Effect of Particulate Matter Exposure and Chronic Cerebral Hypoperfusion on Vascular Inflammation

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No Disclosures
Introduction

• Chronic cerebral hypoperfusion (CCH) is a mediator of vascular disease and is associated with inflammation, oxidative stress, and blood-brain barrier breakdown

• Nano-size particulate matter (nPM) from vehicular exhaust is a potent environmental source of inflammation

• The inflammatory effects of nPM are poorly understood

• The aim of this study is to examine the joint effects of nPM exposure and CCH secondary to bilateral carotid artery stenosis (BCAS) on inflammation in the common carotid arteries of mice.
Methods

• nPM was collected from central Los Angeles, transferred to an aqueous suspension, and re-aerosolized for animal exposure

• Mice were exposed to filtered air or nPM for five hours/day, three-days/week for 10 weeks. 30 days before the end of the nPM exposure, mice underwent either BCAS (microcoil placement on bilateral carotid arteries) or sham surgery

• There were four experimental cohorts: Filter+Sham (n=5), nPM+Sham (n=7), Filter+BCAS (n=5), and nPM+BCAS (n=6)

• Animals were sacrificed, and common carotid arteries were extracted, frozen, cut axially at 20um, and mounted on slides. Immunohistochemical analysis for Intercellular Adhesion Molecule 1 (ICAM-1), an endothelial surface adhesion molecule, was performed.

• Mean ICAM-1 values were computed for each carotid arteries and group averages were compared between the four groups.
nPM and CCH results in activation of endothelial intercellular adhesion molecules 1 (ICAM-1) on endothelial cells of carotid arteries.
Results

• ICAM-1 semiquantitative immunohistochemical analysis demonstrated a significant difference between the 4 groups ANOVA F(3,19)=1.22, p<0.0001.

• On Fisher’s Least Significant Difference (LSD) post hoc analysis, mice exposed to nPM+BCAS had a 70% increase in endothelial ICAM-1 expression compared to nPM (p<0.0001) and a 45% increase in endothelial ICAM-1 expression compared to Filter+BCAS (p<0.003)
Conclusion

• Increased ICAM-1 concentrations on the common carotid arteries of mice exposed to nPM+CCH indicates an inflammatory upregulation of adhesion molecules

• The inflammatory response associated with nPM exposure is exacerbated in the setting of experimental bilateral carotid artery stenosis
Summary Points

• Joint exposure to nano Particulate Matter and Chronic Cerebral Hypopoesfusion results in activation of endothelial intercellular adhesion molecules 1 (ICAM-1) on endothelial cells of carotid arteries

• The inflammatory response associated with nano Particulate Matter exposure is exacerbated in the setting of experimental bilateral carotid artery stenosis