

Metabolic regulation in exercise: mechanisms and experimental models

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During exercise utilization of glucose and fatty acids as well as of glycogen and intramuscular lipids is increased. In skeletal muscle exercise elicits changes in molecular signaling that partly control these changes in fuel utilization; however, the knowledge of this intricate interplay between different molecular signaling pathways is quite limited. In addition, the results of the various experiments are often hard to reconcile because they depend heavily upon whether the results are obtained *in vitro* or *in vivo* and whether experiments have been performed in cells, animals or humans.

The AMP activated protein kinase (AMPK) has received much attention as a master regulator of metabolism in cells, including muscle cells during exercise. AMPK activity is increased in contracting muscle in an intensity dependent manner, but to a smaller extent in women than in men when exercising at the same relative intensity. This is likely due to a higher proportion of oxidative type 1 fibers in muscles in women, and a lesser disturbance of energy status during exercise. Studies with various transgenic and knockout models of AMPK deficiency have yielded conflicting results as to the importance of AMPK for regulation of glucose uptake during exercise, whereas lipid metabolism quite clearly is not regulated by AMPK. In addition, results obtained *in vitro* sometimes do not agree with results obtained *in vivo* likely reflecting the enormous complexity of the *in vivo* whole body exercise response compared to the reductionist models *in vitro*. Finally, the obvious rather large differences between mouse and man are likely also of importance for interpretation of experimental findings.