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n page 533 of this month's issue, in a timely and concise Review, Verstrepen, Reynolds and Fink discuss the genetic mechanisms of variation in fungal adhesins, the cell-surface glycoproteins that allow fungi to adhere to surfaces. Adhesins are known to be involved in the ability of fungi to stick to plastic surfaces and might also have a role in fungal biofilm formation.

The adhesion of fungi to plastic surfaces might sound like a rather abstract concept, but in fact it has important clinical relevance. Over the past 15-20 years, the number of plastic indwelling devices used in modern medicine - such as intravascular catheters, cardiac pacemakers and prosthetic heart valves — has increased enormously. Although such devices have undoubtedly improved the lives of many patients, their frequent use has contributed to the increased incidence of opportunistic fungal infections that has been seen over the same 15-20-year period, particularly in immunocompromised patients.

This increase is not attributable solely to Candida albicans. Candida glabrata was once thought to be a relatively innocuous commensal fungal species, and was typically referred to merely as one of the 'non-albicans Candida', a phrase guaranteed to infuriate C. glabrata researchers. It is now believed however, that C. glabrata is the second most common cause of all bloodstream and mucosal infections in the United States. An increase in the incidence of C. glabrata infections is of particular concern, as this species can be resistant to antifungals such as fluconazole.

The work reviewed by Verstrepen et al. is most straightforward in Saccharomyces cerevisiae, which has a highly tractable genetic system, but the results obtained from this model organism will hopefully give researchers a detailed picture of the mechanisms the pathogenic fungi use to attach to surfaces.



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