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Deciphering cellular and molecular mechanisms that maintain host immune homeostasis with fungi and the breakdown of this homeostatic tolerance during fungal infections disease is a challenge in medical mycology. In fact, the virulence of fungi may be determined by the interaction between fungi and the host immune status and its classification as a commensal microorganism or a pathogen may shift depending on the balance. In addition to the central role of the IL-12/IFN-c-dependent Th1 responses in cell-mediated immune protection against fungi, Th17 cells provide protection and inflammation at mucosal surfaces, and Tregs fine-tune immune responses to prevent damage to the host.

Recent evidence indicates that IL-22-producing cells, employing primitive antifungal effector mechanisms, contribute to antifungal resistance at mucosal surfaces under conditions of defective adaptive immunity. The fact that IL-22 production is driven by commensals points to the need of an integrated, systems biology approach to improve our understanding of the inherent and intimate mechanisms underlying multilevel host–fungus interactions.