



### **Niacin impacts favourably on HDL properties in metabolic syndrome**

Data presented at the Scientific Sessions, American Heart Association Annual Meeting 2007 show that extended-release (ER) niacin has a beneficial effect on HDL vasoprotective qualities in people with the metabolic syndrome.<sup>1</sup>

Recent data suggest that HDL may become functionally defective in people with the metabolic syndrome. This may result in impairment of various atheroprotective properties of HDL.

In this study, researchers investigated the effect of ER niacin on endothelial function, re-endothelialisation capacity by endothelial progenitor cells (EPCs), and the functional capacity of HDL in 30 patients with the metabolic syndrome. Patients were treated with ER niacin 1500 mg/day or placebo for 3 months.

The researchers used a mouse model of carotid injury to assess the re-endothelialisation capacity of EPCs obtained from patients. Endothelial function was assessed by flow-dependent, endothelium-mediated vasodilation measured by ultrasound. HDL was isolated before and after treatment. The capacity of HDL to stimulate endothelial nitric oxide production and reduce superoxide formation was assessed by electron spin resonance spectroscopy.

Treatment with ER niacin significantly raised levels of HDL cholesterol by 16% ( $p < 0.01$ ), whereas there was no change with placebo.

ER niacin treatment improved flow-dependent, endothelium-mediated vasodilation (by 47%,  $p < 0.01$  vs. baseline) and markedly increased the re-endothelialisation capacity of EPCs (by 34%,  $p < 0.001$  vs. placebo). HDL isolated after treatment with ER niacin stimulated endothelial cell nitric oxide production and reduced superoxide formation.

The researchers concluded that treatment with ER niacin improves vasoprotective functions of HDL in patients with metabolic syndrome.

#### **Reference**

1. Sorrentino SA, Besler C, Bahlmann FH et al. Extended-release niacin improves endothelial function, restores re-endothelialization capacity of endothelial progenitor cells and augments vasoprotective properties of HDL in patients with metabolic syndrome. *Circulation* 2007;116:II\_16. Abstract 189.