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OPTIMISING THE EXERCISE PRESCRIPTION FOR FLEXIBLE MITOCHONDRIA

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The healthy metabolic state is characterised by an efficient ability to transition between lipid and carbohydrate oxidation in response to nutritional and physiological cues, and is important to maintain glucose homeostasis. This capacity to adapt fuel oxidation to fuel availability is known as metabolic flexibility. One of the key features of decreased metabolic flexibility is an apparent "stiffness" in mitochondrial substrate selection. That is, mitochondria lose their ability to switch from predominantly lipid oxidation and high rates of fatty acid uptake during fasting conditions to the suppression of lipid oxidation and increased glucose uptake, oxidation, and storage under insulin-stimulated conditions. The proposed central role of mitochondria for the maintenance of metabolic flexibility suggests that metabolic flexibility may be improved by increasing mitochondrial content and function. In this presentation, the relationship between mitochondrial characteristics and metabolic flexibility will first be established before discussing how best to prescribe exercise to improve mitochondrial characteristics. In particular, this presentation will highlight how different exercise prescriptions may be required to improve mitochondrial content and function. For example, the results of cross-sectional studies, as well as training studies involving rats and humans, suggest that training intensity may be an important determinant of improvements in mitochondrial function (as determined by mitochondrial respiration), but not mitochondrial content (as assessed by citrate synthase activity)(Bishop, Granata, & Eynon, 2014; Granata, Oliveira, Little, Renner, & Bishop, 2016a). In contrast, it appears that training volume, rather than training intensity, may be an important determinant of exercise-induced improvements in mitochondrial content (Cesare Granata, Oliveira, Little, Renner, & Bishop, 2016b). Our recent results also indicate that the early molecular events in response to a single bout of exercise differ between high-intensity and high-volume exercise, and this may help to explain the different training responses (C. Granata, Oliveira, Little, Renner, & Bishop, 2017).

Bishop, D. J., Granata, C., & Eynon, N. (2014). Can we optimise the exercise training prescription to maximise improvements in mitochondria function and content? *Biochimica et Biophysica Acta*, 1840(4), 1266-1275

Granata, C., Oliveira, R. S., Little, J. P., Renner, K., & Bishop, D. J. (2016a). Training intensity modulates changes in PGC-1α and p53 protein content and mitochondrial respiration, but not markers of mitochondrial content in human skeletal muscle. *The FASEB Journal*, 30(2), 959-970.

Granata, C., Oliveira, R.S.F., Little, J.P., Renner, K., Bishop, D.J. (2016b). Mitochondrial adaptations to high-volume exercise training are rapidly reversed following a reduction in training volume in human skeletal muscle. *The FASEB Journal,* (10):3413-3423.

Granata, C., Oliveira, R.S.F., Little, J.P., Renner, K., Bishop, D.J. (2017). Exercise-induced modulation of PGC-1α and p53 in enriched subcellular fractions of human skeletal muscle. *Scientific Reports*. 7:44227.

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Time:	3:30pm – 5:00pm
Co-Presenters:	Dr Jonathan Little; Prof David Bishop; Dr Andy Philp
Panel Practitioner:	Mr Daniel Ryan
Session Chairperson:	Prof Martin Gibala