

Lung Tissue Damage under Diesel Exhaust Exposure: Modification of Proteins, Cells and Functions in Just 14 Days

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Abstract : Introduction: Air pollution is a growing global problem which has been shown to be responsible for various adverse health outcomes. Immunotoxicity, such as dysregulated inflammation, has been proposed as one of the main mechanisms in air pollution-associated diseases. Chronic obstructive pulmonary disease (COPD) is among major morbidity and mortality causes worldwide and is characterized by persistent airflow limitation caused by the small airways disease (obstructive bronchiolitis) and irreversible parenchymal destruction (emphysema). Exact pathways explaining the air pollution induced and mediated disease states are still not clear. However, modern societies understand dangers of polluted air, seek to mitigate such effects and are in need for reliable biomarkers of air pollution. We hypothesise that post-translational modifications of structural proteins, e.g. citrullination, might be a good candidate biomarker. Thus, we have designed this study, where mice were exposed to diesel exhaust and the ongoing protein modifications and inflammation in lungs and other tissues were assessed. Materials And Methods: To assess the effects of diesel exhaust a in vivo study was designed. Mice (n=10) were subjected to everyday 2-hour exposure to diesel exhaust for 14 days. Control mice were treated the same way without diesel exhaust. The effects within lung and other tissues were assessed by immunohistochemistry of formalin-fixed and paraffin-embedded tissues. Levels of inflammation and citrullination related markers were investigated. Levels of parenchymal damage were also measured. Results: In vivo study corroborates our own data from in vitro and reveals diesel exhaust initiated inflammatory shift and modulation of lung peptidyl arginine deiminase 4 (PAD4), citrullination associated enzyme, levels. In addition, high levels of citrulline were observed in exposed lung tissue sections co-localising with increased parenchymal destruction. Conclusions: Subacute exposure to diesel exhaust renders mice lungs inflammatory and modifies certain structural proteins. Such structural changes of proteins may pave a pathways to lost/gain function of affected molecules and also propagate autoimmune processes within the lung and systemically.

Keywords : air pollution, citrullination, in vivo, lungs

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