No functional reserve at exhaustion in endurance-trained men?

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TO THE EDITOR: We read with great interest the study by Ferguson et al. (2). The authors remark that the main factor limiting V̇O₂ max in sedentary people is O₂ extraction capacity. However, Saltin’s studies clearly demonstrated that training in deconditioned subjects increases V̇O₂ max by increasing both components: peripheral extraction and cardiac output. The subjects studied in Ferguson et al. had a V̇O₂ max of 4.2 ± 1.0 l/min (22 years old), meaning that about one-third of them could hardly be considered endurance-trained athletes. Then, it is mentioned that energy provision or metabolite accumulation determines the limit of exercise tolerance (LoT). This neglects our recent study showing that neither energy provision nor metabolite accumulation established the LoT during an incremental exercise to exhaustion in normoxia or severe acute hypoxia in healthy physically active subjects (4). Ferguson et al. claim that there is no functional reserve for power generation at LoT, because the power measured at the flywheel was 352 ± 58 W, whereas that measured at the crank during the isokinetic (80 rpm) maximal effort at LoT was 391 ± 72 W (i.e., 12% higher, but P = 0.12). If the comparison were made correctly using the same measurement mode, that is the power measured at the crank, the corresponding values would have been 310 ± 58 vs. 391 ± 72 W (i.e., 26% higher with the isokinetic mode at LoT) and the main conclusion should have been changed to the contrary. In agreement with our findings, in Fig. 3 of Ferguson et al., all subjects increased crank power output from the hyperbolic to the isokinetic mode at LoT: how can this not be statistically significant? Moreover, our data (4) demonstrated that metabolic factors related to lactic accumulation or muscle acidification do not explain task failure, in contrast to the conjecture of Ferguson et al. Furthermore, it seems that verbal encouragement was provided only during the incremental exercise test. Therefore, some subjects may not have given a true maximal effort during the isokinetic sprints as reflected by reduced muscle activation in 6 participants.

To finalize, when Marcora et al. (3) subjects were requested to sprint, 3 s after exhaustion, they achieved much higher pedaling rates and developed much more power output than at task failure. A higher pedaling rate during the final sprint than during exhaustion is of minor relevance for the matter because higher shortening speed requires more ATP. Therefore, Marcora et al. (3) experiment, in agreement with ours, clearly shows that a marked functional reserve exists at exhaustion and that the myosin ATPase can hydrolyze ATP at a much higher rate than at task failure, also implying that ATP was made available at this high rate. The latter necessarily requires that a metabolic reserve exists at exhaustion, as we recently showed by performing muscle biopsies combined with measurement of leg VO₂ by the direct Fick method (1, 4). Thus Ferguson et al. do not have convincing data as to state that, in endurance-trained humans, task failure during incremental exercise occurs because maximal voluntary power cannot be increased above the task requirement.

DISCLOSURES

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AUTHOR CONTRIBUTIONS


REFERENCES