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Aerobic capacity predict skeletal but not cardiac muscle damage after triathlon – the Iron(WO)man study

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This study examines the association between aerobic capacity and biomarkers of skeletal- and cardiac muscle damage among amateur triathletes after a full distance Ironman. Men and women (N = 55) were recruited from local sport clubs. One month before an Ironman triathlon, they conducted a 20 m shuttle run test to determine aerobic capacity. Blood samples were taken immediately after finishing the triathlon, and analyzed for cardiac Troponin T (cTnT), Myosin heavy chain- α (MHC- α), N-terminal prohormone of brain natriuretic peptide (NT-proBNP), Creatin Kinase (CK), and Myoglobin. Regression models examining the association between the biomarkers and aerobic capacity expressed in both relative terms ($\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and absolute terms ($\text{LO}_2 \cdot \text{min}^{-1}$) controlled for weight were fitted. A total of 39 subjects (26% females) had complete data and were included in the analysis. No association between aerobic capacity and cardiac muscle damage was observed. For myoglobin, adding aerobic capacity ($\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) increased the adjusted r^2 from 0.026 to 0.210 (F: 8.927, $p = 0.005$) and for CK the adjusted r^2 increased from -0.015 to 0.267 (F: 13.778, $p = 0.001$). In the models where aerobic capacity was entered in absolute terms the adjusted r^2 increased from 0.07 to 0.227 (F: 10.386, $p = 0.003$) for myoglobin and for CK from -0.029 to 0.281 (F: 15.215, $p < 0.001$). A negative association between aerobic capacity and skeletal muscle damage was seen but despite the well-known cardio-protective health effect of high aerobic fitness, no such association could be observed in this study.

Ultra-endurance races such as the the Ironman triathlon (3.8 km swimming, 180 km cycling and 42.2 km running) has been on the rise since the mid-1980s and the proportion of women participating is currently around 15%. Even though physical activity in general has positive health effects, ultra-endurance races, such as an Ironman triathlon, lead to a rise in biomarkers of skeletal- and cardiac muscle damage¹. However, most of the previous studies in the area have been conducted on elite athletes and the physiological responses to such performances may not be applicable to less trained, amateur athletes². For example, we have previously shown that both female and male amateur athletes have elevated cardiac troponin T (cTnT) well above the clinical reference limit for myocardial infarction after a Ironman triathlon¹ and that another marker for myocardial damage, cardiac specific myosin heavy chain α (MHC- α) follows a two peaked increase post-race. However, the magnitude of the changes were greater in our sample compared to other studies on elite athletes³. Moreover, we were unable to explain the variation of the skeletal and cardiac muscle damage with our models, which included sex, age, body composition and finishing time (r^2 values ranged from 0–22%).

In order to better understand the health implications of the damage observed after ultra-endurance races, and to identify strategies for prevention, it is important to find variables that improve the model of skeletal- and cardiac muscle damage prediction. In this regard, maximum aerobic capacity is of interest since it has been well documented that having a high aerobic capacity is protective against cardiovascular disease in the general population⁴. In addition, the physiological factors leading to high aerobic capacity could also protect the athlete from cardiac and skeletal muscle damage. For instance, higher aerobic capacity has been linked to left ventricular⁵ and cardiac myocyte⁶ adaptations that might minimise cardiac muscle damage. Higher aerobic capacity should in

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theory also limit skeletal muscle damage since it is correlated to training volume⁷ and intensity⁸, which lead to structural adaptations that increase the skeletal muscles' tolerance to stress⁹.

There are several ways of expressing aerobic capacity and the most commonly used is oxygen uptake expressed per body weight, *i.e.* $\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. However, expressing aerobic capacity relative to body weight also implies a proportional relationship between the two variables; if there is no such relationship the ratio becomes invalid¹⁰. Expressing aerobic capacity as a ratio complicates any interpretation of which of the two variables, or if the ratio itself, correlates with the outcome. Entering the two variables that make up the ratio, *i.e.* oxygen consumption ($\text{LO}_2 \cdot \text{min}^{-1}$) and weight (kg), as separate variables when performing statistical analyses is suggested as a solution to overcome the ratio problem¹¹.

In this study we examined the association between aerobic capacity and skeletal- and cardiac muscle damage after a full distance Ironman in a sample of amateur triathletes. Aerobic capacity was expressed both relative to kg body weight ($\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), as well as in absolute terms ($\text{LO}_2 \cdot \text{min}^{-1}$) corrected for weight, in order to see if one is a better predictor than the other. We hypothesize that higher aerobic fitness, independently of how it is expressed, is protective against both cardiac- and skeletal muscle damage.

Methods

Study sample. In all, 55 subjects, who had signed up to participate in an Ironman triathlon in Kalmar, Sweden, were recruited from local sport clubs associated with the Swedish Triathlon Federation. The subjects were approached by advertisements on the local sport clubs Facebook pages as well as by word of mouth. Prior to the race, all participants underwent a clinical examination including an electrocardiogram. None of the subjects had any medical conditions and hence all were included in the study and none was recommended to abstain from participation in the race. Because our sample was not selected at random, there is a risk of selection bias and therefore, we tested for differences regarding age and finishing time from the overall population of Ironman participants (*i.e.* average obtained from 41,000 finishers¹²). There were no significant differences in either age ($p = 0.590$, one sample t-test) or finishing time ($p = 0.780$, one sample t-test) between our sample and the overall Ironman-population.

Ethical considerations. Signed informed consent was obtained from all participants. The study was approved by the Regional Ethics Review Board in Linköping (Dnr 2016/86-31). All research methods were performed in accordance with relevant guidelines and regulations.

Procedure. Approximately one month prior to the Ironman triathlon, all subjects were invited to conduct a 20 m shuttle run test to estimate their aerobic capacity¹³. In total, 42 subjects performed the test. On the same occasion, the subjects were measured for weight and percentage of body fat using bioelectrical impedance analysis on a Tanita BC-545 Body Composition Analyzer (Tanita, Inc., Tokyo, Japan). Height was measured on a SECA 217 stadiometer (SECA medical measurements and scales, Hamburg, Germany).

One week before and within 15 minutes after the race, blood samples were drawn from an antecubital vein by registered nurses. The samples were transferred to the local hospital to be analysed within two hours of being drawn, with the exception of MHC- α which was stored at -80°C and analysed within two months. As markers of skeletal muscle damage, the blood samples were analysed for creatine kinase (CK; reference $< 1.9 \mu\text{kat/L}$) and myoglobin (MG; reference $< 72 \mu\text{g/L}$). Cardiac muscle damage was analysed using cardiac troponin T (cTnT; reference $< 14 \text{ ng/L}$) and the novel marker MHC- α (no available reference values) MHC- α is occurring predominantly in atrial muscle but as well in ventricular muscle¹⁴. N-terminal prohormone of brain natriuretic peptide (NT-proBNP; ref. $< 300 \text{ ng/L}$) was analysed as a marker for cardiac volume overload. Levels of myoglobin cTnT and NT-proBNP were analyzed using an electrochemiluminescence immunoassay on automated analyzers (Cobas e411, Roche Diagnostics GmbH, Mannheim, Germany). CK was analyzed using the multiple-point dry chemistry method (Vitros 5.1 FS Ortho Clinical Diagnostics, Ortho-Clinical Diagnostics, Johnson-Jonson Company, Rochester, USA). The laboratory methods used were standard hospital procedures and were analysed at a laboratory accredited according to Sweden's national accreditation body, Swedac (www.swedac.se). MHC- α concentration was measured with a commercially available ELISA (Cloud-Clone, Houston, Texas, USA) with no reported cross reactivity with other myosins. The biomarkers were chosen because they are commonly used in clinical practice, they are based on recommendations for sports medicine¹⁵ and they allow for comparison with previous research in the field.

Statistics. All analyses were performed using IBM SPSS version 24. Descriptive data are presented as mean \pm standard deviation (SD) or as median an 25th–75th percentile for highly skewed variables. We performed a drop-out analysis in which we investigated if there were any differences in baseline characteristics between those with and without data on aerobic capacity. In the drop-out analysis, independent samples t-test were conducted.

In order to examine the potential influence on aerobic capacity on skeletal- and cardiac muscle damage, we created two sets of models, one in which aerobic capacity is expressed relative to weight ($\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and one in which aerobic capacity is expressed in absolute terms ($\text{LO}_2 \cdot \text{min}^{-1}$). Both sets were analysed in both crude and adjusted analyses. The adjusted models were adjusted for baseline values of the biomarkers, sex, body fat (%) and age of the participants. When aerobic capacity, in absolute terms, was analysed we also entered weight in the model. Furthermore, in the adjusted analyses, a stepwise regression with two steps was fitted. This was done to investigate the independent contribution of aerobic capacity on the dependent variables, *i.e.* the change in the adjusted values of coefficient of determination (r^2). Despite several of the variables being correlated with each other, no sign of severe multicollinearity was observed in any of the models (all variance inflation factors < 5). If the assumption of heteroscedasticity of the residuals was not satisfied, the dependent variables were log transformed.

	Male (n = 29)	Women (n = 10)	Total (n = 39)	p
	Mean \pm SD	Mean \pm SD	Mean \pm SD	
O ₂ (ml/kg/min)	51.5 \pm 6.1	45.5 \pm 5.2	49.9 \pm 6.4	0.009
O ₂ (l/min)	4.0 \pm 0.6	2.8 \pm 0.5	3.7 \pm 0.8	<0.001
Age	42.7 \pm 10.6	37.8 \pm 9.6	41.4 \pm 10.4	0.208
Weight	77.8 \pm 7.1	61.6 \pm 5.4	73.7 \pm 9.8	<0.001
Body fat (%)	14.2 \pm 3.9	22.5 \pm 4.9	16.3 \pm 5.5	<0.001
Cardiac Troponin T (ng/L)	9.2 \pm 4.8	6.7 \pm 4.4	8.6 \pm 4.8	0.159
Myosin Heavy Chain - α (ng/L)	1.1 \pm 0.4	1.6 \pm 0.7	1.3 \pm 0.5	0.007
NT-proBNP (ng/L)	54.2 \pm 17.4	59.2 \pm 21.4	55.5 \pm 18.4	0.466
Creatine Kinase (μ kat/L)	3.8 \pm 4.6	1.9 \pm 0.9	3.3 \pm 4.1	0.216
Myoglobin (μ g/L)	40.9 \pm 29.2	24.9 \pm 5.7	36.8 \pm 26.2	0.097

Table 1. Descriptive statistics and baseline biomarker levels for participants who completed the baseline assessment. P-values are from an independent samples t-test.

	Aerobic capacity relative to weight (mLO ₂ *kg ⁻¹ *min ⁻¹)			Absolute aerobic capacity (LO ₂ *min ⁻¹)		
	Beta	95% CI	r ²	Beta	95% CI	r ²
Cardiac Troponin T (ng/L)	0.01	-0.02-0.05	-0.012	0.04	-0.28-0.36	-0.025
Myosin Heavy Chain - α (ng/L)	-0.05	-0.08--0.01	0.104	-0.60	-0.89--0.31	0.300
NT-proBNP (ng/L)	-0.03	-0.06-0.01	0.036	-0.28	-0.57-0.01	0.072
Creatine Kinase (μ kat/L)	-0.06	-0.10--0.02	0.203	-0.32	-0.65-0.02	0.068
Myoglobin (μ g/L)	-158.6	-283.3--33.9	0.129	-600.1	-1722.5-522.4	0.005

Table 2. Bivariate regression coefficients for aerobic capacity expressed both in relative (mLO₂*kg⁻¹*min⁻¹) and absolute terms (LO₂*min⁻¹). Bold font indicate beta- coefficients significantly different from 0.

Results

In total, 39 finished the race and provided baseline data. On average they had trained for 11.2 \pm 3 hours/week and had finished a median (25th–75th percentile) of 8 (3–19) ultra-endurance races prior to this one. The descriptive data and baseline biomarker levels stratified for sex is shown in Table 1. There were no differences between those with or without baseline assessment data in any of the variables presented in Table 1 (all $p > 0.05$).

In the unadjusted analyses, a negative association between aerobic capacity (mLO₂*kg⁻¹*min⁻¹) and both biomarkers for skeletal muscle damage and MHC- α was observed. No association between aerobic capacity in absolute terms (LO₂*min⁻¹) and the marker for skeletal muscle damage while a negative association for MHC- α were found (Table 2).

In the adjusted analysis, a stepwise regression with two blocks was used to estimate the unique effect of aerobic capacity on the biomarkers (Table 3). Aerobic capacity relative to weight (mLO₂*kg⁻¹*min⁻¹) contributed significantly to the model predicting skeletal muscle damage. For myoglobin, adding aerobic capacity (mLO₂*kg⁻¹*min⁻¹) increased the adjusted r^2 from 0.026 to 0.210 (F: 8.927, $p = 0.005$) and for CK the adjusted r^2 increased from -0.015 to 0.267 (F: 13.778, $p = 0.001$).

A similar picture regarding the independent association between aerobic capacity and the biomarkers was seen when aerobic capacity was entered in absolute terms (LO₂*min⁻¹) in the models (Table 4). For myoglobin, the explained variance increased significantly from an adjusted r^2 value of 0.07 to 0.227 (F: 10.386, $p = 0.003$) and for CK from an adjusted r^2 value of -0.029 to 0.281 (F: 15.215, $p < 0.001$) when adding aerobic capacity to the models.

Discussion

The aim of this study was to investigate the association between aerobic capacity and biomarkers of skeletal and cardiac muscle damage after a full distance Ironman in amateur athletes. We observed that increased aerobic capacity was associated with decreased skeletal muscle damage, while no evidence was found for an association with cardiac muscle damage. These relationships were independent of how aerobic capacity was expressed.

The absence of an association between aerobic capacity and markers for cardiac muscle damage was surprising given the well know cardio-protective health effects of having a high aerobic fitness. We have previously reported that nearly all of the athletes showed pathological values of cTnT (98%) and NT-proBNP (89%) after finishing the race¹⁶ but the clinical value of these observations are under debate. The debate centers on whether the elevated biomarkers seen after ultra-endurance races reflect true pathological changes or if they are a part of the normal physiological response^{17,18}. Given that cTnT leaks into the bloodstream even after rather modest efforts such as after one hour of spinning¹⁹ it might reflect myocyte stress rather than damage in a non-pathological population. For this reason, we included the novel marker MHC- α . While cTnT is a small molecule (37 kDa), this is much larger (224 kDa) and an increase after exercise could hardly be explained by passage through an intact cardiac cell

	Cardiac Troponin T (ng/L)*			Myosin Heavy Chain - α (ng/L)			NT-proBNP (ng/L)*			Creatine Kinase (μ kat/L)			Myoglobin (μ g/L)*		
	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²
Crude models			-0.040			0.605			0.271			-0.040			0.026
Baseline values	-0.002	-0.056-0.052		0.489	0.073-0.905		0.007	-0.033-0.108		0.997	-3.378-5.373		-0.001	-0.010-0.008	
Sex (woman)	0.343	-0.450-1.136		1.532	0.925-2.140		1.173	-1.249-0.477		-26.63	-80.55-27.28		0.271	-0.471-1.013	
Age	0.017	-0.009-0.042		0.028	0.008-0.047		0.007	-0.017-0.039		0.156	-1.605-1.916		0.024	0.001-0.048	
Body Fat (%)	-0.036	-0.100-0.027		-0.086	-0.128-0.043		-0.062	-0.031-0.104		3.011	-1.191-7.212		-0.029	-0.086-0.029	
Full models			-0.041			0.612			0.260			0.342			0.210
Baseline values	-0.006	-0.060-0.049		0.482	0.069-0.895		0.007	-0.006-0.019		-3.506	-7.528-0.516		-0.007	-0.016-0.002	
Sex (woman)	0.439	-0.381-1.259		1.450	0.833-2.067		1.122	0.473-1.771		-64.06	-110.13-18.00		-0.119	-0.839-0.601	
Age	0.020	-0.006-0.047		0.025	0.005-0.045		0.006	-0.017-0.029		-1.374	-2.933-0.185		0.011	-0.012-0.034	
Body Fat (%)	-0.031	-0.096-0.034		-0.091	-0.135-0.048		-0.066	-0.116-0.016		0.955	-2.514-4.424		-0.047	-0.100-0.006	
mLO ₂ *kg ⁻¹ *min ⁻¹	0.022	-0.024-0.069		-0.020	-0.051-0.012		-0.013	-0.050-0.024		-6.378	-9.226-3.53		-0.063	-0.106-0.020	

Table 3. Regression models for the association between aerobic capacity expressed relative to body weight (mLO₂*kg⁻¹*min⁻¹) and the biomarkers. Bold font indicate beta- coefficients significantly different from 0.

	Cardiac Troponin T (ng/L)*			Myosin Heavy Chain - α (ng/L)			NT-proBNP (ng/L)*			Creatine Kinase (μ kat/L)			Myoglobin (μ g/L)*		
	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²	Beta	95% CI	r ²
Crude models			-0.071			0.665			0.284			0.070			0.007
Baseline values	-0.002	-0.058-0.053		0.496	0.084-0.908		0.005	-0.007-0.018		0.987	-3.458-5.432		-0.001	-0.010-0.009	
Sex (woman)	0.318	-0.813-1.450		1.186	0.386-1.985		1.574	0.683-2.466		-31.50	-107.7-44.71		0.484	-0.561-1.529	
Age	0.017	-0.010-0.043		0.024	0.005-0.044		0.012	-0.011-0.036		0.111	-1.743-1.965		0.026	0.002-0.051	
Body Fat (%)	-0.036	-0.102-0.030		-0.080	-0.123-0.037		-0.068	-0.118-0.019		3.089	-1.263-7.442		-0.032	-0.091-0.027	
Weight	-0.001	-0.041-0.039		-0.017	-0.044-0.009		0.020	-0.012-0.051		-0.246	-2.922-2.431		0.011	-0.026-0.047	
Full models			-0.077			0.682			0.286			0.442			0.227
Baseline values	-0.004	-0.060-0.051		0.489	0.081-0.897		0.004	-0.008-0.017		-3.855	-7.958-0.248		-0.008	-0.017-0.002	
Sex (woman)	0.390	-0.757-1.538		1.119	0.320-1.918		1.529	0.633-2.425		-66.61	-128.6-4.6		0.074	-0.885-1.033	
Age	0.020	-0.008-0.048		0.021	0.001-0.042		0.010	-0.013-0.034		-1.497	-3.121-0.127		0.012	-0.012-0.035	
Body Fat (%)	-0.030	-0.098-0.037		-0.086	-0.129-0.042		-0.074	-0.124-0.023		0.959	-2.596-4.515		-0.052	-0.105-0.002	
Weight	-0.015	-0.066-0.036		-0.004	-0.037-0.029		0.032	-0.008-0.072		4.014	1.189-6.839		0.054	0.012-0.096	
LO ₂ *min ⁻¹	0.280	-0.353-0.913		-0.263	-0.674-0.148		-0.247	-0.732-0.239		-88.181	-127.11-49.26		-0.913	-1.490-0.336	

Table 4. Regression models for the association between aerobic capacity expressed in absolute terms (LO₂*min⁻¹) and the different biomarkers. Bold font indicate beta- coefficients significantly different from 0.

membrane or as a normal physiological response. However, as a diagnostic marker, MHC- α has not been thoroughly studied and there are no, with modern technology, established reference values.

One possible explanation for the lack of association between aerobic capacity and cardiac muscle damage may be that subjects with high aerobic capacity exercise at higher aerobic power outputs. Higher aerobic power outputs demand higher cardiac outputs, which in its turn may lead to an increased cardiac stress reflected in both functional and biochemical perturbations²⁰. In fact, the rate of perceived exertion is a reliable predictor of training stress which implicates that athletes automatically adapt the aerobic output to the aerobic capacity. The lack of

positive association between aerobic capacity and cardiac damage could therefore be seen as indirect evidence that aerobic capacity actually is protective of cardiac damage if the required time and power output is constant.

A better predictor for cardiac damage could be the relative intensity, *i.e.* the working heartrate divided by the maximum heartrate which is a commonly used variable to describe intensity and load in endurance athletes. The importance of relative intensity as a predictor of cardiac damage is strengthened by the fact that higher intensity but for shorter duration leads to a greater cTnT release²⁰. Measurement of the athletes working and maximal heartrates should therefore be included in future research. Another factor that should be considered for future research is the indirect measure of aerobic capacity. The 20 m shuttle-run provides reasonably good estimates of aerobic capacity²¹ and is a useful field test since several subjects can be tested simultaneously at a low cost. However, it includes a lot of anaerobic accelerations and decelerations that might become a limiting factor for ultra-endurance adapted athletes. This may have affected the accuracy of the models in this study. However, it's well-known that imperfect measures of independent variables may cause regression attenuation. The 20 m shuttle-run test has relatively high criterion validity against direct measures ($r \sim 0.8$) the attenuation in this study is not of any great concern²².

The association between aerobic capacity and skeletal muscle damage observed in this study could be due to several factors. Having a higher aerobic capacity is correlated to training volume⁷ and intensity⁸, which leads to structural adaptations that increase the skeletal muscles' tolerance to stress⁹. Another aspect that has been linked to skeletal muscle damage, is eccentric (breaking) muscle actions²³. Eccentric muscle action occurs during running, but not during swimming or cycling. In this context, the inter-individual difference in running economy may be the most interesting factor in explaining why we observe a negative association between aerobic capacity and skeletal muscle damage post-race. Running economy is defined as the energy demand for a given velocity of submaximal running and is influenced by several factors, including ground reaction forces, muscle-tendon properties²⁴ and body weight²⁵. Therefore, those with lower aerobic capacity may also have a poorer running economy, *i.e.*, too much movement in the vertical direction. This results in higher ground reaction forces²⁶ with a subsequent increase in eccentric muscle actions and more knee-joint-movement that may lead to skeletal muscle damage. The potential associations between aerobic capacity, running economy and skeletal muscle damage are further supported by the associations between markers of skeletal muscle damage and body weight (Table 4) that were seen in this study. Quantifying the ground reaction force during a race could be done by equipping the athletes with accelerometers which have been shown to be able to measure ground reaction forces²⁷. It is also important to measure during the race rather than at any other occasion since that the running mechanics changes throughout the race²⁸.

In this study, we used two different approaches to analyse the association between aerobic capacity and the biomarkers. This was done out of concern that entering a ratio, in our case aerobic capacity expressed relative to body weight, in the model could be problematic for two reasons. Firstly, the ratio itself may be biased and could cause spurious associations between the ratio and the health related outcomes to be observed^{10,11}. Conversely, a ratio may obscure the true relationship between itself and the health related outcomes. This has been proven by Kronmal, who showed that for another commonly used ratio, the body mass index (weight (kg)/ height² (m²)), the ratio could appear unrelated to the health related outcomes, even though one or both of the variables making up the ratio was related to the same outcomes²⁹. In our study, the two sets of adjusted regression models showed similar results, but they were not identical. In the models where aerobic capacity was expressed in absolute terms a negative association with the biomarkers for skeletal muscle was seen in combination with a positive association between weight and the biomarkers. In theory, this could lead to a situation in which the two variables would be cancelling each other out if they were combined to form a ratio. This was not the case in our study where aerobic capacity, independently of how it was expressed, was negatively associated with skeletal muscle damage. However, the adjusted r^2 -values were higher for the models with aerobic capacity expressed in absolute terms compared to relative terms, even if this difference was small indicating that a slightly clearer picture of the relationship between aerobic capacity and health related outcomes after a full distance Ironman-triathlon emerged.

In this study, the 20 m shuttle-run was used to quantify the aerobic capacity of the triathletes. This is not a direct measure of aerobic capacity, which may have affected the accuracy of the models. However, it provides reasonably good estimates of aerobic capacity²¹ and is a useful field test since several subjects can be tested simultaneously at a low cost. In contrast to most of the previous research in this field, we have included sample that is representative of the general participants and a relatively large proportion of women. This makes the results more relevant in a health context and increases the generalisability of our findings. The major limitation is that our study is an observational study, which limits our ability to draw conclusions regarding causal effects

The growing interest in ultra-endurance events warrants more research into the potential short and long-term health consequences of participating in such races. Most of the literature to date has focused on the acute changes in different biomarkers but information about underlying factors associated with those changes is scarce. In this study, we examined if aerobic capacity predicted the magnitude of cardiac- and skeletal muscle damage post-race. As we found no such association with cardiac muscle damage, our findings indicate that predictors of this remain to be identified so that preventive strategies can be developed. This is important information given the debate whether a marathon, triathlon or ultra-running repeatedly harms the heart by causing heart muscle cell necrosis. There is only circumstantial evidence on both sides of the debate of physiological versus pathological explanations of cTnT leakage. Neither side can so far conclusively prove what the mechanism of cTnT rise really is. On the one hand, it seems counterintuitive that just exercise should be the one and only scenario where increased plasma cTnT levels are not associated with increased risk of adverse outcomes. In all other situations even a subclinical rise of cTnT indicates a higher incidence of subsequent coronary heart disease, heart failure and death³⁰. On the other hand, it seems equally unlikely that elite distance runners who maintain a high fitness over decades would have repeatedly sustained small myocardial infarctions³¹. Further studies using a longitudinal study design is needed to resolve this debate. To avoid excessive skeletal muscle damage, having a high aerobic capacity is

beneficial, possibly linked to structural adaptations of the muscle-tendon unit associated with a higher training dose or with a better running economy.

Conclusion

Aerobic capacity, independently of how it was expressed, was negatively associated with biomarkers for skeletal muscle damage after an Ironman triathlon, but not with biomarkers for cardiac muscle damage. To identify predictors of cardiac muscle damage, future studies could test other predictors, included more potential confounders or analyse more specific biomarkers, or include measures taken during the race if possible. The long term health effects of ultra-endurance races is an area of research which needs further attention.

Data availability

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

All authors (T.D., J.C., L.T.S., J.A. and P.B.) contributed to the conception of the study. P.B. performed the data analysis. Writing – original draft: T.D., J.C., P.B. Writing – review & editing: T.D., J.C., J.A., L.T.S. and P.B. All authors approved the final submitted version of the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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