Role of Mast Cells versus Basophils in IgE-Dependent Local Ear Skin Release of the Serotonin Required to Initiate Contact Sensitivity in Mice

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Key Words

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Our previous work has shown that antigen-specific factors derived from lymphoid cells of contact-sensitized mice induce the initiation of contact sensitivity, by sensitizing local mast cells for the early release of serotonin that activates local vessels to allow local recruitment, infiltration and activation of late-acting, classical, contact sensitivity effector T cells.

In our new experiments, anti-TNP IgE-sensitized non-immune spleen or lymph node cells, or anti-TNP IgE-sensitized purified peritoneal mast cells, or mast cells cultured in vitro with and without fibroblasts, were injected intravenously together with contact sensitivity effector T cells that were depleted of the contact-sensitivity-initiating cells by in vitro treatment with anti-B220 (CD45RA) monoclonal antibody plus complement, and were found to transfer contact sensitivity responses to normal mice, but not to mast-cell-deficient mouse recipients (W/W^v and Sl/Sl^d), defective in manifesting this IgE-dependent contact sensitivity. When mast-cell-deficient mice were restored locally with mast cells by intradermal injection of bone-marrow-derived cultured mast cells, contact sensitivity responses were obtained. This suggested that skin mast cells were required. In vivo treatment of recipients with rat myeloma IgE blocked IgE FcεR in the recipients, and prevented contact sensitivity initiation mediated by TNP-specific IgE-monoclonal-anti-body-sensitized lymphoid cells, but did not prevent elicitation following active contact sensitivity, or adoptive transfer of immune cells.

We concluded that the mast cell binding sites for the antigen-specific contact-sensitivity-initiating factor are different from FcεRI, and that IgE bound to spleen FcεRI-bearing cells (probably basophils) sensitized with IgE in vitro was able to detach from circulating FcεRI+ cells, and then bind to skin mast cell FcεRI, thereby sensitizing for local release of serotonin to initiate contact sensitivity. An alternative hypothesis is that anti-TNP IgE antibodies may have been able to trigger the basophils, via local TNP-antigen available from the challenge, to release

basophil factors (perhaps cy-tokines) that activated local skin mast cells for release of serotonin, thus initiating contact sensitivity.

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