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## *Bifidobacterium longum* alleviates dextran sulfate sodium-induced colitis by suppressing IL-17A response: Involvement of intestinal epithelial co-stimulatory molecules

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Although some bacterial strains show potential to prevent colitis, their mechanisms are not fully understood. Here, we investigated the anti-colitic mechanisms of *Bifidobacterium longum* subsp. *infantis* JCM 1222T, focusing on the relationship between interleukin (IL)-17A secreting CD4<sup>+</sup> T cells and intestinal epithelial co-stimulatory molecules in mice. Oral administration of JCM 1222T to mice alleviated dextran sulfate sodium (DSS)-induced acute colitis. The expression of type 1 helper T (Th1)- and IL-17 producing helper T (Th17)-specific cytokines and transcriptional factors was suppressed by JCM 1222T treatment. Intestinal epithelial cells (IECs) from colitic mice induced IL-17A production from CD4<sup>+</sup> T cells in a cell-cell contact-dependent manner, and this was suppressed by oral treatment with JCM 1222T. Using blocking antibodies for co-stimulatory molecules, we revealed that epithelial co-stimulatory molecules including CD80 and CD40, which were highly expressed in IECs from colitic mice, were involved in IEC-induced IL-17A response. Treatment of mice and intestinal epithelial cell line Colon-26 cells with JCM 1222T decreased the expression of CD80 and CD40. Collectively, these data indicate that JCM 1222T negatively regulate epithelial co-stimulatory molecules, and this effect might be attributed, at least in part, to suppression of IL-17A in DSS-induced colitis.

### Biography

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