



Permeability of rat IgE across rat aortic endothelial cell is enhanced by histamine

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Background: It is a well-known fact that IgE is a key substance that induces an allergic reaction in extravascular tissue. However, it remains to be elucidated how IgE in the circulating blood transfers to the site of the allergic reaction in the extravascular tissue. In the present paper, rat IgE passage through cultured rat aortic endothelial cells (RAEC) was first examined using a dual-chamber system. Second, we examined the effects of histamine, which is thought to affect endothelial permeability, on IgE passage through the RAEC in comparison with the effects of albumin and IgG2a.

Methods: The permeability constant (PC) was used to evaluate the degree of IgE passage through the RAEC.

Results: The value of the PC for rat IgE ($0.58 \pm 0.11 \times 10^{-5}$ cm/s) was lower than that for IgG2a and albumin ($0.88 \pm 0.28 \times 10^{-5}$ and $0.93 \pm 0.26 \times 10^{-5}$ cm/s, respectively) under conditions of non-exposure to histamine. In contrast, the PC of rat IgE was significantly increased by exposure to histamine (10^{-10} mol/L) at 12 h after exposure. However, the PC for IgG2a and albumin were not significantly increased following exposure to histamine. The enhancement by histamine of IgE passage through the RAEC was not inhibited by diphenhydramine, a histamine H1 receptor antagonist, but were inhibited by cimetidine, a histamine H2 receptor antagonist.

Conclusions: On the basis of results from the present study, histamine, acting via H2 receptors, enhances the permeability of rat IgE across the RAEC mono-layer. The increased permeability of endothelial cells induced by histamine may contribute greatly to the transfer of IgE from circulating blood to extravascular tissue.

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