CORE 7. VASCULAR DISEASE: BIOLOGY AND CLINICAL SCIENCE
SESSION TITLE: HYPERTENSION: CELLULAR MECHANISMS OF HYPERTENSION

Abstract 17098: Blood Pressure Response to Controlled Diesel Exhaust Inhalation in Human Subjects is Modified by Functional Variation in TRPV1

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Abstract

Background: Exposure to traffic-related air pollution is associated with increased risk of cardiovascular disease and mortality. Diesel exhaust (DE) inhalation is a model exposure, and we hypothesize that pollutants trigger acute cardiovascular events via neurogenic processes. We evaluated blood pressure (BP) response to DE and whether the effect was modified by genotype for the transient receptor potential receptor, vanilloid type 1 (TRPV1), a non-selective cation channel expressed in the lung and activated by noxious chemical stimuli. The t allele for the TRPV1 single nucleotide polymorphism rs8065080 at position 585 results in Ile substituting for Val, and has been associated with reduced nociception.

Methods: Forty-seven non-smokers, age 18-49 with no prior cardiovascular or pulmonary disease, underwent controlled exposures to each condition [DE at 200 μ g/m^3 $PM_{2.5}$ and filtered air

(FA)] for 120 minutes in a crossover experiment. Exposures were double-blind, randomized to order, and separated by at least 2 weeks. We measured BP pre-exposure, at 5 time points during exposure, and 3, 5, 7 and 24 hours from exposure start. We analyzed pressure change (from pre-exposure values) for each exposure to determine the difference between DE and FA.

Results: Compared with FA, systolic blood pressure (SBP) increased at all time points measured during and after DE exposure (p=0.004); mean effect peaked 30 to 90 minutes after exposure began (3.9 mmHg, p=0.01). No effects were observed for heart rate or diastolic BP. SBP response varied by *TRPV1* Ile585Val genotype, with t/t subjects showing the lowest SBP response to DE (see Figure).

Conclusion: DE exposure was associated with a rapid increase in SBP, with a reduced response in subjects with the Ile-Ile variation of TRPV1. This suggests that TRPV1 activation by a component of DE may be a key step explaining observed vascular effects of air pollution, perhaps by initiating autonomic signaling or neurogenic inflammation.

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