RENAL INFLAMMATION AND OXIDATIVE STRESS TRIGGERED BY CHRONIC PARTICULATE MATTER EXPOSURE

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Particulate matter in the ambient air is composed of extremely small particles with different chemical compositions and properties. Chronic exposure to particulate matter can impair renal function and trigger an inflammatory response which can augment atherosclerotic progression. Although the body's mechanisms to detect particulates and generate a response are not known, evidence suggests that oxidative stress and inflammation play a role in the pathogenesis of chronic particulate matter intoxication. C57BL/6 mice were exposed to 2.5 micrometer particulates (PM 2.5; n=5) or control filtered air (FA; n=5) for ten months. The kidneys were harvested and the renal cortex isolated, snap frozen, homogenized in radioimmunoprecipitation assay buffer and centrifuged to obtain protein extracts. Activities of anti-oxidant and anti-glycation enzymes, expressed as U/mg protein, were assayed by spectrophotometry. Tissue contents of proinflammatory cytokines (TNF-alpha, IL-6) were assessed by immunoblots. Enzyme activities and cytokine contents in the FA and PM 2.5 groups were compared by Student's t test. The activities of glyoxalase 1, glucose-6-phosphate dehydrogenase, glutathione reductase, and glutathione peroxidase trended upward in the PM 2.5 vs the FA group, although the differences were not statistically significant. Interestingly, isocitrate dehydrogenase trended downward in the PM2.5 group. The content of the proinflammatory cytokine IL-6 was lower in the PM 2.5 group than the FA group (P = 0.04), and a downward trend of TNF-alpha content in the PM 2.5 group was noted. The increased activities of antioxidant and anti-glycation enzymes in this pilot study may indicate an adaptive response in the kidney to chronic particulate exposure. These enzymatic adaptations may have contributed to the decreased inflammatory response. Funding provided by the Department of Health and Human Services, National Institute of Health, National Heart, Lung and Blood Institute, SMART Grant 5R25HL007786-25 to Dr. Jamboor K. Vishwanatha, Ph.D.

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