Chronic Social Stress and Susceptibility to Concentrated Ambient Fine Particles in Rats

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Background and Objectives: Epidemiologic evidence suggests that chronic stress may alter susceptibility to air pollution. Persistent spatial confounding, however, may limit the utility of epidemiologic methods to disentangle these effects and cannot identify physiologic mechanisms for this differential susceptibility. Using a rat model of social stress, we compared respiratory response to concentrated fine ambient particles (CAPs), and examined biologic markers of inflammation.

Methods: Twenty-four 12-week-old male Sprague-Dawley rats were randomly assigned to four groups (Stress/CAPs; Stress/Filtered Air (FA); Non-stress/CAPs; Non-stress/FA). Stress group animals were individually introduced into a dominant male's home cage twice weekly. Blood drawn at sacrifice was analyzed for immune and inflammatory markers. CAPs were generated using the Harvard fine particle concentrator, drawing real-time urban ambient fine particles, which enriched concentrations approximately 30 times. CAP/FA exposures were delivered in single-animal phethysmographs, 5 hours/day for 10 days, with respiratory function continuously monitored using a Buxco system.

Results: Stressed animals displayed greater average CRP, TNF-alpha, and white blood cells. Among non-stressed animals, CAP exposures conferred higher flows and volumes, with briefer pauses. Among stressed animals, CAP exposures conferred greater respiratory frequency and lower flows and volumes. Only with both exposures did we observe rapid, shallow breathing patterns, with lower total airflows.

Conclusions: CAP effects on respiratory function differed significantly by stress group. CAPs conferred a shallow, rapid breathing pattern, exacerbated under chronic stress. Blood measures provided evidence of inflammatory responses. Results support epidemiologic findings that chronic stress may alter susceptibility to air pollution and may help elucidate pathways for differential susceptibility.