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Impaired barrier function in patients with house dust mite–induced allergic rhinitis is accompanied by decreased occludin and zonula occludens-1 expression

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ABSTRACT:

Tight junction (TJ) defects have recently been associated with asthma and chronic rhinosinusitis. The expression, function, and regulation of nasal epithelial TJs remain unknown in patients with allergic rhinitis (AR).

We investigated the expression, function, and regulation of TJs in the nasal epithelium of patients with house dust mite (HDM)–induced AR and in an HDM-induced murine model of allergic airway disease.

Air-liquid interface cultures of primary nasal epithelial cells of control subjects and patients with HDM-induced AR were used for measuring transepithelial resistance and passage to fluorescein isothiocyanate–dextran 4 kDa (FD4). *Ex vivo* transtissue resistance and FD4 permeability of nasal mucosal explants were measured. TJ expression was evaluated by using real-time quantitative PCR and immunofluorescence. In addition, the effects of IL-4, IFN-γ, and fluticasone propionate (FP) on nasal epithelial cells were investigated *in vitro*. An HDM murine model was used to study the effects of allergic inflammation and FP treatment on transmucosal passage of FD4 *in vivo*.

A decreased resistance *in vitro* and *ex vivo* was found in patients with HDM-induced AR, with increased FD4 permeability and reduced occludin and zonula occludens-1 expression. AR symptoms correlated inversely with resistance in patients with HDM-induced AR. *In vitro* IL-4 decreased transepithelial resistance and increased FD4 permeability, whereas IFN-γ had no effect. FP prevented IL-4–induced barrier dysfunction *in vitro*. In an HDM murine model FP prevented the allergen-induced increased mucosal permeability.

We found impaired nasal epithelial barrier function in patients with HDM-induced AR, with lower occludin and zonula occludens-1 expression. IL-4 disrupted epithelial integrity*in vitro*, and FP restored barrier function. Better understanding of nasal barrier regulation might lead to a better understanding and treatment of AR.

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