# Passive smoking increases platelet activation in healthy people - ESC Press Release - ESC Congress 2012

**Passive smoking increases platelet activation in healthy people, according to research presented at the ESC Congress 2012 today by Dr Mehmet G. Kaya. The findings suggest that increased platelet activation could be the mechanism by which passive smoking increases the risk of thrombotic events in healthy people.**

“It is well known that passive smoking is harmful for cardiovascular health, but the mechanism has not yet been discovered,” said Dr Kaya. “We investigated the effects of passive smoking on the levels of three parameters – mean platelet volume (MPV), carboxyhemoglobin (COHb) and lactate - in an effort to further understand this mechanism. We also looked at the correlation between the three parameters.”

Mean platelet volume (MPV) is a well established indicator of platelet activation that is increased in acute thrombotic events. It is suspected that carbon monoxide (CO) plays a major role in cigarette smoke-induced cardiovascular diseases. When CO binds with hemoglobin (Hb) in red blood cells it is called COHb. Lactate accumulates in the blood when the supply of oxygen to blood cells is limited.

For the study, 55 healthy nonsmoker volunteers (males 30%; mean age 26±5 years) were prospectively enrolled. Blood samples for measurements of MPV, COHb and lactate were taken at baseline and after spending one an hour in a smoking room.

The blood levels of all three parameters were statistically higher after subjects were exposed to passive smoking (see figure 1). COHb increased from 0.8±0.3% at baseline to 1.2±0.4% after one hour in a smoking room (p=0.001). Lactate increased from 0.70±0.2mmol/L to 2.2±0.9mmol/L (p=0.001). MPV increased from 7.8±0.4 femtolitres (fL) to 8.5±0.6fL (p= 0.001). Dr Kaya said: “These results show that passive smoking increases platelet activation and increases CO and lactate levels in the blood.”

The researchers found significant correlations between MPV and COHb levels (r= 0.55, p=0.0001) and between MPV and lactate levels (r= 0.65, p=0.0001) after smoking. There was also a remarkable relation between COHb and lactate levels after smoking (r= 0.78, p=0.0001). Dr Kaya said: “Previous studies have suggested that the chemicals in cigarette smoke, especially nicotine and CO, increase platelet-activating factor. The correlations found in our study suggest that the CO in cigarette smoke also increases MPV levels. It is likely that lactate levels increased because oxygen levels in the blood dropped as CO increased.”

He concluded: “We have shown that 1 hour exposure to passive smoking increases platelet activation, which could be the mechanism by which it contributes to increased risk of thrombotic events in healthy people. It is likely that prolonged exposure to passive smoking could have even greater effects. Healthy people should avoid exposure to passive smoking so that they do not increase their risk of thrombotic events.”

ENDS

Notes to editors

The authors received no financial support for the research, authorship, and/or publication of this article.

About the European Society of Cardiology www.escardio.org
The European Society of Cardiology (ESC) represents more than 75,000 cardiology professionals across Europe and the Mediterranean. Its mission is to reduce the burden of cardiovascular disease in Europe.

About ESC Congress 2012
The ESC Congress is currently the world's premier conference on the science, management and prevention of cardiovascular disease. ESC Congress 2012 takes place 25-29 August at the Messe München in Munich. The scientific programme is available here.
More information is available from the ESC Press Office at press@escardio.org.

**Authors**

ESC Press Office
ESC Congress 2012
press@escardio.org
33(0)4 92 94 86 27
European Society of Cardiology

**About the European Society of Cardiology**

The European Society of Cardiology (ESC) represents 75,000 cardiology professionals across Europe and the Mediterranean. Its mission is to reduce the burden of cardiovascular disease in Europe.