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Regulation of oxidative phosphorylation during work transitions and bioenergetic background of mitochondrial diseases

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It was proposed that the main mechanism responsible for the regulation of the cell bioenergetic system in skeletal muscle and heart during work transition is each-step activation (ESA) of all oxidative phosphorylation (OXPHOS) complexes, NADH supply and glycolysis in parallel with the activation of ATP usage (actomyosin-ATPase and Ca2+-ATPase) by Ca2+ ions. A widely-validated computer dynamic model involving this mechanism is able to explain numerous, frequently apparently unrelated to each other, properties of the system behavior under various conditions. Computer simulations demonstrate that in intact working skeletal muscle metabolite (PCr, ADP, Pi) concentrations and cytosolic pH begin to change significantly at much higher OXPHOS (complex) activities than the respiration rate (VO2). Consequently, it is postulated that inborn deficiencies of particular OXPHOS complexes and entire OXPHOS as well as ESA dysfunction can lead to mitochondrial myopathies not by compromising mitochondrial VO2 and oxidative ATP production per se, but through increase in cytosolic Pi, ADP and H+ concentrations. This increase can account for such mitochondrial myopathy syndromes as muscle weakness, exercise intolerance (exertional fatigue) and lactic acidosis.

Biography

Bernard Korzeniewski has completed his PhD and Postdoctoral studies from Jagiellonian University. He has published more than 75 papers in reputed journals.

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