

Introduction

FFA

lipotoxicity leading to skeletal muscle dysfunction
→ Trigger by imbalance between ROS Production and antioxidant response?

Methods

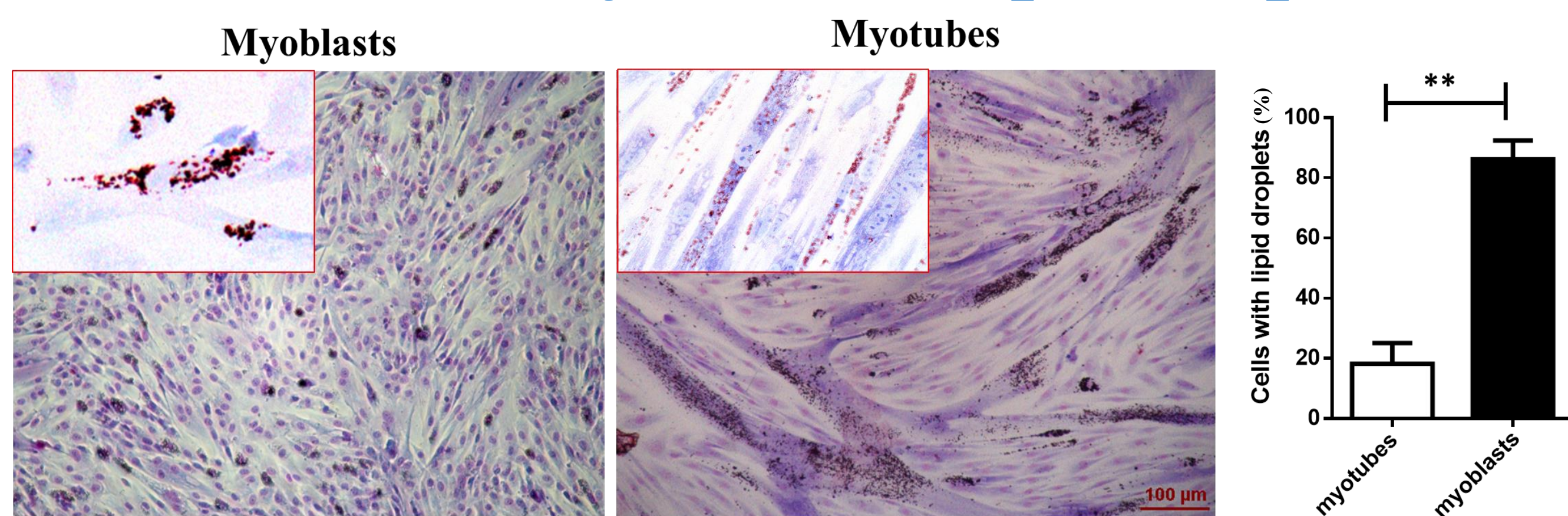
FFA: Palmitate treatment (PA-300μM-24h) on **human** myoblasts and myotubes (after 4 days of diff.)

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

Results

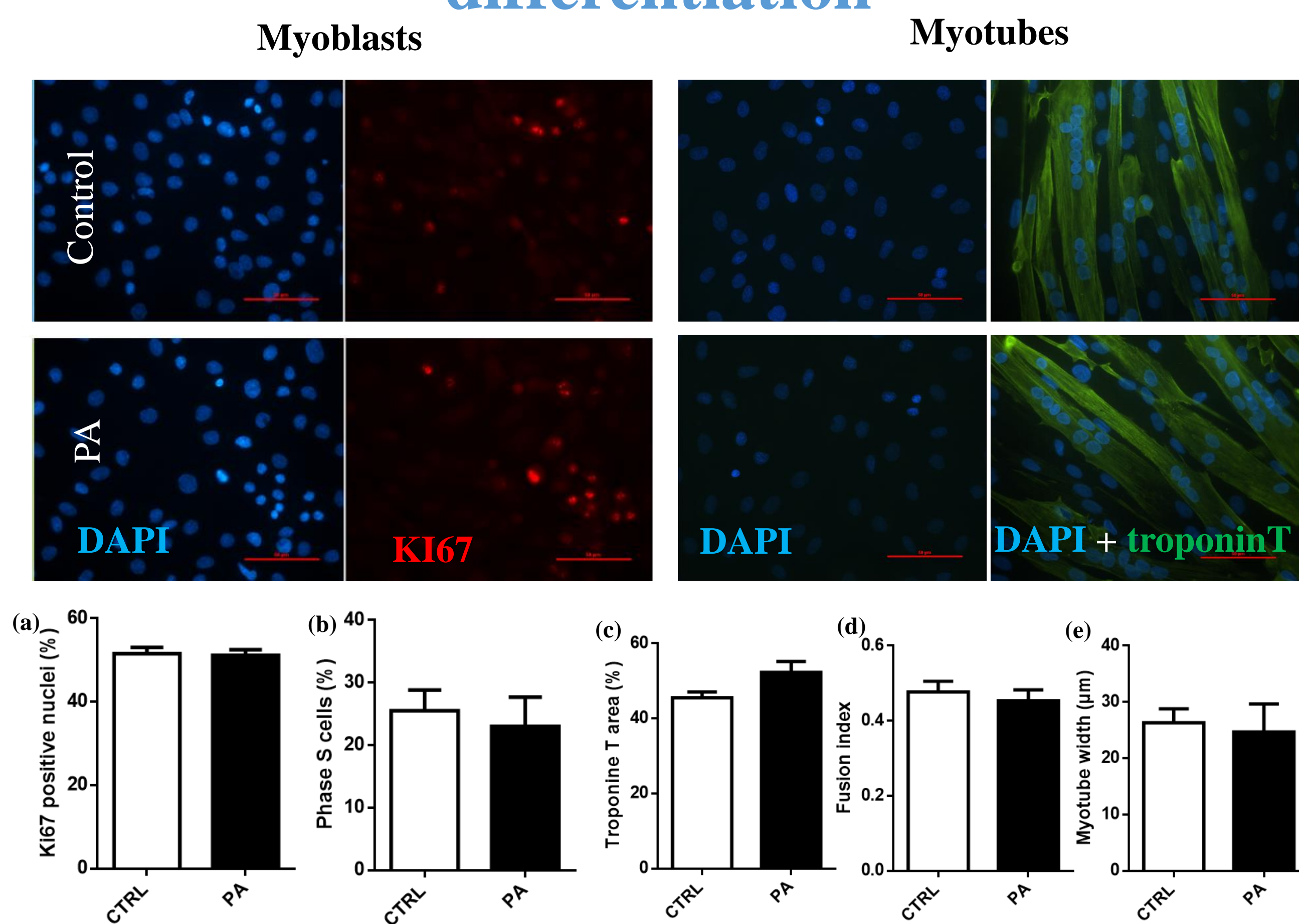
1

Significant accumulation of intramyocellular lipid droplets



The intramyocellular lipid accumulation was labeled by Oil red O (red) staining while cells were counterstained with GEMSA. Lipid droplets are present in myoblasts (20 ± 8%) and even more in myotubes (86 ± 8%). No lipid droplet was found in control cells. Graph illustrated Means ± SEM. N=5

No impact of PA on cell proliferation and differentiation

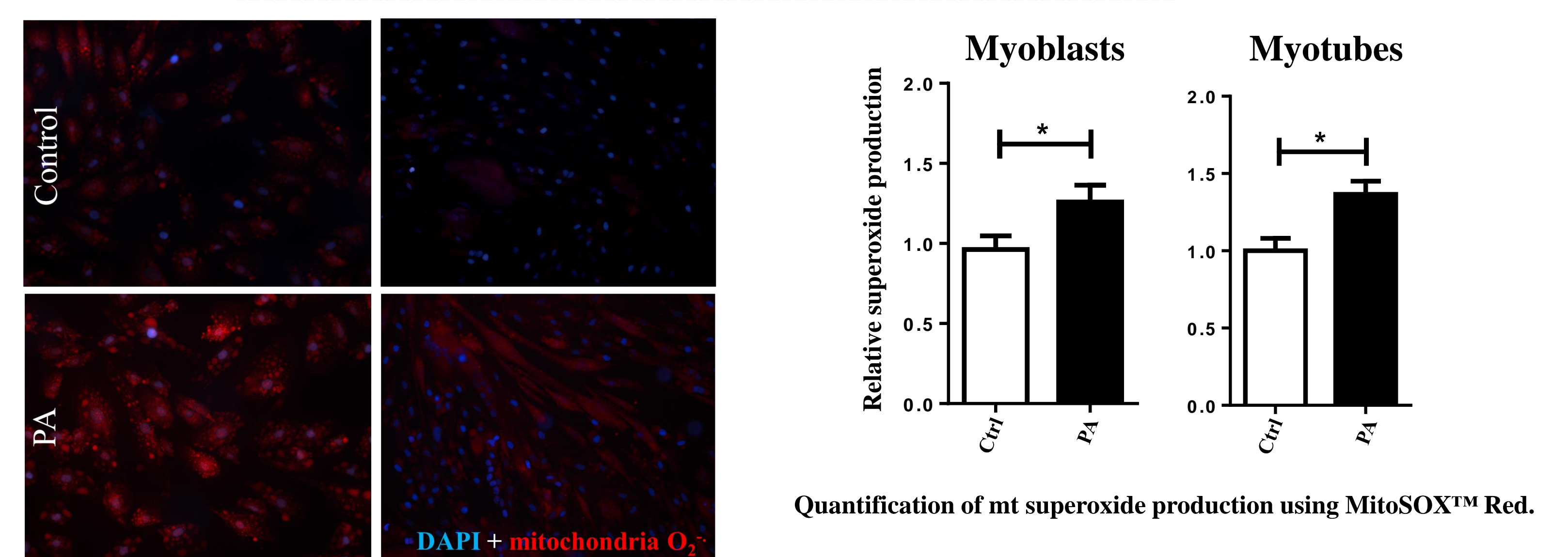


Data did not show any difference in proliferation and differentiation processes of human skeletal cells after 300 μM PA treatment. Bar graphs represent Means ± SEM. N=5

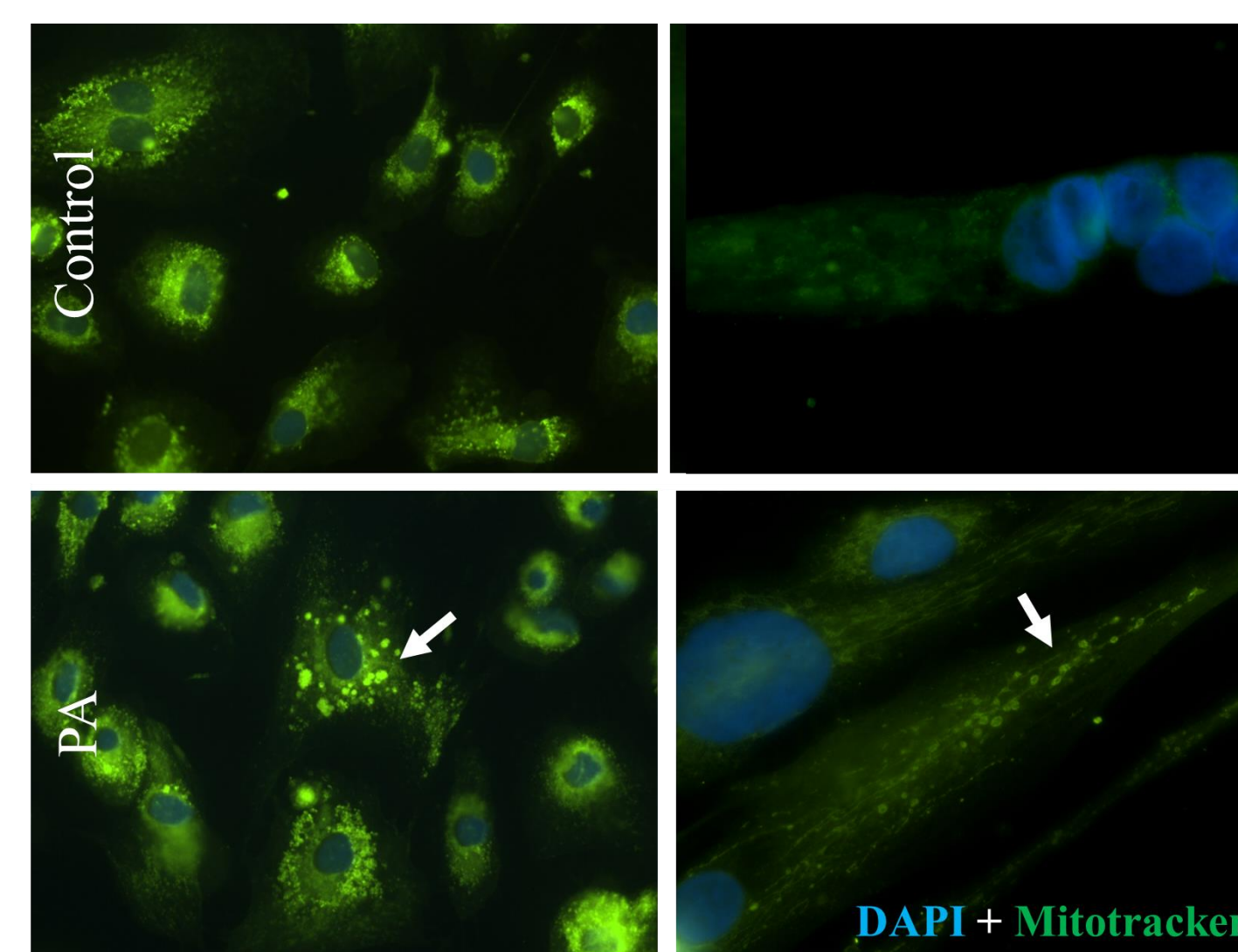
2

Mitochondrial dysfunction in myoblasts and myotubes

A. Mitochondrial superoxide overproduction

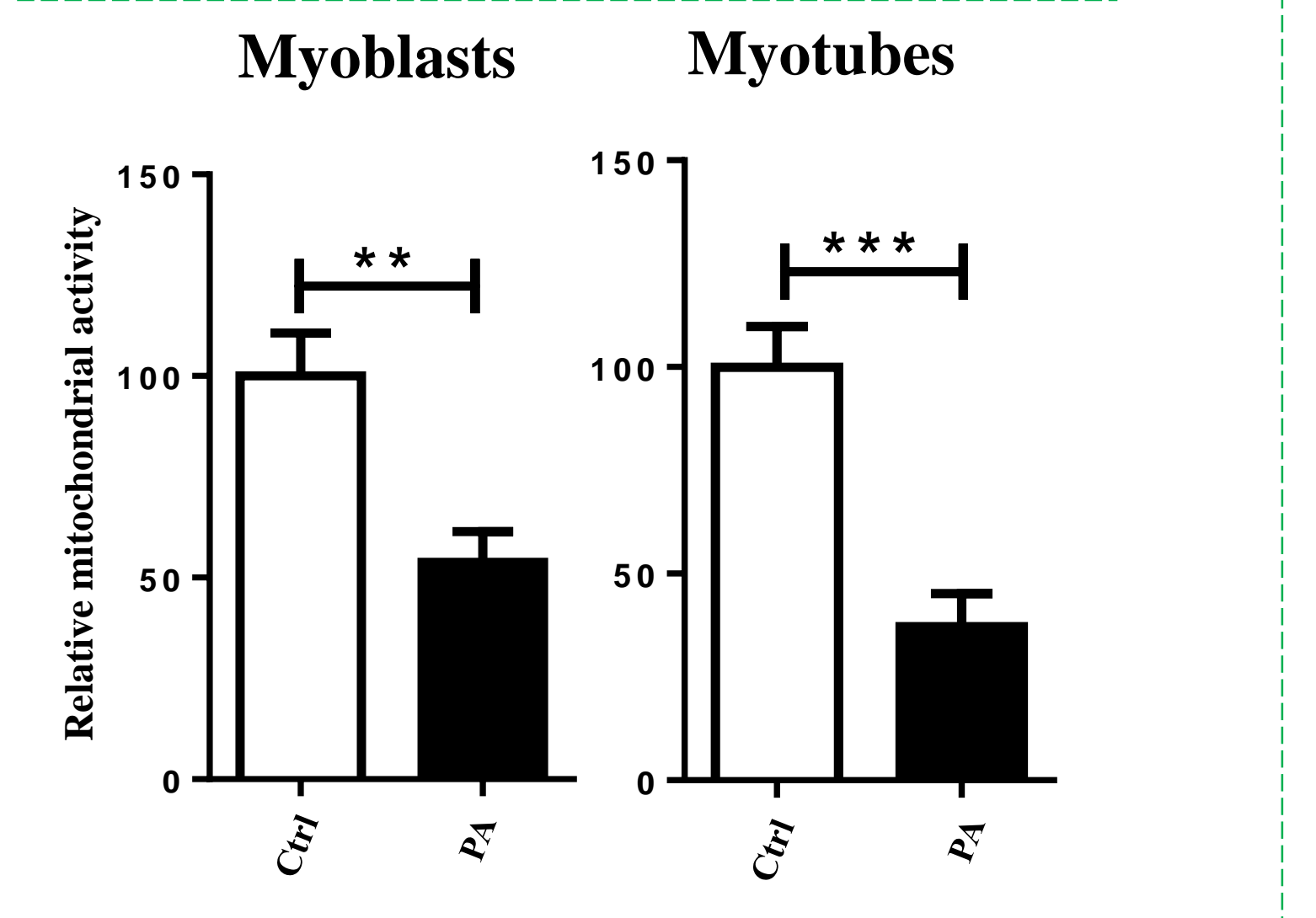


B. Mitochondrial swelling



Swelling of mt was observed using the Mitotracker.

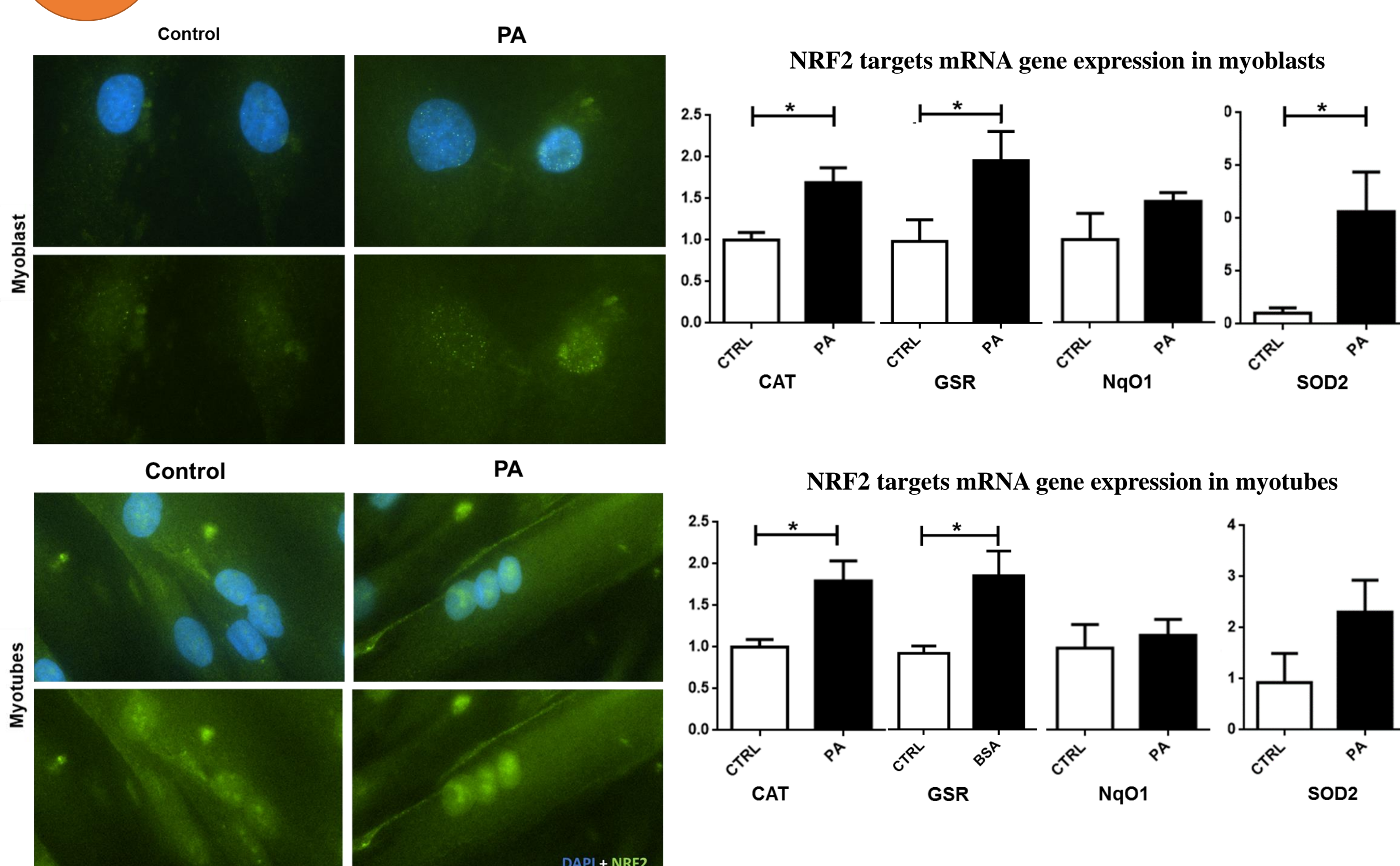
C. Mitochondrial activity



⇒ 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

