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BACKGROUND:

The Candida albicans cell wall is the first point of contact with the host, and its outer surface is heavily enriched in mannoproteins modified through the addition of N- and O-mannan. Previous work, using mutants with gross defects in glycosylation, has clearly identified the importance of mannan in the host-pathogen interaction, immune recognition and virulence. Here we report the first analysis of the MNN1 gene family, which contains six members predicted to act as α-1,3 mannosyltransferases in the terminal stages of glycosylation.

FINDINGS:

We generated single null mutants in all members of the C. albicans MNN1 gene family, and disruption of MNN14 led to both in vitro and in vivo defects. Null mutants in other members of the family demonstrated no phenotypic defects, suggesting that these members may display functional redundancy. The mnn14Δ null mutant displayed hypersensitivity to agents associated with cell wall and glycosylation defects, suggesting an altered cell wall structure. However, no gross changes in cell wall composition or N-glycosylation were identified in this mutant, although an extension of phosphomannan chain length was apparent. Although the cell wall defects associated with the mnn14Δ mutant were subtle, this mutant displayed a severe attenuation of virulence in a murine infection model.

CONCLUSION:

Mnn14 plays a distinct role from other members of the MNN1 family, demonstrating that specific N-glycan outer chain epitopes are required in the host-pathogen interaction and virulence.