Background and Aims: An increased pulse pressure (Δ systolic – diastolic pressure) suggests aortic stiffening. New evidence also suggests that pulse pressure is a more sensitive measure of risk than other indexes of blood pressure in middle-aged and older persons. The objective of this study was to examine the acute effects of both particulate matter (PM) mass and composition on systolic blood pressure, diastolic blood pressure and pulse pressure, among a more susceptible population of elderly persons.

Methods: During the period June 2007 until October 2010, we carried out a panel study in persons living in elderly homes located in the province of Antwerp, Belgium. Blood pressure was measured and a blood sample was collected on two time points. Exposure to in- and outdoor 24 hour mean PM$_{2.5}$ mass concentrations was determined. The elemental content of the collected in- and outdoor PM$_{2.5}$ was measured. Oxygenated polyaromatic hydrocarbons (oxy-PAHs) sorbed onto outdoor PM$_{10}$ were measured.

Results: We recruited 84 persons, 70 % women with a mean age of 83 years (SD: 5.2). Each interquartile range (IQR) increase of 21.1 µg/m³ in 24 hour mean outdoor PM$_{2.5}$ was associated with an increase in systolic blood pressure of 3.9 mmHg (95%CI: 1.6 to 6.2) and in pulse pressure of 4.1 mmHg (95% CI: 1.8 to 6.5), in persons taking antihypertensive medication (n=54), but not in persons not using antihypertensive medication (p for interaction: 0.02). Vanadium, iron and nickel content of PM$_{2.5}$ were significantly associated with systolic blood pressure and pulse pressure, among persons on antihypertensive medication. Similar results were found for indoor concentrations. Of the oxy-PAHs, chrysene-5,6-dione, benzo[a]pyrene-3,6 and benzo[a]pyrene-6,12 were significantly associated with increases in systolic blood pressure and pulse pressure.

Conclusions: In elderly, pulse pressure was positively associated with acute increases in outdoor and indoor air pollution, among persons taking antihypertensive medication. These results might form a mechanistic pathway linking air pollution as a trigger of cardiovascular events.