Abstract

Background Studies have shown a relationship between air pollution and increased risk of cardiovascular morbidity and mortality. Due to the complexity of ambient air pollution composition, recent studies have examined the effects of co-exposure, particularly particulate matter (PM) and gas, to determine whether pollutant interactions alter (e.g. synergistically, antagonistically) the health response. This study examines the independent effects of fine (FCAPs) and ultrafine (UFCAPs) concentrated ambient particles on cardiac function, and determine the impact of ozone (O3) co-exposure on the response. We hypothesized that UFCAPs would cause greater decrement in mechanical function and electrical dysfunction than FCAPs, and that O3 co-exposure would enhance the effects of both particle-types.

Methods Conscious/unrestrained radiotelemetered mice were exposed once whole-body to either 190 μg/m3 FCAPs or 140 μg/m3 UFCAPs with/without 0.3 ppm O3; separate groups were exposed to either filtered air (FA) or O3 alone. Heart rate (HR) and electrocardiogram (ECG) were recorded continuously before, during and after exposure, and cardiac mechanical function was assessed using a Langendorff perfusion preparation 24 hrs post-exposure.

Results FCAPs alone caused a significant decrease in baseline left ventricular developed pressure (LVDP) and contractility, whereas UFCAPs did not; neither FCAPs nor UFCAPs alone caused any ECG changes. O3 co-exposure with FCAPs caused a significant decrease in heart rate variability when compared to FA but also blocked the decrement in cardiac function. On the other hand, O3 co-exposure with UFCAPs significantly increased QRS-interval, QTc and non-conducted P-wave arrhythmias, and decreased LVDP, rate of contractility and relaxation when compared to controls.

Conclusions These data suggest that particle size and gaseous interactions may play a role in cardiac function decrements one day after exposure. Although FCAPs+O3 only altered autonomic balance, UFCAPs+O3 appeared to be more serious by increasing cardiac arrhythmias and causing mechanical decrements. As such, O3 appears to interact differently with FCAPs and UFCAPs, resulting in varied cardiac changes, which suggests that the cardiovascular effects of particle-gas co-exposures are not simply additive or even generalizable. Additionally, the mode of toxicity underlying this effect may be subtle given none of the exposures described here impaired post-ischemia recovery.