Mitochondrial abundance and functionality in cattle undergoing Compensatory Growth

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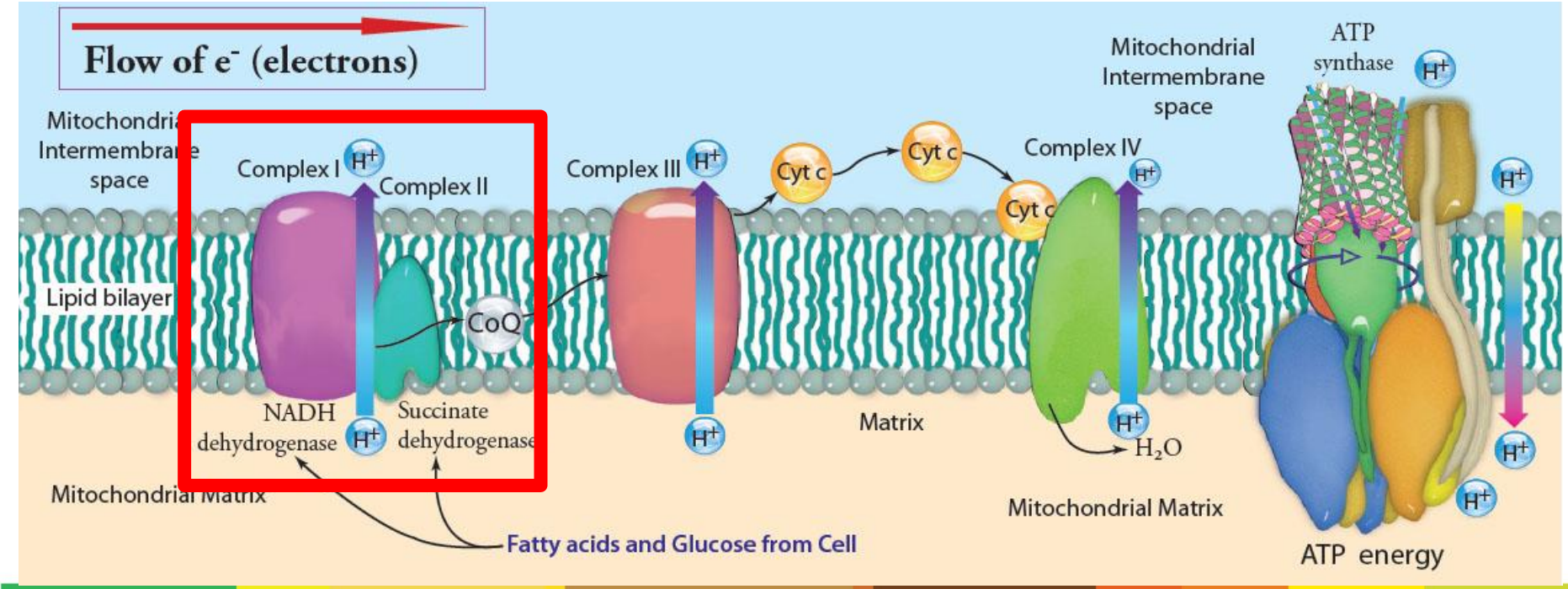
Introduction

- Compensatory growth :
  - *'A physiological process whereby an animal has the potential following a period of under-nutrition to undergo enhanced growth upon re-alimentation, enabling it to achieve its pre-determined inherent growth rate'* (*Hornick et al., 2000*)
- Animals undergoing compensatory growth have a higher FE
- Exploitation of the compensatory growth phenomenon provides a method to reduce feed costs
- Liver is an important metabolic organ :
  - Alterations in the size of the liver have been shown to be directly proportional to dietary intake (*Johnson et al., 1990*)
  - During dietary restriction and subsequent re-alimentation, the liver has repeatedly been shown to be one of the most responsive tissues (*Keogh et al., 2015)*

Mitochondria

- 90% oxygen consumption in body and produce bulk of cellular energy(ATP)
- Key regulators in energy metabolism
- Structure/Function of IMM respiratory chain complexes important for optimal function of mitochondria



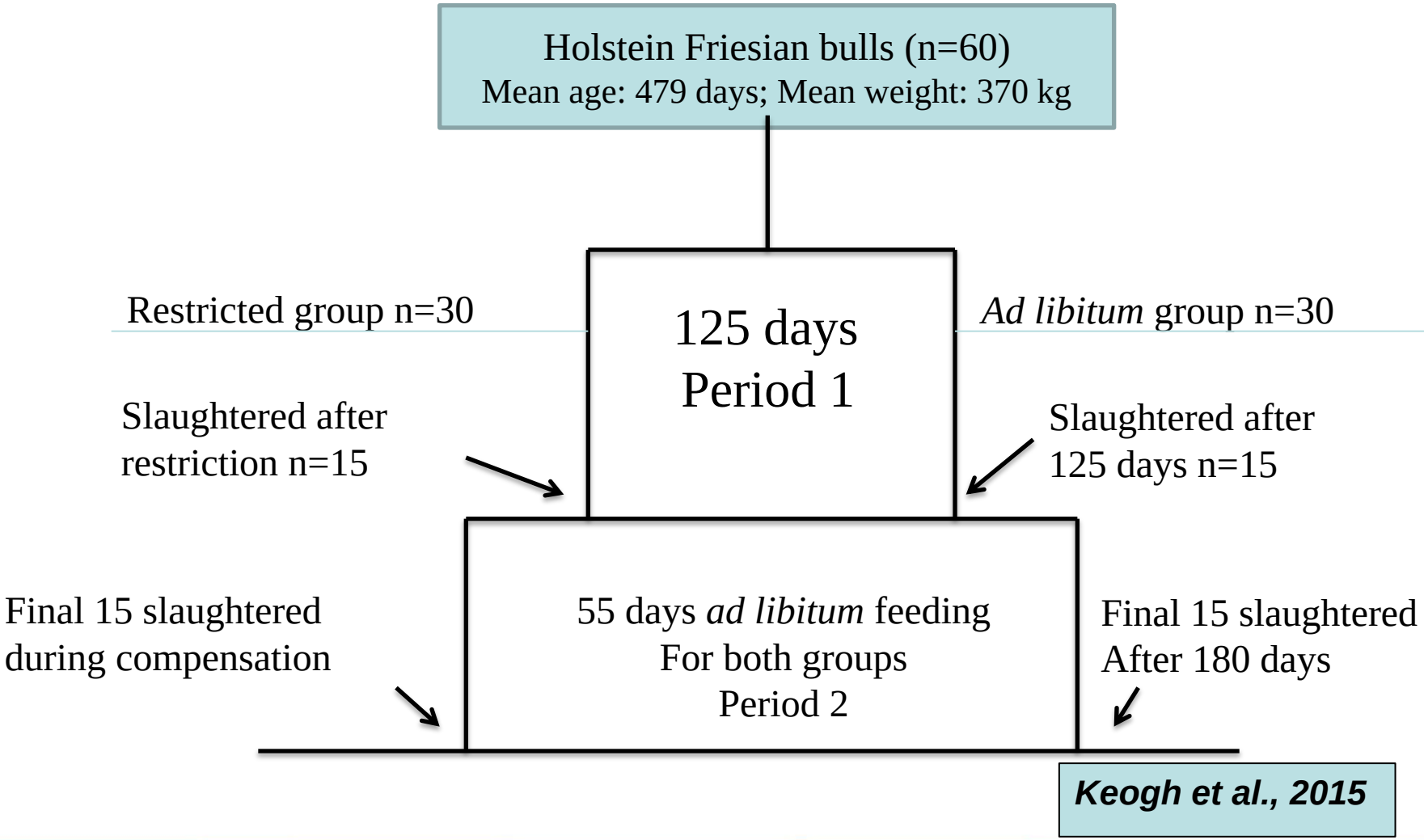
Mitochondria & Compensatory Growth

- Little is known about the role of mitochondrial abundance and complex functionality in relation to animals undergoing CG
- Connor et al. (2010) suggested that improved mitochondrial function may be contributing to CG
- Relationship between CG and Ox-phos in muscle (*Keogh et al.,2016*)

<u>Objective of this study:</u>

- To investigate the premise that mitochondrial abundance and function are contributing to compensatory growth in liver tissue of cattle

Materials and methods - Experimental design



Materials and methods (*Keogh et al., 2015*)

Live animal measurements:

- Bodyweight
  - Recorded every two weeks during Period 1 and every week during Period 2
- Amount of feed offered based on each animals individual bodyweight
- Ultrasonic muscle depths

Slaughter measurements:

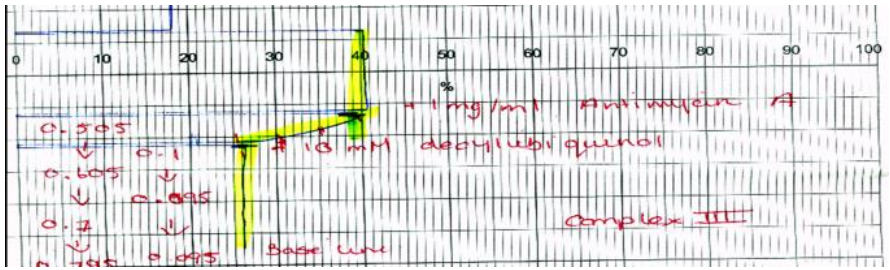
- Non-carcass component weights: liver
- Muscle and liver tissue samples collected at slaughter

Statistical analysis:

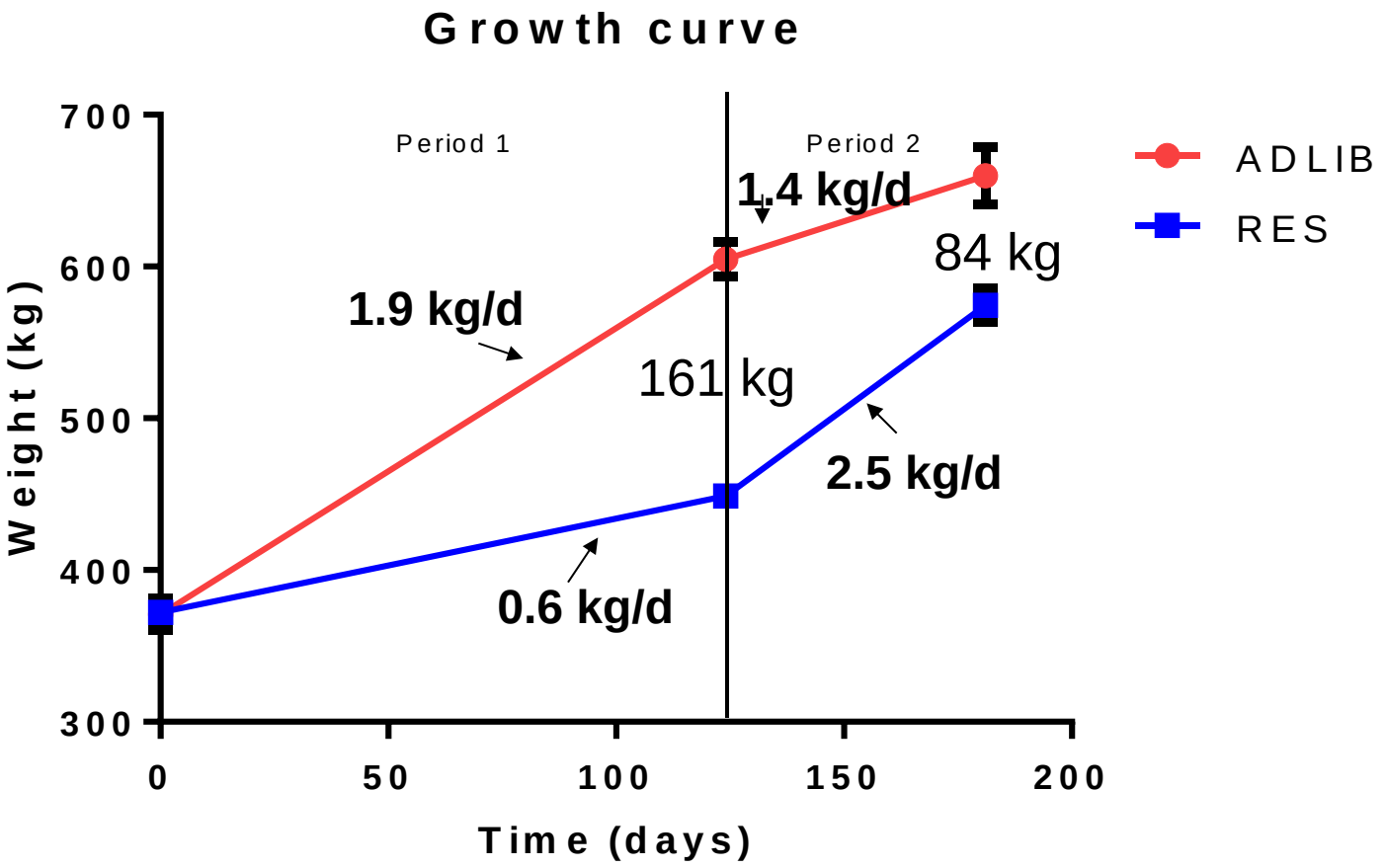
- Data were analysed using the MIXED procedure of SAS
- Main effects: treatment, period, treatment x period
- Random effect: block

Materials and Methods- Laboratory analyses

- Liver homogenates prepared and total protein conc measured – Pierce BCA protein Assay kit
- Enzymatic assays performed using Shimazdu UV-Vis spectrophotometer
  - Citrate Synthase assay
  - Complex 1 activity assay



Results (*Keogh et al., 2015*)



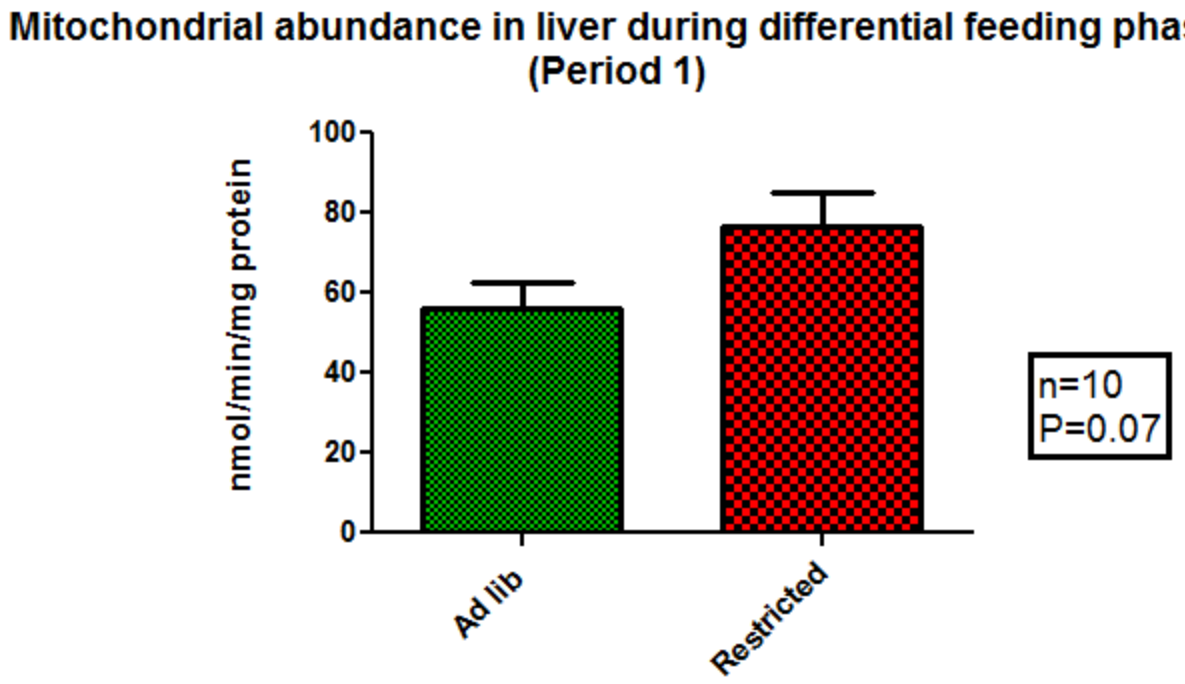
Animals compensated for 48% of their dietary restriction in 55 days of re-alimentation

Results (Keogh et al., 2015)

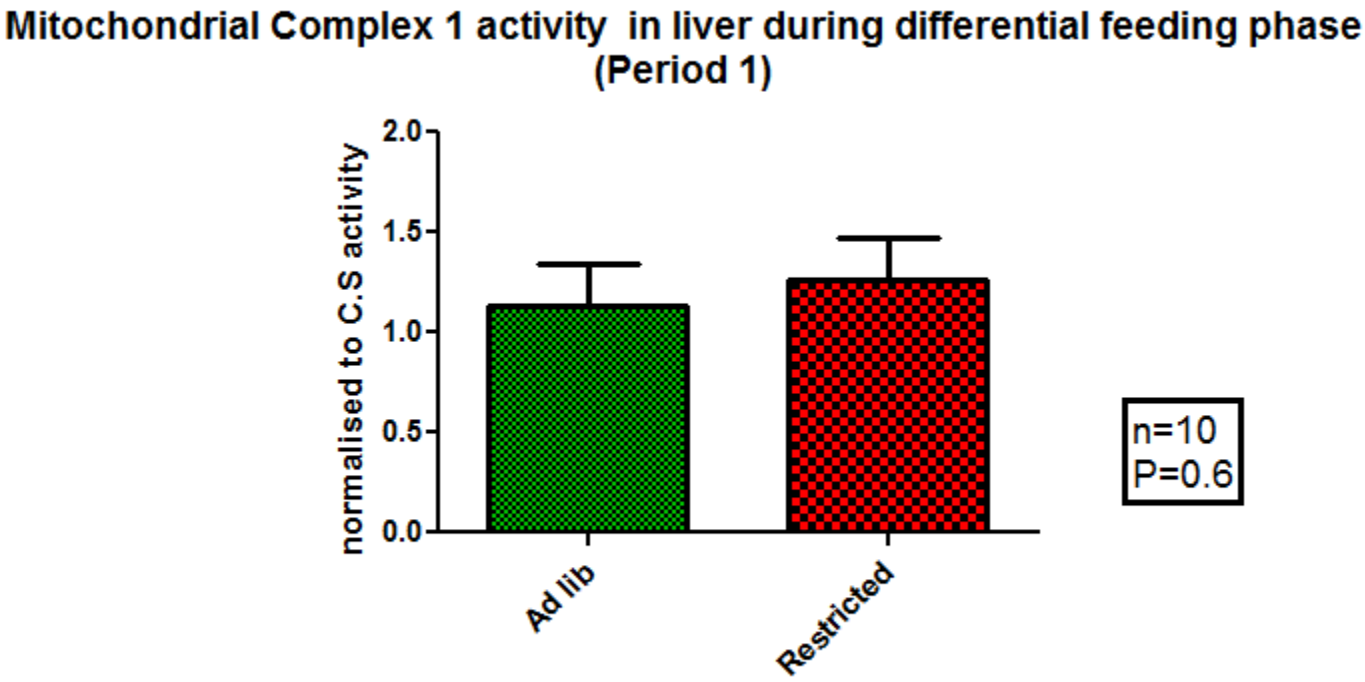
	RES		ADLIB			Significance		
	Period 1	Period 2	Period 1	Period 2	SEM	T	P	T*P
Non-carcass components								
Head	0.0366	0.0344	0.0319	0.0315	0.0005	***	*	†
Liver	0.0096	0.0147	0.0135	0.0131	0.0004	**	***	***
Lungs	0.0066	0.0071	0.0060	0.0057	0.0004	*	NS	NS
KCF	0.0075	0.0102	0.0113	0.0149	0.0006	***	***	NS
Full reticulo-rumen	0.0999	0.0911	0.0883	0.0797	0.0037	**	*	NS
Empty reticulo-rumen	0.0169	0.0219	0.0195	0.0206	0.0007	NS	***	**
Intestines	0.0437	0.0533	0.0472	0.0532	0.0021	NS	***	NS
Omasum	0.0096	0.0137	0.0146	0.0124	0.0006	**	†	***
Front legs	0.0089	0.0084	0.0075	0.0079	0.0001	***	NS	***
Hind legs	0.0124	0.0118	0.0106	0.0106	0.0003	***	NS	NS
Hide	0.0797	0.0803	0.0774	0.0755	0.0016	*	NS	NS

- Metabolically active organs (liver)were most affected by dietary restriction, subsequently compensating first during re-alimentation

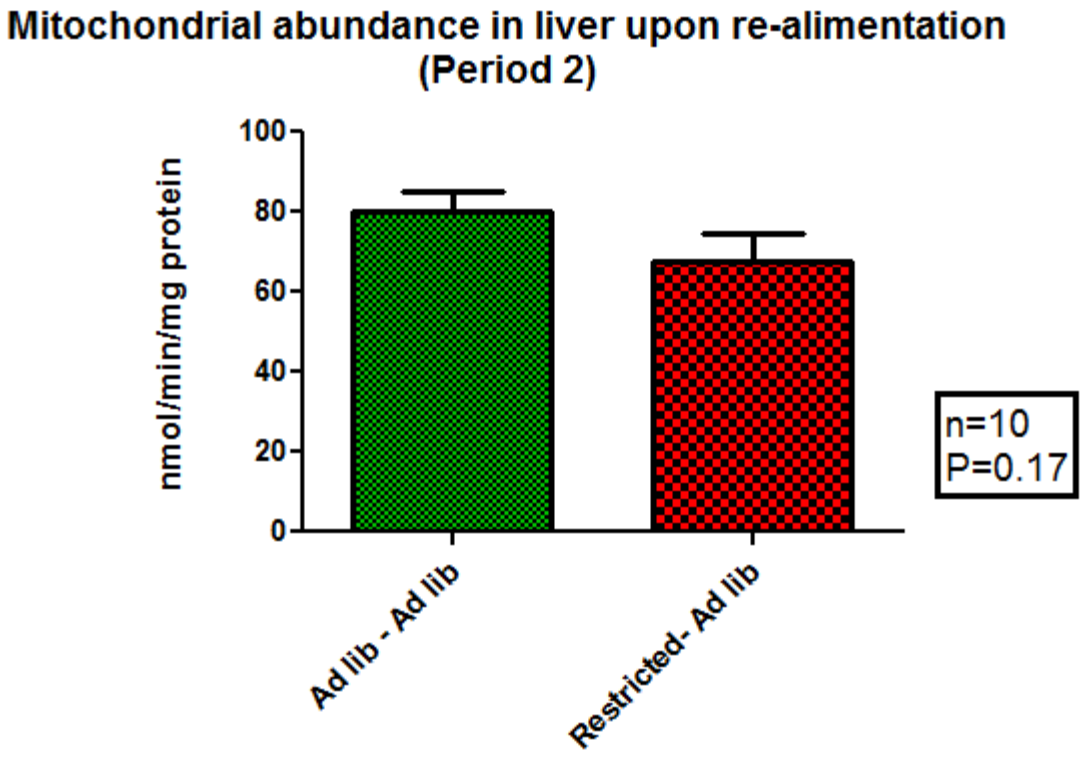
Results – Mitochondrial abundance



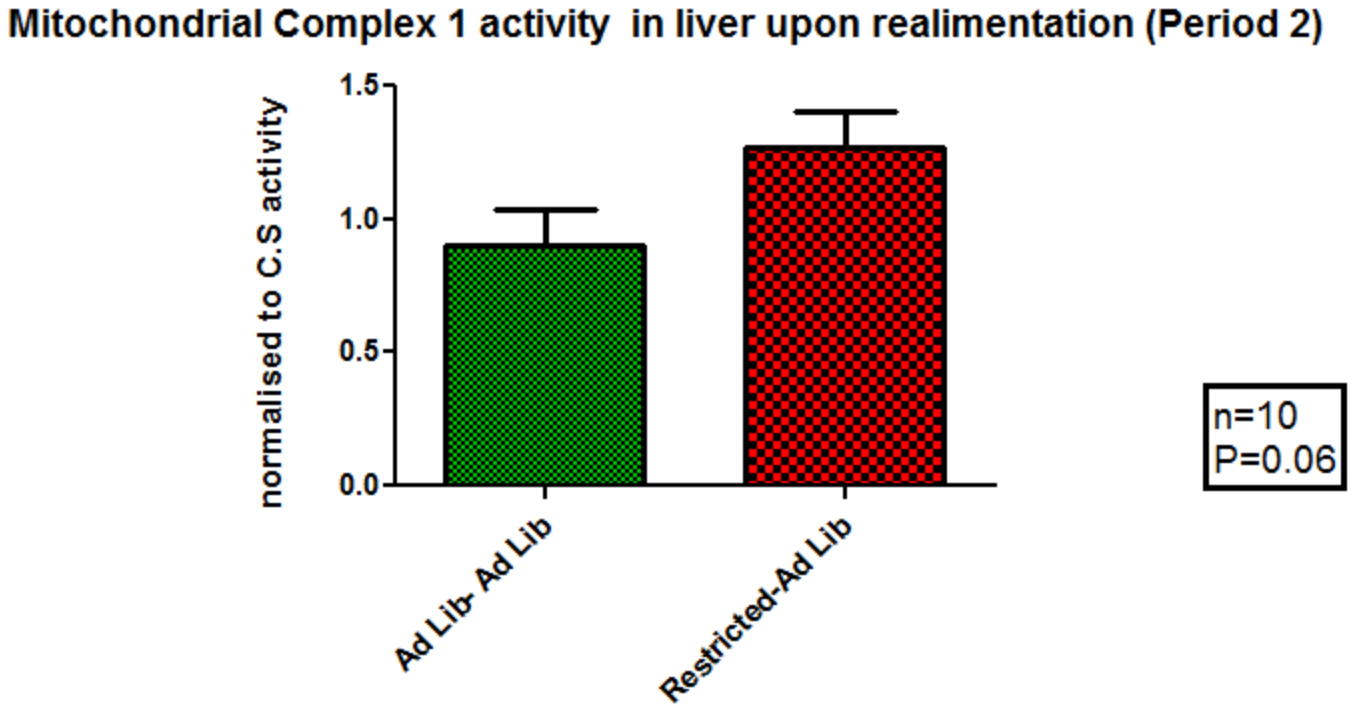
Results- Functionality



Results – Mitochondrial abundance



Results- Functionality



Results

Oxidative phosphorylation RNAseq results (liver)(Keogh et al., 2015)

Analysed within treatment group (RES Period 2 compared to RES Period 1)

- Oxidative phosphorylation was significant ($P < 0.05$)
  - ATP5S, NDUFB9, MT-CYB, COX7B, MT-ND5, UQCR10, NDUFA1, NDUFB7, MT-ND3, ATP5J, MT-CO3, MT-ND2, MT-ND1, SDHB, ATP5O, COX17, COX6B1, UQCRH, NDUFA7, UQCR11, NDUFS8, MT-ATP6, MT-CO1, COX4I1, NDUFB3, SDHA, NDUFB8, COX5B, NDUFA2, NDUFAB1, NDUFB10, NDUFS4, NDUFA4, NDUFB6, UQCRQ, CYB5A, ATP5L, NDUFA3, NDUFA11, ATP5H, ATP5J2, COX8A, MT-CO2, NDUFB1, NDUFB11, NDUFB5, NDUFB4

Overall conclusions

1. Animals had a potential increase in capacity for mitochondrial biogenesis when restricted in order to keep up with an increasing energy demand ,which levels off when animals undergo re-alimentation
2. Greater capacity for mitochondrial energy production was apparent once animals underwent re-alimentation

Science Foundation Ireland sfi For what's next

Thank you!

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Agriculture and Food Development Authority