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Title: Continuous airway inflammation is related to the development of airway remodeling

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Body: In bronchial asthma, airway remodeling leads to refractoriness to treatment. Recent studies suggest that mechanical stress induces remodeling independently of inflammation. For this hypothesis, we examined the effects of continuous mechanical stress and airway inflammation in the mouse. Mice were divided into 3 groups: an airway inflammation group (group A), a methacholine-inhalation-induced mechanical stress group (group B), and a control group (group C). On days 14 to 29 after sensitization by intraperitoneal injection of ovalbumin, the mice in each group inhaled physiological saline, ovalbumin, or methacholine. Counts of eosinophils and other inflammatory cells, smooth-muscle/ basement-membrane thickening, and goblet-cell hyperplasia were compared among the groups. Eosinophil counts increased with time in group A, but not in group B or C. Smooth-muscle thickening was slightly greater in group A and group B than in the control group up to day 10 after sensitization. Group A showed a continuous trend toward increased smooth-muscle thickness up to day 15. In contrast, group B showed a slight decrease in smooth-muscle thickness. The difference was more marked in the peripheral than in the central airway. Basement-membrane thickening progressed with time in group A, but was not evident in group B or group C. Goblet-cell hyperplasia calculated on the basis of mucus scores significantly increased in group A, but was unchanged in group B and group C. Conclusion: Mechanical stress was transiently associated with thickening and proliferation of airway smooth muscle, but this effect decreased with time, suggesting that chronic, continuous airway inflammation plays an important role in remodeling.