Aerobic training workload affects human endothelial cells redox

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Moderate aerobic exercise reduces oxidative stress, intense physical activity may produce the opposite result. At present, the effects of different exercise loads on oxidative stress markers and the response of human cells to different exercise volumes have not been fully elucidated. In this research human (Eahy-926) endothelial cells (ECs), exposed or not exposed to oxidative stress, were conditioned with sera from two groups of triathletes practising at different workloads. Although no differences in functional and hemodynamic variables were observed between the two groups of triathletes, significant changes in some markers for oxidative stress were found in their sera. Thiobarbituric acid reactive substances (TBARS) and superoxide dismutase (SOD) activity were similar, but triathletes practicing the sport at lower volume (T1) had higher serum Nitric Oxide (NO) and lower catalase activity than triathletes performing the training at greater load (T2). The ECs conditioned with serum from T1 (T1-ECs) showed higher survival and proliferation rates and lower senescence levels than the ECs supplemented with T2 (T2-ECs) serum both before and after oxidative stress induction. These effects depended on catalase as demonstrated via enzyme activity inhibition using 3-amino-1,2,4-triazole (ATZ). After oxidative stress induction, Sirt1 activity, a regulator of the oxidative stress response, was significantly increased in the T1-ECs but not in the T2-ECs. Moreover, the T1-ECs required less catalase activity than the T2-ECs to counteract an equal amount of TBARS after H2O2 administration. In conclusion, this study demonstrates that the beneficial effects of aerobic exercise are eliminated when the training is performed at a greater workload. Moreover, we suggest an oxidative stress marker, serum catalase activity, as a valid tool to use in the supervision of changes to exercise volume.

References

Keywords: Exercise training, endothelial cells, exercise workload, Sirt1, catalase.