TACSM Abstract

Exploring the role of BMP7 gene expression in an *In Vitro* model of aging human skeletal muscle.

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ABSTRACT

Sarcopenia is an age-related condition associated with rapid loss of skeletal muscle (SKM) tissue that affects mobility and quality of life of geriatric individuals. Mechanistic Target of Rapamycin (mTOR) and Protein Kinase B (AKT) have significant roles in SKM hypertrophy with responses to DNA damage and repair within SKM. However, mTOR and AKT expression is significantly decreased with age. Upstream of AKT, Bone Morphogenetic Protein (BMP7) is a member of the TGF-β signaling family that has been reported as a positive regulator of muscle hypertrophy through the Bmp–Smad1/5/8 signaling axis. **PURPOSE**: To use an *in vitro* model of aging muscle cells to investigate the role of BMP7 expression on protein synthesis. **METHODS**: Human SKM myoblasts were cultured and grown beyond mature myotube formation (typically day 6) to emulate aged SKM tissue (extracted on day 18). Groups included control cells (D6) and aged SKM myotubes (D18). Total RNA was extracted at the respective time points (days 6 & 18) and gene expression for BMP7, mTOR, and AKT was determined by qPCR. **RESULTS**: BMP7 expression was 7.73 fold greater for D18 compared to D6 ($p < 0.05$). No differences were reported for AKT or mTOR. Data are expressed as fold changes. **CONCLUSION**: BMP7 expression, thought to be a positive regulator of muscle hypertrophy, was increased in the aging muscle cells of our model, despite our hypothesis that it would be decreased. However, BMP7's downstream targets related to increased protein synthesis, mTOR and AKT, did not similarly increase from D6 to D18, which is constant with the phenomena of sarcopenia. This leads us to speculate that there may be additional mechanisms related to BMP7 activation and, despite increased signaling, may block protein synthesis at the level of AKT.