

Publication

Exercise-induced alterations of retinal vessel diameters and cardiovascular risk reduction in obesity

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BACKGROUND: The retinal microcirculation is affected early in the process of atherosclerosis and retinal vessel caliber is an emerging cardiovascular risk factor. Obesity is associated with vascular dysfunction. Here, we investigate the effect of regular exercise on retinal vessel diameters in lean and obese runners. We analyze a possible link to alterations of the nitric oxide (NO)-asymmetric dimethylarginine (ADMA) pathway. METHODS: Retinal vessel diameters were assessed by means of a static vessel analyzer (SVA-T) in 15 obese athletes (OA), 14 lean amateur athletes (AA) and 17 lean elite athletes (EA) following a 10 week training program. ADMA serum levels were detected by ELISA and dimethylarginine dimethylaminohydrolase (DDAH) -1/-2 mRNA-expression in peripheral mononuclear cells (PBMC) was analyzed by real time PCR. RESULTS: At baseline, the mean (\$SD) arteriolar to venular diameter ratio (AVR) was impaired in obese (OA: 0.81ś0.05) compared to lean subjects (AA: 0.87ś0.07; EA: 0.94ś0.05). The individual fitness levels correlated with AVR (rho=+0.66; P<0.001) and the training program improved AVR in all groups (P<0.001), normalising AVR in the obese (OA: 0.86\$0.1). A training-induced arteriolar dilatation was found in OA (P=0.01), which was accompanied by a significant decrease of ADMA levels (0.56ś0.12-0.46ś0.12 µmoll(-1); P<0.028). DDAH-1 mRNA levels in PBMC increased in all groups (P<0.01). CONCLUSIONS: Cardiovascular fitness and body composition affect retinal vessel diameters. Regular exercise reverses the subclinical impairment of the retinal microvasculature in obesity by inducing retinal arteriolar dilatation. The NO/ADMA pathway may play a key role in the training-induced improvement of microvascular function, which has the potential to counteract progression of small vessel disease.

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