**Introduction**

Evidence reveals that skeletal muscle hypertrophy is achieved through increased protein synthesis and/or reduced protein breakdown, but the molecular mechanisms underlying these changes are not fully understood. A crucial factor contributing to muscle hypertrophy is the mechanical stress on the muscle fibers, which is known to stimulate muscle growth. Understanding the underlying mechanisms of muscle hypertrophy is essential for developing effective strategies to treat muscle wasting diseases, such as muscular dystrophies.

**Hypothesis**

We hypothesize that the synergistic effects of glucose and methylglyoxal on ROS generation and the viability of cultured H9c2 myoblast cells.

**Methods**

**Measurement of H9c2 cell viability after HG/MG/Met Treatment:**

- **Cell Culture:** H9c2 rat myoblasts were incubated with 25 µM non-methylated glucose (HG), 1200 µM methylglyoxal (MG), and 10 mM metformin (Met) for 24 hours.
- **Viability Measurement:** Cell viability was measured using the CCK8 kit (Dojindo Molecular Technologies, Inc.) according to the manufacturer's instructions. Absorbance at 450 nm was measured using a spectrophotometer.

**Measurement of ROS generation after HG/MG/Met Treatment:**

- **Measurement Method:** ROS generation was measured using the DCF-DA dye (Invitrogen) according to the manufacturer's instructions. Fluorescence was measured using a plate reader.

**Results**

- **Cell Viability:** Cell viability was measured and compared to control cells. *p < 0.05 vs. control.
- **ROS Generation:** ROS generation was measured and compared to control cells. "mg/mg/met" showed a significant decrease compared to control cells.

**Conclusions**

The data suggest that high concentrations of methylglyoxal, not glucose, induce H9c2 cell death and mitochondria in young cells from the synergistic effect of methylglyoxal-induced oxidative stress and hyperglycemia. This suggests that methylglyoxal-induced oxidative stress and hyperglycemia may play a role in the development of diabetic complications such as diabetic cardiomyopathy.

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**References**