Rapamycin enables specific inhibition of *de novo* protein synthesis

Alexander Rosén*, Emil Rindom and Mark Bayley.

Introduction

- Traditionally protein synthesis has been inhibited with cycloheximide
- Cycloheximide inhibits nearly all protein synthesis and is thus often terminal.
- *De novo* protein synthesis is mainly regulated by mTORC1
- A major upregulation of *de novo* protein synthesis takes plays during SDA.
- The compound Rapamycin is a specific inhibitor of mTORC1

Research questions

Can the post prandial increase in de novo protein synthesis be specific inhibited by rapamycin?

METHODS

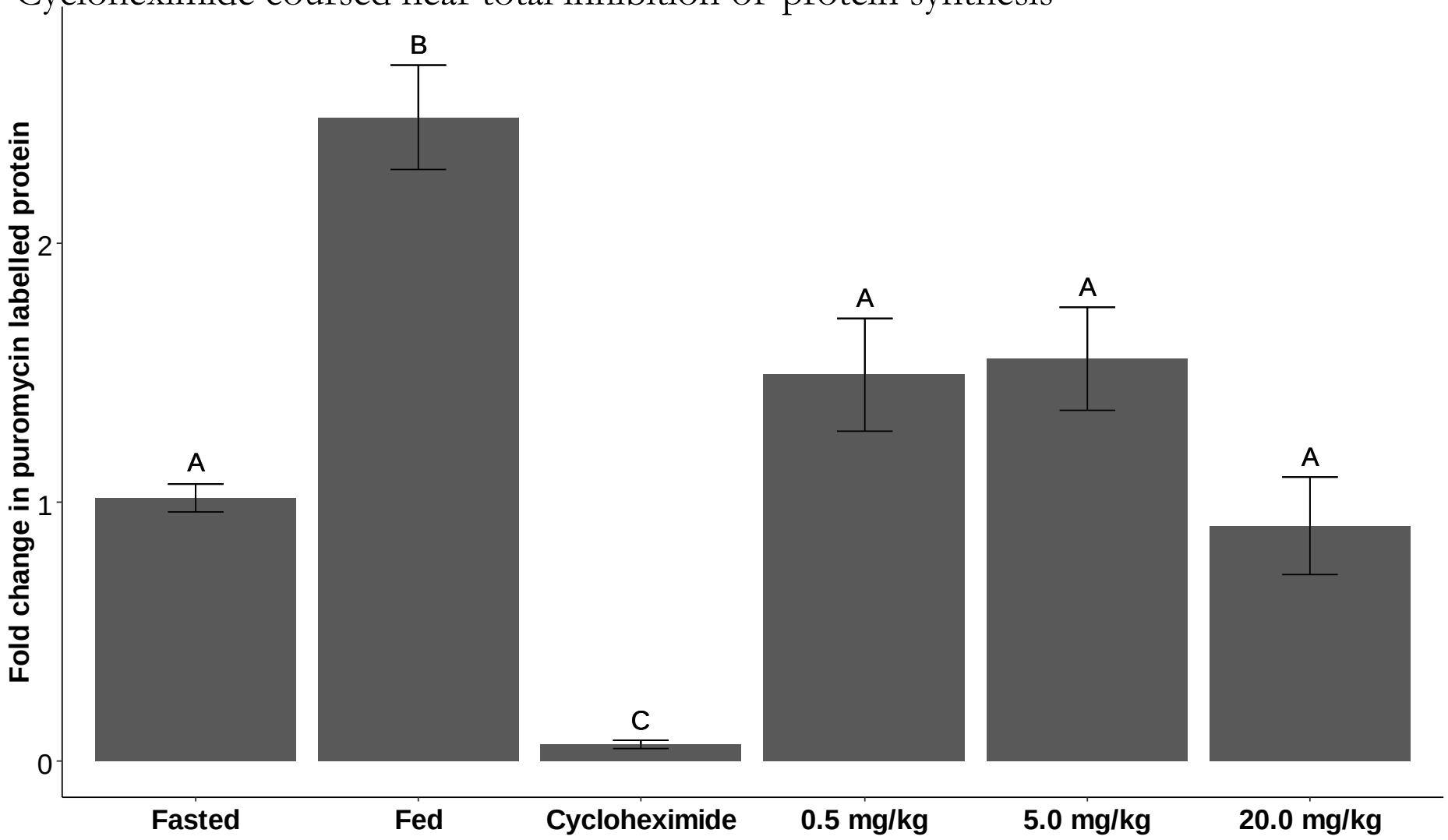
- Force feed 3% bodymas
- Intra-peritonal (IP) injection with Rapamycin
- Protein synthesis measured with IP puromycin using the SUnSET method

Experimental tanks

Experimental animal: *Perca fluviatilis*

RESULTS

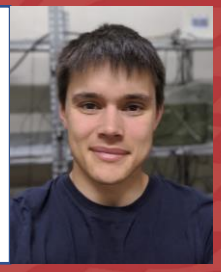
- Rapamycin significantly reduced the post prandial increase in protein synthesis.
- 20 mg/kg Rapamycin reduced post prandial protein synthesis to pre feeding level.
- Cycloheximide coursed near total inhibition of protein synthesis



DISCUSSION

- Rapamycin appears to allow for the specific inhibition of upregulated *de novo* protein synthesis while not affecting background turnover.
- The use of Rapamycin should replace Cycloheximide in studies examining only upregulated *de novo* protein synthesis such as during the SDA response.

***Presenting author**
Alexander Rosén
PhD-student
Section for Marine Living Resources
DTU aqua
Denmark



DTU Danmarks Tekniske Universitet