| ID No. | 346 |
|---|---|
| 研究課題名 | 腸管 TLR の機能解析 |
| 研究代表者 | 辻　典子　　（産業技術総合研究所・上級主任研究員） |
| 研究組織<br>受入教員<br>研究分担者 | <br>三宅　健介　　（東京大学医科学研究所・教授）<br>福井　竜太郎　（東京大学医科学研究所・助教） |

研究報告書

Double-stranded RNA of lactic acid bacteria (LAB) is recognized by dendritic cells (DCs) via endosomal-TLR3 and benefits the anti-inflammatory response through induction of interferon-β (IFN-β). However, how such IFN-β impacts T cell immune responses, and how immune homeostasis is better maintained in the presence of commensal or food-derived LAB is unknown. Here we show that LAB enhances interleukin-12 (IL-12) secretion by DCs and differentiation of IFN-γ-producing T cells in an IFN-β-dependent manner. We demonstrated that IFN-β secreted in response to LAB increased IFN regulatory factor 1 (IRF1) and IRF7 mRNA, which contribute to *Il12p35* expression. It was clarified that $CD11c^{+}CD11b^{-}CD8\alpha^{+}CD103^{+}$ DCs in Peyer's patches mainly induced Th1 cell differentiation through IFN-β production in response to LAB. The resultant induction of IFN-γ production in $CD4^{+}$ T cells also occurs *in vivo*, where oral administration of LAB enhances Th1 immunity or suppresses Th2 immune responses. Th1 polarization and maintenance of *Foxp3* expression by $CD4^{+}$ T cells due to TLR3-mediated IFN-β production may thus confer anti-allergic or anti-inflammatory activity by commensal or probiotic LAB.