Lipids as Modifiable Risk Factors of Environmental Lung Disease: A Systematic Review of the Good, the Bad and the Misunderstood

E. Caraher, G. Crowley, S. Haider, R. Lam, S. Kwon, A. Nolan; Pulmonary, Critical Care and Sleep Medicine, New York University School of Medicine, New York, NY, United States.

RATIONALE The mechanisms by which some patients develop obstructive airways disease (OAD) following environmental or toxic exposures is not well understood. Our group has identified metabolic biomarkers of particulate matter associated lung disease. Of particular interest, lipids and their metabolites are associated with loss of FEV1 in firefighters exposed to particulates at the site of WTC destruction, which adds to increasing evidence of an association between lipids and OAD. Metabolomics is the study of small molecules using targeted or untargeted techniques to elucidate pathways active in the pathogenesis of disease. Lipid-associated pathways, once validated, could provide a metabolomics fingerprint of OAD useful in both diagnosis, assessment and treatment. OBJECTIVES Previous reviews have focused on metabolomics technique. This systematic review will provide a comprehensive overview of the commonly active lipid pathways in exposure associated OAD identified in metabolomics studies in the past 10 years. METHODS Data Source: PubMed (8/15/17) Data Extraction: Databases were searched for: metabolomics or lipidomics AND (particulate matter OR smoke OR ozone OR polycyclic aromatic hydrocarbons) AND lung. Additionally, only papers in English and published in the last 10 years were included. The final systematic review will be registered with PROSPERO. RESULTS/SYNTHESIS A total of 46 studies were identified through the search above and 4 through other sources, Figure 1. Upon screening, 5 papers were excluded as they did not look at pulmonary endpoints, 2 papers were excluded as their focus was lung cancer, 3 papers did not use metabolomics and 1 paper lacked lipid analysis. Of the final 43 papers, 27 were animal studies, 11 were human studies and 1 compared plasma/BAL metabolomes in humans and mice. The specimens analyzed in human studies included induced sputum (n=1), human basal cell isolates (n=1), blood (n=7), BAL (n=1) and exhaled breath condensate (n=1). Commonly identified lipids altered following exposures included sphingolipids, phospholipids and polyunsaturated fatty acids. CONCLUSIONS With a greater understanding of the pathways commonly active in OAD, future work can focus on elucidating the metabolomics fingerprint of OAD and dissecting the pathways identified to inform the use of targeted therapeutics. FUNDING NHLBI R01HL119326, CDC/NIOSH U01-OH011300

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