increasing level of tobacco smoke exposure. In addition, systolic and diastolic blood pressure and BMI were independent predictors of the aortic elasticity indices. In multivariable models, cotinine level (P = .020) and systolic blood pressure (P < .001) were inversely associated with AC and directly related to SI (cotinine level, P = .005; systolic blood pressure, P = .0003). **CONCLUSIONS:** These data suggest that passive smoking is associated with decreased aortic elasticity in children, indicating early arterial changes.

**EXPERIMENTAL RESEARCH OF SIMVASTATIN IN REVERSING PULMONARY VASCULAR REMODELING IN VIVO AND IN VITRO**

Submitted by Hanmin Liu
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**INTRODUCTION:** Simvastatin was predicted to be a potential inhibitor to pulmonary vascular remodeling. This novel reversion induced by simvastatin has remained an uncertain mechanism.

**OBJECTIVE:** Our goal was to explore the role of simvastatin as a potential inhibitor of pulmonary vascular remodeling.

**METHODS:** We established a neointimal pulmonary hypertensive rat model receiving monocrotaline after pneumonectomy. Simvastatin was administered after the operation. Hemodynamic and vascular remodeling corresponding indices were detected. GATA-6, a gene transcription factor, was evaluated in vivo. Proliferation and the cellular cycle were assessed in cultured vascular smooth muscle cells (VSMCs). α-SM-actin, F-actin, and paxillin were detected to evaluate the phenotype changes.

**RESULTS:** Neointimal changes developed in 88.5% of right lung intraacinar arteries after pneumonectomy and monocrotaline administration. Mean pulmonary artery pressure, the right ventricle/(left ventricle + S) ratio, and media wall thickness significantly increased in rats that had pneumonectomy and were treated with monocrotaline but decreased significantly in simvastatin-treated rats. The expression of GATA-6 markedly decreased in these rats and was significantly upregulated after receiving simvastatin. In vitro, the proliferation was significantly downregulated in VSMCs with simvastatin compared to that with platelet-derived growth factor. α-SM-actin increased significantly, and F-actin or paxillin was downregulated in simvastatin-treated rats.

**CONCLUSIONS:** Our data indicate that simvastatin is most likely a pulmonary vascular remodeling inhibitor, which may reverse the proliferation of VSMCs and phenotype changes. Simvastatin can also upregulate GATA-6 expression in lung tissue.
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