

Changes of cardiac biomarkers after ultradistance and standard-distance triathlon

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Triathlon is becoming more popular sport due to recognition of the positive effects of triathlon. Generally, participants in these strenuous activities are well considered as healthy individuals pursuing a healthy lifestyle. However, there is still controversy on the beneficial effects of prolonged exercise training and endurance sport events. The duration of exercise required to elicit cardiac dysfunction and the mechanisms underlying this phenomenon have not been fully elucidated. There are only limited data in literature for exercise-induced changes of cardiac and muscle damage biomarkers in athletes participating in different triathlon distances. Monitoring cardiac and muscle damage biomarkers in triathletes participating in different triathlon distances will help researchers, coaches, and athletes better understand how to design

training cycles minimizing overtraining and injury risk. Therefore, the purpose of the present study was to examine for evidence of blood biomarkers during triathlon events of two different distances such as standard- and ultradistance triathlon in male triathletes. The results of the present study showed that ultradistance showed greater muscle damage markers such as creatine kinase, myoglobin and lactate dehydrogenase than standard-distance. We also found that the distance of triathlon did not lead to an increase of troponin T in male triathletes.

Keywords: Endurance sport events, Cardiac dysfunction, Muscle damage biomarkers, Triathlon, Troponin T

INTRODUCTION

Aerobic exercise is well recognized with a lower risk of cardiac disease. Recently, many people participate in a variety of aerobic activities such as triathlon and marathon. Acute and endurance exercise substantially increase in maximum oxygen consumption, cardiac output, stroke volume, and systolic blood pressure. Among these, triathlon is becoming more popular sports due to recognition of the positive effects of these. Generally, participants in these strenuous activities are well considered as healthy individuals pursuing a healthy lifestyle. However, there is still controversy on the beneficial effects of prolonged training and endurance sport events. Prolonged strenuous exercise leads to an elevation of cardiac specific biomarkers such as troponin T (cTnT), troponin I (cTnI), MB-creatine kinase (CKMB), and N-terminal pro brain natriuretic peptide (NT-pro BNP) in healthy endurance athletes (Park et

al., 2014). cTnT and cTnI are highly sensitive and specific markers to detect myocardial damage in the presence of peripheral muscle damage (Thygesen et al., 2007). After myocardial damage, troponins are released from cardiomyocytes and are detected within 3–10 hr in peripheral blood (Traiperm et al., 2012). Myoglobin and creatine kinase (CK) indicating exercise-induced skeletal muscle damage are well found after triathlon events in triathletes (Park et al., 2014). Monitoring of CK obtains an information about the state of the muscle and provides an information on the physical status of the athlete (Brancaccio et al., 2007) especially during the endurance events. For the precise detection of myocardial muscle damage, more cardiac specific biomarkers such as CKMB and cardiac troponins have been identified (Agewall et al., 2011; Thygesen et al., 2007).

Generally, full-distance triathlon consists of a 3.8-km swimming, 180.2-km cycling, and 42.2-km running and a half-distance tri-

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athlon consists of a 1.9-km swimming, 90-km cycling, and 21-km running. This distance is known as ultradistance (UD) triathlon. Olympic triathlon consists of a 1.5-km swimming, 40-km cycling, and 10-km running and is known as standard-distance (SD) triathlon. It may be the unique physiological demand during triathlon events of different distances. Previous studies investigated blood biomarkers and cardiac dysfunction following ultratriathlon (Arecas et al., 2015; Danielsson et al., 2017; Whyte et al., 2000) and also evaluated cardiac biomarkers following standard triathlon (Park et al., 2014). The duration of exercise required to elicit cardiac dysfunction and the mechanisms underlying this phenomenon have not been fully elucidated. There are only limited data in literature for exercise-induced changes of cardiac and muscle damage biomarkers in athletes participating in different triathlon distances. Monitoring of cardiac and muscle damage biomarkers in triathletes participating in different triathlon distances will help researchers, coaches, and athletes to reduce overtraining and even sports injury (Lee et al., 2017). Therefore, the purpose of the present study was to examine for evidence of blood biomarkers during triathlon events of two different distances such as SD and UD in male triathletes.

MATERIALS AND METHODS

Subject and design

Experimental design was implemented with a pre, post, and recovery measurement to determine the changes of blood biomarkers after events of different triathlon distances in amateur triathletes. This study received ethical approval (DIRB-201706-HR-R-017) from University of Dong-Eui Institutional Review Board. A signed informed consent was obtained from all participants.

Blood was taken from an antecubital vein. Three days before the race, baseline blood samples were collected after an overnight fasting (8–10 hr). Postrace blood samples were collected in the supine position immediately and three hours after following the triathlon.

Eight healthy amateur male triathletes (age, 39.12 ± 4.61 years) who regularly participated in triathlon training and race were recruited from triathletes. All triathletes participated in standard- and ultratriathlon race and also completed two races. During the study, all athletes consented to exercise normally, maintain normal diet, weight status, and avoid the use of medications known to affect exercise performance time, cardiac markers, and energy substrates responses. Physical characteristics of the participants are given in Table 1.

Table 1. Characteristics of subjects and performance

Characteristic	Value
Age (yr)	39.12 ± 4.61
Weight (kg)	66.87 ± 5.6
Height (m)	176.0 ± 2.9
Career (yr)	3.25 ± 1.16
UD finish time (min)	817.1 ± 139.7
SD finish time (min)	162.1 ± 12.7

Values are presented as mean ± standard deviation. UD, ultradistance; SD, standard-distance.

Measurements

Blood samples were taken from an antecubital vein 3 days before the race, immediately, and three hour after the complete race. Blood samples for plasma analyses were collected into 5- to 8-mL vacutainer tubes containing lithium heparin. Samples were frozen at -80°C for later analysis. Blood samples were assayed for markers of muscle damage, hsTnT, and energy substrates by assay kit and spectrophotometry. CK activity and CKMB activity were analyzed with Modular System P (Roche Diagnostics GmbH, Mannheim, Germany). Myoglobin was analyzed with Elecsys Modular Analytics E170 (Roche Diagnostics GmbH). cTnT was analyzed with Elecsys Modular Analytics E170 (Roche Diagnostics GmbH). Although the detection limit for cTnT is 0.01 ng/mL, the recommended diagnostic threshold for myocardial damage is >0.3 ng/mL.

Statistical analysis

For statistical calculation, the IBM SPSS ver. 18.0 (IBM Co., Armonk, NY, USA) were used. All data are expressed as the mean ± standard deviation. The differences between baseline values and the postexercise values were established for all biomarkers using the Students *t*-test. Comparison between time points for each group was performed using independent *t*-test. For all statistical tests, the threshold for statistical significance was set at $P \leq 0.05$.

RESULTS

Table 2 shows the changes of cardiac biomarkers after different triathlon distances. CK levels were significantly higher at postrace than at prerace in all distance ($P < 0.001$). CK levels were also significantly higher in UD than in SD at post and recovery among the groups ($P < 0.001$). Myoglobin levels were significantly higher at postrace than at prerace in UD ($P < 0.001$) and in SD ($P < 0.01$). CKMB levels were significantly higher at postrace than at prerace in all distance ($P < 0.001$). However, CKMB levels were not sig-

Table 2. Changes in biomarkers of skeletal and cardiac muscle damage before, immediately after and 48 hr after the race

Variable		Pre	Post	Recovery
CK (mg/dL)	UD	189.67 ± 34.84	1,128.07 ± 247.62**	388.83 ± 62.17
	SD	168.00 ± 37.39	466.53 ± 181.60**	246.08 ± 83.40
	<i>P</i> -value	0.928	0.000	0.002
CKMB (mg/dL)	UD	2.39 ± 0.66	9.07 ± 2.51**	3.53 ± 0.95
	SD	2.09 ± 0.65	7.48 ± 2.81**	2.65 ± 0.84
	<i>P</i> -value	0.393	0.252	0.072
Myoglobin (µg/L)	UD	38.22 ± 4.68	1,467.47 ± 1,002.56**	68.97 ± 26.96
	SD	27.46 ± 4.33	369.19 ± 275.07*	36.42 ± 9.05
	<i>P</i> -value	0.000	0.010	0.006
LDH (mg/dL)	UD	262.40 ± 23.11	552.48 ± 51.64**	323.08 ± 49.24
	SD	228.45 ± 32.39	294.74 ± 24.99*	248.00 ± 32.02
	<i>P</i> -value	0.030	0.000	0.003
cTnT (ng/mL)	UD	0.01 ± 0.00	0.02 ± 0.01	0.03 ± 0.03
	SD	0.01 ± 0.00	0.01 ± 0.01	0.03 ± 0.02
	<i>P</i> -value	-	0.445	0.873

Values are presented as mean ± standard deviation.

UD, ultradistance; SD, standard-distance; CK, creatine kinase; CKMB, MB-creatine kinase; LDH, lactate dehydrogenase; cTnT, troponin T.

P* ≤ 0.01. *P* ≤ 0.001.

nificantly different among distance. Myoglobin levels were also significantly higher in UD than in SD at post and recovery among distance (*P* < 0.01). LDH levels were significantly higher at postrace than at prerace in UD (*P* < 0.001) and in SD (*P* < 0.01). LDH levels were also significantly higher in UD than in SD at post (*P* < 0.001) and recovery (*P* < 0.01) among the groups. cTnT levels were not significantly different among the groups and time points. Table 3 shows the changes of cortisol and free fatty acid (FFA) after different triathlon distances. Cortisol levels were significantly higher at postrace than at prerace in all groups (*P* < 0.001). Cortisol levels were also significantly higher in UD than in SD at post (*P* < 0.01), but were not significantly different among the groups at recovery. FFA levels were significantly higher at postrace than at prerace in all distance (*P* < 0.01). FFA levels were also significantly higher in UD than in SD among the groups at post (*P* < 0.01) and recovery (*P* < 0.05).

DISCUSSION

The previous studies have demonstrated that the duration of triathlon are the main factors affecting physiological responds (Olcina et al., 2018; Suzuki et al., 2006). In this study, we investigated the acute effects on biomarkers related to skeletal and cardiac muscle damage in a sample of male triathletes according to dif-

Table 3. Changes in biochemical variables before, immediately after and 48 hr after the race

Variable		Pre	Post	Recovery
Cortisol (µg/dL)	UD	23.04 ± 2.94	30.52 ± 6.14**	14.18 ± 2.65
	SD	20.97 ± 4.57	25.08 ± 3.39**	13.26 ± 4.22
	<i>P</i> -value	0.300	0.046	0.610
FFA (mmol/L)	UD	303.12 ± 101.79	2,346.50 ± 475.69**	248.25 ± 47.06
	SD	352.00 ± 101.99	1,703.25 ± 196.82**	338.25 ± 98.01
	<i>P</i> -value	0.354	0.003	0.035

UD, ultradistance; SD, standard-distance; FFA, free fatty acid.

***P* ≤ 0.001.

ferent distance triathlon such as ultradistance and standard-distance. CK and myoglobin indicating exercise-induced skeletal muscle damages were found after triathlon events in triathletes (Park et al., 2014). Lactate dehydrogenase (LDH) levels can be increased by UD and are generally used as markers of muscle and liver damage (Suzuki et al., 2006). The results of the present study showed that UD showed greater muscle damage markers such as CK, myoglobin and LDH than SD.

Prolonged strenuous exercise leads to an elevation of cardiac specific biomarkers such as cTnT and CKMB in healthy endurance athletes (Park et al., 2014). CKMB also showed increases after different distance races compared with the pre levels. In contrast to these elevated markers, cTnT levels were not increases after different distance races compared with the pre levels. Though previous studies showed cTnT release in marathon or ultramarathon runners (Jassal et al., 2009), there was no release of cTnT after different distance races compared with the prelevels in our study. Some studies showed that the relative intensity is correlated with cTnT release such that higher intensity but shorter duration leads to a greater release (Fu et al., 2009; Stewart et al., 2016). Other studies suggested that there was no cardiac dysfunction after an Ironman-distance triathlon (Leischik and Spelsberg, 2014). We also found that the distance of triathlon did not lead to an increase of cTnT in male triathletes. Further study on specific mechanism of cardiac dysfunction is needed in this area.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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