

Replicating Intermittent Fasting in Human Skeletal Muscle Cells: A Pilot Study

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ABSTRACT

Type 2 diabetes (T2D), the most common form of diabetes (90-95% of diagnoses), is marked by decreased insulin sensitivity (insulin resistance) or a defect in insulin secretion. T2D disrupts nutrient signaling where the body cannot maintain adequate blood glucose levels. Inability to receive glucose in skeletal muscle due to insulin resistance in T2D results in oxidative stress and increased muscle atrophy. Properly regulated glucose uptake is pivotal for healthy aging and maintenance of the skeletal muscle system.

PURPOSE: The purpose of this study is to examine the effects of nutrient deprivation on human skeletal muscle metabolism, with an emphasis on oxidative stress and atrophy markers in healthy and T2D cell models. **METHODS:** Healthy human skeletal muscle myoblast cells (HSMM) and diabetic human skeletal muscle myoblast cells (DHSMM) (Lonza Inc, Walkersville MD) were cultured in a 37°C with 5% CO₂ incubator in a T-75 flask. At confluency, 10⁴ cells were transferred into four 24-well plates and were incubated for 48h with standard culture media (Lonza Inc, Walkersville MD). The cells were then incubated for 12 or 24 h in media containing varying serum concentrations: 5%, 10%, and 15%. The media contained either fetal bovine serum (FBS) (Lonza Inc, Walkersville MD) or pooled human serum (HS) from either healthy or diabetic patients (Doctors Regional, Corpus Christi TX). Following the 24 hours, cell viability and density were determined, and sandwich enzyme-linked immunosorbent assay kits (RayBiotech, Norcross GA) were performed to measure the amount of superoxide dismutase (SOD1) present in each sample. **RESULTS:** A treatment effect was found using T2D HS which had a significant influence on mean SOD1 levels (range of SOD1 pg/mL; p=0.0423). There was no significant effect of time between 12h and 24h (p=0.1100). In the FBS models, a significant effect of concentration HSMM is seen (p=0.0263). Incubation time had little effect on FBS DHSMM (p=0.2671) and HSMM (p=0.2780) models. **CONCLUSION:** As serum concentration increases, the level of SOD1 present in the samples also increases. This suggests that treatment concentration may influence the activity of SOD1. This may be due to exogenous SOD1 already present in the serum. However, we did not assess the rate of appearance and decay of SOD1 already present in the serum. Incubation time shows little difference in all models. These results suggest that compositional environment can influence SOD1 levels and that a higher concentration may promote oxidative stress more so than a lower concentration environment.