The Effect of Dietary Lipids on the Pathology of Salmon Pancreas Disease

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Background

- The application of the correct dietary profile is important in maintaining health and welfare in aquaculture
- Previous studies indicated that PD was a nutritional defect associated with the vitamin E-selenium deficiency (Ferguson *et al*., 1986) and deficiency of these two nutrients may be important in the pathogenesis of the PD condition (Bell *et al*., 1987).
- More recently it was suggested that oxidative stress/lipid peroxidation may induce the pathology in SAV infections (Ferguson 2006).

- The role of PUFA’s in managing fish health is a growing area of research interest
- Alteration of PUFA (n-6/n-3) effects fatty acid composition of fish tissues as well the fish immune response
- However, this is has not been fully explored

Aim

To assess whether the application of

- low or high lipid with low or high antioxidant (Experiment 1)
- differing PUFA ratios (Experiment 2)

in the diet would help reduce the severity of lesions and improve the recovery of the fish from SAV 1 infection

Experiment 1

Experimental Design

Rainbow trout were fed with **five different diets** (1 control {E} and 4 experimental {A-D}) for 6 weeks

Dietary information

	Lipid level	Lipid (%)	Antioxidant level
E	Control	22%	Low antioxidants
A	Low Lipid	18%	Low antioxidants
B	Low Lipid	18%	High antioxidants
C	High Lipid	26%	Low antioxidants
D	High Lipid	26%	High antioxidants

Experimental fish were reared in triplicate tanks and controls were in a single tank.

Fish were infected i.p. with SAV 1

Samples were obtained at Day 0 and 5, 15, 30 d.p.i.

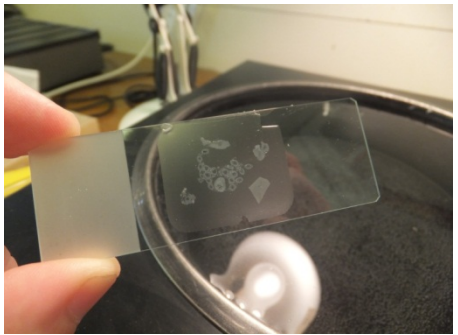
SAMPLE COLLECTION for both experiments

- Weight/Length
- Tissues for histopathology
  -Pancreas, Heart, Skeletal, muscle, Kidney, Liver, Spleen, Gill
- Tissues for viral load study
- Tissues for total fatty acids, TBAR analysis
- Serum for CPK, Vit E, Se analysis

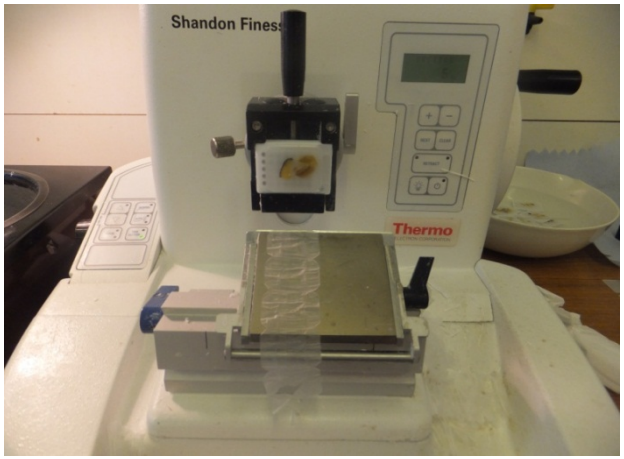
- Tissues embedded in paraffin wax sectioned at 5µm and stained with H&E

- Tissue damage was measured according to a scoring system developed by Christie *et al* 2007; modified by Herath, 2010.

- Scoring system (blind scoring) Degeneration
  Inflammation

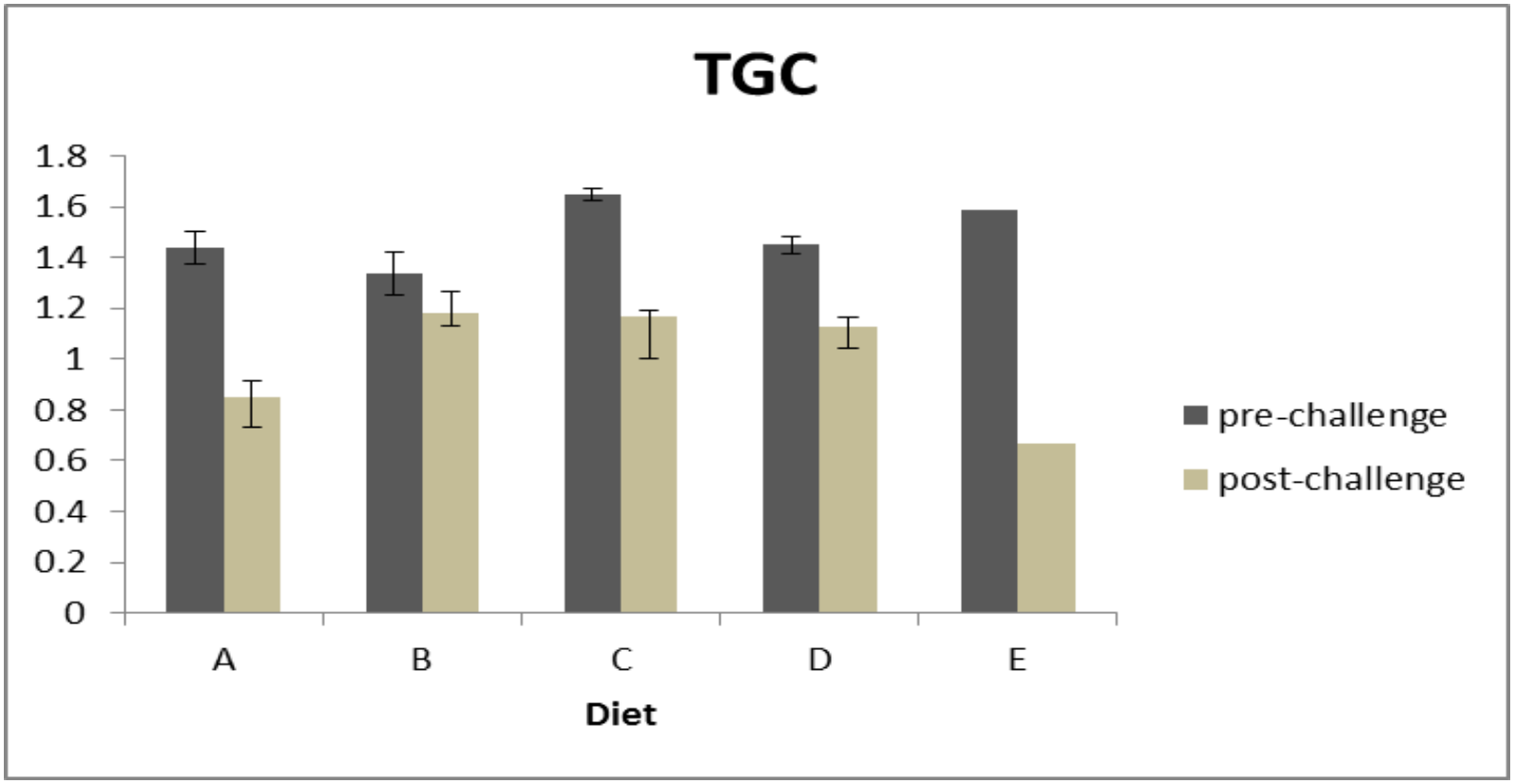


0-3
0 Normal
1 Mild
2 Moderate
3 Severe



Results

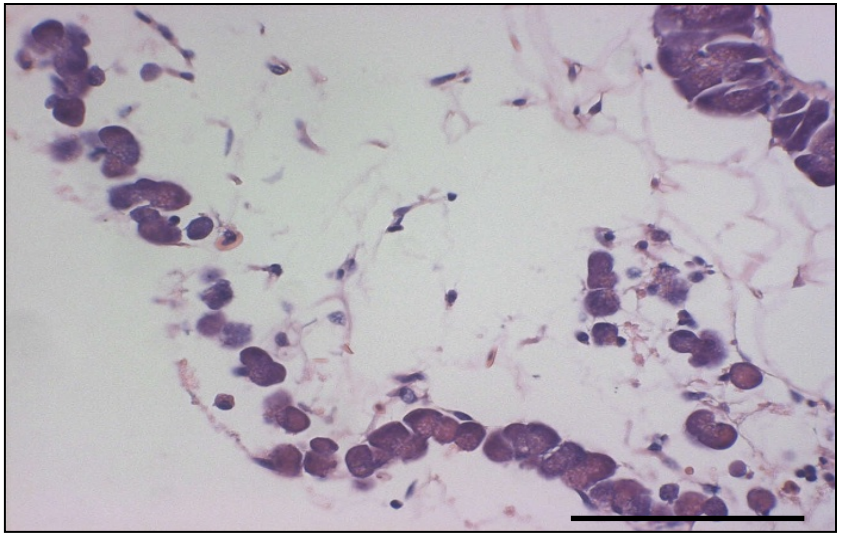
Thermal Growth Coefficient

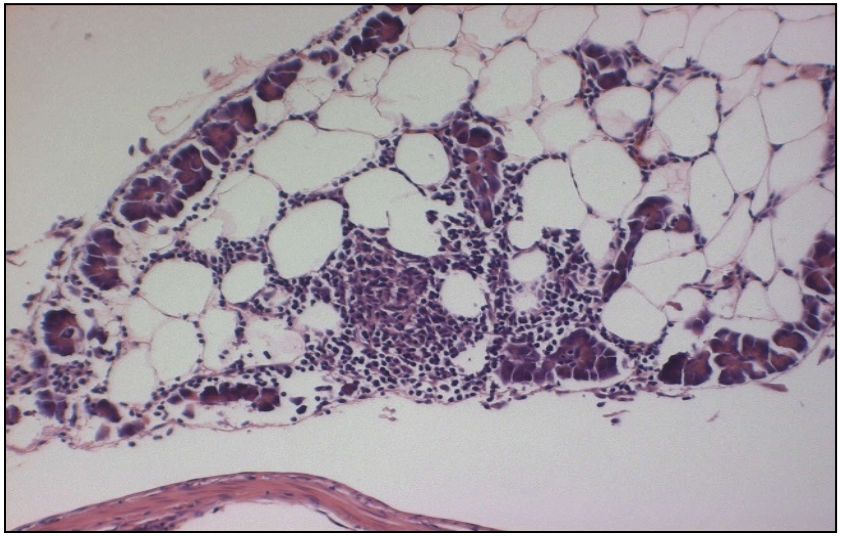


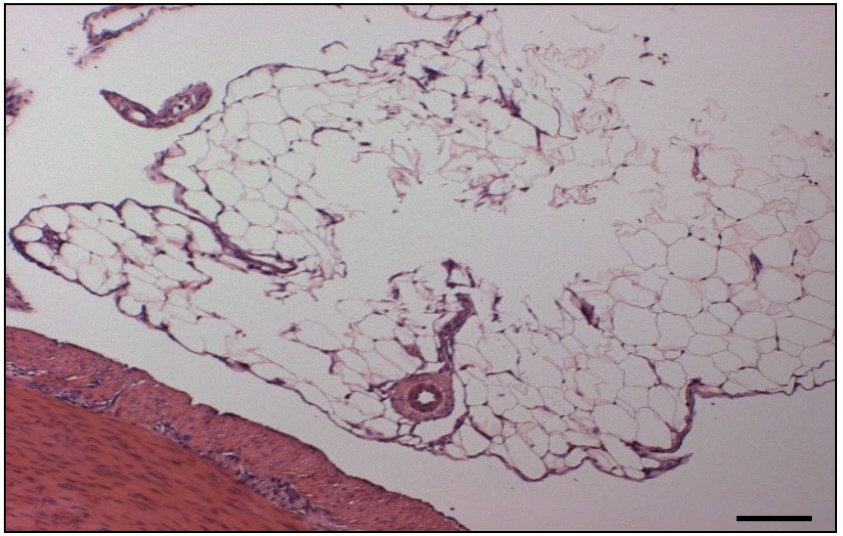
Diet	Lipid level	Antioxidant level
E	Control	Low antioxidants
A	Low Lipid	Low antioxidants
B	Low Lipid	High antioxidants
C	High Lipid	Low antioxidants
D	High Lipid	High antioxidants

Results

Histopathology findings - pancreas

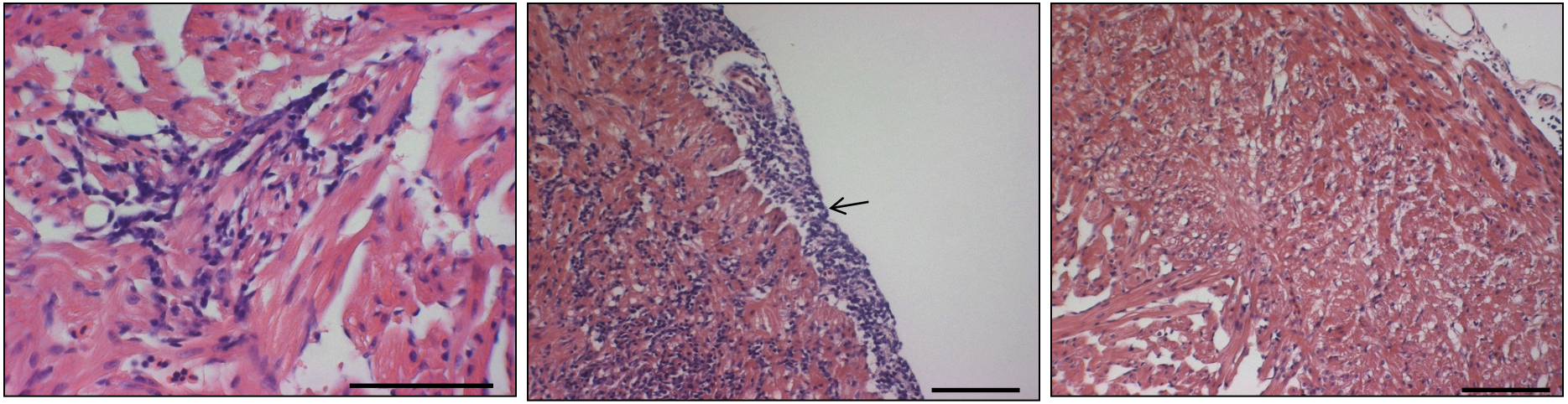






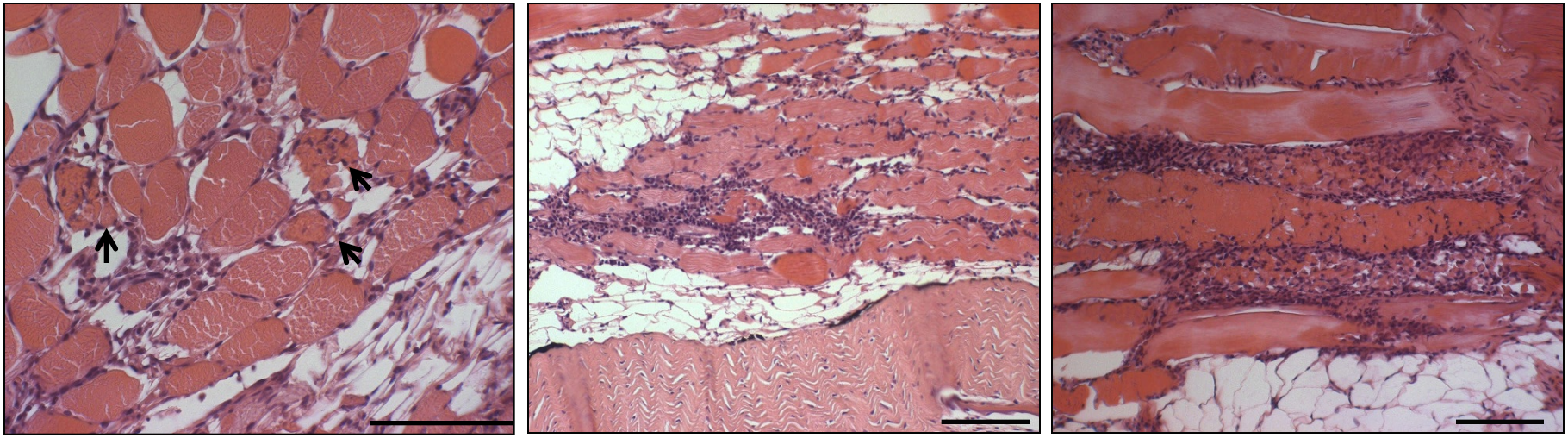
Results

Histopathology findings – Heart



Results

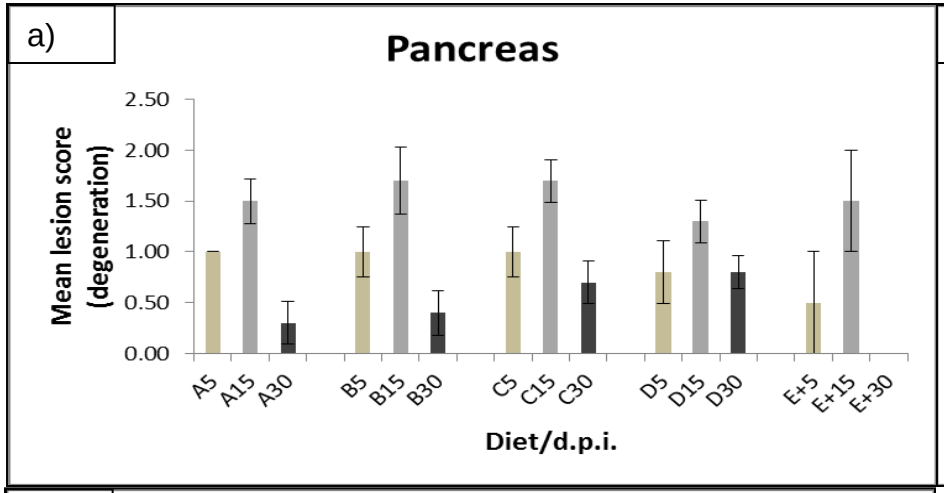
Histopathology findings – Skeletal muscle



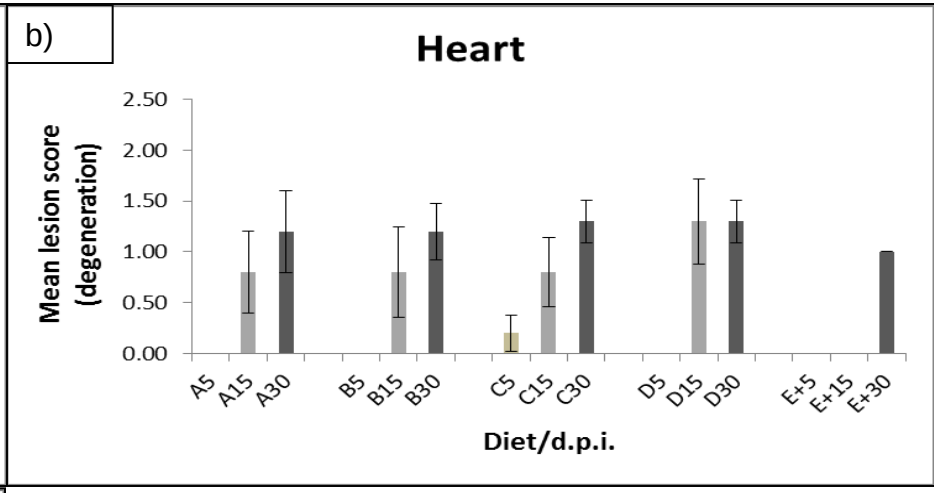
Results

Mean lesion score for degeneration (0-3)

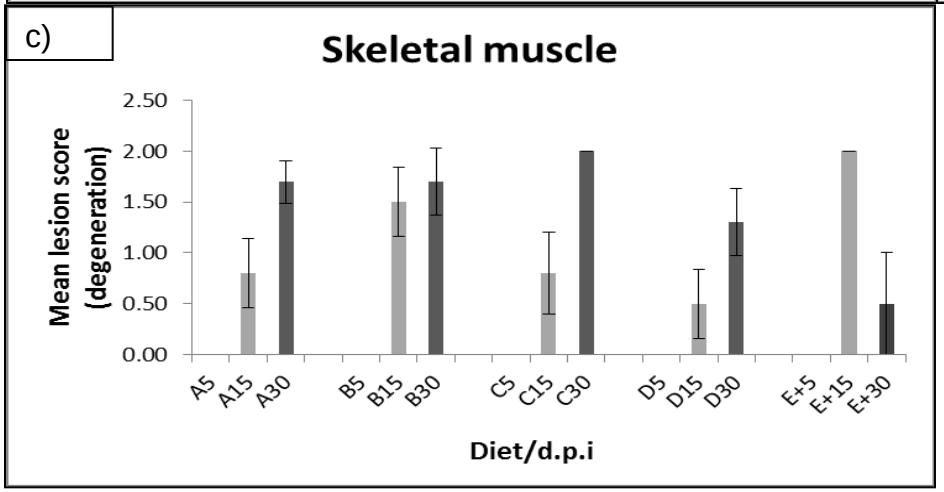
a)



b)



c)

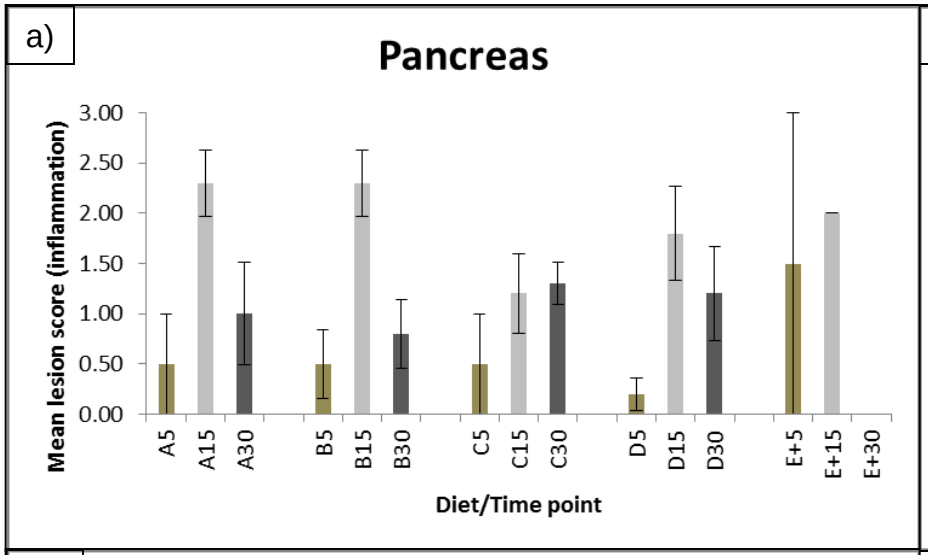


Diet	Lipid level	Antioxidant level
E	Control	Low antioxidants
A	Low Lipid	Low antioxidants
B	Low Lipid	High antioxidants
C	High Lipid	Low antioxidants
D	High Lipid	High antioxidants

Results

Mean lesion score for inflammation (0-3)

a)



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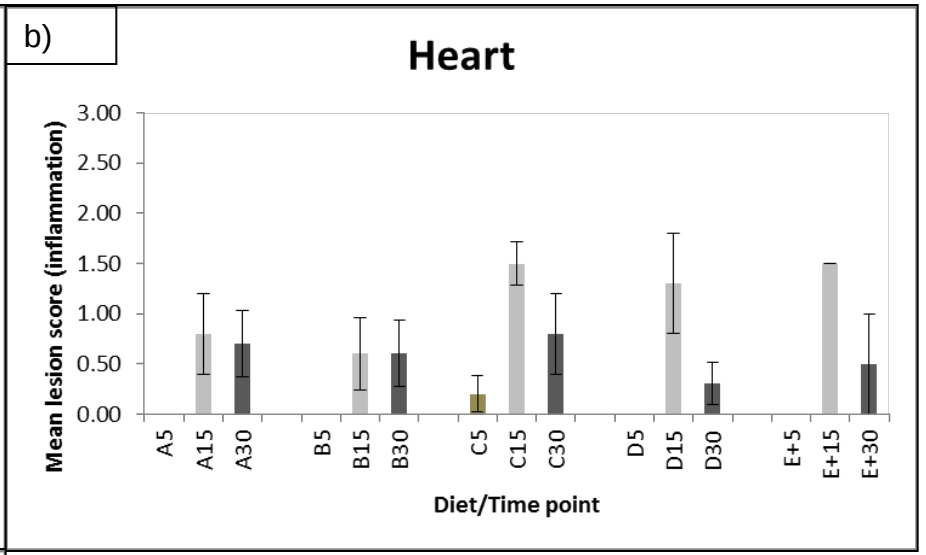

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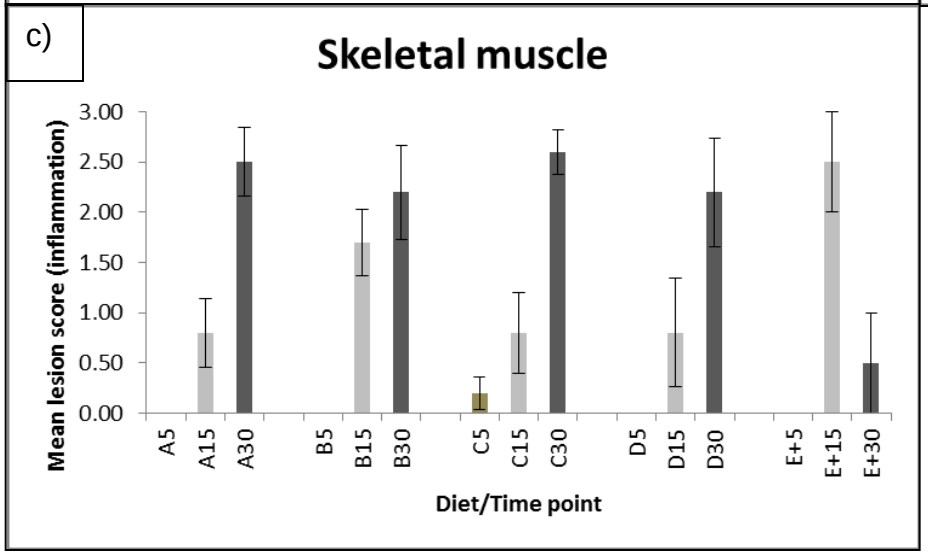


Diet	Lipid level	Antioxidant level
E	Control	Low antioxidants
A	Low Lipid	Low antioxidants
B	Low Lipid	High antioxidants
C	High Lipid	Low antioxidants
D	High Lipid	High antioxidants

Discussion

Experiment 1

- No mortalities were recorded throughout the period of the experiment.

- Fish fed with low, or high lipid or diets supplemented with low, or high antioxidant all showed evidence of infection with PD.

- Fish fed with different diets have similar patterns of pathology.

Discussion

Experiment 1

- Increasing antioxidant concentrations and altering high and low fat in the diet could be a solution ?
  - Need further information
  - Role of lipid peroxidation in viral pathogenesis

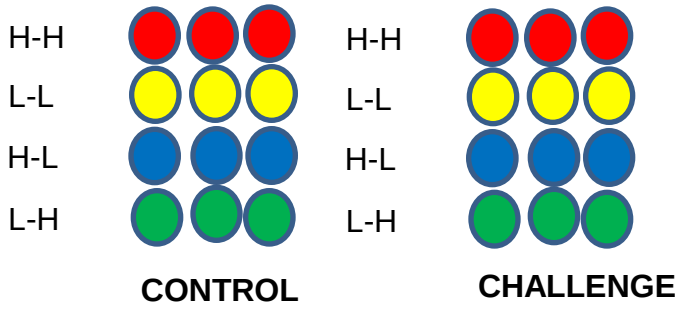
Experiment 2

Experimental Design

- Rainbow Trout fed with two different diets
    - High n-3 PUFA
    - Low n-3 PUFA for **4 weeks**

- 28 fish from each tank were challenged with SAV-1 and transferred into challenge facility
- Two additional dietary regimes introduced (in both control and challenge)
    - High-Low n-3 PUFA
    - Low-High n-3 PUFA
- All regimes performed in triplicate
- Sampled 2 fish/tank for pathology

    at 0, 5, 15, 30, 45 d.p.i



Similar sampling regime to Experiment 1

Experiment 2

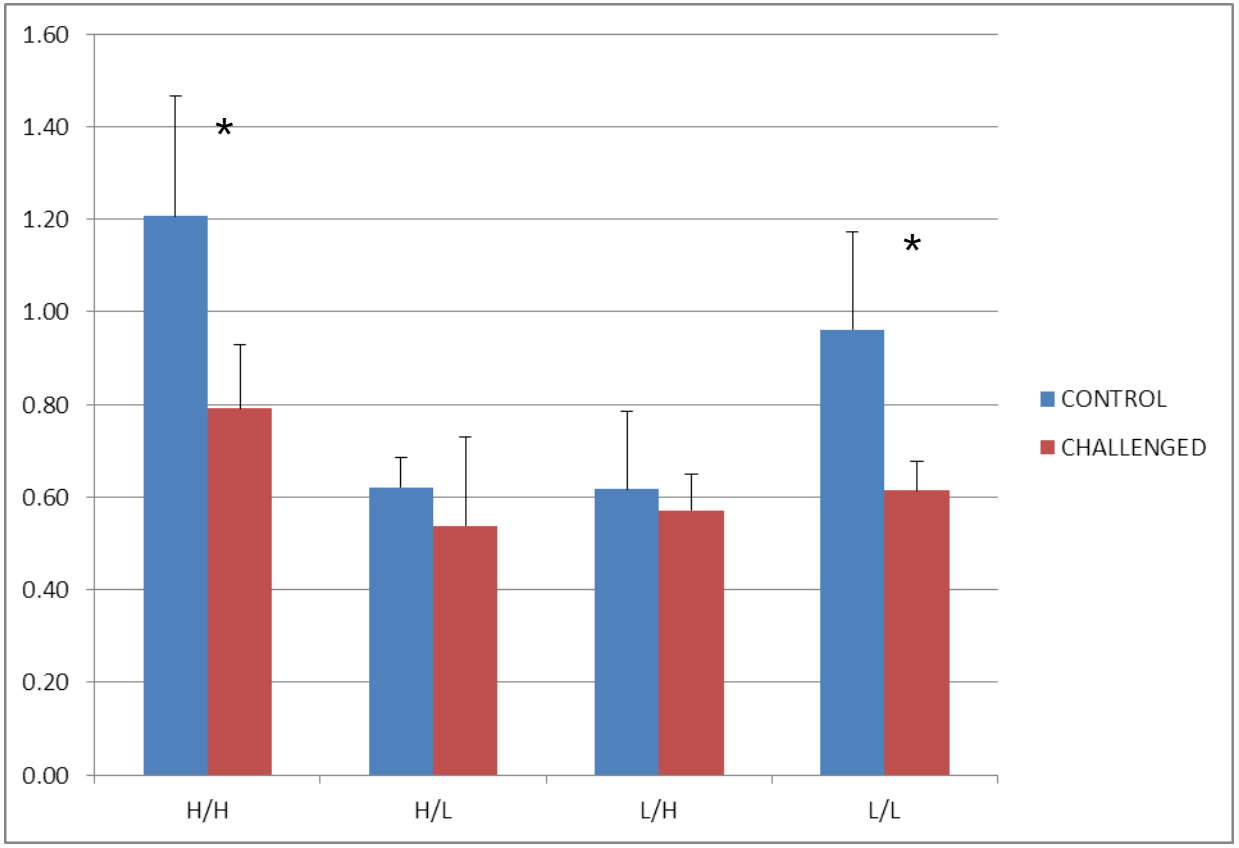
Dietary information

	Diet H	Diet L
Fatty Acid	%	%
Total saturated	25.53	14.24
Total monounsaturated	43.51	54.28
Total n-6 PUFA	7.29	16.55
Total n-3 PUFA	**22.43**	**14.44**
16:2;16:3;16:4	1.23	0.49
Total PUFA	**30.95**	**31.48**
Total	**100**	**100**

Experiment 2

Results

Thermal Growth Coefficient

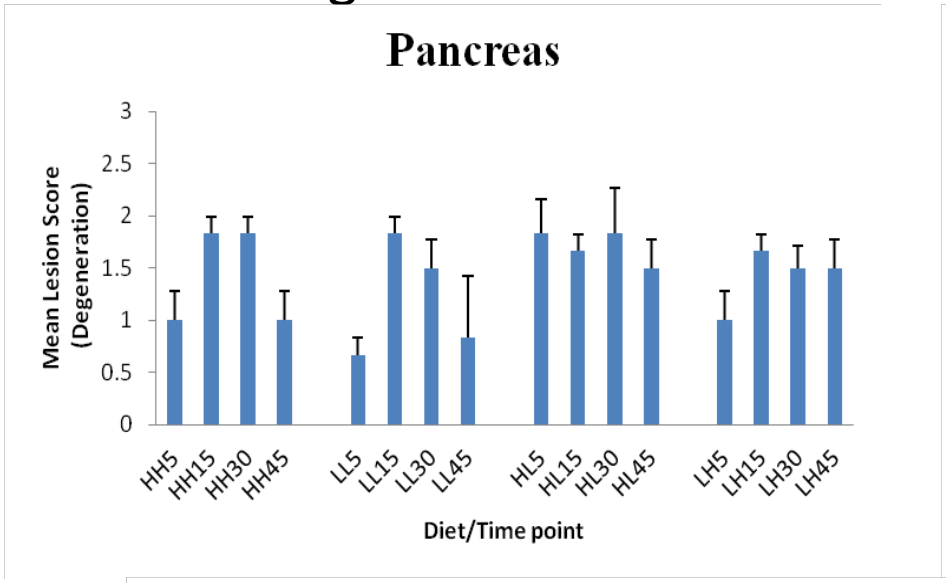


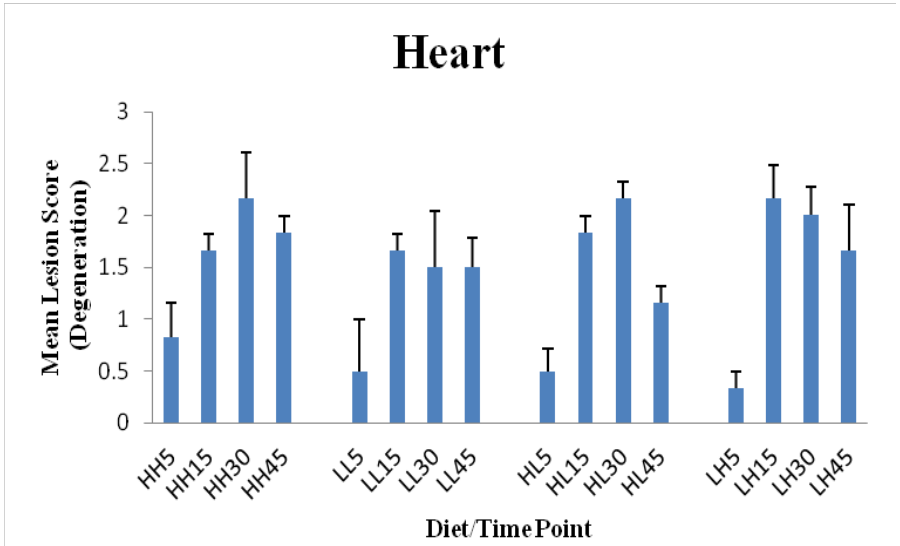
H/H	High to high
L/L	Low to low
L/H	Low to high
L/L	High to low

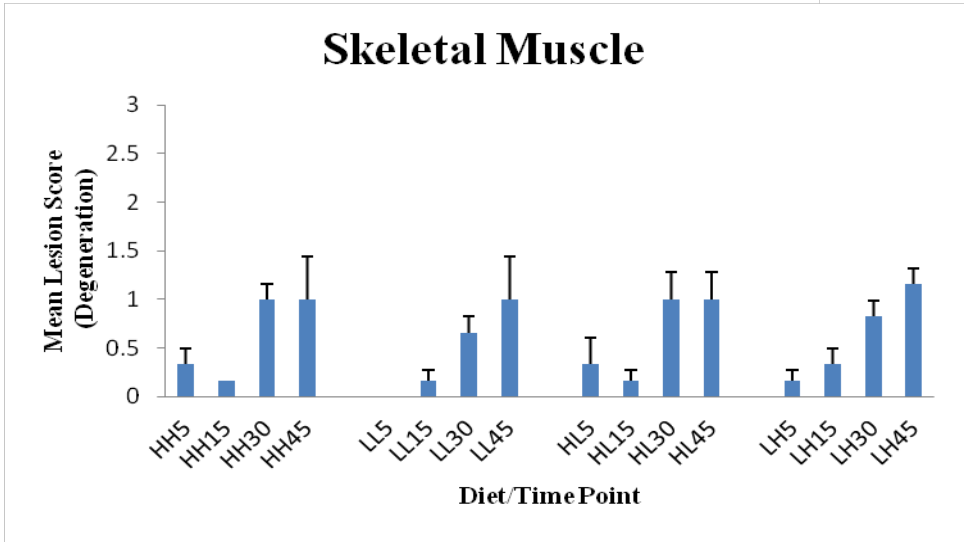
Experiment 2

Results

Tissue Degeneration





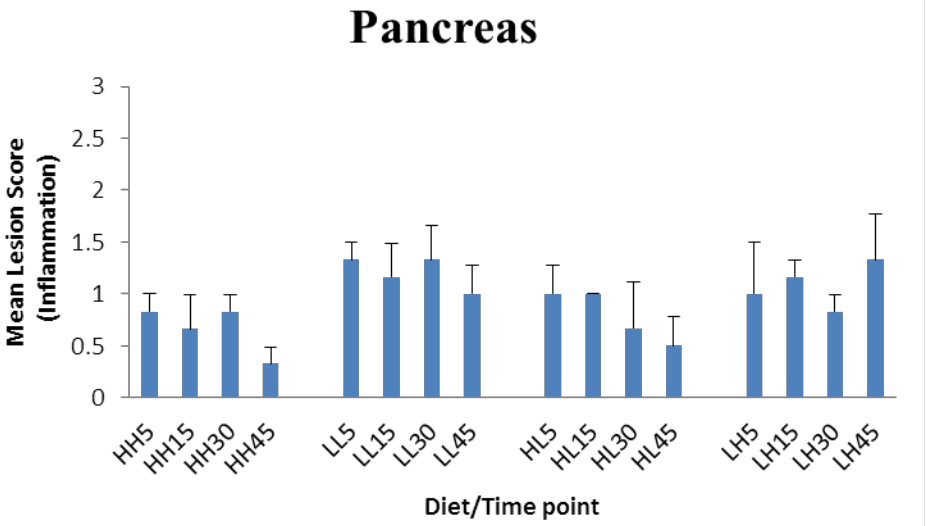


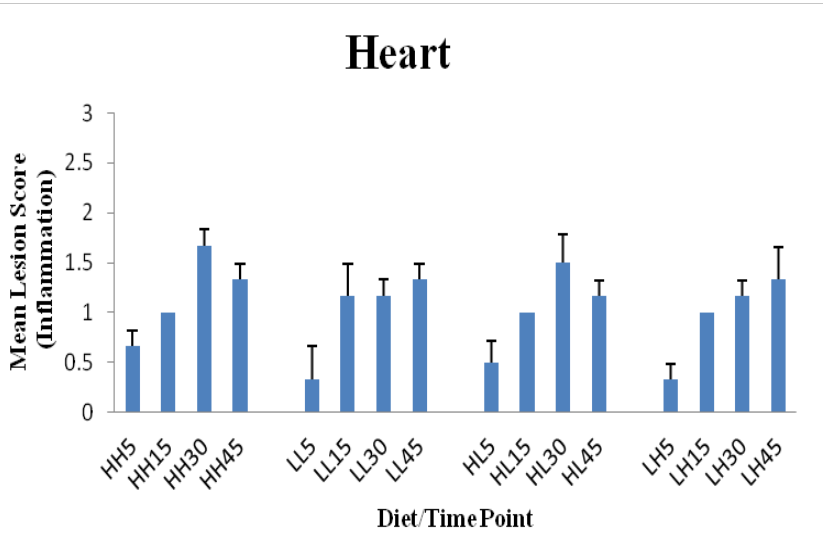
H/H	High to high
L/L	Low to low
L/H	Low to high
L/L	High to low

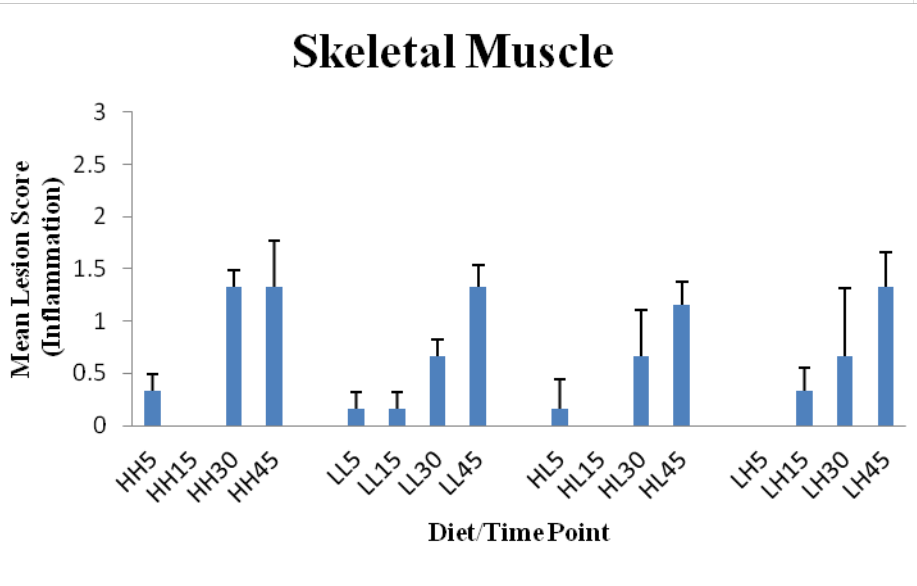
Experiment 2

Results

Tissue Inflammation







H/H	High to high
L/L	Low to low
L/H	Low to high
L/L	High to low

Discussion

Experiment 2

- Consistent regimes appear to perform better
- HH regime (high fish oil) group had significantly lower inflammation levels
- Switched regimes-no effect ?
- Extension of pre-feeding and trial periods ?
- Is pathology scoring sensitive enough to detect any differences between the diets ?

Thank you!