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The β-glucan receptor Dectin-1 is a member of the C-type lectin family and functions as an innate pattern recognition receptor in antifungal immunity. In both mouse and man, Dectin-1 has been found to play an essential role in controlling infections with Candida albicans, a normally commensal fungus in man which can cause superficial mucocutaneous infections as well as life-threatening invasive diseases. Here, using in vivo models of infection, we show that the requirement for Dectin-1 in the control of systemic Candida albicans infections is fungal strain-specific; a phenotype that only becomes apparent during infection and cannot be recapitulated in vitro. Transcript analysis revealed that this differential requirement for Dectin-1 is due to variable adaptation of C. albicans strains in vivo, and that this results in substantial differences in the composition and nature of their cell walls. In particular, we established that differences in the levels of cell-wall chitin influence the role of Dectin-1, and that these effects can be modulated by antifungal drug treatment. Our results therefore provide substantial new insights into the interaction between C. albicans and the immune system and have significant implications for our understanding of susceptibility and treatment of human infections with this pathogen.