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The immune system is essential to maintain homeostasis with resident microbial populations, ensuring that the symbiotic host-microbial relationship is maintained. In parallel, commensal microbes significantly shape mammalian immunity at the host mucosal surface, as well as systemically. Candida albicans is an opportunistic pathogen that lives as a commensal on skin and mucosa of healthy individuals. Little is known about its capacity to modulate responses toward other microorganisms, such as colonizing bacteria (e.g., intestinal microorganisms). The aim of this study was to assess the cytokine production of PBMCs induced by commensal bacteria when these cells were primed by C. albicans. We show that C. albicans and β-1,3-glucan induce priming of human primary mononuclear cells and this leads to enhanced cytokine production upon in vitro stimulation with TLR ligands and bacterial commensals. This priming requires the β-1,3-glucan receptor dectin-1 and the noncanonical Raf-1 pathway. In addition, although purified mannans cannot solely mediate the priming, the presence of mannosyl residues in the cell wall of C. albicans is nevertheless required. In conclusion, C. albicans is able to modify cytokine responses to TLR ligands and colonizing bacteria, which is likely to impact the inflammatory reaction during mucosal diseases.