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INHIBITION OF ENDOTHELIAL MICROPARTICLES-MEDIATED OXIDATIVE STRESS BY BERBERINE AND ITS RELATION WITH ENDOTHELIAL FUNCTION IN HUMANS

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Background Circulating endothelial microparticles (EMPs) lead to impaired endothelial vasodilation accompanied by increased oxidative stress. The authors previously demonstrated that berberine protects endothelial function which is

associated with decreased circulating EMPs, but the underlying mechanisms are not clear up to date. Here, the authors investigate the effect of berberine on endothelial microparticles-mediated oxidative stress and its relation with endothelial function in humans.

Methods Twelve healthy subjects received 1-month berberine therapy and 11 healthy subjects served as control. Endothelium function was assessed by flow-mediated vasodilation (FMD) and sublingual nitroglyceride-mediated vasodilation (NMD), as well as circulating EMPs and serum malondialdehyde (MDA) were measured before and after therapy, respectively. In vitro, human umbilical vein endothelial cells (HUVECs) were stimulated by EMPs with or without presence of antioxidant compound apocynin, and berberine. Intracellular reactive oxygen species (ROS), nitric oxide (NO) production and NADPH oxidase 4 (Nox4) protein expressions were detected, respectively.

Results Decreased serum MDA, circulating CD31+/CD42-MPs and improved endothelium-dependent vasodilation are observed in the berberine group but not in the control group. The EMPs in vitro led to increased ROS production and Nox4 protein expression in HUVECs associated with reduced NO synthesis, which was reversed by the presence of apocynin or berberine, respectively.

Conclusions The results show that berberine-induced decline in oxidative stress by circulating CD31+/CD42- microparticles contributes to amelioration of endothelial function in healthy subjects.