

## Air Pollution and Proteomics



### Chin Kook Rhee

**Organization** Department of Internal Medicine, Seoul St. Mary's Hospital, The Catholic University of Korea  
**Current Position** Professor

#### Educational background

2007-2013	Ph.D., The Catholic University
2005-2007	M.A., Internal Medicine, The Catholic University
1996-2002	M.D., The Catholic University

#### Professional experience

Present	Editorial Board Member of AJRCCM
Present	Chairperson of APRS Research Committee
Present	Head of ASPR COPD Assembly
Present	Steering Committee Member of ISAR
Present	Deputy Editor of TRD

Air pollution is a leading global risk factor for respiratory morbidity and mortality, contributing significantly to asthma, chronic obstructive pulmonary disease (COPD), and other chronic respiratory disorders. While epidemiologic evidence has firmly established the association between fine particulate matter (PM), nitrogen oxides, and ozone with impaired lung function and exacerbations, recent advances in proteomics and metabolomics are now unraveling the molecular mechanisms underlying these associations. Studies of airway macrophages demonstrate that black carbon deposition, derived from PM<sub>2.5</sub> and cigarette smoke, correlates with indoor pollutant exposure and is linked to worse lung function, increased exacerbation risk, and impaired quality of life in COPD. Life-course metabolomic studies further reveal that air pollution exposure alters amino acid, lipid, and energy metabolism, with distinct metabolic signatures predicting accelerated lung function decline and higher incidence of chronic respiratory diseases. Controlled human exposure studies provide mechanistic evidence: diesel exhaust exposure in COPD patients increases fibrinogen and thromboxane metabolites, reflecting heightened systemic prothrombotic and inflammatory responses. Complementary work highlights extracellular vesicles (EVs) as novel mediators of pollutant injury. Environmental pollutant-derived EVs carry dysregulated proteins and microRNAs that disrupt epithelial barrier integrity, promote neutrophilic inflammation, and drive airway remodeling, thereby contributing to asthma, COPD, fibrosis, and pulmonary hypertension. Collectively, proteomic and metabolomic profiling provide unprecedented insights into how environmental exposures translate into molecular injury and clinical outcomes. These approaches not only deepen understanding of pathogenesis but also enable discovery of biomarkers for exposure, susceptibility, and disease progression. Integration of omics with exposome data holds promise for early detection, risk stratification, and development of targeted interventions. Air pollution proteomics thus bridges environmental exposure with molecular phenotyping, paving the way for precision respiratory medicine and advancing strategies to mitigate the global burden of pollution-related lung disease.