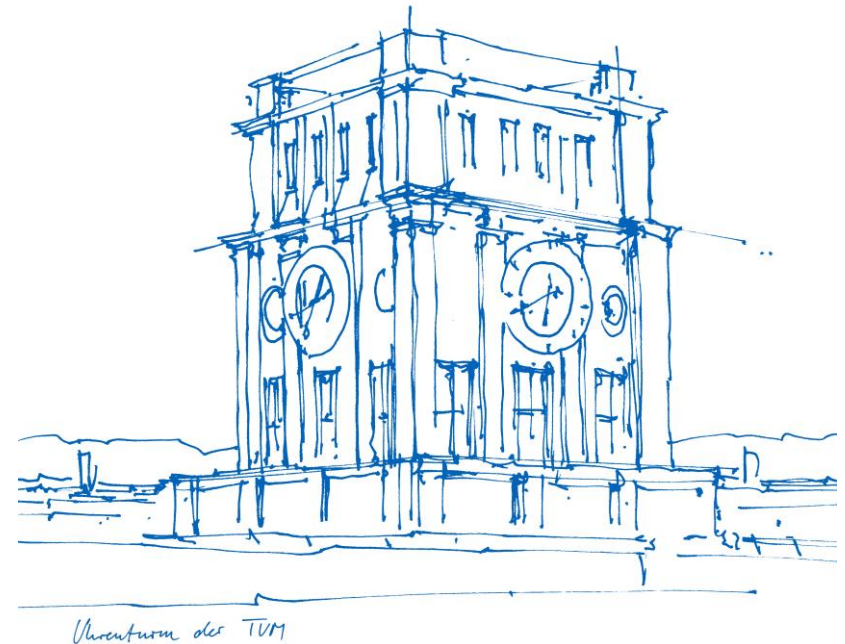


# Source depot and dietary status influence adipocyte oxidative phosphorylation capacity

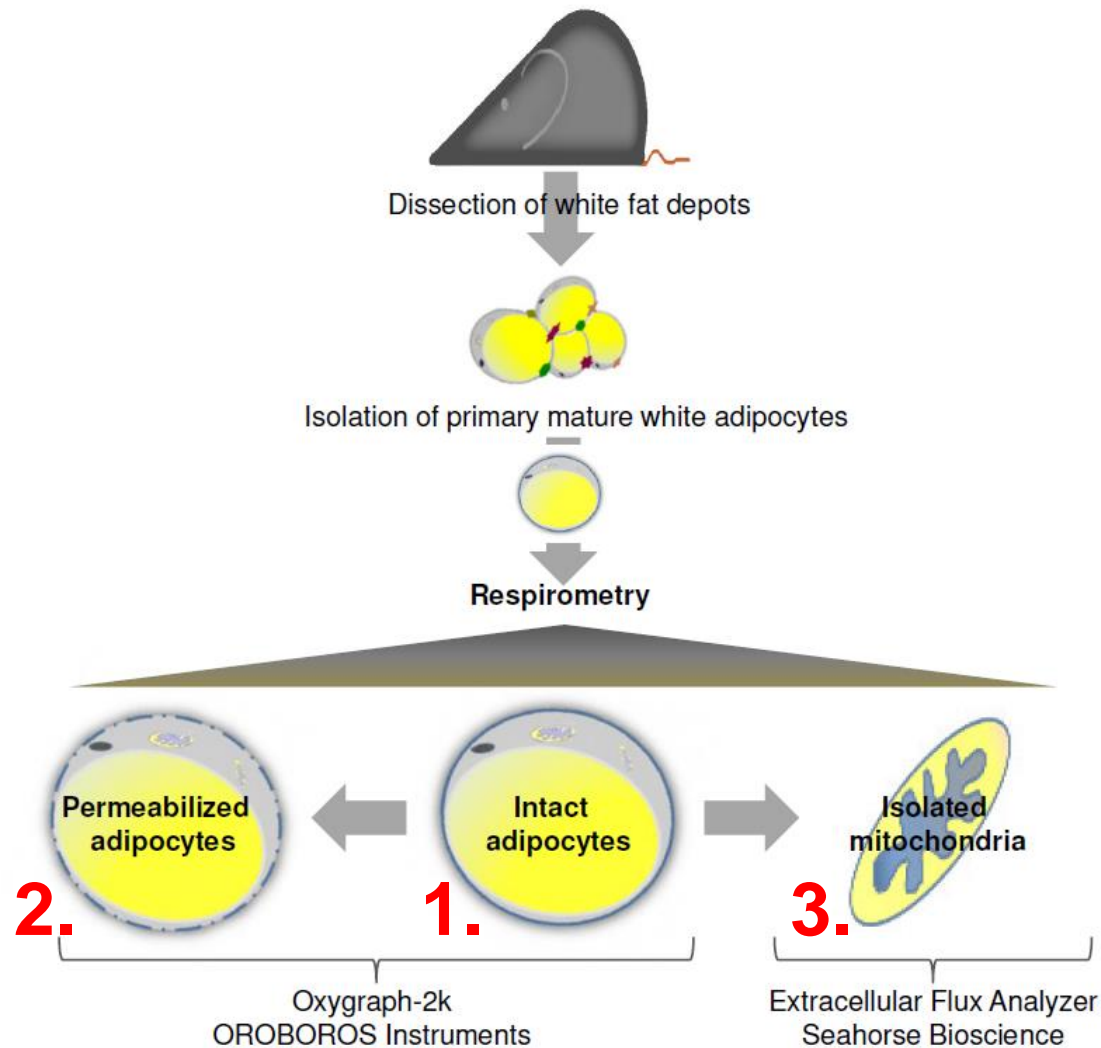
Dr. Tobias Fromme

Chair for Molecular Nutritional Medicine  
Technical University of Munich  
TUM School of Life Sciences  
Germany

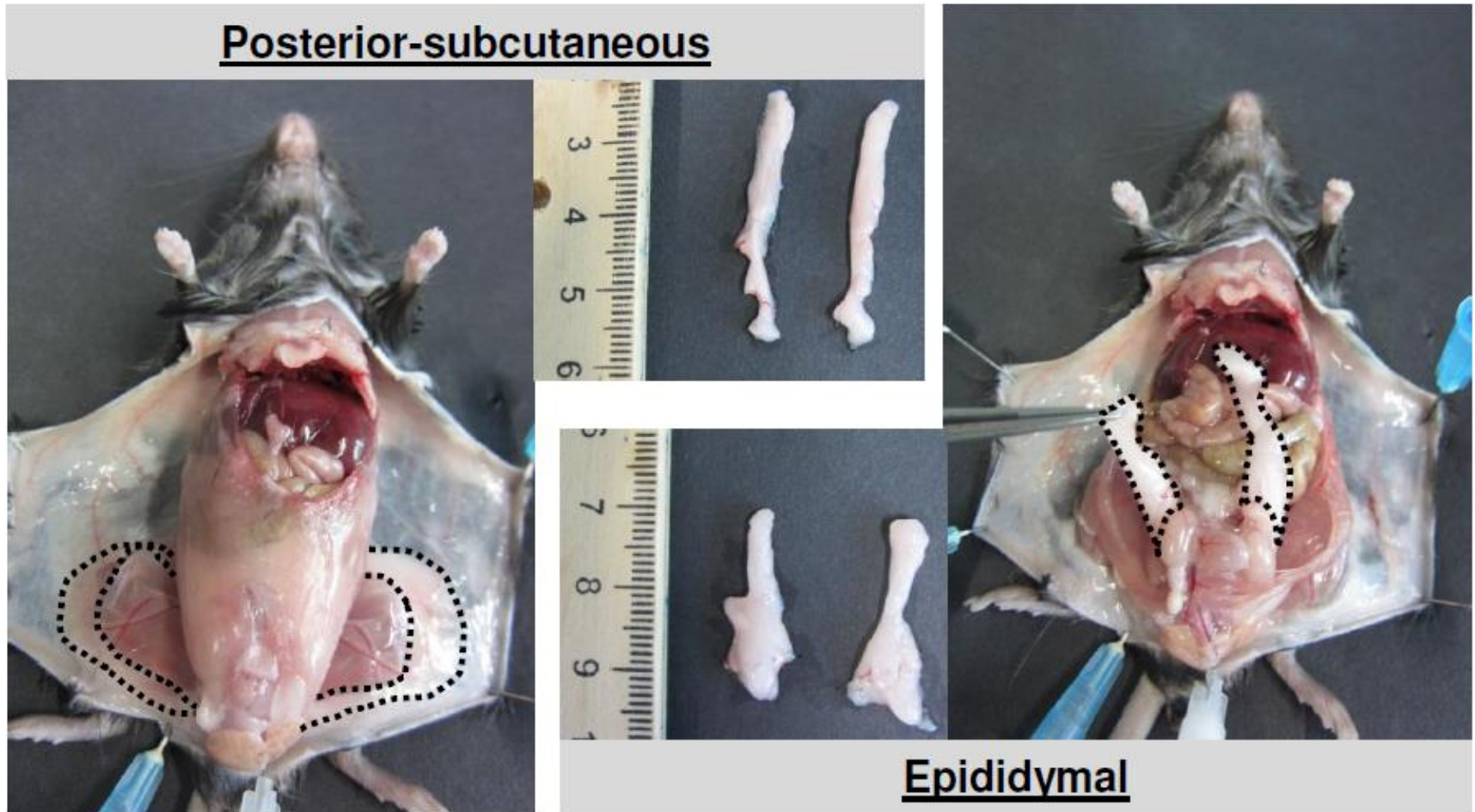
*Barcelona, March 21<sup>st</sup>, 2017*



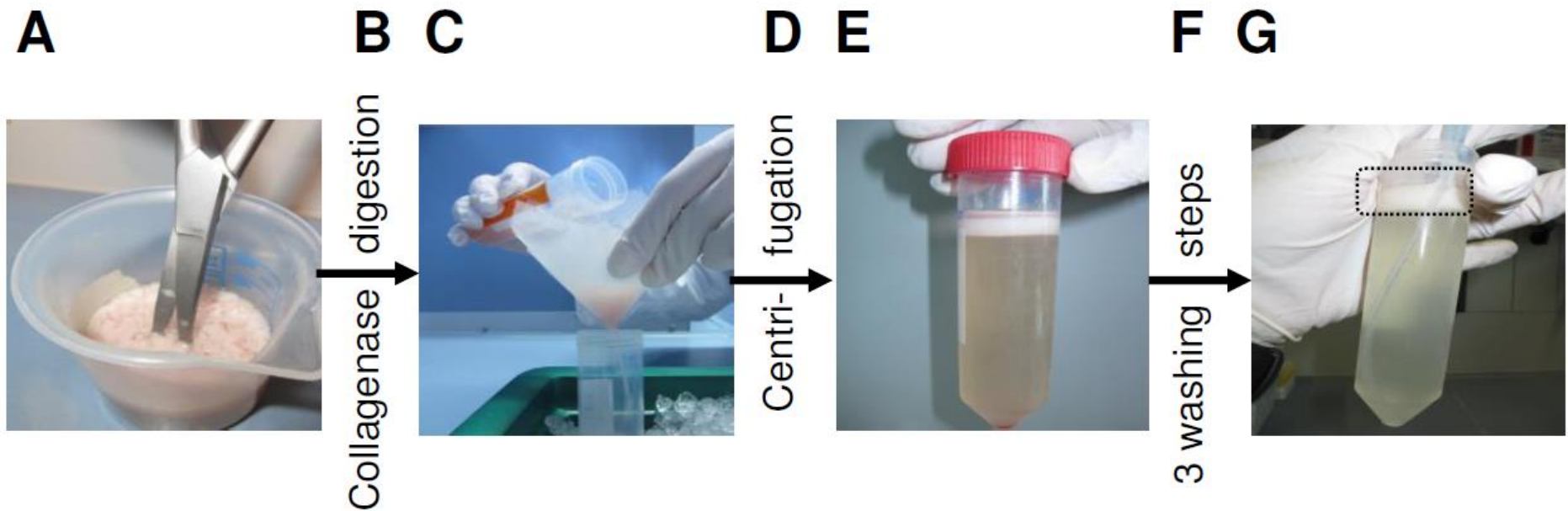
# Three options to characterize mitochondrial bioenergetics



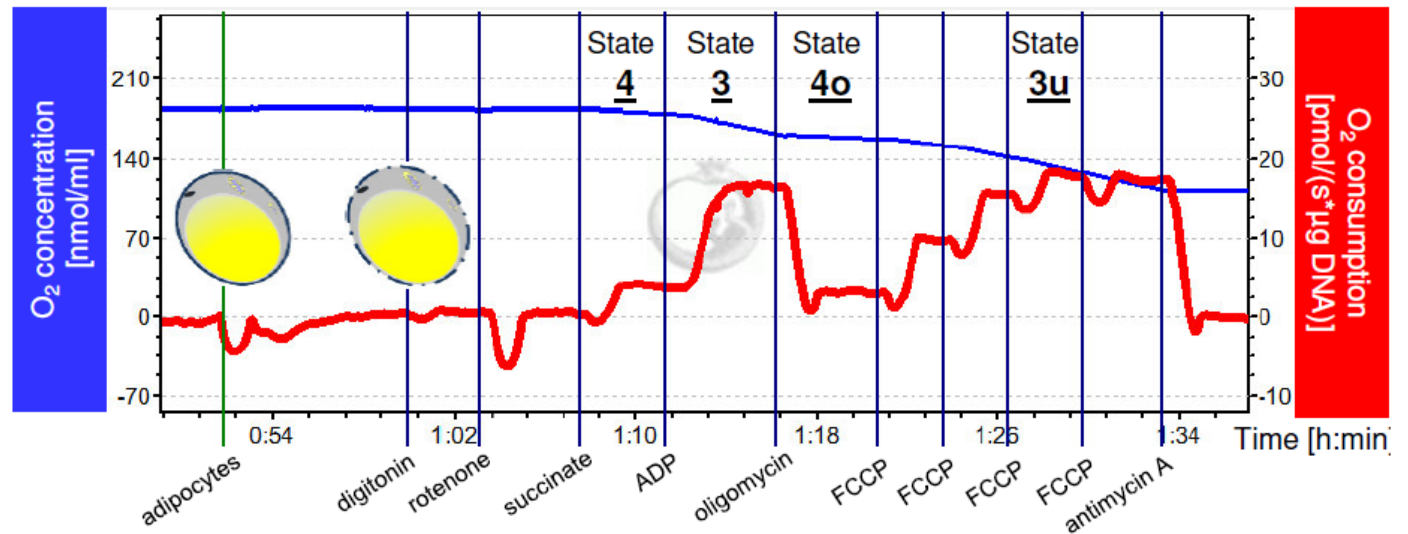
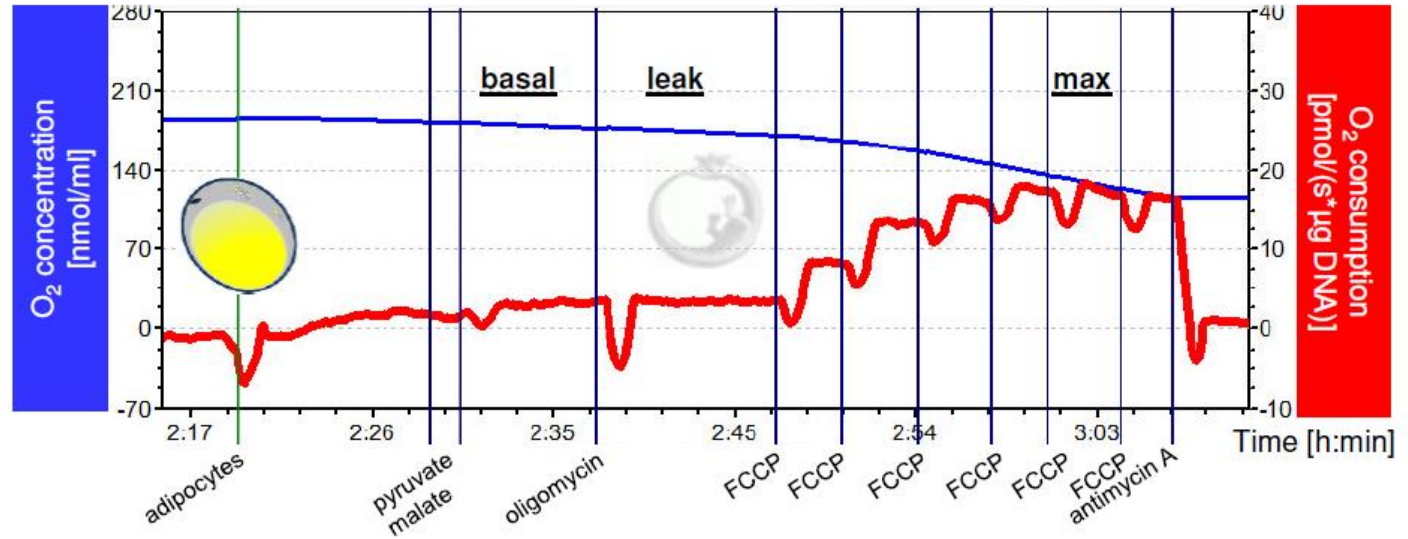
## Two source depots: subcutaneous vs. intraabdominal



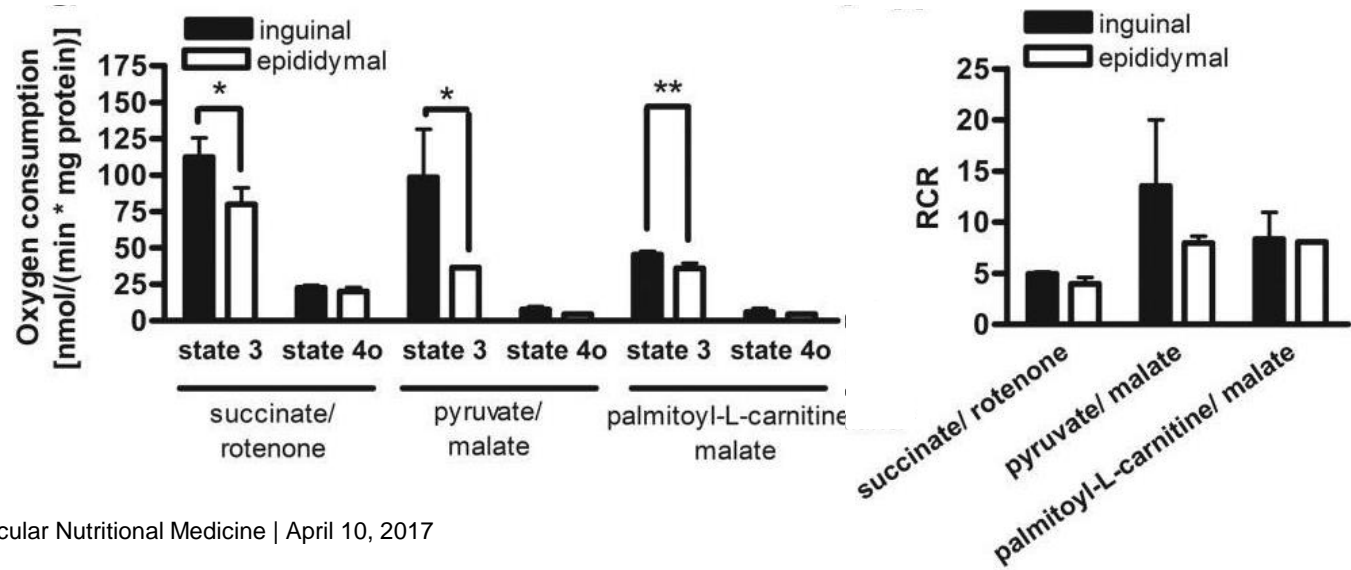
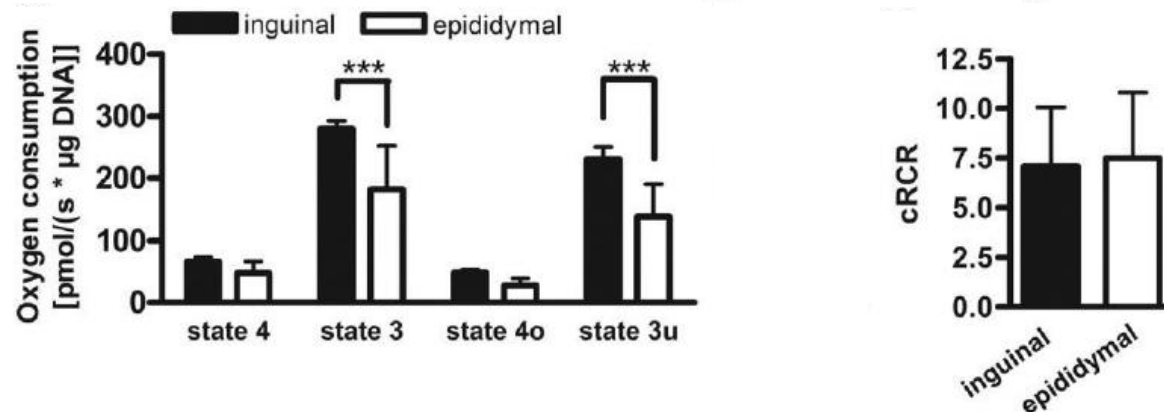
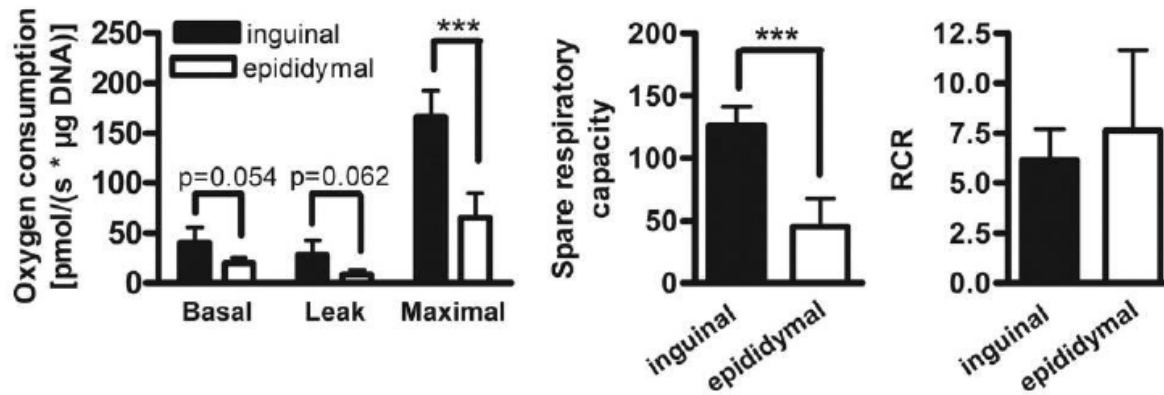
# Isolation of mature adipocytes



## Respirometry of intact vs. permeabilized fat cells

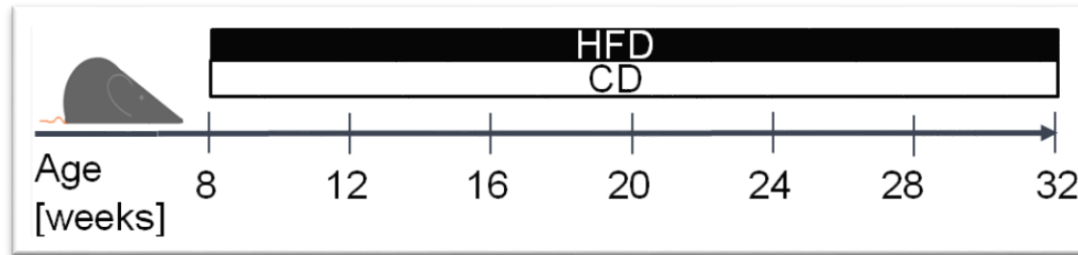






# Model: diet induced obesity + decreased glucose tolerance

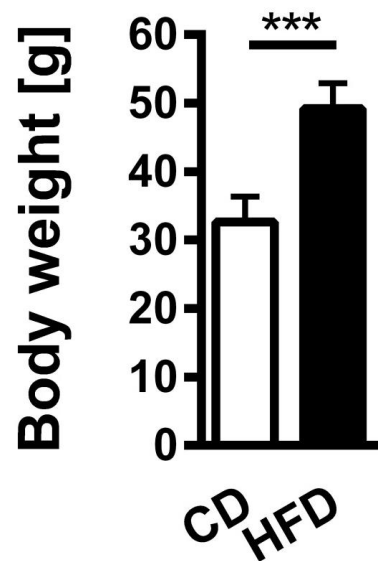
C57BL/6N  
mice



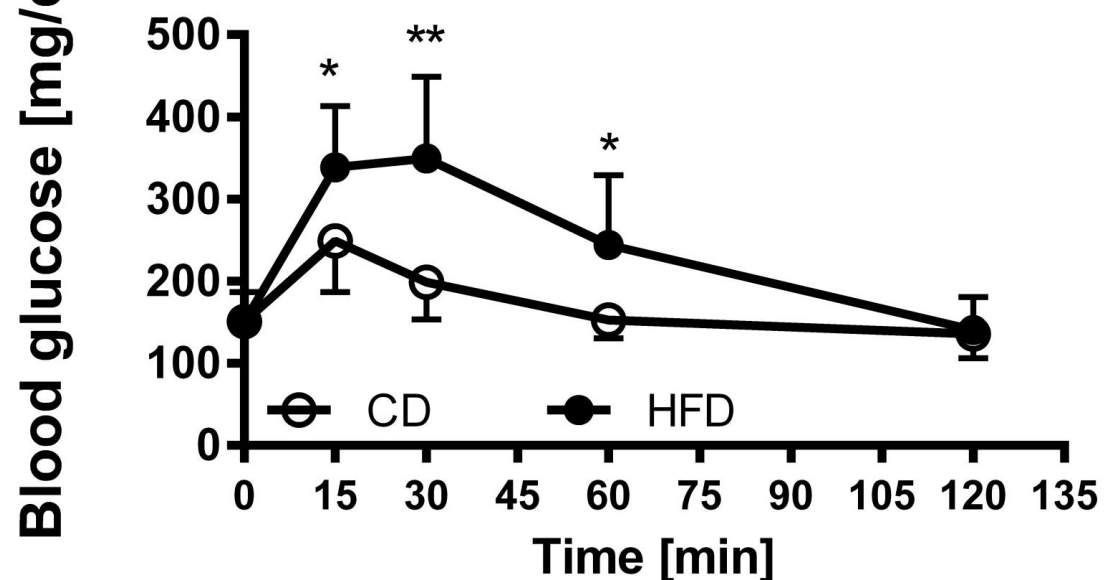
n = 38

n = 41

body mass

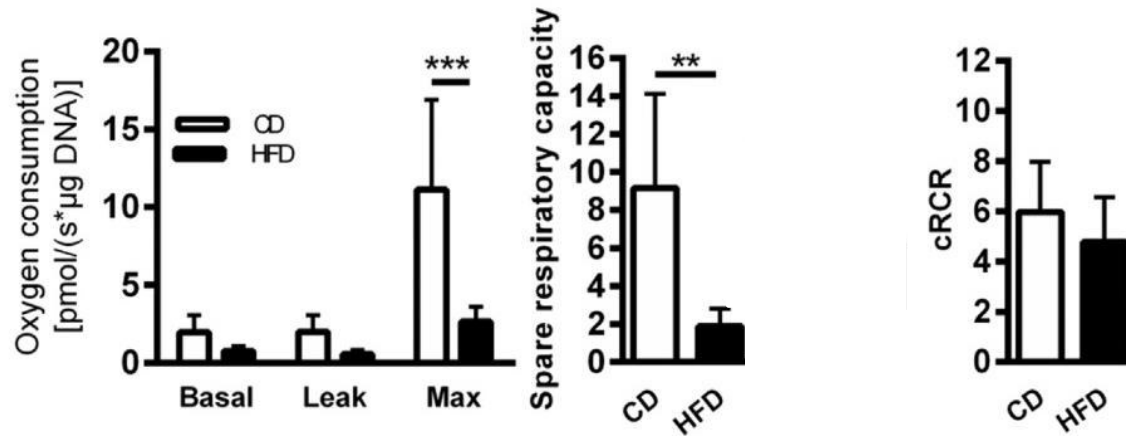


oral glucose tolerance test

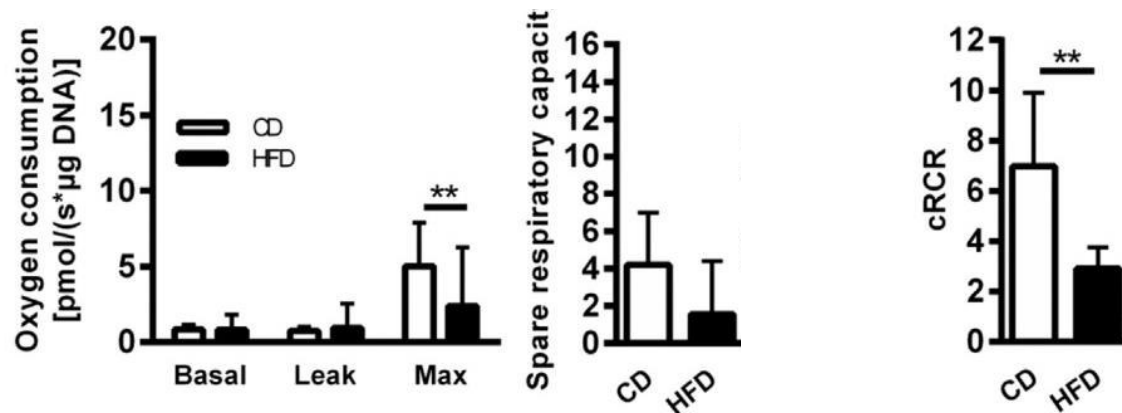


# Obesity reduces adipocyte OXPHOS capacity adipocytes

inguinal

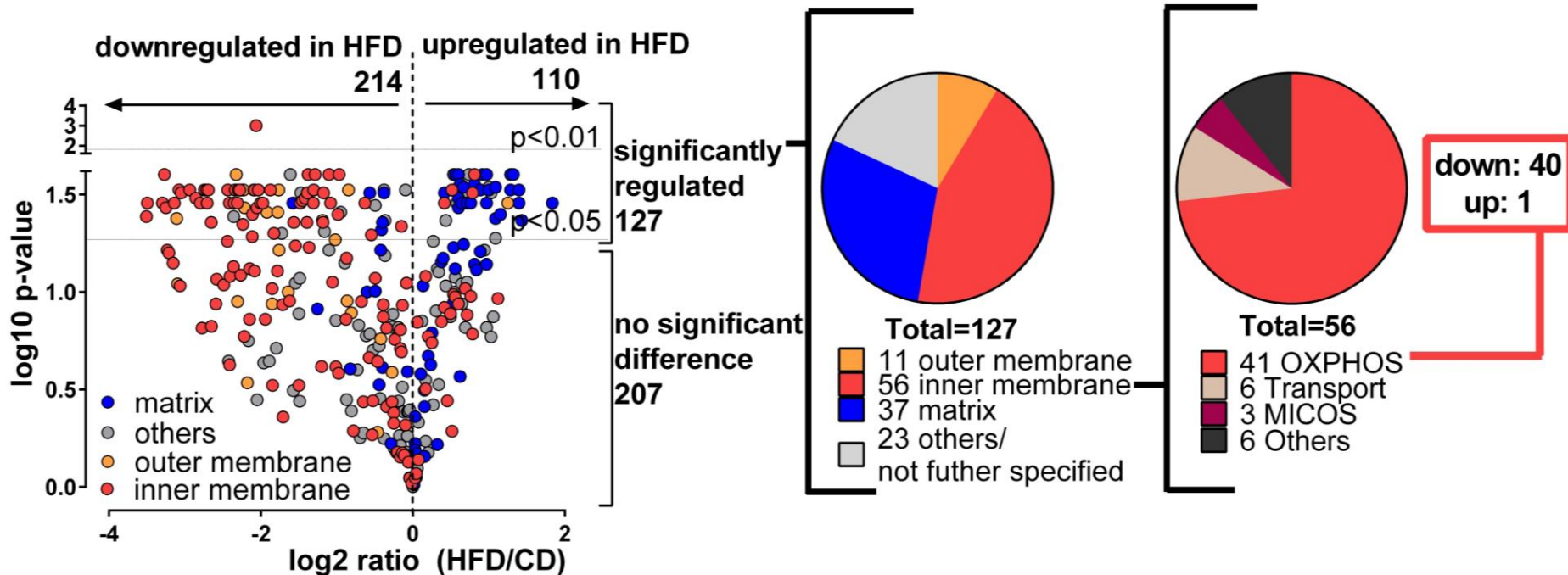


epididymal

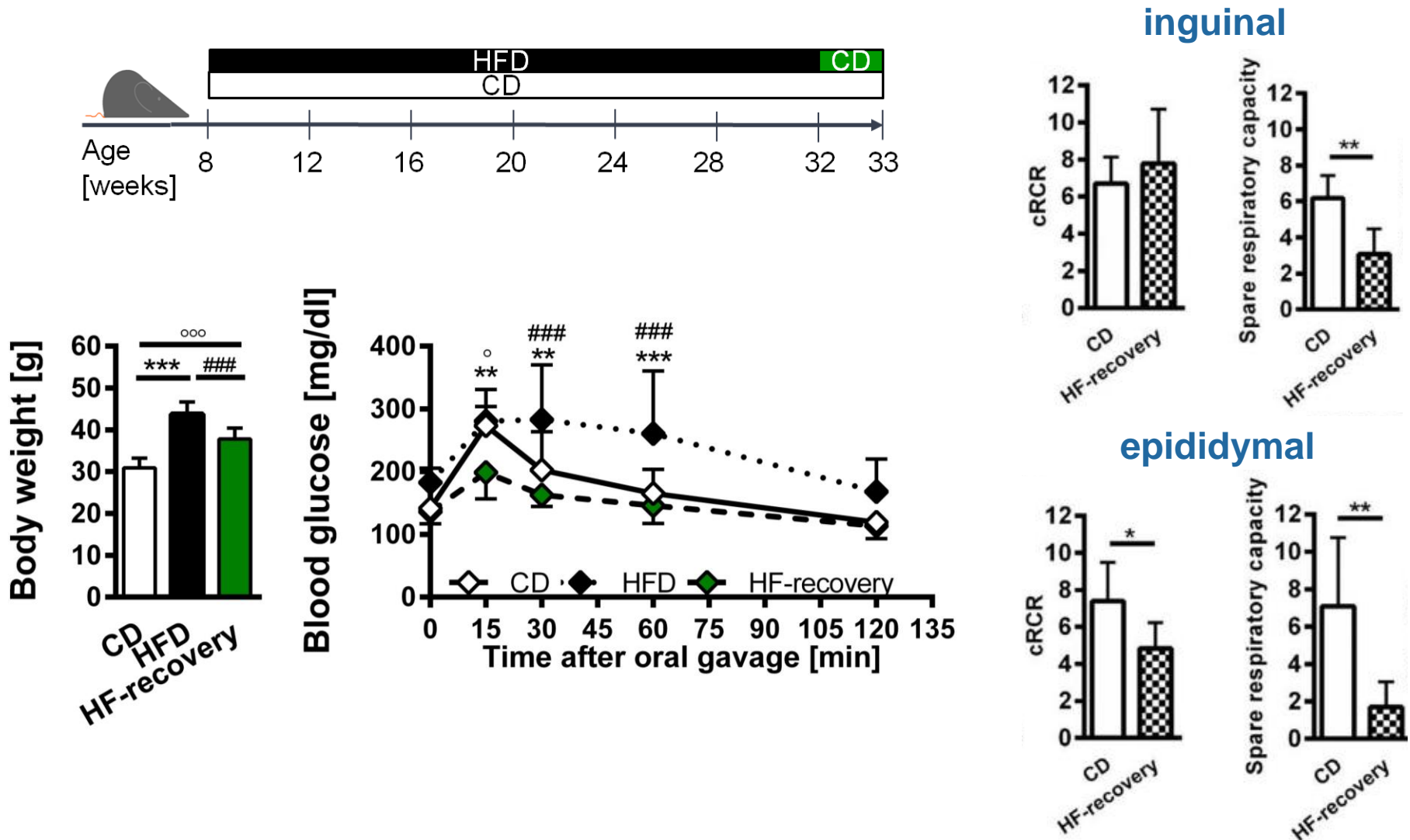




# Obesity reduces adipocyte OXPHOS capacity adipocytes



# Reduced OXPHOS capacity does not cause glucose intolerance



# Summary

Bioenergetic profiling on the level of...

-intact adipocytes

-permeab. adipocytes

-isolated mitochondria

...yields complementary data. There is no one optimal level.

Reduced OXPHOS capacity in adipocytes is a hallmark of obesity.

Reduced OXPHOS capacity in adipocytes does not cause glucose intolerance (alone).

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