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# Hypoxia- reoxygenation induces neutral lipid accumulation in cardiomyocytes

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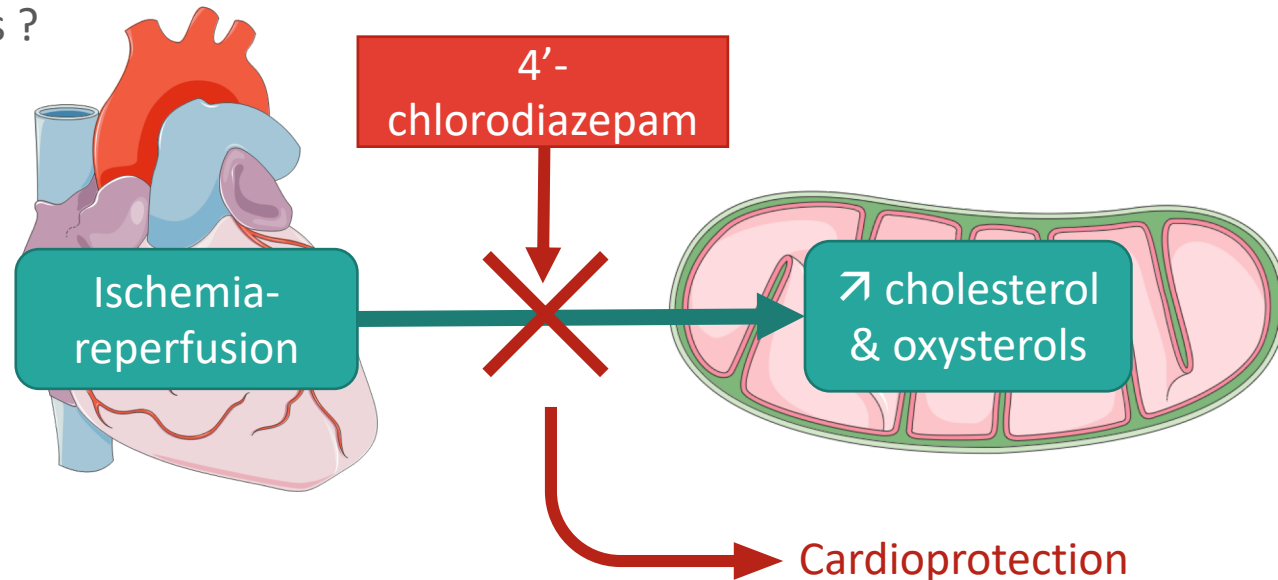
# Conflits d'intérêts / Statement of Financial Interest

Je n'ai pas conflits d'intérêts à déclarer\*

*\* I do not have any potential disclosure to report*

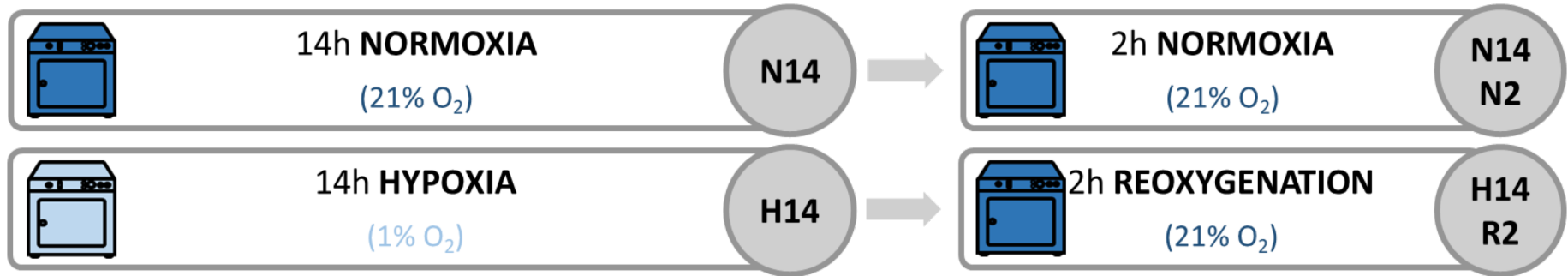
# Introduction

- Circulating lipids = cardiovascular risk factors but direct physiopathological role on cardiomyocytes ?

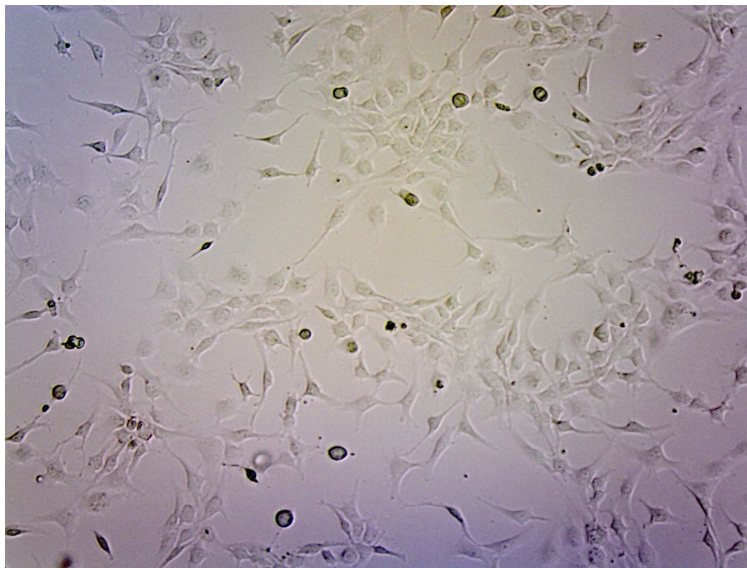


- Distribution and the trafficking of lipids in cardiomyocytes during cardiac ischemia-reperfusion ?
  - **Goal : to investigate the dynamic of lipids during hypoxia-reoxygenation in a cellular model.**

# Material & methods

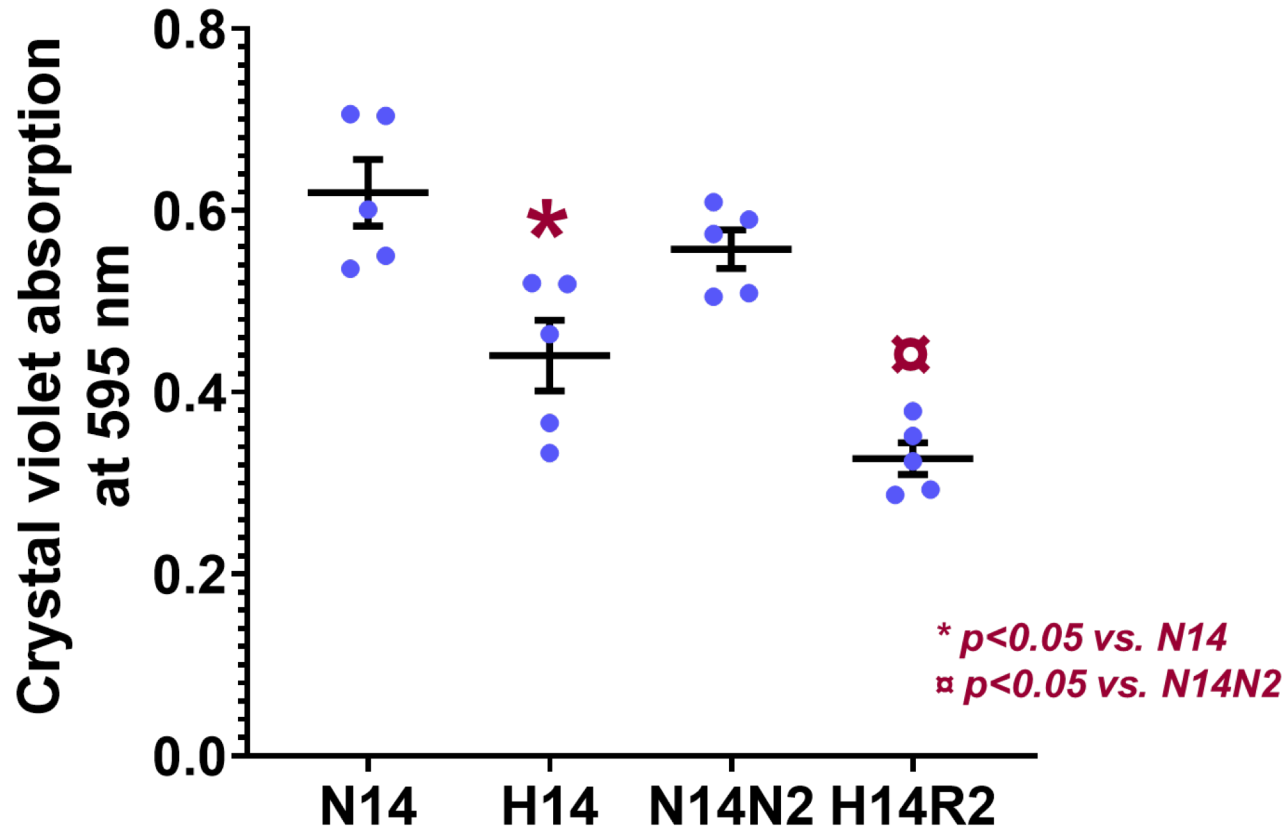


AC16 cells



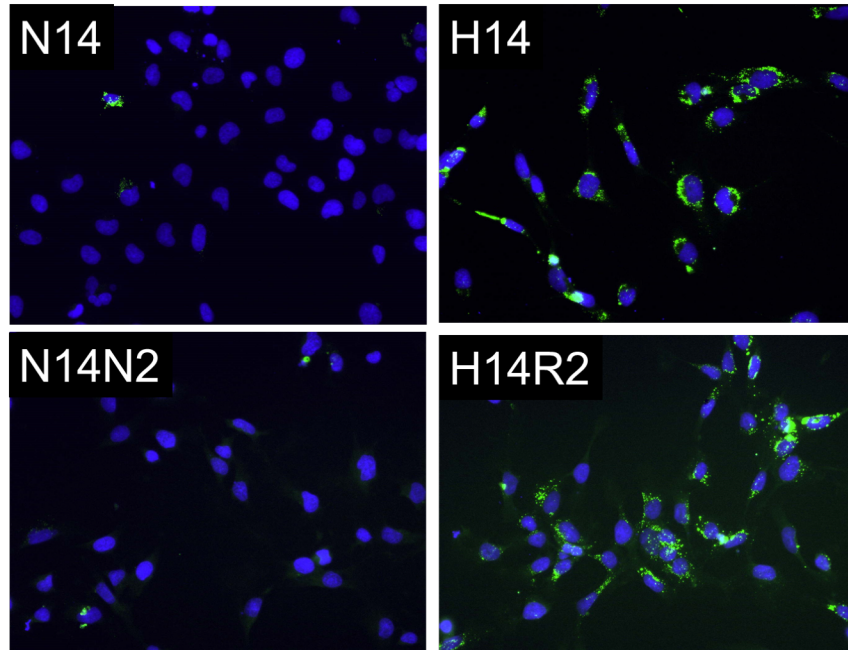
- **Mortality** (crystal violet assay)
- **Cytosolic lipid droplet** formation (Bodipy)
- **Plasma membrane cholesterol** (D4-mCherry)
- **Whole cell neutral lipids** (thin layer chromatography)

# Results

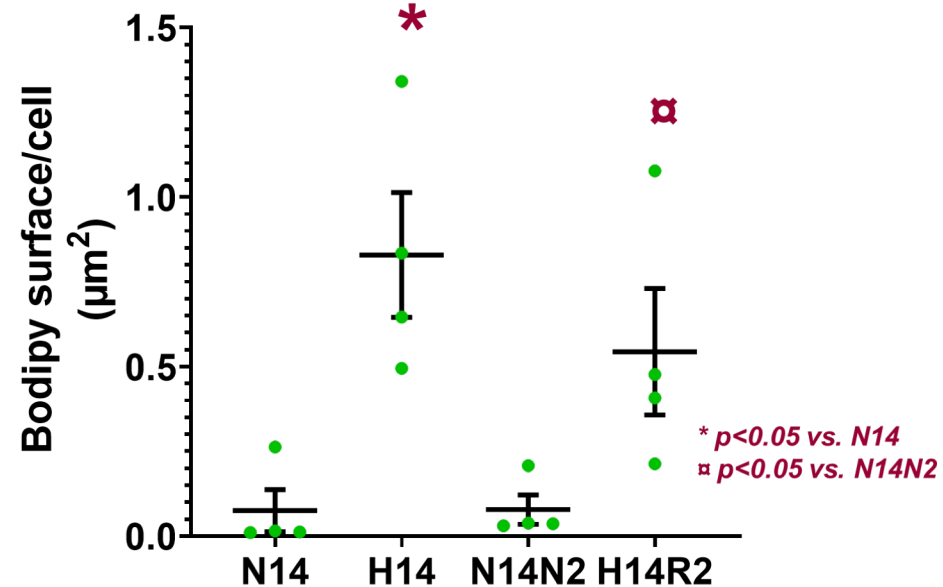


Hypoxia-reoxygenation induces cell mortality

# Results

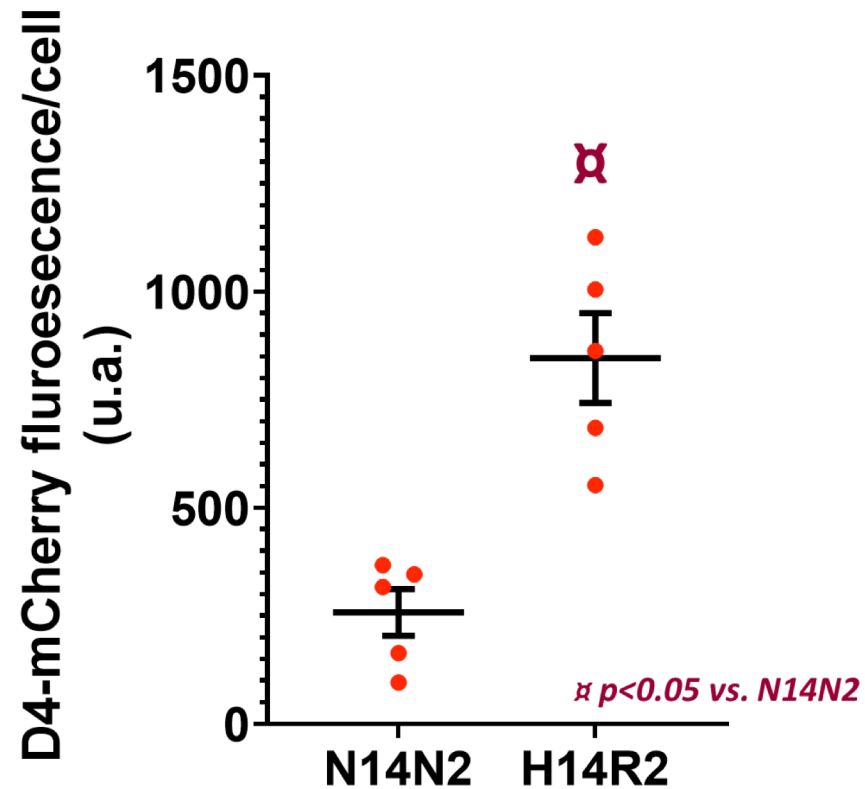
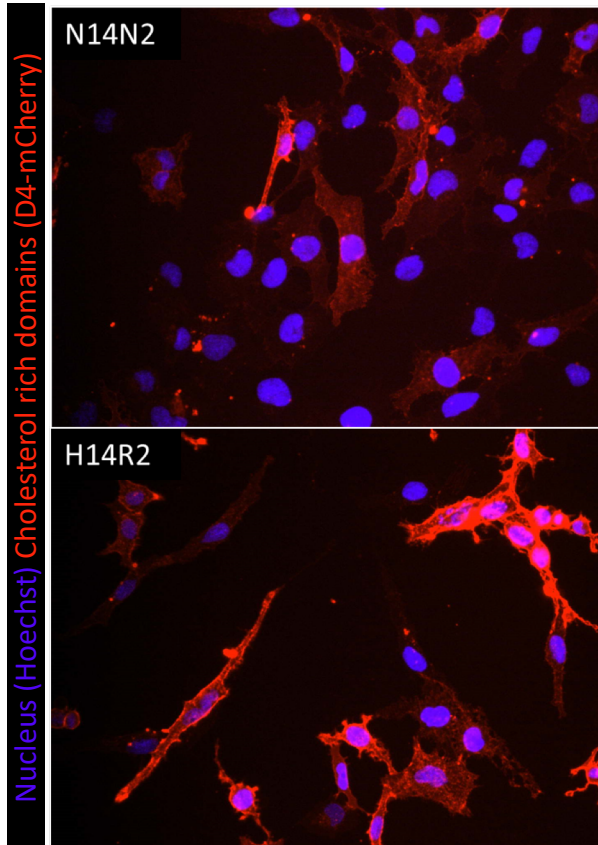


AC16 cells stained with Bodipy (lipid droplets) Hoechst (nucleus)



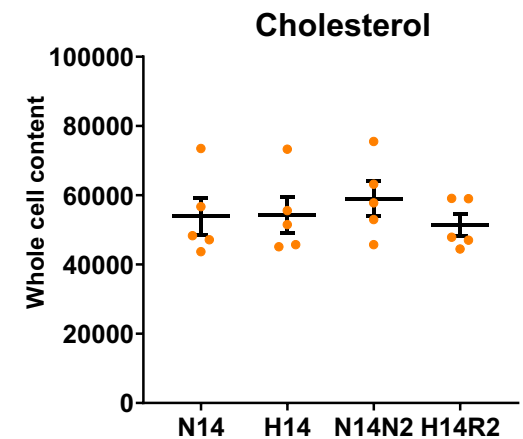
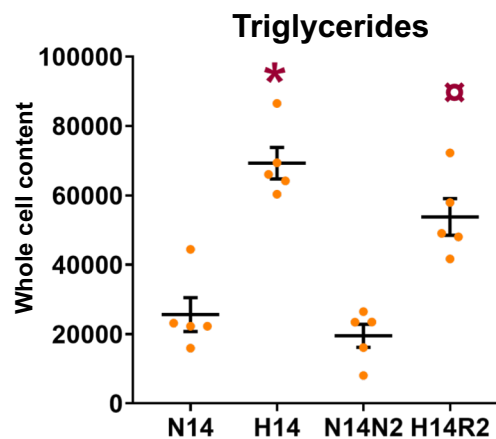
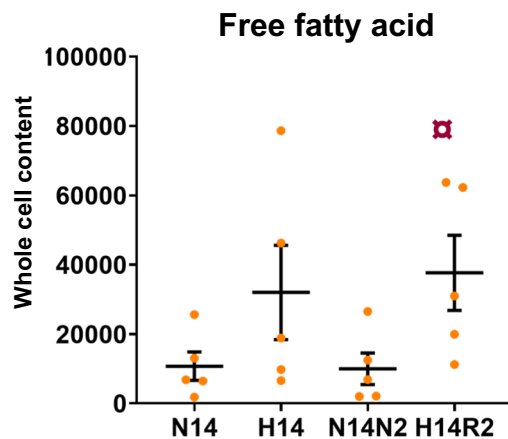
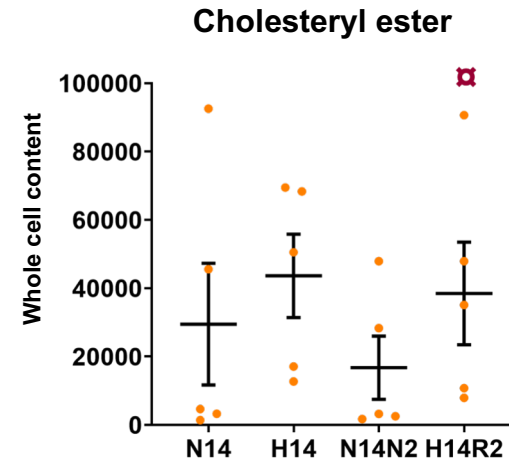
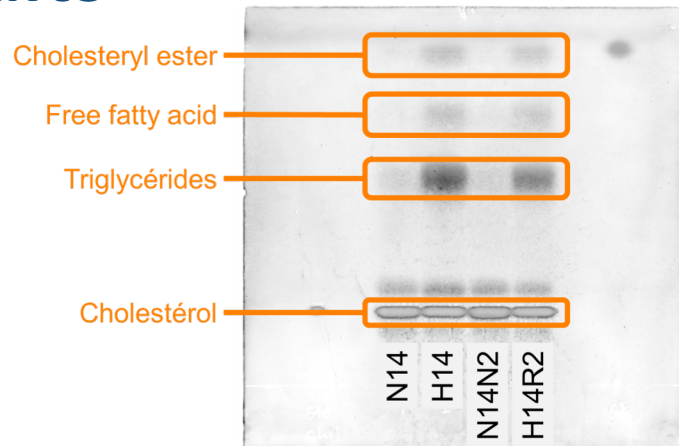
Hypoxia-reoxygenation induces lipid droplet formation

# Results



Hypoxia-reoxygenation increases cholesterol-rich domains in membranes

# Results



Hypoxia-reoxygenation induces an accumulation of neutral lipids

\*  $p < 0.05$  vs. N14

■  $p < 0.05$  vs. N14N2



## Conclusion & perspectives

Hypoxia-reoxygenation induces cytosolic neutral lipid and plasma membrane cholesterol accumulation in cardiomyocytes. Whether this change of lipid distribution is responsible for mitochondrial cholesterol accumulation at reperfusion needs to be determined.