

## SUMMARY

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The responses of central systems to oxygen deprivation have been well characterised while adaptations in peripheral systems, such as skeletal muscles, have presented confounding variations. Several reasons for these discrepancies are purported, amongst them being the duration of exposure to hypoxia and variations in fibre composition. Moreover, in real-life high altitude situations there may be a combination of factors which have the ability to modify or alter the effect of hypoxia. This study investigates the effect of short duration hypoxia *per se* on substrate utilisation in different types of skeletal muscles.

Muscles were selected for their different substrate utilisation capacity, the premise being that hypoxia might differentially effect aerobic and anaerobic biochemical pathways. I studied activities of phosphofructokinase (PFK) and lactate dehydrogenase (LDH) as indicators of the glycolytic pathway; citrate synthase (CS) as an index of total oxidative capacity of the muscle cell and hydroxyacyl dehydrogenase (HAD) as indicative of the capacity for fatty acid oxidation. Enzyme activity ratios were calculated to elucidate the degree of reliance on a particular substrate, be it aerobic or anaerobic, fat or carbohydrate.

Analysis of PFK and LDH enzymatic activity confirmed the status of *extensor digitorum longus* (EDL) as a fast muscle, reliant on glucose metabolism for normal energy requirements. Soleus muscle was least reliant on glucose for normal energetic requirements and, as determined by the LDH / HAD ratio, had a greater potential for fat utilisation than either gastrocnemius or EDL. Soleus also had a greater capacity for aerobic metabolism (LDH:CS) than the other muscles.

Exposure to acute hypoxia (48 hours of continuous exposure to 11% O<sub>2</sub>) did not favour a rapid adaptation for enhanced reliance on anaerobic glycolysis in any of the muscles, nor did it alter oxidative capacity. We therefore conclude that oxygen deprivation, of an acute nature and in a controlled laboratory environment, affected neither aerobic nor anaerobic metabolism in skeletal muscles regardless of fibre type, over the time course of the present study.

## OPSOMMING

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Alhoewel die aanpassing van kardiaale spier op suurstof deprivasie kenmerkende karaktertrekke het, is bevindings omtrent adaptasies in die perifere skeletspiere, teenstrydig. Verskeie redes vir hierdie variasies word aangegee, onder andere die tydsduur van blootstelling aan hipoksie en spierveseltipe. Bowendien, in normale situasies van blootstelling aan hoogte-bo-seespieel, mag daar 'n kombinasie van faktore wees wat die vermoë het om die effek van hipoksie te varieer of te verander. Hierdie studie ondersoek die effek van korttermyn hipoksie *per se* op substraat verbruiking in onderskeie tipes skeletspiere.

Spesifieke spiere is geselekteer omrede hulle kapasiteit vir substraat verbruiking kenmerkend verskil. Die vooropgestelde idee is dat aerobiese en anaerobiese biochemiese bane verskillend deur hipoksie beïnvloed mag word. Ons het die aktiwiteit van fosfofruktokinase (PFK) en laktaatdehidrogenase (LDH) as indikatore van die glikolise baan bestudeer, sitraatsintase (CS) as 'n indeks van oksidatiewe kapasiteit van die spiersel en hidrosie-asiel-dehidrogenase (HAD) as indikatief van vetsuur oksidasie. Die ensiem aktiwiteitsverhouding is bereken om lig te werp op die graad van vertrou op 'n spesifieke substraat - hetsy aerobies of anaerobies, vet of koolhidraat.



Die analise van PFK en LDH ensiem aktiwiteit bevestig die status van *extensor digitorum longus* (EDL) as 'n vinnige spier wat afhanklik is van glukose metabolisme vir normale energie verbruiking. Soleus spiere was die minste afhanklik van glukose vir normale energie verbruiking en het 'n vergrote kapasiteit, soos bepaald deur die LDH / HAD verhouding, vir die gebruik van vette as gastrocnemius of EDL. Soleus het egter ook 'n vermeerderde oksidatiewe vermoë (LDH:CS) as die ander spiere.

Blootstelling aan akute hipoksie (11% O<sub>2</sub> onafgebroke vir 48 uur) het nie 'n vinnige adaptasie ten gunste van afhanklikheid aan anaerobiese glikolise in enige van die spiere begunstig nie, en daarmee saam ook nie die oksidatiewe kapasiteit verander nie. Ons gevolgtrekking is dus dat akute suurstof deprivasie in 'n gekontroleerde laboratorium omgewing nie, oor die tydperk van die huidige eksperiment, aerobiese of anaerobiese metabolisme in skeletspiere affekteer nie, ongeag die spierveseltipe.

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