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캔디다 감염증에서 IL-33에 의한 감염내성과 면역관용의 유도

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IL-33 Enhances Host Tolerance to Candida Albicans Kidney Infections Through Induction of IL-13 Production by CD4+ T Cells

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Susceptibility to systemic Candida albicans infection is determined not only by immune resistance but also by the ability to control Candida-induced immunopathologies. We previously showed that exogenous IL-33 can increase resistance to peritoneal C. albicans infection by regulating multiple steps of the neutrophil anti-Candida response. Here, using a mouse model of systemic candidiasis, we observed that IL-33 administration limited fungal burden and inflammation and increased survival. In kidneys, IL-33 seemed to directly act on neutrophils and CD4+ T cells: IL-33 administration enhanced fungal clearance by increasing neutrophil phagocytic activity without which Candida proliferation was uncontrollable. On the other hand, IL-33 stimulated CD4+ T cells to produce IL-13, and it in turn drove the polarization of macrophages toward the M2 type. Furthermore, the absence of IL-13 abolished IL-33-mediated polarization of M2 macrophages and renal functional recovery. In addition, IL-33 and IL-13 acted synergistically to increase M2 macrophage polarization and its phagocytic activity. Overall, this study identifies IL-33 as a cytokine that is able to induce resistance and tolerance, and suggests that targeting resistance and tolerance simultaneously with therapeutic IL-33 may benefit patients with systemic candidiasis.

Key Words: IL-33, 감염 내성, 면역 관용
IL-33, Resistance, Tolerance