Effects of concentrated ambient particles on heart rate, blood pressure, and cardiac contractility in spontaneously hypertensive rats.

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摘要

Abstract

Epidemiological studies have shown that particulate matter (PM) air pollution is associated with cardiovascular mortality and morbidity, especially for particles with aerodynamic diameters under 2.5 microm (PM(2.5)). Recent studies have revealed an association between PM pollution and autonomic functions including heart rate (HR), blood pressure (BP), and heart-rate variability. However, the association and linking mechanisms have not been clearly demonstrated in animal studies. Utilizing a novel approach that employs a mixed-effects model to overcome the problems of variations in diseased animals and circadian cycles, we have previously demonstrated an association between concentrated PM(2.5) and changes of HR and BP in pulmonary hypertensive rats. The objective of this study is to test the plausibility of this methodology and to demonstrate the particle effects under different pathophysiology. The feasibility of cardiac contractility (measured as QA interval, QAI) as an indicator for PM toxicology was also explored. Four spontaneously hypertensive (SH) rats were repeatedly exposed to concentrated PM(2.5) during spring and summer. The mass concentration of particles during the 5 h of exposure was 202.0 +/- 68.8 (mean +/- SE) and 141.0 +/- 54.9 microg/m(3) for spring and summer experiments, respectively. During spring exposures, the maximum increase of HR and mean BP noted at the end of exposure were 51.6 bpm (p <.001) and 8.7 mm Hg (p =.002), respectively. The maximum decrease of QAI noted at the same time was 1.6 ms (p =.001). Though a similar pattern was demonstrated during summer exposures, the responses were less prominent. We conclude that concentrated PM(2.5) may increase HR and mean BP and decrease QAI in SH rats. Our results also show that QAI may be used as an indicator in PM toxicology.