Universität Basel

Research Project

UMDF - Stiftung

Third-party funded project

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The PGC-1a protein is a master regulator of mitochondrial biogenesis and oxidative metabolism. Accordingly, aberrant changes in the relative levels of PGC-1a in skeletal muscle or other tissues result in pathological consequences due to inadequate mitochondrial function. For example, mice that lack a functional PGC-1a gene in muscle display metabolic abnormalities and muscle damage, reminiscent of some of the symptoms of patients suffering from mitochondrial myopathies. In contrast, therapeutic elevation of PGC-1a in skeletal muscle of transgenic mice reduces muscle atrophy and ameliorates muscle wasting in COX10 knockout animals, a mouse model for a mitochondrial myopathy.<?xml:namespace prefix = o ns = ”urn:schemas-microsoft-com:office:office” />

Our project aims at A.) studying the effects of altered PGC-1a on mitochondrial function in skeletal muscle, B.) elucidating the mechanisms that underlie muscle damage in the absence of PGC-1a as well as the therapeutic effect of elevated PGC-1a on muscle wasting, respectively, and C.) estimating the protective effect of PGC-1a on suppressing reactive oxygen species production that could cause muscle damage. Ultimately, our goal is to gain a better understanding of the role of mitochondria in human disease. Furthermore, we hope that in the future, the findings from this study facilitate the therapeutic use of PGC-1a in patients suffering from diseases associated with aberrant mitochondrial function, in particular individuals suffering from mitochondrial myopathies.

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