1546409

Pulmonary Exposure to Fine Particulate Matter From Oil Combustion Power Plants Decreases Myocardial Ischemic Tolerance in Rats

Golomb, E[1]; Korach, A[2]; Schneider, A[2]; Houminer, E[2]; Nyska, A[3]; Kodavanti, U[4]; Schwalb, H[2]; Shapira, O[2]

[1]Shaare Zedek, Jerusalem, Israel; [2]Hadassah, Jerusalem, Israel; [3]Tel Aviv University, Tel Aviv, Israel; [4]US Enviromental Protection Agency, Reseach Triange, USA

Background: Short term exposure to respirable ambient particulate matter (PM) triggers ischemic cardiac events (Pope et al., Circulation 2006: 114: 2443). This has been associated with oxidative stress, inflammation, and acceleration of atherosclerosis in compromised mouse models. An alternative / additional mechanism could involve an adverse effect of PM on cardiomyocyte ischemic tolerance.

Aim: To assess ischemic tolerance of rat heart following pulmonary exposure to oil combustion source PM enriched with vanadium.

Methods: Healthy male SD rats (300±20 g) were exposed to a single intratracheal instillation of saline or different doses of soluble vanadium-rich oil combustion fly ash PM, collected from a power plant in Boston, MA (HP-10) containing: carbon based particles, sulfate, iron, nickel, and vanadium. Myocardial slices were exposed in vitro to simulated ischemia/reoxygenation 48h later. Mitochondrial function (mitochondrial dehydrogenases redox activity) in these slices was assessed by the MTT colorimetric assay.

Results: Rats exposed to the PM, at 2.5 mg/kg, did not exhibit toxic effect by histological assessment, and had no effect on dehydrogenase activity in oxygenated conditions. However, simulated ischemia/reoxygenation, significantly decreased dehydrogenases activity by 29%, compared to controls ($p<0.05$). A lower dose of HP-10 (0.5 mg/kg) cause no effect on the MTT assay in either condition.

Conclusions: Recovery of cardiac ischemic events depends not only on the extent and duration of the ischemic stimulus, but also on the myocardial intrinsic ischemic tolerance. Thus, inhaled PM may cause occult cardiotoxicity, reflected by normal histology and mitochondrial function during normoxia, but an impaired response to ischemia / reoxygenation.