

Mitochondrial Biology in sporadic Inclusion Body Myositis

A Dissertation

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In

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by

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ABSTRACT

Sporadic Inclusion Body Myositis (sIBM) is an inflammatory muscle disease that strikes individuals at random and accounts for approximately 1/3 of all idiopathic inflammatory myopathies. It is characterized by progressive weakness of distal and proximal muscles and is the most common muscle disorder in individuals over 50 years of age. Currently, there is no known cause, cure, or enduring treatment for sIBM, although a number of theories as to its cause have been proposed. One theory proposes that activation of the inflammatory/ immune response is the primary trigger resulting in muscle degeneration and protein abnormalities, while an alternative theory suggests that sIBM is a degenerative muscle disease with abnormal pathogenic protein accumulation, in particular Abeta, being a primary cause that triggers an inflammatory/ immune response. Mitochondrial abnormalities have been observed in skeletal muscle from patients diagnosed with the disease, however the role of the mitochondria in disease pathology is still unclear. The aim of this dissertation was to evaluate: 1) the role of the mitochondria in the development of sIBM and 2) the role of amyloid beta on mitochondrial function in skeletal muscle. A better understanding of the role of the mitochondria in the development of sIBM may help to identify novel prevention and/ or treatment strategies.

Keywords: Amyloid beta; Inclusion Body Myositis; Mitochondria