Abstract


Biotin status affects nickel allergy via regulation of interleukin-1beta production in mice.

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BACKGROUND: Biotin, a water-soluble B complex vitamin, is possibly involved in chronic inflammatory diseases, although the detailed mechanisms are unclear.

OBJECTIVE AND METHODS: In this study, we investigated the effects of biotin status on nickel (Ni) allergy in mice. Mice were fed a basal or biotin-deficient (BD) diet for 8 wk and sensitized with an intraperitoneal injection of NiCl(2) and lipopolysaccharide. Ten days after sensitization, NiCl(2) was intradermally injected into pinnas and ear swelling was measured. For in vitro analysis, we cultured a murine macrophage cell line, J774.1, under a biotin-sufficient (C, meaning control) or BD condition for 4 wk and analyzed interleukin (IL)-1 production.

RESULTS: Significantly higher ear swelling was induced in BD mice than C mice. Adaptive transfer of splenocytes from both C and BD mice induced Ni allergy in unsensitized mice. Regardless of donor mice, ear swelling was significantly higher in BD recipient mice than C recipient mice. Ni allergy was not induced in either C or BD IL-1(-/-) mice. Splenocytes from BD mice produced a significantly higher amount of IL-1beta than those from C mice. Production and mRNA expression of IL-1beta were significantly higher in BD J774.1 cells than in C cells. Biotin supplementation inhibited the augmentation of IL-1beta production in vitro. In vivo supplementation of biotin in drinking water dose-dependently decreased ear swelling in C and BD mice.

CONCLUSION: These results indicate that biotin status affects Ni allergy in the elicitation phase via the upregulation of IL-1beta production in mice, suggesting that biotin supplementation may have therapeutic effects on human metal allergy.

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