Carvalho A, Giovannini G, De Luca A, D'Angelo C, Casagrande A, Cunha C, Iannitti RG, Romani L. Dectin-1 isoforms contribute to distinct Th1/Th17 cell activation in mucosal candidiasis. *Cell Mol Immunol*. *3*: 276-86.

The recognition of β-glucans by dectin-1 has been shown to mediate cell activation, cytokine production and a variety of antifungal responses. Here, we report that the functional activity of dectin-1 in mucosal immunity to Candida albicans is influenced by the genetic background of the host. Dectin-1 was required for the proper control of gastrointestinal and vaginal candidiasis in C57BL/6, but not BALB/c mice; in fact, the latter showed increased resistance in the absence of dectin-1. The susceptibility of dectin-1-deficient C57BL/6 mice to infection was associated with defects in IL-17A and aryl hydrocarbon receptor-dependent IL-22 production and in adaptive Th1 responses. In contrast, the resistance of dectin-1-deficient BALB/c mice was associated with increased IL-17A and IL-22 production and the skewing towards Th1/Treg immune responses that provide immunological memory. Disparate canonical/noncanonical NF-κB signaling pathways downstream of dectin-1 were activated in the two different mouse strains. Thus, the net activity of dectin-1 in antifungal mucosal immunity is dependent on the host's genetic background, which affects both the innate cytokine production and the adaptive Th1/Th17 cell activation upon dectin-1 signaling.