Lipotoxicity and oxidative stress induced by palmitate in human muscle cells

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Introduction

- Lipotoxicity leading to skeletal muscle dysfunction
- Trigger by imbalance between ROS Production and antioxidant response?

Methods

FFA: Palmitate treatment (PA-300μM-24h) on human myoblasts and myotubes (after 4 days of diff.)

Cells were treated with PA complexed to BSA and compared to cells treated with BSA only (control condition)

Results

1. Significant accumulation of intramyocellular lipid droplets

No impact of PA on cell proliferation and differentiation

2. Mitochondrial dysfunction in myoblasts and myotubes

A. Mitochondrial superoxide overproduction

B. Mitochondrial swelling

Swelling of mit was observed using Mitotracker.

C. Mitochondrial activity

MITT Cell Assay was used to measure the mit complex II activity.

> PA treatment induces an important alteration of mitochondrial activity along with increased production of mitochondrial O2− and mitochondrial swelling.

3. NRF2 expression & Antioxidant balance

NRF2 translocation was observed after PA treatment along with antioxidant response in myoblasts and myotubes as attested by activation of NRF2 targeted genes.

Conclusion

Acknowledgments: We acknowledge the platform for immortalization of human cells from the Institute of Myology (Paris) for providing the immortalized myoblasts.

This work was financially supported by a grant of the ABMM Téléthon.