Candida glabrata persistence in mice does not depend on host immunosuppression and is unaffected by fungal amino acid auxotrophy.

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Details

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Abstract

Candida glabrata has emerged as an important fungal pathogen of humans, causing life-threatening infections in immunocompromised patients. In contrast, mice do not develop disease upon systemic challenge, even with high infection doses. In this study we show that leukopenia, but not treatment with corticosteroids, leads to fungal burdens that are transiently increased over those in immunocompetent mice. However, even immunocompetent mice were not capable of clearing infections within 4 weeks. Tissue damage and immune responses to microabscesses were mild as monitored by clinical parameters, including blood enzyme levels, histology, myeloperoxidase, and cytokine levels. Furthermore, we investigated the suitability of amino acid auxotrophic C. glabrata strains for in vitro and in vivo studies of fitness and/or virulence. Histidine, leucine, or tryptophan auxotrophy, as well as a combination of these auxotrophies, did not influence in vitro growth in rich medium. The survival of all auxotrophic strains in immunocompetent mice was similar to that of the parental wild-type strain during the first week of

infection and was only mildly reduced 4 weeks after infection, suggesting that C. glabrata is capable of utilizing a broad range of host-derived nutrients during infection. These data suggest that C. glabrata histidine, leucine, or tryptophan auxotrophic strains are suitable for the generation of knockout mutants for in vivo studies. Notably, our work indicates that C. glabrata has successfully developed immune evasion strategies enabling it to survive, disseminate, and persist within mammalian hosts.

Involved units

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Topics

Interactions with immune cells (MPM)

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