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Specific metals in PM2.5 associated with HRV in the elderly with coronary heart disease: a community-based panel study

Exposure to PM2.5 is linked with cardiovascular mortality and morbidity, and the underlying biological mechanisms considering metals have raised increasingly concern. It has been an inevitable issue to assess associations of specific metal constituents with risk for cardiovascular health. To evaluate the exposure to specific chemicals of PM2.5 from various sources and their cardiac effects, a panel study was conducted with four repeated measurements of elderly participants' HRV and PM2.5 species in urban Beijing. Multiple metals and other PM2.5 bound chemicals were characterized for PM2.5 source apportionment and personalized exposure assessment. Five sources were identified with specific chemicals as the indicators: oil combustion (1.1%, V & PAHs), secondary particle (11.3%, nitrate & sulfate), vehicle emission (1.2%, Pd), construction dust (28.7%, Mg & Ca), and coal combustion (57.7%, As & Se). In addition, multiple metals including Pb, Ni, Zn, Cu, Cd, Cr, and Sb presented relatively high loadings in relevant sources. As observed, each IQR increases in exposure to PM2.5 significantly decreased rMSSD by 11.1% (95% CI: -19.6%, -1.9%) at lag 0 and 14.3% (95% CI: -26.2%, -0.21%) at lag 3. There were a 1.26-fold to 2.51-fold decrease in rMSSD with elevated exposure to Pb, Ni, Zn, Cu, Cd, Cr, and Sb at lag 0 compared to those of PM2.5 mass. While those of vehicle emission (Pd) and coal combustion (Se) had a 2.47-fold and 2.23-fold at lag 0 respectively, and those of oil combustion (V) with 1.82fold at lag 3 compared to those of PM2.5 mass. Increased exposures to specific sources/constituents of PM2.5 disrupt cardiac autonomic function in elderly and have a stronger adverse effect than PM2.5 mass. In the stratified analysis, medication and gender modify the associations of specific chemicals from variable sources with HRV. The findings of this study provide evidence on the roles of influential metals of ambient air PM2.5 and their sources in terms of their adverse cardiovascular health effects.

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