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Poster Sessions

Allergic activation of RBL-2H3 mast cells with L-Asparaginase and its inhibition by anti-IgE antibody

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L-Asparaginase (L-ASP) derived from *E. coli* is a key drug in the treatment of childhood acute lymphoblastic leukemia (ALL). L-ASP often causes hypersensitivity reactions, including anaphylaxis. We reported that a neutralizing antibody (Ab) of IgE was effective to inhibit L-ASP-induced allergy in the mouse model. In the present study, we used RBL-2H3 cells to establish *in vitro* model of L-ASP allergy.

Male BALB/c mice were sensitized by L-ASP with Al(OH)₃ on days 0 and 14. Cyclophosphamide (CY) was i.p. administrated on days -2 and 12. The serum was collected on day 26. Total IgE level in the serum was measured by ELISA. RBL-2H3 was sensitized by the serum and stimulated by L-ASP to determine β -hexosaminidase (β -Hex) release *in vitro*.

L-ASP sensitization increased serum IgE level, which was enhanced by CY at 150 mg/kg. When RBL-2H3 was sensitized by L-ASP-sensitized serum *in vitro*, L-ASP stimulated β -Hex release from the cells. The serum of CY-treated mice induced higher β -Hex release than normal. Anti-IgE Ab inhibited allergic β -Hex release both *in vitro* and *ex vivo*.

From the present results, L-ASP sensitization induced IgE production *in vivo* and this serum induced β -Hex release from RBL-2H3 cells. Using cell lines expressing Fc ϵ RI, it can be possible to evaluate L-ASP allergy and efficacy of anti-IgE Ab *in vitro*.