Thermally governed local blood flow regulation by the capillary pumps and CO₂ is the basis of the improved work efficiency of the trained muscle bed

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to the editor: Dr. Barrett-O’Keefe and colleagues (1) reported an exceptionally important conclusion that the increased work efficiency of trained muscle stems exclusively from the muscle bed. We fully understand the difficulties explaining these valuable results and are in agreement with the final conclusions made that the changes in skeletal muscle work efficiency are multifactorial in nature. At the same time, we believe that the difficulties of explaining the results stem from one of the greatest fallacies in physiology that local blood flow is regulated by the metabolic demand via the arterioles (4, 5). In Ref. 5, we were the first to express the opinion that striated muscle cells are extremely vulnerable to overheating during exercise and maximally necessitate thermal regulation. We proved that these cells represent thermal engines with internal combustion, which makes especially obvious the need of single cooling, not in bulk, because ~80% of their cell plasma is sequestered within the myofibrils, hampering the intracellular convective heat transfer. The necessary immediately reacting cooling can be ensured only by the directly adherent capillaries. Thus local blood flow is not regulated by oxygen demand, but by the heat and CO₂ produced by the cell metabolism. It was Krogh (3, p. 469) who expressed the ingenious assumption that the increase in number of opened capillaries during exercise may be brought about to meet requirements other than oxygen demands of the muscle that cannot be achieved by the supplying arterioles. Unfortunately, the lack of success in finding the mechanism of capillary blood flow regulation brought to the forgetfulness of Krogh’s far-sighted opposition to the arteriolar regulation and this absurd theory continues to triumph almost an entire century later. It continues even after it was confirmed that O₂ supply is always redundant, around half of it being supplied by diffusion from the entire arterial tree (2).

With the above in mind, we firmly believe that the important results reported by Dr. Barrett-O’Keefe et al. could become a good start for discussion on the validity of the arteriolar local blood flow regulation. From the data reported, unchanged muscle mass, unchanged lactate efflux from the exercising muscle, and the duration of each set, it could be concluded that the training was aerobic, which stimulates only angiogenesis, but not protein synthesis. According to our theory of capillary pump heat- and CO₂-regulated local blood flow, mentioned above, angiogenesis takes place to meet the increased cooling requirements at maximal exercise. For the submaximal one, however, the capillary density becomes overexpressed. This leads to a lower fiber temperature, decreased metabolism (which is temperature dependent), and decreased heat and CO₂ production, which both govern the muscle capillary blood flow, explaining its reduction. In other words, the myocyte engine works more efficiently at relatively lower temperature, reduced heat production, and energy loss. On the other hand, the increased capillary density ensures more uniform oxygen supply and its more effective utilization.

disclosures

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author contributions

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references


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