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DOCTORAL THESIS

Effects of maximal intermittent exercise in normoxic and hypoxic environments on the release of cardiac biomarkers and the potential mechanism

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Abstract

The purposes of this study were 1) to investigate the release of cardiac biomarkers resulting from acute bouts of maximal intermittent exercise in a laboratory-based setting and set up an exercise-induced cardiac biomarker release (EICBR) model; 2) to compare the changes in cardiac biomarkers in normoxic and hypoxic environments and determine the effects of hypoxia; 3) to investigate the changes in oxidative stress biomarkers resulting from acute bouts of maximal intermittent exercise in normoxic and hypoxic environments at multiple time points; and 4) to observe the relationship between oxidative stress and EICBR and explore the hypothesis that lipid peroxidation triggers the release of cardiac biomarkers from the cytosolic pool.

The maximal oxygen consumption (VO_{2max}) and the corresponding velocity of VO_{2max} (vVO_{2max}) of ten well-trained male marathon runners (age 22.1±2.6 y, body mass 64.0±4.9 kg and height 177.3±3.9 cm) was determined under normoxic (FIO_2=21.0%, VO_{2max}_N=64.72±5.63 $ml \cdot kg^{-1} \cdot min^{-1}$ and vVO_{2max}_N=18.2±1.0 $km \cdot h^{-1}$) and hypoxic (FIO_2=14.4%, VO_{2max}_H=62.16±6.74 $ml \cdot kg^{-1} \cdot min^{-1}$ and vVO_{2max}_H=16.7±0.7 $km \cdot h^{-1}$) conditions in two experimental trials. One set of conditions was tested in each trial. The order in which each participant faced each trial was selected at random and the trials were separated by 72 h.

The ten participants also completed three maximal intermittent exercise protocols, under normoxic (trial N, FIO_2=21.0%), absolutely hypoxic (trial AH, FIO_2=14.4%) and relatively hypoxic (trial RH, FIO_2=14.4%) conditions. The order in

which the participants faced the three conditions was once again selected at random and the protocols were separated by at least 7 d. Each bout of maximal intermittent exercise in trials N and AH consisted of a hard run of 16.4±0.9 $km \cdot h^{-1}$ (90% vVO_{2max}_N) for 2 min, followed by an easy run of 9.1±0.5 $km \cdot h^{-1}$ (50% vVO_{2max}_N) for 2 min with a 2% slope. In trial RH, each bout of exercise consisted of a hard run of 15.0±0.6 $km \cdot h^{-1}$ (90% vVO_{2max}_H) for 2 min, followed by an easy run of 8.4±0.3 $km \cdot h^{-1}$ (50% vVO_{2max}_H) for 2 min with a 2% slope. Each of the three trials consisted of 23 bouts of maximal intermittent exercise, performed over 92 min.

Measurements of the serum of the antecubital venous blood were performed pre- and post- (0 h, 2 h, 4 h and 24 h) exercise. The measurements were taken at five time points for each of the three conditions. The cardiac damage biomarkers of high sensitivity cardiac troponin T (hs-cTnT) and cardiac troponin I (cTnI) and the oxidative stress biomarkers of malondialdehyde (MDA), lipid hydroperoxide (LH), superoxide dismutase (SOD), catalase (CAT), glutathione (GSH) and total antioxidant capacity (TAOC) were analysed. Heart rate (HR) and arterial oxygen saturation (SaO_2) were recorded before and during exercise.

Due to the skewed distribution of the data (P<0.05), a non-parametric Friedman's test was used to compare the differences in the levels of hs-cTnT and cTnI between pre- and post-exercise and at each time point for the three conditions. MDA, LH, SOD, CAT, GSH, TAOC and HR were normally distributed (P>0.05) and were analysed using one-way repeated ANOVA tests. Pearson's product moment correlation coefficients were used to determine the degree of association between the

peak levels of hs-cTnT and cTnI, and MDA, LH, SOD, CAT, GSH and TAOC.

In trial N, the level of hs-cTnT was elevated 0 h post-exercise (9.628±3.797 pg·ml^{-1} was significantly different from the pre-exercise level of 5.118±1.857 pg·ml^{-1}, *P*=0.005), reached its peak level 2 h post-exercise (24.290±18.628 pg·ml^{-1} was significantly different from the pre-exercise level, *P*=0.005) and returned to the baseline level at 24 h post-exercise (5.978±1.849 pg·ml^{-1}). The peak levels of hs-cTnT (N, AH 37.001±31.995 pg·ml^{-1}, RH 28.614±23.628 pg·ml^{-1}) and cTnI (N 0.0375±0.0437 ng·ml^{-1}, AH 0.0475±0.0533 ng·ml^{-1}, RH 0.0345±0.0375 ng·ml^{-1}) did not significantly differ under the three conditions.

In trial AH, the peak levels of hs-cTnT (2 h, 4 h) and cTnI (2 h, 4 h) were highly related to the MDA_0h and the TAOC_24h. In trial RH, the peak levels of hs-cTnT (2 h, 4 h) and cTnI (2 h, 4 h) were highly related to the TAOC_4h.

It was concluded that maximal intermittent exercise can be used to trigger EICBR. The stimulus of hypoxia did not induce more cardiac damage in this exercise model. Maximal intermittent exercise potentially triggers EICBR through oxidative stress, especially lipid peroxidation.

Keywords: *cardiac biomarkers, hs-cTnT, cTnI, oxidative stress, hypoxia*

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