OP45-1 Heat Shock Protein, αB Crystallin, Can Prevent the Arrhythmogenic Effect of Ambient Particulate Matter by Attenuating the Repolarization Gradient and Triggered Activity

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Introduction: Ambient particulate matter (PM) can increase cardiac arrhythmias via oxidative stress. This study was performed to reveal that the heat shock protein, αB-crystallin, could prevent the arrhythmogenic effect of PM.

Methods: Using optical mapping, arrhythmic mechanism were evaluated in Langendorff-perfused Adult Sprague-Dawley rat heart after infusion of diesel exhaust product (DEP) and αB-crystallin.

Results: In neonatal rat cardiomyocytes, DEP provoked cell death and ROS generation in dose dependent manner. In Langendorff-perfused rat hearts, DEP infusion of 12.5 mg/L (n=12) prolonged action potential duration (APD90) at only the base of left ventricle (LV) from 101±14 ms to 152±22 ms (p=0.001) increasing apicobasal APD differences from 4±8 ms to 54±25 ms (p=0.003). Pretreatment of αB-crystallin (1 mg/kg, n=9) with a delivery system (TAT-protein transduction domain) prevented the DEP-induced APD prolongation (106±10 ms, p=0.83) and apicobasal APD differences (1.5±1.5 ms, p=0.83). Compared with DEP alone, pretreatment of αB-crystallin decreased triggered activity (67% vs. 11%, p=0.02) and apicobasal reentry (82% vs. 6%, p=0.001). The ventricular tachyarrhythmia (VT) was observed in 9 (75%) and 4 (44%) hearts after DEP alone and pretreatment of αB-crystallin (p=0.04), respectively.

Conclusions: Heat shock protein, αB-crystallin, can prevent arrhythmogenic effect of PM by suppressing apicobasal repolarization gradient and triggered activity.

Keywords: particulate matter, heat shock protein, oxidative stress