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E-letters

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Brief Report

[Helicobacter pylori infection prevents allergic asthma in mouse models through the induction of regulatory T cells](#)

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The role of interleukin 10 in the prevention of allergic asthma in mouse models with Helicobacter pylori infection

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Dear Editor,

We read with great interest the recent contribution by Arnold et al. (1) They reported that *Helicobacter pylori* (HP) infection prevents allergen-induced asthma in mouse models through the induction of regulatory T (Treg) cells, providing experimental evidence that the adoptive transfer of purified Treg populations was

sufficient to transfer protection from infected donor mice to uninfected recipients. However, not only in mouse models but also in humankind, inverse correlation between allergy and HP infection exists (2). We would like to specify a possible mechanism of HP protecting from allergy.

According to a previous study by Codolo et al. (3), systemic HP neutrophil-activating protein (HP-NAP) significantly resulted in both reduction of total serum IgE and increase of interleukin 12 (IL-12) plasma levels. Mucosal administration of HP-NAP was equally successful as the systemic delivery in reducing eosinophilia, IgE, and T helper type 2 (Th2) cells cytokine levels in bronchoalveolar lavage. In contrast, Windle et al. (4) found that IL-10 production by lamina propria lymphocytes was significantly higher in the HP positive group after stimulation with HP-NAP ($P < 0.05$) than in the HP negative group. Bontems et al. (5) also showed that IL-10 concentration was increased in HP-infected children compared with adults ($P < 0.05$). Furthermore, Harris et al. (6) demonstrated that HP infection in the children was associated with markedly increased levels of gastric IL-10 messenger RNA, suggesting that gastric Treg cell responses down-regulate the inflammation and ulceration induced by HP in children. IL-10, originally named “cytokine synthesis inhibitory factor”, is an major agent in the resolution of asthmatic inflammation, inhibiting eosinophilia by suppression of IL-5 and granulocyte macrophage-colony stimulating factor, by direct effects on eosinophil apoptosis, and by effects on cell proliferation through down-regulation of IL-1 (7). It is thought to occur through the upregulation of suppressor of cytokine signaling-3. After stimulation by allergen, IL-10-secreting Treg cells inhibit cytokine secretion by allergen-specific Th2 cells in an IL-10-dependent manner (8). The treatment of allergen-injection immunotherapy may induce IL-10-secreting Treg cells, leading to suppression of Th2 responses (9).

Therefore, there is a possibility when a patient is infected by CagA+ HP, IL-10 stimulated by HP-NAP may play a crucial role in preventing asthma through the induction of Treg cells. However, further studies are necessary to elucidate the exact molecular role of IL-10 and specific correlation between “hygiene hypothesis” or “disappearing microbiota hypothesis” including HP and asthma incidence in the future.

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