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Contact urticaria is recognized as the wheal and flare reaction at a site from direct contact with a chemical or protein agent. Ongoing studies have proposed that gene silencing may have a promising future in finding optimal treatment of a variety of disease; hence, the aim of the study was to investigate the effect of RNA interference-mediated E-selectin (SELE) gene silencing on cell adhesion molecule expression and on cell-cell adhesion in vascular endothelial cells (VECs) in a mouse model of immunologic contact urticaria (ICU). Following the successful establishment of mouse models of ICU induced by antidinitrophenol immunoglobulin E (IgE) combining 2,4-dinitrofluorobenzene challenge, enzyme-linked immunosorbent assay and immunohistochemistry were used to measure the levels of IgE, interleukin 4 (IL-4), interferon- γ (IFN- γ), and histamine as well as the positive expression rate of SELE, respectively. The siRNA- SELE vector was constructed and transfection efficiency was estimated prior to performing quantitative reverse-transcription polymerase chain reaction and Western blot assay to determine the relative expression of SELE, eosinophil cationic protein (ECP), intercellular adhesion molecule 1 (ICAM-1), L-selectin (CD62L), and the alpha chain of leukocyte function-associated antigen-1 (CD11a). Adhesion assay was then performed to assess the cell adhesion ability in VECs. Elevated levels of IgE, IL-4, IFN- γ , and histamine and increased positive expression rate of SELE were indicative of successful establishment of mouse models of ICU. Furthermore, the relative expression levels of SELE, ECP, ICAM-1, CD62L, and CD11a were highest in the OE- SELE group. Besides, cell adhesion ability of VECs was notably promoted. Collectively, the current study define the potential role of SELE silencing as an inhibitor to ICU development by inhibiting cell adhesion ability of VECs ¹⁾.

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