

Abstract

Short-term exposure to air pollutants has been linked to acute cardiovascular morbidity and mortality. Even in the absence of overt signs or symptoms, pollutants can cause subtle disruptions to internal compensatory mechanisms, which maintain homeostatic balance in response to various environmental and physiological stressors. We hypothesized that a single exposure to acrolein, a ubiquitous gaseous air pollutant, would decrease the sensitivity of baroreflex (BRS), which maintains blood pressure by altering heart rate (HR), modify cardiac electrophysiological properties and increase arrhythmia in rats. Wistar-Kyoto normotensive (WKY) and spontaneously hypertensive (SH) rats implanted with radiotelemeters and a chronic jugular vein catheter were tested for BRS using phenylephrine and sodium nitroprusside 2 days before and 1 h after whole-body exposure to 3 ppm acrolein (3 h). HR and electrocardiogram (ECG) were continuously monitored for the detection of arrhythmia in the pre-exposure, exposure and post-exposure periods. Whole-body plethysmography was used to continuously monitor ventilation in conscious animals. SH rats had higher blood pressure, lower BRS and increased frequency of AV block as evidence by non-conducted p-waves when compared with WKY rats. A single exposure to acrolein caused a decrease in BRS and increased incidence of arrhythmia in both WKY and SH rats. There were minimal ECG differences between the strains, whereas only SH rats experienced irregular breathing during acrolein. These results demonstrate that acrolein causes immediate cardiovascular reflexive dysfunction and persistent arrhythmia in both normal and hypertensive animals. As such, homeostatic imbalance may be one mechanism by which air pollution increases risk 24 h after exposure, particularly in people with underlying cardiovascular disease.

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