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**Title:** Second hand smoke exposure impairs CD39 expression and function in the lung

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**Body:** Chronic second hand smoke (SHS) exposure is the main risk factor for non-smokers to develop chronic obstructive pulmonary disease COPD. The mechanisms behind the chronic inflammation and lung destruction are not completely understood. In response to injury vascular and blood cells release ATP that is implicated in regulating cellular and immune responses. Plasma membrane ecto-nucleotidase CD39 hydrolyzes ATP to ADP and AMP. Ecto-5'nucleotidase CD73 hydrolyzes AMP to adenosine. Here we investigated the contribution of CD39/73 expressing inflammatory cells in lung remodeling in response to SHS exposure. Sprague Dawley rats were exposed to SHS in a smoking chamber (total particulate matter levels 115 mg/m<sup>3</sup>). The expression levels of CD39 and CD73 in the inflammatory cells and lung tissue were determined by flow cytometry, real time PCR and Western blot analysis. Chronic second hand smoke exposure resulted in the development of emphysema in rats as measured by MLI (87.2 ± 1.5 mm after 2 month of exposure versus 73.4 ± 1.1 mm in room air exposed controls). The CD39 expression was significantly downregulated in the whole lung tissue on mRNA and protein levels. Moreover, CD39 expression was decreased in the lung tissue from COPD patients. Cigarette smoke extract in vitro almost abolished CD39 and CD73 expression in the alveolar macrophages and vascular endothelial cells. Second hand smoke exposure impairs ectonucleotidase expression in the lungs and leads to accumulation of extracellular ATP that confers increased proinflammatory responses leading to the development of emphysema. Funded by AHA 0735388N, 11GRNT7520020, FAMRI CIA 072053, Emphysema Research Fund and Bixler Family Foundation.