The Effects of Chronic Ozone Exposure in Mice on Skeletal Muscle Mitochondrial Proteins

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Ozone is an urban air pollutant known to induce localized and systemic inflammation, characterized by the production of reactive oxygen and nitrogen species. This inflammatory cascade is associated with increases in expression of tumor necrosis factor alpha (TNFα) and nitric oxide (NO) and decreases expression of peroxisome proliferator-activated receptor-γ coactivator-α (PGC1-α), a regulator of mitochondrial biogenesis. Ozone exposure has also been reported to impair mitochondrial function and respiration. Skeletal muscle has a high density of mitochondria and myoglobin, a hemoprotein that facilitates oxygen transport and scavenges NO. We speculate that ozone-induced systemic inflammation leads to altered mitochondrial function in skeletal muscle. To test this, we analyzed the effects of chronic ozone exposure on myoglobin, TNFα, and PGC1-α expression in skeletal muscle. Female C57BL/6 mice were exposed to filtered air or 1.5 ppm ozone for 2 h, 2x/week for 6 weeks. The quadriceps and gastrocnemius muscles were collected 24 h following the final exposure and snap frozen in liquid nitrogen. Tissue protein was quantified using a bicinchoninic acid (BCA) assay. Myoglobin, TNFα, and PGC1-α were analyzed by western blotting, and densitometry assessed using ImageJ. A Student's t-test was performed to compare densitometry means with statistical significance set at $p>0.05$. PGC1-α was found to be significantly decreased in the gastrocnemius ($p=0.048$) following ozone exposure, but not in the quadriceps. Conversely, ozone had no effect on myoglobin or TNFα expression in either muscle. These findings suggest that chronic ozone exposure differentially affects skeletal muscle mitochondria, potentially through the regulation of PGC1-α. Supported by NIH grants ES033698, ES005022, ES02072, MRF-836930, T32-832715 and ASPET summer internship.