Highlights

1.- First study on the antioxidant system in newborns from pregnant women consuming oily fish

2.- Appropriate intake of oily fish during pregnancy avoids an imbalance of n-3/n-6 ratio

3.- Oily fish is an adequate way to provide n-3 LC-PUFA during pregnancy

1   **Fatty acid status and antioxidant defence system in mothers and their newborns**

2   **after salmon intake during late pregnancy**

3

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29 **Abbreviations used:** AA (arachidonic acid), ALA (α-linolenic acid), CAT (catalase),

30 CoQ_{10} (coenzyme Q_{10}), $CoQ_{10}H_2$ (reduced coenzyme Q_{10}), DHA (docosahexaenoic

31 acid), DPA (docosapentaenoic acid), EPA (eicosapentaenoic acid), GPx (glutathione

32 peroxidase), GR (glutathione reductase), GSH (reduced glutathione), GSSG (oxidized

33 glutathione), HPLC (high pressure liquid chromatography), HPLC-EC (high pressure

34 liquid chromatography coupled to an electrochemical detector), LA (linoleic acid), LC-

35 PUFA (long-chain polyunsaturated fatty acid), MUFA (monounsaturated fatty acid),

36 ROS (reactive oxygen species), Se (selenium), SiPS (Salmon in Pregnancy Study), SOD

37 (superoxide dismutase).

Abstract

Objective: The aim of the present study was to assess the maternal and newborn status of erythrocyte fatty acids and the antioxidant defence system after the intake of two portions of salmon per week during late pregnancy.

Research Methods and Procedures: Pregnant women ($n=123$) were randomly assigned to continue their habitual diet which was low in oily fish (control group, $n=61$) or to consume two 150-g salmon portions per week (salmon group, $n = 62$) from 20 week of gestation until delivery. Fatty acids, selenium and glutathione concentrations and antioxidant defence enzyme activities were measured in maternal erythrocytes at 20, 34 and 38 weeks of pregnancy, and in cord erythrocytes collected at birth. Plasma concentrations of antioxidant molecules were also measured.

Results: Compared with the control group, consuming salmon had little effect on erythrocyte fatty acids in either mothers or newborns. Components of the antioxidant defence system did not differ between groups. Glutathione peroxidase activity and the concentrations of tocopherols, retinol and coenzyme Q_{10} were significantly lower in cord blood compared to maternal blood at week 38 in both groups.

Conclusion: Maternal and newborn erythrocyte fatty acids are little affected by the intake of two portions of salmon per week during the second half of pregnancy, although erythrocyte DHA might be increased in newborns. Maternal and newborn antioxidant defence systems are not impaired by intake of salmon from 20 weeks gestation.

Keywords: omega 3, fatty acids; fish oils; pregnancy; newborn; antioxidants

Introduction

The requirements for the long chain polyunsaturated fatty acids (LC-PUFA) arachidonic acid (AA, C20:4 n-6) and docosahexaenoic acid (DHA, C22:6 n-3) are especially high during the last trimester of pregnancy and the first weeks of extra-uterine life, because of their accretion into the growing brain and other tissues[1,2]. AA and DHA can be formed by elongation and desaturation of the essential precursors linoleic acid (LA, C18:2 n-6) and α-linolenic acid (ALA, C18:3 n-3), respectively, but foetal fatty acid-desaturase enzymes are unable to supply sufficient LC-PUFA until 16 weeks after birth[3]. Therefore, foetal LC-PUFA must be supplied from the maternal circulation and so are ultimately derived from the maternal diet.

The increased foetal demand for LC-PUFA is indicated by a concomitant decrease in the relative concentrations of DHA and AA in the maternal plasma as pregnancy progresses[4,5]. Fish oils, rich in n-3 LC-PUFA DHA and eicosapentaenoic acid (EPA, 20:5 n-3), may enhance maternal, foetal and neonatal PUFA status. Findings from several studies have shown that dietary intakes of n-3 LC-PUFA of ≥2.6 g/d significantly increase the n-3 LC-PUFA status in both pregnant women and their newborns[6,7,8]. Nonetheless, this increase may be accompanied by a reduction of n-6 LC-PUFA towards the end of pregnancy[8,9,10,11] and this is not desirable.

The United Kingdom Government recommends that pregnant women consume one or two portions of oily fish each week as a source of n-3 LC-PUFA[10]. It is not clear whether consumption of fish as a whole food delivering n-3 LC-PUFA affects the n-3 LC-PUFA status of mothers and their newborns. In this regard, Sanjurjo *et al*.[11] observed higher status of EPA and DHA and lower status of AA in mothers with high dietary intake of oily fish in relation to those with lower consumption, with similar findings in newborns. Currently, no intervention studies apart from the Salmon in

87   Pregnancy Study (SiPS)[12] have investigated the effect of higher oily fish intake in

88   pregnant women whose consumption of oily fish was normally low. In SiPS, the intake

89   of two portions of salmon per week (equivalent to a daily intake of about 500 mg

90   EPA+DHA) resulted in an enhanced status of plasma EPA and DHA in pregnant

91   women and a higher status of EPA and DHA in the umbilical cord blood plasma than

92   seen in the control group[12].

93   It is known that LC-PUFA are good substrates for lipid peroxidation, and so a diet

94   high in *n*-3 LC-PUFA could contribute to oxidative stress[13]. However, several

95   mechanisms exist to protect against peroxidative damage. These mechanisms involve

96   exogenous vitamins and trace elements as well as endogenous enzyme systems[14]. In

97   SiPS, maternal oxidative stress markers remained unaffected after consumption of two

98   portions of salmon per week[15]. Further, maternal retinol and selenium (Se) levels

99   were significantly higher in the group supplemented with salmon than in the control

100 group[16]. To our knowledge, there are no studies on the effect of increased maternal

101 oily fish intake on the antioxidant defence system in newborns.

102   Therefore, the aims of the present study, as part of SiPS, were to examine the effect

103 of increased salmon consumption from week 20 of pregnancy until delivery on

104 erythrocyte fatty acids in pregnant women and their newborns and on the antioxidant

105 defence system in the newborns' blood.

106

107 **Materials and Methods**

108 The study design, the characteristics of the pregnant women, aspects of their diet, and

109 compliance have been described in detail elsewhere[12]. In brief, 123 pregnant women

110 residing in or near Southampton, UK were enrolled in the study. The inclusion criteria

111 were: age 18 to 40 years; <19 wk gestation; healthy, uncomplicated, singleton

pregnancy; having a baby at risk of atopy; consuming < two portions of oily fish per month, excluding tinned tuna; and not taking fish oil supplements either currently or in the previous three months. All procedures were approved by the Southampton and South West Hampshire Research Ethics Committee (07/Q1704/43). The study was conducted according to the principles of the Declaration of Helsinki, and all the women provided written informed consent. SiPS is registered at www.clinicaltrials.gov (NCT00801502).

Study design

The recruited women were randomly assigned to one of two groups. Women in the control group (n=61) were asked to continue their habitual diet, and women in the salmon group (n=62) were asked to incorporate two portions per week of farmed salmon (150 g/portion) into their diet from study entry (20 wk of pregnancy) until delivery. SiPS was powered according to an anticipated increase in maternal plasma phosphatidylcholine EPA content. It was calculated that a sample size of 50 women/group would have 93% power to detect a 50% higher plasma phosphatidylcholine EPA content in the salmon group than in the control group[12]. The farmed salmon used in the SiPS were raised at Skretting Aquaculture Research Centre, Stavanger, Norway using dietary ingredients selected to contain low levels of contaminants. Each 150 g salmon portion contained (on average) 30.5 g protein, 16.4 g fat, 0.57 g EPA, 0.35 g docosapentaenoic acid (DPA, C22:5 n-3), 1.16 g DHA, 3.56 g total n-3 PUFA, 4.1 mg α-tocopherol, 1.6 mg γ-tocopherol, 6 μg vitamin A, 14 μg vitamin D_3, and 43 μg selenium. The full fatty acid composition of the salmon is shown in Table 1. Contaminants constituted <12.5% of the FAO/WHO provisional tolerable weekly intake for dioxin and dioxin-like polychlorobiphenyls, <11.5% for arsenic,

137   <0.00000008% for cadmium, 0.0000025% for mercury and <0.00000002% for lead[12]

138   Fifteen women were unable to complete the study (as a result of preterm delivery,

139   withdrawal due to fatigue, a busy schedule or an unspecified injury), leaving a total of

140   54 women in each group at the end of the study; 101 blood samples were collected at

141   birth (*n*=50 in the control group and *n*=51 in the salmon group)[12].

142

143   **Analytical procedures**

144   Fasting maternal venous blood samples were collected for analysis at 20 wk of

145   gestation, before the intervention started, at 34 wk, and at 38 wk. Blood samples were

146   obtained from the umbilical vein after cord clamping, immediately after delivery. All

147   samples were added to heparin and centrifuged. Plasma and washed erythrocytes were

148   immediately frozen and stored at -80ºC.

149   *Erythrocyte fatty acid profile*

150   Erythrocyte fatty acids were transmethylated using acetyl chloride[17]. Hexane-

151   resuspended methylated fatty acids were injected into a Hewlett Packard HP5890 Series

152   II chromatograph (Hewlett Packard, Palo Alto, CA, USA), with a capillary column (60

153   m x 32 mm inner diameter; 20 μm film thickness) impregnated with SP2330 FS

154   (Supelco, Bellefonte, CA, USA). Running conditions were as described elsewhere[18].

155   Fatty acid methyl esters were identified by comparison of retention times with those of

156   authentic standards run previously.

157   *Enzymatic antioxidants*

158   Haemoglobin (Hb) concentration in the blood samples was determined by the

159   colorimetric cyanmethemoglobin method. Erythrocyte catalase (CAT) activity was

160   assayed as described by Aebi[19]. Erythrocyte superoxide dismutase (SOD) activity

161   was assayed according to McCord & Fridovich[20]. Erythrocyte glutathione reductase

162 (GR) activity was assayed by the method of Carlberg & Mannervik[21]. Erythrocyte

163 glutathione peroxidase (GPx) activity was assayed according to Flohé & Günzler[22].

164 *Non-enzymatic antioxidants*

165 Plasma concentrations of α- and γ-tocopherol, retinol and coenzyme Q_{10} (CoQ_{10}) were

166 determined by high pressure liquid chromatography coupled to an electrochemical

167 detector (HPLC-EC)[23], after extraction with 1-propanol. Beta-carotene was also

168 determined after extraction with 1-propanol in an HPLC system attached to a multi-

169 wavelength ultraviolet detector set at 450nm. All compounds were identified by

170 predetermining the retention times of individual standards.

171 *Erythrocyte selenium and glutathione*

172 Erythrocyte Se was determined by inductively-coupled plasma mass spectrometry[24]

173 on an Agilent 7500 ICPMS. Se concentration was calculated using an external standard.

174 Erythrocyte glutathione content was measured by HPLC with fluorescence detection at

175 420 nm, as described by Cereser *et al*.[25].

176

177 **Statistical analysis**

178 Values are presented as mean ± standard error of the mean (SEM). Prior to statistical

179 analysis all variables were checked for normality using the Kolmogorov-Smirnov test.

180 The homogeneity of the variances was estimated using Levene's test. In pregnant

181 women, a general linear model of variance for repeated measures was performed to

182 assess differences among times and between groups and the interactions between group

183 and time. When Mauchly's test indicated that the assumption of sphericity was violated,

184 the Greenhouse-Geisser correction was applied for univariate analysis. When the

185 Greenhouse-Geisser correction was less than 0.05, we used multivariate ANOVA tests,

186 which do not depend on the assumption of sphericity. A one-way ANOVA was applied

187   to evaluate the effects of time (20, 34, and 38 wk) within each group, and *a posteriori*

188   Bonferroni tests were used for the comparison among multiple means. To evaluate

189   differences between two groups, Student t-test was performed. Correlations between

190   parameters were estimated by computing Pearson's and Spearman ρ correlation

191   coefficients. *P* values <0.05 were considered statistically significant. All statistical

192   analyses were performed with SPSS 15.0 for Windows.

193

Results

195   As reported previously[12], the two groups did not differ in maternal age, height, or

196   weight or in infant birth weights or skin prick test positivity. Additionally, the

197   percentages of EPA and DHA in plasma phospholipids decreased during pregnancy in

198   the control group[12]. This decline did not occur in the salmon group; indeed, the

199   percentages of EPA and DHA increased so that both were higher in the salmon group

200   than in the control group at weeks 34 and 38[12].

201

Maternal erythrocyte fatty acids

203   During pregnancy there was a significant increase in the proportion of palmitic acid in

204   erythrocytes in both groups and of AA, lignoceric acid and DHA (*P* 0.005 to <0.001) in

205   the control group. The proportion of ALA decreased in both groups, while LA and DPA

206   decreased only in the salmon group (*P* 0.012 to <0.001) (Table 2). There were no

207   changes for other fatty acids during pregnancy. The percentage of maternal erythrocyte

208   fatty acids did not differ between the control and salmon groups at any time point.

209

Umbilical erythrocyte fatty acids

211   Erythrocytes from newborns in the salmon group had a significantly lower proportion of

9

lignoceric acid than did erythrocytes from their mothers ($P=0.020$), whereas the proportions of ALA, EPA, DHA, *n*-3 PUFA and *n*-3 LC-PUFA were significantly higher in new born than in their mothers (P 0.036 to 0.005) (Table 2).

Relationships between maternal and newborn erythrocyte fatty acid

We observed negative correlations between maternal and newborn erythrocyte *n*-6 PUFA in both control [r=-0.391, *P=0.025*] and salmon [r=-0.516, *P=0.003*] groups. Both, DHA and saturated fatty acid (SFA) proportions were positively correlated between maternal and newborn erythrocytes in the salmon group [r=0.384, *P=0.030* and r=0.356, *P=0.045*, respectively] while MUFA and total PUFA were negatively correlated only in the control group [r=-0.350, *P=0.046* and r=-0.488, *P=0.004*, respectively].

Maternal and newborn enzymatic antioxidant defence system

Table 3 shows the antioxidant enzyme activities in both groups. GPx activity was significantly lower in cord blood from newborns than in their mothers, both in control and salmon groups ($P<0.001$).

Maternal and newborn non-enzymatic antioxidant defence system

There were no significant differences between the two groups or between maternal blood at 38 weeks and newborn blood for Se, oxidised glutathione (GSSG), reduced glutathione (GSH) or total glutathione concentrations (data not shown). There were no significant differences between the two groups for maternal blood at 38 weeks or for newborn blood α-tocopherol, γ-tocopherol or CoQ_{10} concentrations (data not shown). The concentration of retinol was significantly higher in mothers in the salmon group

237   compared to those in the control group (P=0.002). Compared with maternal blood, cord

238   blood from newborns had significantly lower concentrations of tocopherols, retinol and

239   CoQ_{10} in both the control (P 0.035 to <0.001) and the salmon (P 0.014 to<0.001)

240   groups.

241

242   *Relationship between maternal and newborn antioxidant defence system*

243   Combining data for both control and salmon groups, SOD activity and Se concentration

244   correlated positively and significantly between mothers and newborns (r=0.697,

245   *P*<0.001*,* n=69 and r=0.603, *P*<0.001, n=55, respectively). No correlations were found

246   for CAT, GR, GPx, α-tocopherol, γ-tocopherol, β-carotene, retinol, CoQ_{10}, or GSH. In

247   contrast, mothers concentrations of γ-tocopherol, β-carotene and CoQ_{10} correlated

248   negatively with SOD activity in newborns (r=-0.439, *P*<0.001*, n*=70; r=-0.291,

249   *P*=0.014*, n*=71 and r=-0.296, *P*=0.014, *n*=69, respectively). When separating data

250   according to group, significant positive correlations were observed between maternal

251   and newborn for Se concentration and SOD activity in both control [r=0.469, *P*=0.010*,*

252   *n*=29, and r=0.706, *P*<0.001*, n*=34, respectively] and salmon [r=0.622, *P*=0.001*, n*=26,

253   and r=0.689, *P*<0.001*, n*=35] groups. In addition, levels of α-tocopherol were positively

254   correlated between mother and newborn in the group eating salmon [r=0.516, *P*=0.005*,*

255   *n*=28].

256

257   Discussion

258   In normal pregnancies, there is a physiological insulin resistance during the last

259   trimester that promotes maternal lipolysis, to ensure the provision of fatty acids to the

260   foetus[26]. Among these fatty acids, *n*-3 LC-PUFA are the most important, because

261   they are essential for normal foetal brain development[1] and for visual acuity[2].

262 Additionally, adequate levels of *n*-6 LC-PUFA are also necessary during early

263 development[27,28]. In the present study, erythrocyte AA proportions increased during

264 pregnancy and percentages in either maternal or newborn erythrocytes were not

265 different between groups. This suggests that the provision and incorporation of AA are

266 not being limited by the increased intake of n-3 LC-PUFA in the salmon group.

267 Decreased AA and increased DHA proportions in both plasma and erythrocytes have

268 been reported towards the end of pregnancy in women receiving *n*-3 LC-PUFA

269 supplements in some studies[8-11]. However, other studies showed no impact of DHA

270 supplementation [29,30]. One likely reason for these discrepancies is the quantity of

271 DHA used in those studies. Regarding SiPS, the *n*-3 LC-PUFA provided was equivalent

272 to a daily intake of about 500 mg EPA plus DHA, and levels of both DHA and AA

273 increased in erythrocytes during pregnancy. Velzing-Aarts *et al.*[31] suggested that a

274 dose of 500 mg *n*-3 LC-PUFA/d during pregnancy significantly increased neonatal *n*-3

275 LC-PUFA status without affecting *n*-6 LC-PUFA. Similarly, supplementation of

276 pregnant women with 570 mg DHA/d significantly increased plasma and erythrocyte

277 DHA levels in newborns without a reduction in *n*-6 LC-PUFA[32]. The observations

278 made in the current study are consistent with the findings made in pregnant women

279 taking supplements providing about 500 mg DHA/day.

280 In addition, we observed that maternal erythrocyte and plasma phospholipid,

281 EPA and DHA appear to respond differently during pregnancy. In particular, in the

282 control group, DHA declined in plasma phospholipids but increased in erythrocytes,

283 while EPA declined in plasma phospholipids and did not change in erythrocytes. This

284 may be because plasma phospholipids are more metabolically active and are involved in

285 the processes of preferential transfer of *n-3* LC-PUFA to the developing foetus[33].

286 Conversely, an enhanced status of plasma phospholipid EPA and DHA was detected in

mothers and their newborns in the salmon group in SiPS[12]. Additionally, percentages of EPA and DHA along with total *n*-3 PUFA and *n*-3 LC-PUFA in cord erythrocytes in the salmon group were significantly higher compared to mother's erythrocytes at 38 weeks of gestation.

To our knowledge, no studies have directly investigated the effect of *n*-3 fatty acids from oily fish on the newborns' antioxidant defences. It is well known that birth is a situation of increased stress and free radical generation[34] in which the newborn is highly exposed to oxygen, which may be difficult to control[35]. However, endogenous antioxidant enzymes, as well as vitamins and trace elements, are responsible for the detoxification of deleterious oxygen radicals[14]. Some studies have shown that cord plasma contains significantly lower levels of antioxidant vitamins and soluble factors than maternal plasma[34-36], which may be due to the lower amount of lipids present in the cord blood that limit the ability to transport such factors[34]. In the current study, although cord blood and maternal GSSG and GSH concentrations were similar in both groups, the enzyme GPx showed lower activity in cord erythrocytes than in mothers' erythrocytes. When we made comparisons between groups, we observed that antioxidant enzyme activities in cord blood were not different; additionally, concentrations of vitamins and soluble factors were similar in both groups of newborns. Hence, salmon consumption (twice per week) seems not to affect these molecules in the newborn.

We observed a significant negative correlation for *n*-6 PUFA between maternal and newborn erythrocytes in both groups, the same results were seen in supplemented [7,37] and unsupplemented[38,39] women. Moreover, this negative relationship was also evident for MUFA and PUFA in the control group. In contrast, there was a positive correlation for SFA and DHA in the salmon group. Likewise, significant correlations

312   were observed between maternal and umbilical cord blood for SOD activity and Se

313   concentration. In addition, associations of plasma α- and γ-tocopherol concentrations

314   between mothers and their newborns were observed, these correlations are in

315   accordance with those observed previously by other authors[34-36].

316   It is important to highlight that there are external factors that may have affected the

317   results obtained in the present study. Different changes during pregnancy in each

318   woman, changes associated with the evolution of pregnancy, the previous nutritional

319   status of the mother and the characteristics of the foetus (growth, weight, etc.) are

320   aspects that could affect the influence of LC *n*-3 PUFA, and these factors cannot be

321   controlled. The precise nutrient composition of salmon could also affect the results; this

322   composition depends on the diet fed to the salmon and may differ between each salmon.

323   Also, the exact way that women incorporated salmon into their diet and what the salmon

324   replaced could vary and this may influence the results obtained.

325

Conclusions

327   Limited attention has been given to the antioxidant defence system of the foetus in

328   relation to maternal LC-PUFA exposure and, never before, when the source of n-3 LC

329   PUFA is oily fish. The present study demonstrates that the consumption of farmed

330   salmon twice a week from week 20 of pregnancy until delivery (providing about 500

331   mg of EPA + DHA/week) did not impair the antioxidant defence system and did not

332   alter erythrocyte fatty acid composition in newborns or their mothers.

333

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462

1   **Table 1.** Fatty acid composition of the salmon

Fatty acid	Percentage of salmon fatty acids
Myristic acid (14:0)	2.25 ± 0.09
Palmitic acid (16:0)	12.25 ± 0.27
Palmitoleic acid (16:1n-7)	2.52 ± 0.08
Stearic acid (18:0)	3.32 ± 0.11
Oleic acid (18:1n-9)	33.01 ± 0.36
Vaccenic acid (18:1n-7)	2.70 ± 0.01
Linoleic acid (18:2n-6)	11.60 ± 0.15
α-Linolenic acid (18:3n-3)	7.37 ± 0.32
Eicosenoic acid (20:1n-9)	2.79 ± 0.13
Docosadienoic acid (20:2n-6)	1.15 ± 0.2
AA (20:4n-6)	1.30 ± 0.05
EPA (20:5n-3)	3.53 ± 0.16
Nervonic acid (24:1n-9)	0.36 ± 0.01
DPA (22:5n-3)	2.09 ± 0.04
DHA (22:6n-3)	7.11 ± 0.11
Others	6.65 ± 0.04

2   AA, arachidonic acid; DHA, docosahexaenoic acid; DPA, docosapentaenoic acid; EPA, eicosapentaenoic

3   acid

4

Table 2. Fatty acid profile of erythrocyte membranes in newborns and their mothers consuming their habitual diet (control group) or including salmon twice per week (salmon group) beginning at week 20 of gestation.

	Group							
	Control (n=33)				Salmon (n=32)			
	20 weeks	34 weeks	38 weeks	Newborn	20 weeks	34 weeks	38 weeks	Newborn
%								
Palmitic acid (16:0)	24.02±0.32[a]	25.96±0.51[b]	25.00±0.58[ab]	25.24±0.63	23.35±0.33[a]	25.68±0.51[b]	26.00±0.59[b]	24.78±0.39
Stearic acid (18:0)	16.50±1.24	15.01±0.26	14.86±0.74	14.74±0.28	16.50±1.26	15.37±0.27	16.04±0.75	15.01±0.30
Oleic acid (18:1n-9)	14.37±0.48	14.40±0.34	14.42±0.40	15.34±0.32	13.71±0.48	14.10±0.34	13.95±0.41	14.75±0.33
Linoleic acid (18:2n-6)	8.35±0.38	7.49±0.34	7.27±0.44	7.33±0.51	8.15±0.39[a]	7.21±0.35[ab]	6.73±0.45[b]	6.87±0.47
α-Linolenic acid (18:3n-3)	0.26±0.03[ab]	0.29±0.02[a]	0.21±0.02[b]	0.24±0.03	0.24±0.03[ab]	0.30±0.02[a]	0.19±0.02[b]	0.27±0.03*
AA (20:4n-6)	10.65±0.51[a]	10.58±0.61[a]	12.94±0.62[b]	12.57±0.55	11.45±0.52	11.33±0.63	12.44±0.63	13.29±0.39
EPA (20:5n-3)	0.49±0.06	0.47±0.04	0.50±0.04	0.41±0.04	0.49±0.06	0.51±0.04	0.36±0.04	0.49±0.05*
Lignoceric acid (24:0)	2.92±0.16[a]	3.95±0.19[b]	3.53±0.17[b]	3.29±0.14	3.08±0.16[a]	3.72±0.20[b]	3.58±0.17[ab]	3.05±0.12*
Nervonic acid (24:1n-9)	6.62±0.29	6.68±0.23	6.57±0.23	6.53±0.18	7.20±0.30	6.55±0.23	6.63±0.24	6.02±0.19
DPA (22:5n-3)	1.69±0.11	1.57±0.12	1.59±0.14	1.55±0.14	1.92±0.11[a]	1.52±0.12[b]	1.47±0.14[b]	1.69±0.14
DHA (22:6n-3)	3.58±0.29[a]	4.57±0.35[ab]	5.26±0.34[b]	5.30±0.32	4.33±0.29	4.74±0.36	5.02±0.34	6.10±0.28*
SFA	39.23±0.78	41.75±0.99	39.81±1.29	37.86±1.46	38.31±0.79	40.20±1.00	38.78±1.31	38.32±1.34
MUFA	24.00±0.99	22.76±0.26	22.22±0.56	22.07±0.29	24.45±1.01	23.07±0.26	23.44±0.57	21.90±0.35
PUFA	30.53±1.04	30.54±0.59	31.02±0.81	31.67±0.76	30.54±1.06	30.08±0.60	29.47±0.82	31.84±0.83
n-6 PUFA	24.77±0.85	23.93±0.53	23.67±0.69	24.41±0.64	23.80±0.86	23.31±0.54	22.62±0.70	23.56±0.69
n-3 PUFA	5.76±0.40	6.61±0.45	7.35±0.42	7.26±0.41	6.74±0.41	6.77±0.45	6.85±0.43	8.28±0.39*
n-6 LC-PUFA	1.63±0.10	1.44±0.10	1.58±0.12	1.34±0.08	1.50±0.10	1.45±0.10	1.53±0.12	1.51±0.09
n-3 LC-PUFA	5.76±0.40	6.61±0.46	7.35±0.42	7.26±0.41	6.74±0.41	6.77±0.45	6.85±0.43	8.28±0.39*

Values are presented as mean weight % of total fatty acids ± SEM.

8   Values for maternal erythrocyte not sharing a common superscript letter are significantly different (within a group). There were no significant

9   differences between groups.

10   *Significantly different from mothers at 38 wk gestation, $p < 0.05$.

11   AA, arachidonic acid; DHA, docosahexaenoic acid; DPA, docosapentaenoic acid; EPA, eicosapentaenoic acid; LC-PUFA, long chain

12   polyunsaturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.

13

14   **Table 3.** Enzymatic antioxidant activities in newborns and their mothers consuming their habitual diet (control group) or including salmon twice

15   per week (salmon group) beginning at week 20 of gestation.

	Group			
	Control (n=34)		Salmon (n=35)	
	38 weeks	Newborn	38 weeks	Newborn
CAT (nmol/l . g Hb)	2.93±0.14	3.36±0.64	3.00±0.16	2.84±0.12
SOD (U/mg Hb)	1.52±0.14	1.72±0.15	1.43±0.13	1.47±0.14
GR (U/g Hb)	3.44±0.27	3.59±0.15	3.46±0.29	3.75±0.18
GPx (U/g Hb)	519±38	332±16*	604±42	382±20*[†]

16   Values are mean ± SEM.

17   [*]Significantly different from mothers at 38 wk gestation.

18   [†]Significantly different from control group

19   CAT, catalase; GPx, glutathione peroxidase; GR, glutathione reductase; SOD, superoxide dismutase

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