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Institution: The Johns Hopkins Bloomberg School of Public Health

Title: Targeting AP1 Proteins in COPD

ABSTRACT I

It is well documented that MAP kinases play a central role in mediating the cellular responses initiated by various extracellular stimuli. Various MAP kinase modules display remarkable specificity and functional importance in controlling gene expression, apparently by distinctly activating downstream effector transcription factors, including AP-1 family proteins. AP-1, a dimeric transcription factor, is essential for various cellular events, ranging from proliferation and differentiation. Several studies, including ours, have demonstrated a differential activation of AP-1 family member expression by other toxicants such as cigarette smoke. However, little is known about the contribution and the expression levels of AP-1 proteins to the development and progression of toxin-induced respiratory pathogenesis in COPD. We are using genetic mouse models to understand the roles of AP1 proteins the cigarette smoke induced emphysema/COPD. We hypothesize that AP-1 proteins modulates oxidative stress through ht regulation of antioxidant gene expression and a dysfunctional AP-1 signaling may contributes to COPD. The specific objective of this pilot study is to analyze the expression levels of AP-1 proteins in the non-COPD and COPD tissues to compliment our on going studies with genetic mouse models. The results of this study may provide may have important implications for understanding the role of AP1 proteins in COPD and enable us to target MAP kinase/AP1 signaling in lung diseases such as COPD.

Groups:

10 Subjects per group. Four groups.

COPD: FEV1% >80%, FEV1% 50-80%, FEV1% <50%

IPF: FVC% <50%

Types:

Formalin Fixed (10 slides/subject)

RNA Later (1 aliquot/subject)

Flash Frozen (1 aliquot/subject)