

Short-term smoking cessation reverses endothelial damage

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Eight weeks of smoking cessation reverses the endothelial damage caused by smoking, according to research presented at the ESC Congress today by Dr. Yasuaki Dohi from Japan. Serotonin remained elevated, suggesting eight weeks of cessation is insufficient to reverse the risk of myocardial infarction.

Dr Dohi said: "Smokers are twice as likely to have a heart attack as people who have never smoked. Quitting smoking is the most important thing people can do to reduce their risk of cardiovascular disease. But until now, studies have not examined whether the increased risk caused by smoking is completely reversed after smoking cessation."

The current study investigated how the [vascular system](#) is altered by smoking and whether the changes can be normalised by smoking cessation. The researchers focused on the effects of smoking and smoking cessation on arterial [endothelial function](#) and circulating serotonin concentration.

Both [endothelial dysfunction](#) and serotonin contribute to the development of atherosclerosis. Serotonin released from platelets induces platelet aggregation, which initiates blood coagulation and contractions in arteries especially those with damaged endothelium.

Smoke from cigarettes contains [toxic molecules](#) including nicotine, carbon monoxide and hydrogen cyanide which may cause and promote atherosclerosis via endothelial dysfunction and increased activity of [blood coagulation](#).

The study included 27 apparently healthy [male smokers](#) aged 40±8 years and 21 age-adjusted non-smokers (40±7 years). Endothelial function was assessed by flow mediated dilation and peripheral arterial tonometry (PAT). Both methods assess endothelial function as the ability to dilate arteries through the release of endothelium-derived relaxing factors.

Dr Dohi said: "As expected, smoking damaged arterial endothelial function and increased plasma serotonin levels."

Only 21 subjects agreed to stop smoking for 8 weeks. Smoking cessation was confirmed in 11 out of the 21 subjects by measuring serum levels of cotinine, the principal metabolite of nicotine. Smokers who completely attained smoking cessation had a significantly increased PAT ratio (P

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