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Hyperthermia decreases mitochondrial ATP synthesis in equine skeletal muscle

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Hyperthermia and acidosis commonly occur concurrently with fatigue, meaning either or both could be major contributors to fatigue. We hypothesised that acidosis and hyperthermia induce fatigue by decreasing the rate of mitochondrial function. Eight adult, unfit, rested Thoroughbred racehorses were used. Mitochondria isolated from fresh semitendinosus muscle biopsies were analysed using high-resolution respirometry under control conditions (38 °C and pH=7.0) as well as hyperthermia (43 °C), acidosis (pH=6.5) and the combination of both. pH levels were achieved through the titration of lactic acid into the reaction chamber. Respiration was supported with saturating concentrations of pyruvate, glutamate, malate, succinate, and ADP. Data were analysed using 2-way RM-ANOVA. $P \leq 0.05$ was considered significant. There was a significant pH × temperature interactive effect on maximal mitochondrial respiration in which hyperthermia at pH=7.0 caused increased respiration, but at pH=6.5 caused decreased respiration. Acidosis and hyperthermia both increased leak respiration and decreased calculated respiration efficiency, with the combination causing a greater decrease in efficiency than the effects of the two factors alone. Hyperthermia decreased ATP synthesis by 43% during maximal respiration and resulted in a 51% decrease in ratio of ATP produced to oxygen atoms consumed. There was no significant effect of acidosis on ATP synthesis. Hyperthermia decreased the rate of ATP synthesis via increased leak of protons across mitochondrial membranes during respiration. Hyperthermia-induced decrease in oxidative phosphorylation efficiency potentially creates a vicious cycle of increased mitochondrial respiration and thus heat production to maintain ATP synthesis, resulting in further loss of mitochondrial efficiency and ultimately leading to fatigue.

Mitochondrial metabolism changes following diet and exercise modifications in type 1 PSSM (PSSM1)

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Polysaccharide storage myopathy (PSSM) constitutes an inherited form of glycogen storage disorder and is a cause of exertional rhabdomyolysis. A previous study analysing mitochondrial function via high-resolution respirometry (HRR) of PSSM type-1 horses showed significant bioenergetics impairment with decreased electron transfer capacity. We hypothesised that appropriate management of PSSM type-1 horses may influence mitochondrial bioenergetics. Therefore, microbiopsies were sampled from gluteus medius as well as triceps brachii muscles. We performed HRR measurements in duplicate on two confirmed PSSM type-1 horses before and after 6 months adaptation. During this period, horses were fed a low-carbohydrate (<10% non-structural carbohydrates), fat-supplemented diet and sustained a daily aerobic exercise. After this period, a 1.5 fold change in complex II-sustained respiration using pyruvate & glutamate as substrates and malate as co-substrate was recorded in both horses. A lesser effect was noticed when glutamate (with malate) was the sole substrate used. Also, the effect seemed to be enhanced in the triceps brachii vs gluteus medius muscle which leads to the hypothesis of a different use of pyruvate depending on the fibre type composition. Since the metabolic stress in form of muscle damage is more important during submaximal exercise, the use of protocols using fatty acids as substrates for HRR is the next step. This descriptive case study suggests a modification in energy conversion after PSSM1 management. The decreased metabolic stress impacting mitochondrial function may enhance mitochondrial respiratory capacity after a 6-months period of adapted nutrition and exercise.