

Impact of strenuous exercise on the release of cardiac biomarkers



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Background: The aim of this study was to investigate the impact of intense exercise, represented by different endurance races, in relationship with oxidative stress and cardiac markers. In a second time, we tried to demonstrate if oxidative stress induced by physical activity is a physiological or pathological process, and to establish some issues to diagnose the risk of sudden death in athletes.

Materials and Methods:

- Four populations were compared
- a control group of 16 participants “sedentary” (37 ± 4,39 years old)
- a group of 24 semi-marathon runners (41 years ± 8,76 years old)
- a group of 28 marathon runners (44,1 ± 8,37 years old)
- a group of 33 ultra-trail runners (45,8 ± 8,7 years old)

Three blood tests were drawn

- one just before
- one just after
- the last three hours after the end of the race

Different oxidative and stress and cardiac biomarkers were measured:

- Myeloperoxidase
- Reduced Glutathion
- Oxydized Glutathion
- Lipid peroxyde
- Creatine kinase isoform MB
- C-reactive protein
- Highly Sensitive Troponin T
- Natriuretic peptide (NT-proBNP)

All automated assays were performed according to the manufacturer’s specifications.

The ultra-trail runners will be subject to an echocardiography and an ECG pre- and post-race.

For statistical analysis, STATISTICA 10 software was used. We performed a non-parametric test of Kruskal-Wallis for independent sample and a Friedman ANOVA for paired samples.

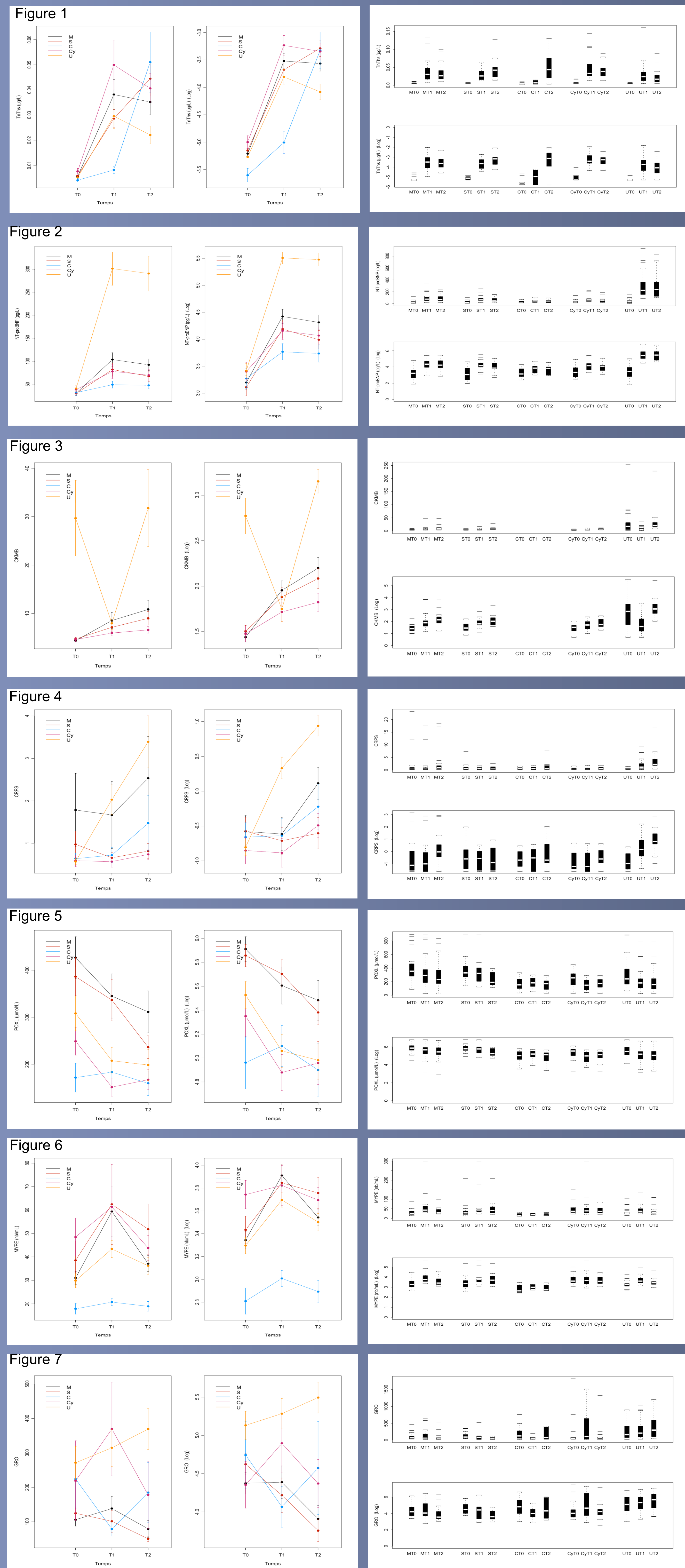
Results and discussion: We observe an increase of troponin T and natriuretic peptide but with a different kinetic than the kinetic obtained for a myocardial infarction (Fig 1 and 2). Also, we note an increase of creatine kinase isoform MB (Fig 3) and C-reactive protein (Fig 4) during the race. There is a decrease in lipidic peroxidation during exercise (Fig 5).

Myeloperoxidase increased during exercise, but the release is less important according to the level of training of the runners (Fig 6).

GSH/GSSG ratio seems to remain stable during the race but it could increase during the 24 hours post-race (Fig 7).

Medical imaging in ultra-trail runners present cardiac adaptations to endurance training, as left ventricular hypertrophy (LVH) and incomplete right bundle branch block (IRBBB). A decrease of systolic and diastolic volumes of the left ventricle and a decrease of longitudinal strain were observed by echocardiography at the end of the race.

Legend: All these figures represents the kintetic of the significative blood biomarkers (cardiac and metabolic) released during an international cycling race



Conclusions: Endurance races induce the increase of oxidative stress objectified by different biomarkers increase, but a cell necrosis is not specially observed. In fact, the increase of the cardiac markers during endurance races but may be explained by a transient modification of myocyte permeability, with a release of pool cytosolic. These races may induce micro-muscle damages causing the appearance of an inflammatory process explaining our observations of markers of inflammation.

For the medical imaging, it was observed a myocardial adaptation to training and a transient impairment of ventricular function due to dehydration.

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