Endothelial progenitor cells (EPCs) have been shown to significantly contribute to neovascularization and have emerged as a novel risk factor for cardiovascular disease (CVD) because of their role in maintaining endothelial function. Reactive Oxygen Species (ROS) are small molecules or ions that oxidize other cells and cause cellular damage. This is particularly important in the cells of the vascular walls as this oxidative stress to endothelial cells contributes to endothelial dysfunction and progression of CVD. Antioxidant enzymes present within the cell are capable of slowing or preventing the oxidation of other molecules. Studies have shown that there is an increase in CFU-EC after a bout of endurance exercise in both active and sedentary individuals, but the effects of acute and chronic exercise on the redox state of these putative EPCs have not been studied. This study tested whether (i) there would be increased expression of antioxidant genes and a decreased expression of ROS-producing genes in EPCs of active compared to inactive young men, and (ii) an acute bout of exercise altered expression of these genes.

**Hypothesis**

It was hypothesized that chronic and acute endurance exercise would favorably affect the expression of genes involved in cellular oxidative stress in EPC colony forming units (CFU-EC).

**Methods**

- Groups consisted of healthy young men and were matched for age, BMI, body composition, and the standard CV risk factor profile. Active individuals reported performing more than 4h/week endurance exercise, whereas inactive individuals reported less than 20 min/d on less than 2 d/week endurance exercise.
- CFU-EC were cultured from blood drawn before (baseline) and after 30 min of exercise at 75% of maximal oxygen uptake in active (n = 8) and inactive (n = 8) men.
- Total RNA was isolated from CFU-EC after 5 days in culture. mRNA for antioxidant (PPAR-delta, MnSOD, Gpx1, CuZnSOD) genes and pro-oxidant (Xanthine Oxidase) gene were assessed using semiquantitative RT-PCR. Data are expressed as fold-difference from baseline values for the active group.

**Results**

The strongest differences between groups of young men at baseline were observed for PPAR-delta (40% higher in the inactive group) and MnSOD (21% higher in the inactive group). The strongest differences after exercise were in PPAR-delta (10-20% increase from baseline in both groups) and Xanthine Oxidase (40% increase in the inactive group, and 28% decrease in the active group).

**Discussion**

(i) Expression of antioxidant genes were expected to increase after acute exercise in both active and inactive groups; instead data indicate that levels only increased in the inactive group, whereas antioxidant expression unexpectedly fluctuated within the active group.

(ii) As expected, expression of XO decreased as a result of acute exercise in active individuals. However, expression within the inactive group rose, possibly implicating even higher oxidative stress after exercise.

Overall, our data suggest that the acute effect of endurance exercise on oxidative stress-related genes may be modified by training status.

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