kidneys and livers of *Candida*-infected transgenic mice. Notably, in the kidneys, a total loss of the largest human CEACAM1 isoform was observed. However, the overwhelming immune response induced in the systemic infection model likely covered any CEACAM1-specific effects in the transgenic animals. In vitro studies using bone marrow-derived neutrophils from both mouse strains also revealed no differences in their reaction to *C. albicans*. In conclusion, in contrast to bacterial pathogens interacting with CEACAM1 on different mucosal surfaces, the human CEACAM1-transgenic mice did not reveal a role of human CEACAM1 in the in vivo candidiasis models used here. Further studies and different approaches will be needed to reveal a putative role of CEACAM1 in the host response to *C. albicans*.

Beteiligte Forschungseinheiten

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