PROBIOTICS EFFECTS ON MITOCHONDRIAL PLASTICITY FROM HEAD-KIDNEY LEUCOCYTES COMPROMISED BY FATTY LIVER DISEASE DEVELOPED IN *Sparus aurata*

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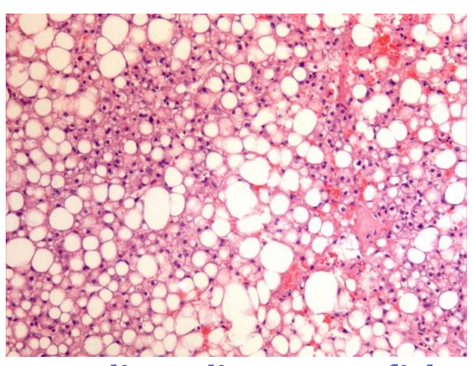
Introduction

Unbalanced diets

Inactivated SPPdp11



Fatty liver disease on fish

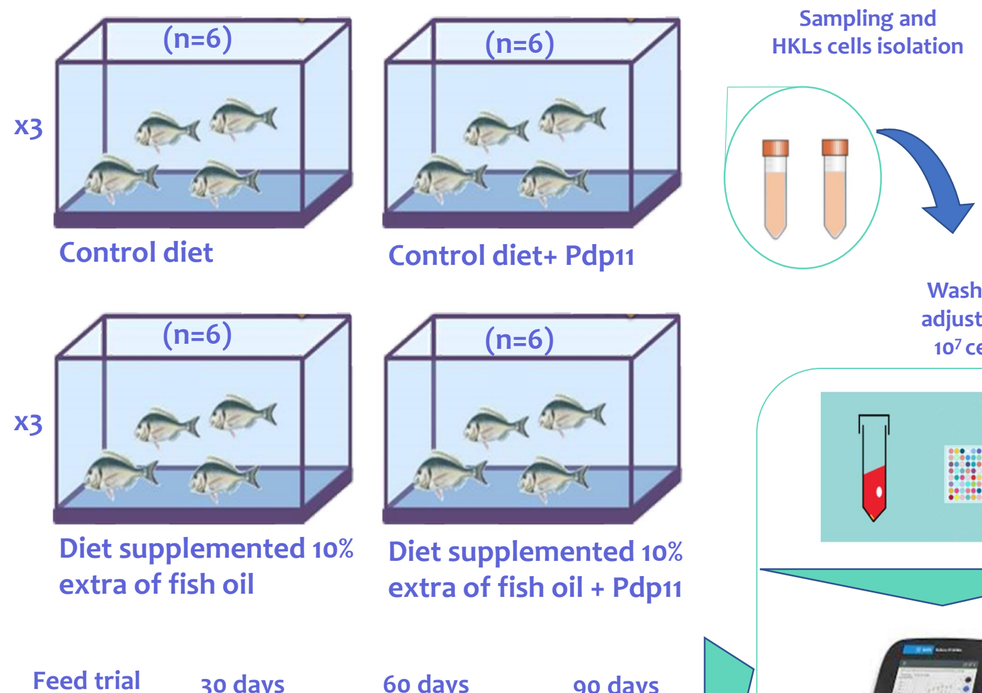
Reduced growth
Poor feed conversion
Metabolic disease
Immune system compromised

?

[1-3]

Material and Methods

Sparus aurata specimens



Objectives

The present work studies the way in which the different components of the probiotic SpPdp11 affect the metabolic syndrome in seabream. Two diet will be used to produce fatty liver disease on fish: Diet supplemented with an extra of 10% fish oil and the other will be Diet supplemented with an extra of 10% fish oil + inactivated Pdp11. The effect on mitochondrial plasticity in head-kidney leukocytes (HKLs) from sea bream cells will be evaluated.

Results

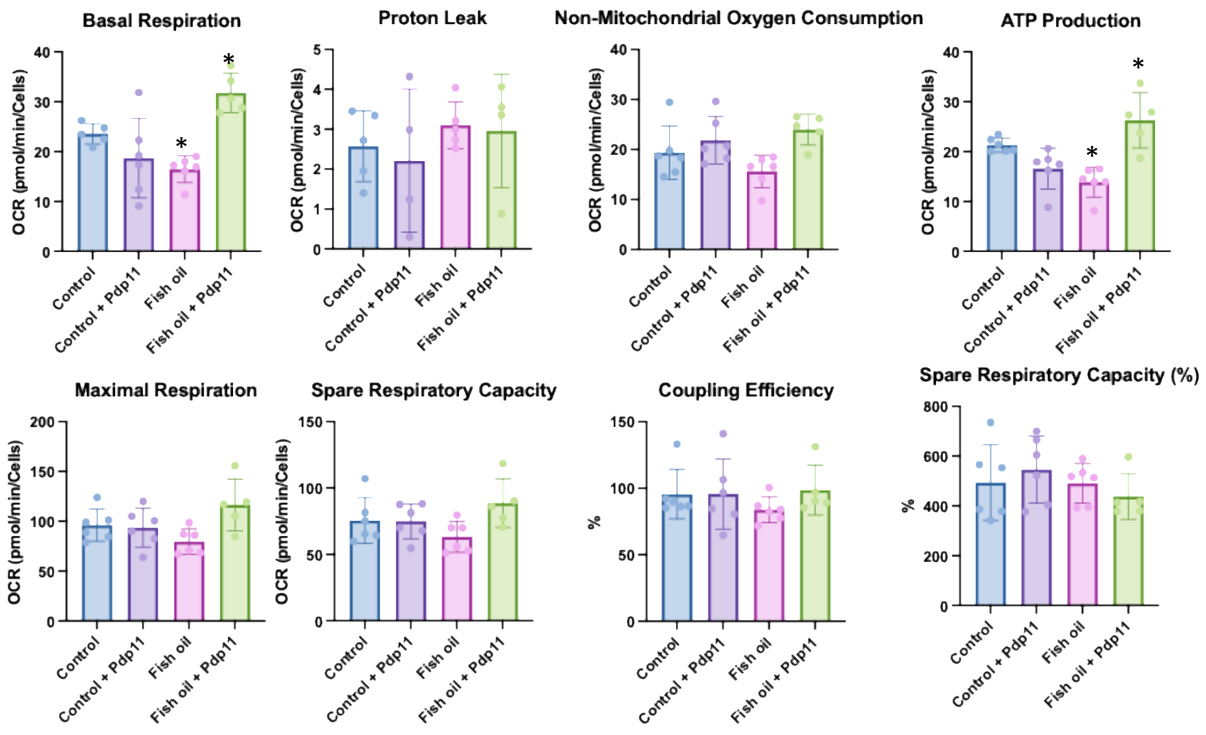


Figure 1. Cell respiratory capacities in head-kidney leukocytes from *Sparus aurata after 3 months of feed trial*. Oxygen consumption rates (OCR) were measured following overnight cell proliferation. The Mito Stress Test was conducted following the injection of the test drug, which consisted of the sequential additions of oligomycin (1.5 µM), FCCP (0.5 µM), and rotenone + antimycin A (0.5 µM). The different parameters were calculated using Wave 2.6.3 software and the Seahorse XF Mito Stress Test Report Generator tool, according to the Agilent Technologies procedures. Absolute OCR values are shown (means ± SEM, n = 6 wells). Statistical differences respect to the control group are denoted by asterisks ($p<0.05$)

Conclusions

The mitochondrial plasticity from HKLs were affected by the unbalanced diet after 3 months, pointing the basal respiration and ATP production as key parameters. Besides, both basal respiration and ATP production resulted increased in cells from fish feed the unbalanced diet + Pdp11.

Overall, our results point to inclusion of this probiotic can reduce or mitigate the impact of unbalanced diet, suggesting also it can influence the mitochondrial plasticity from head-kidney leucocytes, which have a main role in cellular response.

[5]

Acknowledgments

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