# Alteration in claudin-4 contributes to airway inflammation and responsiveness in asthma

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Abstrak

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PURPOSE:
Claudin-4 has been reported to function as a paracellular sodium barrier and is one of the 3 major claudins expressed in lung alveolar epithelial cells. However, the possible role of claudin-4 in bronchial asthma has not yet been fully studied. In this study, we aimed to elucidate the role of claudin-4 in the pathogenesis of bronchial asthma.

METHODS:
We determined claudin-4 levels in blood from asthmatic patients. Moreover, using mice sensitized and challenged with OVA, as well as sensitized and challenged with saline, we investigated whether claudin-4 is involved in the pathogenesis of bronchial asthma. Der p1 induced the inflammatory cytokines in NHBE cells.

RESULTS:
We found that claudin-4 in blood from asthmatic patients was increased compared with that from healthy control subjects. Plasma claudin-4 levels were significantly higher in exacerbated patients than in control patients with bronchial asthma. The plasma claudin-4 level was correlated with eosinophils, total IgE, FEV1% pred, and FEV1/FVC. Moreover, lung tissues from the OVA-OVA mice showed significant increases in transcripts and proteins of claudin-4 as well as in TJ breaks and the densities of claudin-4 staining. When claudin-4 was knocked down by transfecting its siRNA, inflammatory cytokine expressions, which were induced by Der p1 treatment, were significantly increased.

CONCLUSIONS:
These findings thus raise the possibility that regulation of lung epithelial barrier proteins may constitute a therapeutic approach for asthma.