

## Exercise Attenuated Plasma Oxidized Low-density Lipoprotein and Myeloperoxidase

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### ABSTRACT

Oxidized low-density lipoprotein (Ox-LDL) is a major contributing factor of atherosclerosis. Elevated levels of several intracellular enzymes such as myeloperoxidase (MPO), 12-lipoxygenase (LOX-12), and 15-lipoxygenase (LOX-15) can accelerate the LDL oxidation. The current study investigated the effects of 4-weeks of moderate intensity walking exercise on plasma ox-LDL, MPO, LOX-12 and LOX-15 in overweight or obese men and women. Twenty seven (13 males and 14 females) overweight or obese (defined as BMI > 25.0 kg/m<sup>2</sup>) participants were randomly assigned to either exercise (N=15) or control (N=12) group. The exercise group performed 60-min walking (3 days/week) on a treadmill at 70 % of HR<sub>max</sub> for 4 weeks, whereas the control group did not exercise. Overnight fasting blood samples were collected before and after the study protocol to determine plasma ox-LDL, LOX-12, LOX-15, and MPO. A 2 X 2 [groups (exercise and control) X time (pre and post)] factorial MANOVA was used to determine the significant interaction. If significant interactions were found, follow-up simple effects tests were performed. The main effects for groups and time were tested using ANOVA. A  $p < 0.05$  was set for the statistical significance. The main effects for groups and time were not significant. However, the significant group by time interaction was found in ox-LDL ( $p = 0.041$ ) and MPO ( $p = 0.037$ ), while LOX-12 and -15 remained unchanged. The follow-up simple effect tests showed that only the exercise group significantly lowered ox-LDL ( $p = 0.032$ , from  $44.73 \pm 9.64$  to  $38.51 \pm 8.44$  U/L) and MPO ( $p = 0.010$ , from  $31.48 \pm 9.77$  to  $23.09 \pm 5.58$  ng/mL) following the 4-weeks of exercise intervention. The results of the current study suggest that the 4-weeks of moderate intensity walking exercise in overweight or obese men and women may prevent the LDL oxidation by lowering plasma MPO, which is known to trigger the inflammatory process of atherosclerosis.