

Effects of Ultramarathon Running on Mitochondrial Function of Platelets and Oxidative Stress Parameters: A Pilot Study

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Table 1: Flux control ratios and flux control efficiencies before competition (PRE), immediately after the race (POST) and 24 h after finishing (REC)

	PRE	After		p	η^2	Change (%)	
		POST	REC			Δ PRE-POST	Δ PRE-REC
Flux control ratios							
R	0.250 ± 0.061	0.291 ± 0.064	0.246 ± 0.049	0.226	0.257	+16.34	-1.84
PM _R	0.327 ± 0.054	0.334 ± 0.067	0.334 ± 0.030	0.854	0.031	+2.07	+2.18
PM _L	0.199 ± 0.029	0.256 ± 0.040	0.223 ± 0.047	0.036	0.566	+28.37*	+11.82
PM _P	0.452 ± 0.051	0.484 ± 0.058	0.454 ± 0.087	0.789	0.046	+7.01	+0.43
PGM _P	0.566 ± 0.053	0.611 ± 0.048	0.563 ± 0.080	0.384	0.174	+7.93	-0.46
Flux control efficiencies							
1-PM _L /PM _P	0.556 ± 0.069	0.450 ± 0.095	0.501 ± 0.095	0.005	0.731	-19.18*	-9.91
1-PM _P /PGM _P	0.202 ± 0.051	0.212 ± 0.085	0.199 ± 0.062	0.835	0.035	+5.25	-1.25
1-S _E /PGMS _P	0.332 ± 0.033	0.350 ± 0.039	0.323 ± 0.055	0.846	0.041	+8.23	+1.85

Values are shown as means ± SD, p values of ANOVA and η^2 (effect size) of both flux control ratios and flux control efficiencies PRE, POST and REC. Percental changes PRE-POST and PRE-REC are given. Drop-outs are not included in statistics. *Significant change in PRE-POST post hoc test, p = 0.05.

Table 2: Abbreviations

JO ₂	Respiratory O ₂ flux
Mitochondrial fluxes	
R	JO ₂ of ROUTINE respiration depending on endogenous substrates
PM _R	JO ₂ of ROUTINE respiration in the presence of pyruvate and malate
PM _L	JO ₂ of LEAK respiration in the presence of pyruvate and malate
PM _P	JO ₂ of OXPHOS in the presence of pyruvate and malate
PGM _P	JO ₂ of OXPHOS in the presence of pyruvate, malate, glutamate
PGMS _P	JO ₂ of OXPHOS in the presence of pyruvate, malate, glutamate, succinate
S	JO ₂ of the CI-linked electron transfer pathway state
Flux control ratios	
R	R normalized by PGMS _P
PM _R	PM _R normalized by PGMS _P
PM _L	PM _L normalized by PGMS _P
PM _P	PM _P normalized by PGMS _P
PGM _P	R normalized by PGMS _P
Flux control efficiencies	
1-PM _L /PM _P	PM _L normalized by PM _P . Step analysis of ADP titration
1-PM _P /PGM _P	PM _P normalized by PGM _P . Step analysis of glutamate titration
1-S _E /PGMS _P	S _E normalized by PGMS _P . Step analysis of Rotenone titration

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Platelet mitochondrial function and race performance

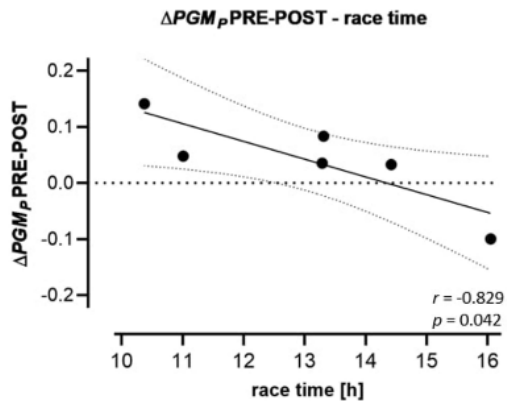


Figure 1. Regression (95 % confidence intervals) of change in PGM_p PRE-POST to race performance. Changes of FCRs of the NADH-linked OXPHOS state (substrates: PGM) PRE-POST to race time of the participants.

Correlation between respirometry parameters and markers of muscle injury

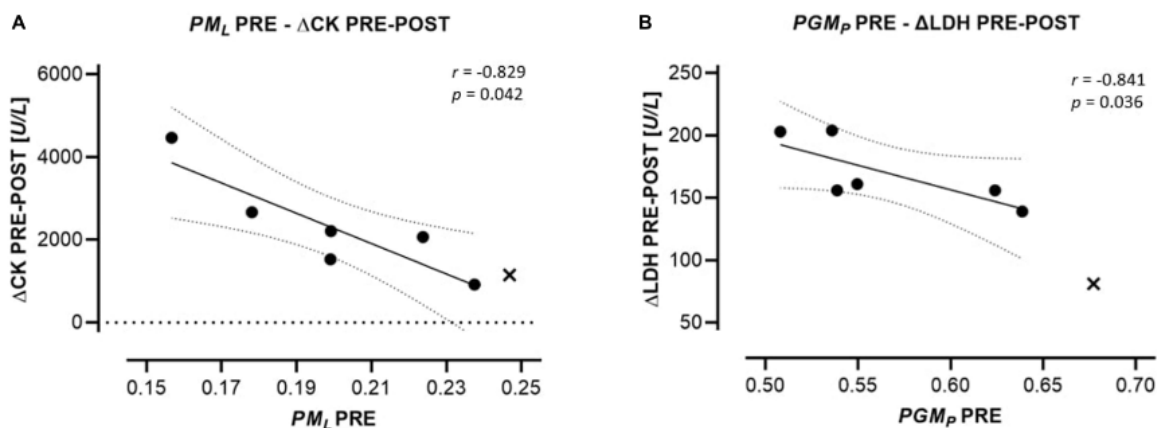


Figure 2. Regression (95 % confidence intervals) of HRR parameters to changes in indirect markers for tissue damage. **(A)** FCRs of the LEAK state (PM_L) PRE to changes of creatine kinase (CK) PRE-POST. **(B)** FCRs of the NADH-linked OXPHOS state (substrates: PGM) to changes of lactate dehydrogenase (LDH) PRE-POST.

Ultramarathon running induced an increase in LEAK O_2 flux of platelet (PLT) mitochondria. There were inverse correlations between race time and N-linked substrate state PRE-POST, and changes in CK and LDH levels were significantly correlated to PLT mitochondrial LEAK and N-linked respiration pre-race. Increase in the relative N-linked respiration in faster runners might suggest PLT Complex I as an indicator of physical fitness. The higher PLT LEAK pre-race and diminished increase of CK during the race may represent a prophylactic preconditioning. Furthermore, ultramarathon runners showed increased intrinsic uncoupling ($1-PM_L/PM_p$) post-race compared to pre-race, which could be interpreted as protection against thromboembolism formation.

Reference: Hoppel F, Calabria E, Pesta DH, Kantner-Rumplmair W, Gnaiger E, Burtscher M (2021) Effects of ultramarathon running on mitochondrial function of platelets and oxidative stress parameters: a pilot study. *Front Physiol* 12:632664.

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