Characterisation of the immune response induced by *Candida parapsilosis*:

The role of cell wall *N*-mannan and Dectin-1 independent recognition

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Introduction

Fig.1. Model of N-linked glucans in wild type (A) and *C. parapsilosis* *och1Δ/och1Δ* null mutant (B). Pérez-García et al. Front. Microbiol. 2016

The Dectin-1 receptor displayed dispensable role in the recognition of wild-type *C. parapsilosis*

Fig. 3.B Flow cytometry analysis of immune cell subsets of kidney

Fig. 3.C Fungal burden of wt and Dectin-1^{-/-} mice after wt *C.par* GA1 and CDC 317 infection

The *Cpoch1Δ/Δ* strain is attenuated in virulence in the wild-type mouse model

Fig. 2.A The fungal burden of *CpWT* and *Cpoch1Δ/Δ* strain in wild-type mice

Fig.2.C Flow cytometry analysis of immune cell subsets of kidney

The *Cpoch1Δ/Δ* phenotype is independent from the Dectin-1 receptor

Fig. 4. A The fungal burden of *Cpoch1Δ/Δ* in C57BL/6 and Dectin-1^{-/-} mice.

Fig.4.B Flow cytometry analysis of immune cell subsets of kidney

Conclusions

The *Cpoch1Δ/Δ* null mutant showed decreased virulence in the wild-type mouse model of systemic infection, indicating that the *N*-mannan content in the cell wall regulates the activation of the innate immune cells and the cytokine response against *C. parapsilosis*.

Interestingly, our observations suggest the Dectin-1 independency in the recognition of wild-type *C. parapsilosis* in *in vivo* experiments.

Similarly, the attenuated virulence of the *N*-mannan mutant strain is not exclusively associated with the presence of the Dectin-1. This results propose another receptor, which could explain the immune modulating role of the *C. parapsilosis* cell wall components.