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**Title:** Apolipoprotein A1 (ApoA1) abrogate cigarette smoke induced emphysema in mice

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**Body:** Rationales: Apolipoprotein A-1 (ApoA1) have anti-inflammatory and antioxidant properties as well as cholesterol efflux. Objectives: To determine if the expression of human ApoA1 within the lung protect against the development of emphysema Methods: Transgenic human ApoA1 mice(ApoA1 TG) were exposed to CS for 6 month and compared to control transgenic mice. Other ApoA1 TG mice were treated with intratracheal elastase in order to generate emphysema. Measurement; Lung inflammation, oxidative injury was measured in the lung. Emphysema was determined by measuring the mean linear intercept(Lm). Proinflammatory cytokines in the BALF were measured by ELISA and analysis of apoptosis using the TUNEL assay. Results: Compared with control TG mice, ApoA1 TG mice had significantly less lung inflammation, oxidative damage and apoptosis as well as decreased levels of proinflammatory cytokines. ApoA1 attenuated the development of emphysema in both the smoke-induced and elastase-generated models. Conclusions: Overexpression of ApoA1 prevents CS and elastase induced emphysema in mice. Augmentation of ApoA1 in the lung could be effective for the prevention or treatment of emphysema/COPD.