MIP/MTMR14 and Muscle Aging

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It is well known that the human aging process is associated with a progressive loss of muscular strength. Characteristic of this decline in muscle performance is the loss of skeletal muscle mass (sarcopenia) that occurs even in the healthy elderly. Indeed, humans can lose as much as 40% of their muscle mass from age 20 to 60. This significant loss of muscle mass and strength has important health and social implications as muscle weakness can contribute to an increased risk of falls and a loss of independence. Therefore, improving our understanding of the mechanisms responsible for sarcopenia and the age-related loss of muscle strength is important.

The findings of Romero-Suarez et al. [1] are exciting and set the stage for future experiments to further examine the molecular role that MIP/MTMR14 plays in the age-related decline in skeletal muscle mass and function. Prior studies by Shen, et al. [2] suggest that MIP/MTMR14 deficiency elevates PtdIns(3,4)P2 and PtdIns(3,5)P2 levels in the sarcoplasmic reticulum. These phosphoinositide derivatives appear to promote calcium leak, contractile dysfunction, and protein catabolism. It remains to be seen whether PtdIns(3,4)P2 and PtdIns(3,5)P2 accumulate in the SR of aged muscles. This is an intriguing possibility. A detailed understanding of the cellular function of MIP/MTMR14 and the molecular events leading to sarcopenia may lead to clinical countermeasures to slow the age-related loss of muscular strength.

REFERENCES