reduced in various cardiovascular conditions such as chronic heart failure, post-infarction and hypertension. Given air pollution can alter autonomic balance, we hypothesized that exposure to air pollutants would also alter BRS and increase the risk of adverse cardiovascular events. Conscious unrestrained Wistar-Kyoto (WKY) and spontaneously hypertensive (SH) rats implanted with an intravenous catheter and radiotelemeters were administered increasing doses of phenylephrine (PE-vasoconstrictor) and then sodium nitroprusside (SNP-vasodilator) while HR response, BP, and electrocardiogram (ECG) were continuously measured. This BRS test was done one day before and one day after exposure to either air or 3ppm acrolein (3hrs). Before exposure, PE caused a dose-dependent increase in BP, which resulted in decreasing HR in WKY rats; SNP caused a dose-dependent decrease in BP and resulting HR increases. SH rats had a similar BRS response; however, the reflexive changes in HR due to BP increase/decrease were attenuated at the higher doses. Twenty-four hours after acrolein, BRS was decreased in WKY rats and further blunted in SH rats. SH rats had significantly more arrhythmias than WKY rats during and after exposure to acrolein. Although SH rats had higher baseline BP, there were no other ECG differences between strains. In conclusion, a single exposure to a toxic air pollutant alters the body's ability to reflexively regulate cardiovascular function and predisposes to adverse cardiac events. (This abstract does not reflect EPA policy)

PL 862 CHARACTERIZATION OF SOY BIODIESEL EXHAUST AND TOXICOLOGICAL EFFECTS IN MICE.

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Although biofuel use across the world is increasing, very little is known about possible health effects resulting from biofuel exhaust (BE) from this relatively new source of transportation fuel. The U.S. EPA has instigated an *in vivo* screening approach in rodents to examine whether BE can induce lung and cardiopulmonary responses in healthy and compromised animal models. In our combustion facility, biofuel exhaust is generated by a 0.32 L Yanmar engine driving a 3.8 kW Pramac generator with a constant load of 3 kW. Initial studies tested soy-based biodiesel, either 100% (S100) or a 20% mix with conventional petrodiesel (S20). Organic solvent extracts of S100 fuel were composed of about 70% methyl esters (e.g. linoleate, oleate) and 15% organic acids (e.g. hexadecanoic). Exhaust from combustion of S100 or S20 was diluted to target concentrations of 0, 50, 150, or 500 $\mu g/m^3$ as determined by TEOM. Average CO, NO (ppm) at the 500 $\mu g/m^3$ level were 12.3, 18.7 (S100) and 13.9, 13.2 (S20), respectively, while SO_2 and NO_2 were not above instrument background (<1 ppm). Female Balb/cJ mice (8/group) were exposed whole body to these emissions 4 hr/d, for 1 d, 5 d, or 4 wk (5 d/wk), and necropsied 2 or 24 hr after each of these exposures. Lung inflammation was minimal as neutrophils in BAL fluid were <3% with both S20 and S100 at all concentrations and time points. However, significantly fewer BAL macrophages (62% of control) were found 2 hr after 5 d exposure to 500 $\mu g/m^3$ S20. At the same time point, MIP-2α levels in BAL fluid were significantly higher in mice exposed to 500 $\mu g/m^3$ S100 (mean ± SE: 52 ± 5 vs. 12 ± 6 pg/ml at 0 level). No significant changes in BAL protein, LDH, albumin, or NAG were found. Future studies comparing these effects of soy BE with those generated by exposure to exhaust from other biodiesel preparations or pure petrodiesel will enable a thorough understanding of the toxicology of BE. (This abstract does not represent U.S. EPA policy.)

PL 863 HYPOTENSIVE AND BRADYCARDIC RESPONSES TO INHALED O3 AND AMBIENT FINE PARTICLES ARE ENHANCED IN RATS ON A HIGH-FRUCTOSE DIET.

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People with diet-induced cardiometabolic disorders (diabetes, hypertension) may be more susceptible to the adverse cardiovascular effects of air pollution. The present study was designed to determine if a high-fructose diet affects cardiovascular responses to inhaled air pollutants. We used a mobile air research laboratory located in an industrial area of Dearborn, MI, to expose male Sprague Dawley rats, to filtered air (FA), or the combination of ozone (O3; 0.5 ppm) and concentrated ambient fine particles (CAPs; 400 μg/m3). Rats were fed a normal (ND) or high-fructose diet (HFD) for 8 weeks prior to exposure and during exposure. Inhalation exposures were 8h/day for 9 days (4-5 days/week). Heart rate (HR), heart rate variability (standard deviation; SDNN), and diastolic, systolic and mean arterial blood pressures were collected by radiotelemetry every 5 minutes during exposures. FA-exposed rats fed a HFD had elevated systolic and mean arterial pressures compared to ND rats (10 and 9% greater, respectively). HR was not different between FA-exposed ND and HFD rats. Coexposure to O3/CAPs caused hypotension regardless of diet. O3/CAPs-induced decreases in systolic and mean pressures (decreases of 2 and 3%, respectively) were modest in ND rats compared to HFD rats (6 and 5%). O3/CAPs also induced marked bradycardia in both ND and HD rats. Decreases in HR were greater in HFD (7%) compared to ND-fed rats (3%). Furthermore, SDNN was increased in HFD, but not in ND rats exposed to O3/CAPs. Observed hypotensive and bradycardic responses to O3/CAPs are consistent with a nasal irritant-induced trigeminocardiac reflex. Mechanisms underlying HFD modification of these cardiovascular changes are yet to be determined. Funded by US EPA RD83479701.

PL 864 TOXICITY OF DAY/NIGHT URBAN SAN JOAQUIN VALLEY, CALIFORNIA PARTICULATE MATTER IS SIZE AND SEASON DEPENDENT.

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There is strong evidence for an association between exposure to ambient particulate matter (PM) and adverse cardiopulmonary health effects both nationally and in the highly impacted San Joaquin Valley (SJV) of California. However, the physical and chemical characteristics of PM and the mechanisms responsible for increased morbidity and mortality remains unclear. Diurnal and temporal variability in PM sources and atmospheric processes may influence PM composition and therefore toxicity. We hypothesized that seasonal and diurnal differences in source contributions and photochemical activity would influence the cardiopulmonary toxicity of SJV ultrafine (UF, mean mass aerodynamic diameter (MMAD) < 170nm) and submicron fine (SMF, MMAD < 1μm) PM. Acute cardiopulmonary inflammatory responses were measured in the lungs and blood of healthy male Balb/C mice following oropharyngeal aspiration of 50 μg of summer/winter, UF/SMF, and day/night PM in 50 μl Hank's balanced salt solution (HBSS) or HBSS alone. Results indicate that PM toxicity was significantly dependent on particle size and season and was not influenced by diurnal trends. In general, UF PM elicited significantly more pulmonary inflammation compared to SMF PM regardless of season or time of day. PM size-dependent toxicity demonstrated a more pronounced difference between winter UF and SMF PM compared to summer UF and SMF PM. In contrast, circulating neutrophils in peripheral blood were higher in mice exposed to summer but not winter day UF compared to SMF PM. These findings suggest that particle chemical composition strongly influences PM toxicity, possibly due to seasonal PM sources prevalent in winter compared to summer rather than diurnal impacts. Understanding seasonal impacts on PM-induced adverse respiratory and systemic health effects can lead to more effective targeting of sources for improved control of PM pollution within the SJV and protection of public health.

PL 865 AIRWAY SENSORY IRRITATION STUDY OF BORIC ACID AND SODIUM TETRABORATE PENTAHYDRATE IN MICE.

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The objective of this study was to evaluate the potential for boric acid (BA) and sodium tetraborate pentahydrate (SB) to produce respiratory rate depression resulting from airway sensory irritation when administered to male Swiss-Webster mice by head-only inhalation exposure. The respiratory depression of BA, SB, and calcium oxide (CaO) was evaluated in a 30-minute exposure study in mice (ASTM E981-04). BA was administered to 12 groups (6 exposure levels) of 4 male mice via head-only inhalation exposure as a dust aerosol at concentrations ranging from 221 to 1174 mg/m3 (maximum achievable concentration). SB was administered to 8 groups (6 exposure levels) at concentrations ranging from 186 to 1704 mg/m3 (maximum achievable concentration). CaO was administered to 5 groups (4 exposure levels) at concentrations ranging from 3.5 to 30 mg/m3. Exposure to a mean BA concentration of 1096 mg/m3 resulted in a 19% reduction in respiratory rate (RR), graded as slight irritation. A 9% reduction in RR was recorded at a concentration of 221 mg/m3, graded as no irritation. A maximum SB exposure concentration of 1704 mg/m3 resulted in a 33% reduction in RR, graded as moderate irritation. The lowest concentration of 186 mg/m3 resulted in a RR reduction of 11%, graded as no irritation. It was not possible to produce a 50% respiratory depression at the concentrations of BA and SB that were achievable. The response to CaO aerosol in mice appeared to represent pulmonary irritation rather than upper airway sensory irritation. The standard method for the mouse sensory irritation test has

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