

Energy metabolism of human muscle and training

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The first general analysis of the pathways of central energy metabolism was done in the early 1980s by Heinrich in collaboration with Selkov's group (Dybnik et al., 1980; Heinrich et al., 1980). Their model incorporated the most essential kinetic properties of the glycolytic enzymes, which allowed them to define the principal regulatory mechanisms and properties of central energy metabolism. Here we use a model similar to this basic model (scheme in Figure 1) to analyse biochemical differences in central energy metabolism between two groups of people trained differently. The model considers that energy for muscle contractions is provided mainly from glycogen, which feeds glycolytic flux by providing ATP energy in the immediate vicinity of contracting myofibrils (Shulman and Rothman, 2001), and substrate for the tricarboxylate cycle.

As there are variations in the pathways linked to energy production, physical training could affect muscle metabolism and performance in several ways that vary essentially with the exercise protocol and the intervals of rest. The experimental study analysed revealed difference in performance and metabolite concentrations, but the enzyme activities that were available for measurement were found to be similar (Parra et al., 2000). The data alone could not adequately explain the biochemical basis of different effects of training, and so we applied a more complex analysis using a mathematical model. Once the known information and data measured before training (Parra et al., 2000) had been incorporated in the model we used it to simulate the changes measured after each of the two training programmes. The parameter changes necessary for simulating the data identified the key metabolic changes responsible for the effect of training programmes.

The switch from rest to exercise two orders of magnitude increases ATP consumption while keeping the ATP concentration almost constant, as a result of high sensitivity to the concentrations of ADP, AMP and inorganic phosphate. These products of ATP consumption affect glycogen phosphorylase, glyceraldehyde 3-phosphate dehydrogenase and lactate dehydrogenase, the main targets for glycolytic flux regulation during exercise, because of their ability to sense the concentrations of NADH and products of ATPase activity. ATPase activity, together with the adenylate kinase reaction, determines the concentrations of AMP and inorganic phosphate, which regulate the glycolytic flux affecting glycogen phosphorylase; ADP and inorganic phosphate stimulate glyceraldehyde 3-phosphate dehydrogenase. Previously the mechanism of mitochondrial function regulation by the products of ATP hydrolysis under intensive contraction workloads were explored in detail (Wu et al., 2007). Our quantitative analysis completes the picture of energy metabolism regulation by the products of ATP hydrolysis stimulated by intensive exercise. It has shown that the local concentration changes must be much higher than the averaged values measured; this assumes existence of strong local diffusional barriers for small molecules in cytosol, as suggested now by various experiments.

Our analysis of the experimental data revealed that the short periods of training increased energy consumption at rest, without significant improvement in energy production, whereas the long periods of training programme increases transport of NADH reducing equivalents into mitochondria and pyruvate utilization in the tricarboxylate cycle. Recent studies that demonstrated increase of tricarboxylate cycle enzyme activities after resistance training indirectly confirm the latter conclusion. These changes assume that in parallel there is physiological remodelling of the oxygen transport system.

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Figure 1: Scheme

