Post-prandial effects of a meal rich in long-chain omega-3 fatty acids on indicators of cardiovascular risk

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Introduction

Evidence from epidemiological studies indicates that the regular consumption of oily fish may be protective against the risk of cardiovascular disease. The benefits appear to be related to the content of long-chain omega-3 fatty acids (LC n-3 PUFA), specifically eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Current UK dietary guidelines therefore recommend the consumption of two portions of fish per week, one of which should be oily (1), which equates to 0.45g LC n-3 PUFA per day. Although there is limited information about intakes of EPA and DHA in Scotland, recent studies show that they are consistently below recommendations (2). Further review of the dietary intake data indicates that the consumption of oily fish is sporadic and inconsistent (3) despite attempts to promote regular intake.

Several of the mechanisms involved in the development of cardiovascular disease (CVD) involve endothelial function. Post-prandial hyperlipidaemia has been linked to an increased risk of CVD (4), which is largely attributed to the transient (2-6 hour) decrease in endothelial function (5). Changes in endothelial function have also been shown to be associated with superoxide production (6), implicating oxidative stress as a possible mechanism for endothelial dysfunction. The long-term effects of LC n-3 PUFA on oxidative stress and inflammation are well established, however little is known about their immediate post-prandial effects. Identifying the possible benefits of consumption of a single meal rich in LC n-3 PUFA may provide a new perspective on which to promote dietary changes.

The aim of this pilot project was therefore to identify post-prandial changes in markers of cardiovascular risk, assessed by measurement of arterial compliance, whole blood fatty acid profile, plasma glucose and insulin, markers of endothelial dysfunction, oxidative stress and antioxidant status in response a test meal naturally rich LC n-3 PUFA compared with a control meal.

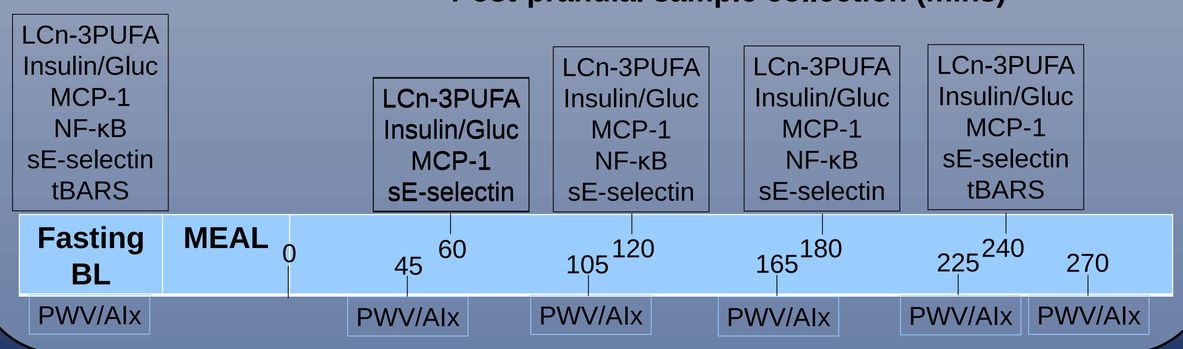
Study Design

Healthy male participants were randomized to receive one of two pasta meals varying in their content of LC n-3 PUFA. Routine LCn-3 PUFA intake was assessed by food frequency questionnaire (FFQ) (2).

	Test meal: Salmon	Control meal: Ham
Energy (kcal)	968	970
Protein (% kcal)	23	23
Fat (% kcal)	51.5	51.5
LC n-3 PUFA (g)	9.7	<0.2

Arterial compliance was assessed by pulse wave velocity (PWV) and augmentation index (AIx) using a portable Vicorder monitor (Skidmore Medical, UK). Whole blood fatty acid levels were measured using the Omega Bloodcount Test ™. This test involves collecting a 50µl blood sample on a Whatman 903® neonatal STD card and subsequent analysis of fatty methyl esters by gas-liquid chromatography and has previously been validated against red blood cell polar lipid fatty acids (7). Endothelial dysfunction was assessed by the measurement of MCP-1. Other measurements included changes in the inflammatory markers NF-κB and sE-selectin and changes in antioxidant status assessed by overall antioxidant concentration in plasma. Blood samples were taken at fasting baseline and at intervals for four hours post-prandially.

Post-prandial sample collection (mins)



Project Outcomes

Seven healthy male participants were recruited to the study. Participant characteristics are given below.

	Mean (± SD)	Range
Age (years)	29.6 (± 7.0)	21 – 40
BMI (kg/m^2)	23.0 (± 1.3)	20.8 – 24.6
Waist circumference (cm)	78.4 (± 4.4)	71.5 – 84.4

All participants were below the threshold for increased risk of metabolic complications, such as cardiovascular disease, according to the WHO cut-off points for waist circumference (>94cm).

Average intakes of EPA, DPA, DHA and total LC n-3 PUFA (EPA + DPA + DHA) as assessed by the FFQ are given below

Fatty acid intake (g/d)	Mean (± SD)
EPA	0.20 (± 0.17)
DPA	0.04 (± 0.03)
DHA	0.30 (± 0.19)
Total LC n-3 PUFA	0.54 (± 0.38)

Despite mean intakes of LC n-3 PUFA being close to current UK recommendations of 0.45g/d (1), only 2 participants (29%) exceeded the recommended intake, with one participant having almost 300% of the recommended intake.

Arterial compliance

No significant differences were observed in PWV in response to the meals or between the meals (Fig. 1).

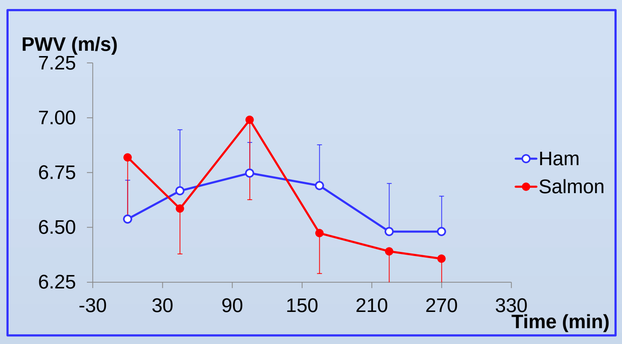


Fig. 1 Response in pulse wave velocity (PWV)

After correction for heart rate of 75 bpm (AIx@75), AIx reduced significantly post-prandially (p=0.001). In addition, for the control meal AIx@75 at 225 mins was significantly lower than at baseline (p<0.05, Fig. 2).

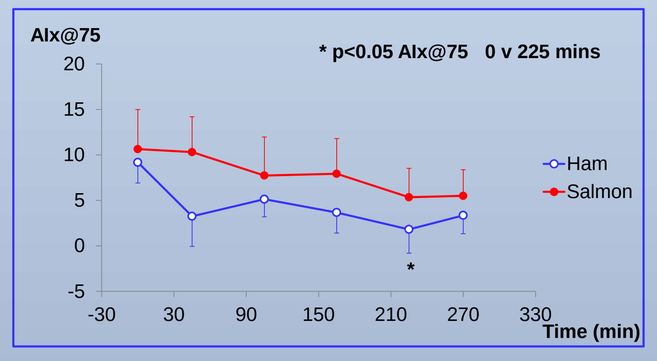


Fig. 2 Response in augmentation index at a heart rate of 75 bpm (AIx@75)

There was no significant difference in MCP-1, as a marker of endothelial dysfunction, over time or between the meals (Fig. 3)

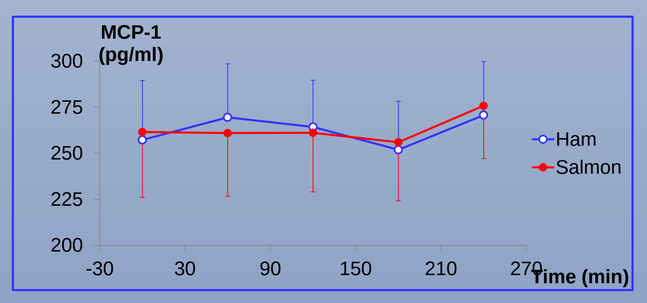


Fig. 3 Response in MCP-1

Whole blood fatty acid profile

Baseline total whole blood LC n-3 PUFA levels (EPA+DPA+DHA) were consistent with previous data (2) and also correlated significantly with routine intakes as assessed by the FFQ ($r=0.84$, $p<0.02$).

In response to the test meal, total LC n-3 PUFA levels were sustained, whereas levels fell significantly following consumption of the control meal ($p<0.05$, Fig. 4)

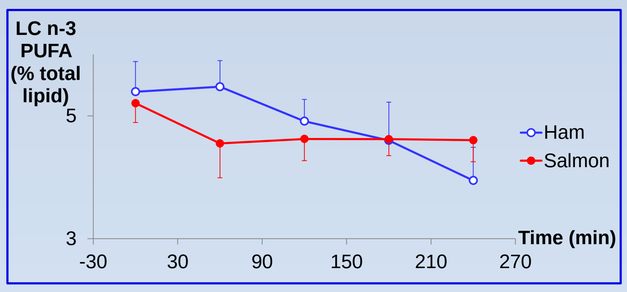
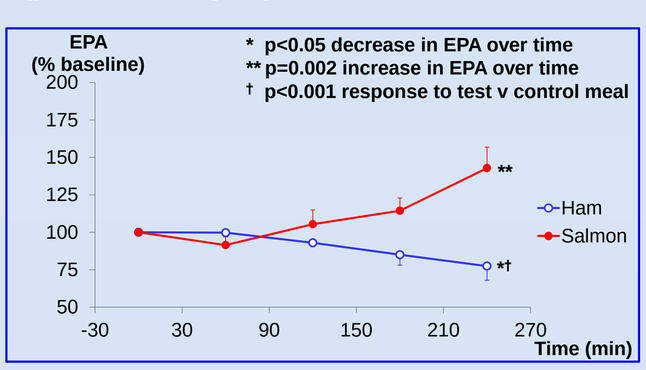


Fig. 4 Post-prandial response in whole blood total LC n-3 PUFA

As a percentage of baseline levels, whole blood EPA levels fell significantly for the control meal ($p<0.05$) but increased significantly for the test meal ($p=0.002$), indicating a significant difference between the responses to each of the meals ($p<0.001$, Fig. 5)

Fig. 5 Post-prandial response in whole blood EPA level



There were no significant differences in DPA levels between the meals. However, as a percentage of baseline, whole blood DHA levels fell significantly for both the control meal ($p=0.01$) and the test meal ($p<0.05$) (Fig. 6)

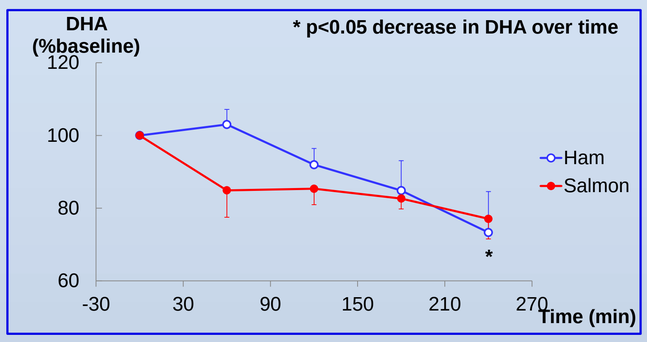


Fig. 6 Post-prandial response in whole blood DHA level

Plasma glucose and insulin

No significant differences were seen in the responses in plasma insulin (Fig. 7) or glucose (Fig. 8) to the test meals.

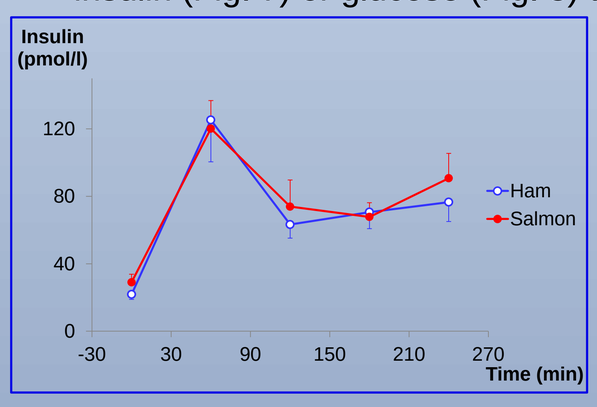


Fig. 7 Response in plasma insulin

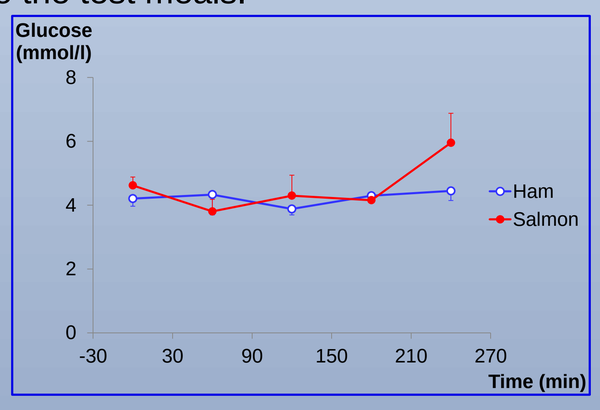


Fig. 8 Response in plasma glucose

Antioxidant status and levels of oxidative stress

There were no significant differences in antioxidant status, measured as the overall antioxidant plasma concentration based on a Trolox standard, following consumption of either of the meals (Fig. 9). However, antioxidant status decreased in 6/7 participants in response to the control meal, whereas it decreased in just 2/7 participants in response to the test meal.

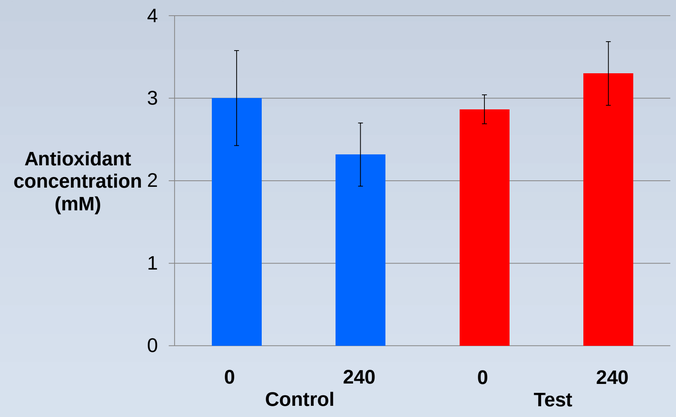


Fig. 9 Post-prandial response in antioxidant status

No significant differences were observed in the post-prandial levels of sE-selectin as a marker of oxidative stress (Fig. 10)

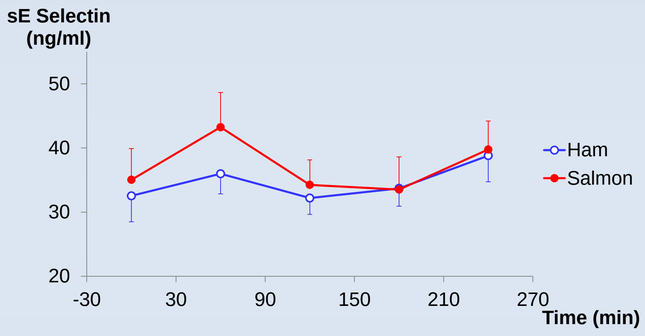


Fig. 10 Post-prandial response in sE-selectin

Due to constraints relating to the volume of blood that could be sampled, insufficient protein was recovered from the samples for NF-κB to allow meaningful analysis

Conclusions

- Routine intakes of LC n-3 PUFA were relatively high, which also reflected that this study population were at a particularly low risk of cardiovascular disease.
- A single meal rich in LC n-3 PUFA significantly increased whole blood EPA and total LC n-3 PUFA levels for at least 4 hours post-prandially.
- The response in PWV was not influenced by the type of meal eaten, however there was evidence that the ham meal significantly reduced AIx three hours post-prandially. This may have been a direct effect of the nitrate content of the ham, which was subsequently assessed to be 4.2μM in comparison to an undetectable level in the salmon (3).
- Other markers of cardiovascular disease were not significantly influenced by consumption of a single meal rich on LC n-3 PUFA, although antioxidant status did show a trend towards improvement.
- This pilot study provides preliminary data on which to develop future studies to comprehensively study post-prandial effects of LC n-3 PUFA in a larger study group and potentially those at increased rick of cardiovascular disease.

References:
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The salmon was generously donated by Scottish Sea Farms Ltd, Oban, Scotland
Funding was provided through a joint funding initiative from the Institute for Health & Welfare Research at RGU and QMU