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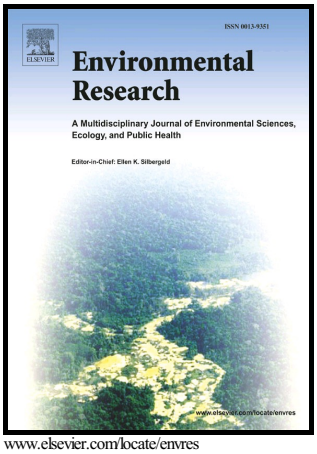

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Stress axis variability is associated with differential ozone-induced lung inflammatory signaling and injury biomarker response

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ABSTRACT

Ozone (O_3), a ubiquitous urban air pollutant, causes adverse pulmonary and extrapulmonary effects. A large variability in acute O_3-induced effects has been observed; however, the basis for interindividual differences in susceptibility is unclear. We previously demonstrated a role for the hypothalamic-pituitary-adrenal (HPA) stress axis and glucocorticoid response in acute O_3 toxicity. Glucocorticoids have important anti-inflammatory actions, and have been shown to regulate lung inflammatory responses. We hypothesised that a hyporesponsive HPA axis would be associated with greater O_3-dependent lung inflammatory signaling. Two genetically-related rat strains with known differences in stress axis reactivity, highly-stress responsive Fischer (F344) and less responsive Lewis (LEW), were exposed for 4h by nose-only inhalation to clean air or 0.8 ppm O_3, and euthanized immediately after exposure. As expected, baseline (air-exposed) plasma corticosterone was significantly lower in the hypo-stress responsive LEW. Although O_3 exposure increased plasma corticosterone in both strains, corticosterone remained