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Relationship Between Serum Adiponectin and Plasma Fatty Acids Composition In Off-Shore (Rig) Workers In Bayelsa State, Nigeria

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ABSTRACT :*The concentration and type of fat in the diet influence the development of obesity and related inflammatory activity. Knowledge of the possible influence of dietary habits on serum adiponectin, a molecule with putative anti-inflammatory properties, maybe helpful in preventing atherosclerosis and type 2 diabetes.The relationship between dietary fat (inferred from plasma fatty acid composition from gas-liquid chromatography) and serum adiponectin (measured by competitiveradioimmunoassay) was evaluated in 70 adult healthy off-shore (Rig) workers.The proportion of saturated fatty acids in plasma was significantly associated with serum adiponectin (r = -0.23; P = 0.01).Specifically, percentage of palmitic acid (C16:0) was significantly associated with lower adiponectin concentration (r = -0.30; P = 0.001), particularly among women (r = -0.37; P =0.01) and non-smokers (r = -0.32; P = 0.005). Percentage of myristicacid (C14:0) was also significantly associated with lower adiponectin among non-smokers (r = -0.26; p = 0.02) and women (r = -0.40; P = 0.01).The other fatty acids were not significantlyassociated with adiponectin except for eicosanoic acid (C20:1 w-9), which was significantly and positively associated with serum adiponectin in all individuals (r = 0.22; P = 0.01). This latter association was most significant in smokers (r = 0.42; P = 0.006). In a multivariate regression analysis to predict serum circulating adiponectin, after controlling for age, body mass index, waist-to-hip ratio, and the individual remaining fatty acids, the percentages of palmitic acid (P = 0.005) andeicosanoic acid (P = 0.02) contributed independently (8% and 5%, respectively) to adiponectin variance. Among non-smokers, the percentage of palmitic acid (p = 0.01) and w-3 fatty acids contributed 10% and 9% respectively, to adioponectin variance. Among smokers, the percentage of eicosanoic acid (P = 0.03) contributed to 12% of adiponectin variance, independently of body mass index, age, waist-to-hip ratio, and the remaining individual fatty acids.Saturated and w-3 fatty acids of dietary origin (inferred from plasma fatty acid composition) are associated with serum adiponectin concentration in healthy industrial (Rig) workers in Bayelsa State, Nigeria. The proportion of eicosanoic acid also appears to be positively associated with serum adiponectin. The knowledge of how these interactions occur may be helpful in the planning of dietary measures aimed at the modulation of inflammatory activity.*

KEYWORDS: *Serum-Adiponectin, Plasma-fatty-acids, off-shore (Rig)-workers, Bayelsa-state, Nigeria.*

I. INTRODUCTION

Adipose tissue has been known to secrete a myriad of proteins that are increasingly recognized to influence the metabolism of proteins, carbohydrates and lipids [1]. Adiponectin (also variously called Acrp30 or AdipoQ in mice) is a 244-amino acid protein synthesized and secreted exclusively by adipose tissue [2, 3]. This hormone circulates at relatively high concentrations and has a half-life of several hours [4]. This rate of turnover of this hormone is consistent with its role as a regulator of metabolic processes, such as modulation of fat partitioning, among other actions. Administration of adiponectin and/or its globular head portion stimulates free fatty acid oxidation in muscle [5]. High fat feeding leads to decreased Acrp30 secretion in mice [6], whereas caloric restriction leads to higher circulating concentrations of adiponectin in mice and humans [7]. Circulating adiponectin concentrations are also inversely correlated to triglyceride [8, 9] and intramyocellular lipid stores [10].

It has been reported that plasma concentrations of adiponectin are lower in obesity and type 2 diabetes [8, 11] and that adipose tissue Acrp30/adiponectin mRNA expression is decreased in obese ob/ob mice and obese humans [12].The mechanisms underlying the observed close association between plasma adiponectin

concentration and lipid metabolism are being elucidated [13, 14]. In vitro and in vivo studies in rodents have shown that adiponectin prevents lipid accumulation in skeletal muscles in parallel to lowering blood glucose and improving insulin action [14]. Some observations suggest that adiponectin could play a role in counteracting the development of diet-induced insulin resistance [6, 7]. It is important to note that these actions appear to be independent of the presence of obesity: adiponectin-null mice showed diet-induced insulin resistance despite increases in body weight similar to those in control mice [15, 16].The interaction among diet-induced insulin resistance, adiponectin concentrations, and lipid metabolism could be exerted at the level of the inflammatory cascade. The amount and quality of fat in the diet seem to be of related inflammatory activity [17]. A low proportion of long-chain unsaturated fatty acids and a high proportion of saturated fatty acids in the diet have been associated with impaired insulin action [18].Adiponectin possess anti-inflammatory [19] and anti-atherogenic properties [20, 21]. On the other hand, highly unsaturated fatty acids, w-3 fatty acids in particular, are also receiving increasingattention as potential anti-inflammatory agents [22] because these dietary fatty acids appear to modulate the release of different cytokines [23, 24].How dietary fat may impact on peripheral adiponectin concentration, or vice versa, is unknown. One potential pathway is activation of the nuclear receptor peroxisome proliferator-activated receptor-y (PPAR-y) [1]. In fact, fatty acids activate PPAR-y [25], and pharmacologic activation with PPAR-y agonists leads to increased plasma adiponectin concentrations [26, 27].
Knowledge of the possible influence of dietary habits on circulating adiponectin concentrations could be helpful in delineating dietary measures aimed at preventing type 2 diabetes. However, studies of the associations etween dietary habits in relation to the development of chronic diseases are hampered by severalmethodologic problems, includingimprecision of dietary surveys [18]. One way to monitor the type of fat in the diet is to record the fatty acid composition in plasma [18]. Our aim was to evaluate the relationship or association between dietary habits, as inferred from plasma fatty acid composition, and circulating adiponectin (serum adiponectin) in off-shore (Rig) "healthy" workers especially as this group of individuals are on special company-provided meals.

II. SUBJECTS AND METHODS

We evaluated 70 individuals as part of the periodic medical examination for offshore (Rig) industrial workers in the Niger Delta region (BayelsaState, in particular) of Nigeria. This particular examination also involved screening for metabolic syndrome and assessment of non-classical cardiovascular risk factors in the working population, given the fact that they are all exposed to the same special company-provided offshore meals. All the workers involved are blacks and from Niger Delta region of Nigeria (particularly, Bayelsa State). All those involved in the study reported to maintaining a steady body weight for at least 3 months before the study. None was taking any medication or had any evidence of metabolic disease apart from obesity.Inclusion criteria for the study were (1) a body mass index (BMI) < $40kg/m^2$, (2) absence of any systemic disease, and (3) absence of any infection in the previous month. The study was approved by the Niger Delta University Teaching Hospital ethics committee, and informed consent was obtained from all the participants and management of the companies involved.Body mass index (BMI) and waist-to-hip ratio (WHR) were measured in all participants. BMI was calculated as weight (in kilograms) divided by the height (in meters) squared. The waist-to-hip ratio was gotten by measuring the waist circumference with a soft tape placed midway between the lowest rib and the iliac crest and dividing the value gotten with that of the hip circumference gotten using the same tape to measure the widest part of the gluteal region.Smokers were defined as any person consuming at least one cigarette a day in the previous 6 months. Resting blood pressure (BP) was taken from all participants using the Accouson's Mercury Sphygmomanometer after participants has been in a sitting position for a minimum of 15min. The blood pressure was read thrice in the right arm by the same investigator and the mean of the three measurements was recorded for this study. All the female participants were pre-menopausal, had regular menstrual cycles and were studied in their follicular phase. Liver disease and thyroid dysfunction were specifically excluded from this study by biochemical workup.Following the method of Lepage and Roy [28], we precisely weighed 100µl of plasma obtained after a 12-hour fast into an Ethylene Diamine Tetra-acetic Acid (EDTA) specimen container and diluted the plasma with methanol-benzene (4:1 by volume). Slowly added acetyl chloride (200µl) over a period of 1 min. after transesterification, we dried the pooled solvent extracts under a gentle stream of nitrogen at room temperature. Residues were dissolved in 500µl of hexane, an aliquot chromatographed as methylesters on a 30-m fussed-silica column (0.25min i.d.). Analysis was performed on a Hewlett-Packard 5890 gas chromatograph equipped with a flame ionization detector. The column temperature was held at 80^0C for 3min and then increased in a stepwise fashion to a plateau of 220^0c. The injection port and detector temperatures were 250 and 270^0C, respectively. Helium was used as carrier gas. An internal standard consisting of 50µg of pentadecanoic acid (C15:0) was precisely weighed and added to the serum.

Serum adiponectin concentrations were measured by competitive Radioimmunoassay (LINCO Research Inc.). Samples were diluted 500-fold (10µl of plasma plus 4990µl of assay buffer) before the assay. The detection limit of the method is 2µg/L. the intra-and interassay coefficient of variations (CVs) were <5%.Fasting plasma glucose concentration was measured in duplicate by the glucose oxidase method. The specimen for this assay was collected in the fluoride oxalate specimen container. Serum insulin concentration for all participants was measured in duplicate by a monoclonal immunoradiometric assay (IRMA) (Medgenix Diagnostics). The lower limit of detection for insulin was 4.0mIU/L. The intraassay CV was 5.2% at a concentration of 10mIU/L and the interassay CV was 6.9% at 14mIU/L. The fasting insulin resistance index [Homeostasis model assessment (HOMA) was calculated with the formular: HOMA = fasting glucose (mmol/L) X fasting insulin (mIU/L) / 22.5. HOMA correlates well with insulin resistance index calculated by the minimal model approach: r = 0.79; P <0.0001 [29].Total plasma cholesterol was measured by the reaction of cholesterol esterase/cholesterol oxidase/peroxidase (Enzymatic method). Total triglycerides were measured through the reaction of glycerol-3-phosphate oxidase and peroxidase (Enzymatic method).

III. STATISTICAL METHODS

Descriptive results for continuous variables are expressed as the mean (SD). Before statistical analysis, guassian distribution and homogeneity of variances were tested. Variables that did not fulfill these tests (individual fatty acids and ratios, HOMA, adiponectin) were log-transformed. The relationships between variables were analyzed by simple correlation analysis (Pearson, r). We set statistical significance at $P \leq 0.05$ and have based our discussion on these relationships.We also constructed a stepwise multivariate linear regression analysis to predict adiponectin concentrations, taking into consideration those variables with statistical association of at least $P < 0.01$ on univariate analysis. Because in a previous work we found that smoking affected the relationship between inflammatory markers and metabolic variables [30], we also examined whether smoking (defined as at least I cigarette/day in the previous 6 months) affected adiponectin relationships.

IV. RESULTS

The main characteristics and serum fatty acid composition of the study participants are shown in Tables 1 and 2. The absolute fatty acid concentration did not differ significantly between men and women. Serum adiponectin was significantly higher in women than men (P = 0.005; Table 1). The proportion of saturated acids tended to be lower and the proportion of oleic acid (C18:1 ω-9) tended to be higher among the female workers (Table 1). The proportion of eicosanoic acid (C20:1 ω-9) was significantly higher in women than men (Table 1). Plasma adiponectin correlated negatively with BMI (r = -0.21; P = 0.02), WHR (r = -0.25; P = 0.001) and fasting triglycerides (r = -0.36; P = 0.0001) and tended to be associated with HOMA value (r = -0.14; P = 0.06). Serum adiponectin was not significantly associated with age, systolic or diastolic blood pressure, fasting glucose, or cholesterol.

The proportion of saturated fatty acids was significantly associated with circulating adiponectin concentrations (r=-0.23; P =0.01; Table3). Particularly, the proportion of palmitic acid (C16:0) was significantly inversely associated with adiponectin concentration (r=-0.30; P =0.001). This association wassignificantly mainly among women (r = -0.37; P = 0.01) and in non-smokers (r = -0.32; P = 0.005). The proportion of myristicacid (C14:0) was also significantly inversely associated with adiponectin among non-smokers (r = -0.26; P =0.02) and in women (r=-0.40; P =0.01)The other individual fatty acids were not significantly associated withadiponectin except for eicosanoic acid (C20:1ω-9), which was significantly and positively associated with adiponectin in all participants (r=0.22; P =0.01). This latter association was most significant in smokers (r=0.42; P =0.006). C20:1 ω-9 was the only fatty aid that was significantly increased in smokers compared with non-smokers (Table2). Total monounsaturated ω-9 fatty acids were also significantly associated with adiponectin among smokers (Tables 3). The proportion of ω-3 fatty acids tended to be positively associated with adiponectin in non-smokers (r=0.20; P=0.06).The ratio of saturated (ω-3) fatty acids correlated positively with both HOMA value (r = 0.18; P=0.02)and serum adiponectin concentrations (r=0.18; P =0.02).We performed several multivariate regression analyses to predict circulating adiponectin concentrations. In these models, we considered as independent variables those individual fatty acids that showed a relationship with at least P<0.1 on univariate analysis. After controlling for age, BMI, WHR (which persisted in the model), and the individual remaining fatty acids, only the proportions of palmitic acid (C16:0; P=0.005) and eicosanoic acid (C20:1ω-9; P=0.03) contributed independently to adiponectin variance (8% and 5% respectively).Among non-smokers, and after again controlling for age, BMI, WHR (which persisted in the model),and the remaining individual fatty acids, only the proportions of palmitic acid (C16:0; P =0.01) and ω-3 fatty acids contributed to adiponectin variance (10% and 9%, respectively).

Among smokers, only the proportion of eicosanoicacid (C20:1 ω-9; P =0.02) contributed to adiponectin variance (12%), independently of BMI, age, WHR, and the remaining individual fatty acids. Of the remaining factors, only WHR persisted in the model.

V. DISCUSSION

The proportions of fatty acids in plasma mirror the dietary fat composition. The relationships between the amount of polyunsaturated fatty acids in the diet and the corresponding proportions of the same fatty acids in plasma lipids are often strong [18]. This is usually true for essential fatty acids, such as linolenic and linoleic acids. However, most other types of fatty acids can be synthesized by humans from precursors, particularly saturated fatty acids.

Adiponectin was found to be negatively associated with the main saturated fatty acid, palmitic acid (C16:0). In an in-vitro study, saturated fatty acids induced activation of nuclear factor-kB, an important mediator in the production of several cytokines [31], whereas docosahexanoic acid (DHA; C22:6) counteracted these effects [31,32]. The possible actions of saturated fatty acids on adiponectin production have not been evaluated. Our findings suggest that increased intake of saturated fatty acids and increased endogenous transformation of fatty acids that leads to increased concentrations of saturated fatty acids in plasma are associated with decreased serum adiponectin concentrations. This study could not derive the cause and consequence but it could be hypothesized that decreased serum adiponectin concentrations amplify the proinflammatory action of saturated fatty acids.Adiponectin was positively associated with the proportion of C20:1 ω-9. This association was especially significant in smokers (P = 0.006). Although BMI tended to be lower with increased proportions of C20:1ω-9, the association remained significant after controlling for BMI (r = 0.24, P = 0.006). Significantly increased products of delta-9 desaturation and significant increases in C20 elongation products have been observed in rats with reduced food intake [33]. On the other hand, smoking has been found to be associated with a decreased proportion of essential fatty acids [34], and essential fatty acid deficiency leads to a characteristic increase in ω-9 fatty acids [35]. Since caloric restriction can lead to higher circulating concentrations of adiponectin in mice and humans [7], it is tempting to speculate that reduced food intake leads to increased adiponectin and C20:1ω-9 concentrations concomitantly.We found, secondly that those individuals with an increased proportion of C18:3 ω-9 had concomitantly decreased serum adiponectin concentrations. The content of this fatty acid is typically very small in diet. It reflects increased endogenous desaturation of linoleic acid by delta-6 desaturase, increasing the proportion of C18:3 ω-6, probably as a consequence of a low proportion of linoleic acid in the diet [18]. Our findings suggest that low intake of this essential fatty acid could lead to increased C18:3 ω-6 and concomitant decreased adiponectin concentrations.

We showed that the proportion of ω-3 fatty acids in general, and DHA in particular, were highest in those individuals with increased circulating adiponectin. In non-smokers, this association persisted after controlling for age, BMI, WHR, and the remaining individual fatty acids. Interestingly, two putative anti-inflammatory molecules, ω-3 fatty acids and adiponectin, seem to be linked. The same processes and dietary habits that increase the concentrations of ω-3 fatty acids lead to concomitant increasedadiponectin concentrations. Novel functional sets of lipid-derived mediators with anti-inflammatory action generated from ω-3 fatty acids have been increasingly recognized [22]. The association of the proportions of ω-3 and adiponectin described here seems particularlyimportant in light of findings in American women: increased intake of the ω-3 polyunsaturated fatty acids eicosapentanoic (C20:5 ω-3) and DHA (C22:6 ω-3) was associated with reduced risk of thrombotic diseases [26]. In parallel with these observations, the authors of in-vivo and in-vitro studies in humans reported that supplementation witheicosapentanoic acid and DHA appeared to reduce cytokine production [23, 24, 31, 36, 37, 38]. The above relationships were most likely to have a dietary explanation because food is the major source of these fatty acids [18]. Adiponectin also has anti-atherogenic properties and inhibits proliferation of vascular smooth muscle cell [16]. Inflammation in the vessel wall plays a major role in the initiation and progression of atherosclerosis [39, 40]. Decreased adiponectin concentrations have been observed in patients with coronary artery disease [8]. The findings of the present study suggest that all these associations are interrelated events.An association between concentrations of fasting plasma triglycerides and serum adiponectin has also been observed [9]. Other studies have shown that adiponectin promotes lipid oxidation in humans, with a subsequent decrease in intracellular lipid content in human muscle [10]. These results are consistent with animal data: Adiponectin was shown to enhance lipid oxidation and decrease the concentration of muscle triglycerides [5, 13].Our study is limited by the fact that our findings are hypothesis-generating. It will be necessary to demonstrate that adiponectin concentrations can be modulated by ingestion of certain fatty acids or by changing of serum fatty acid composition. In addition, fatty acids were measured in plasma even when it is well known that fatty acids were also major components of cell

membranes.Fatty acids measured in cell membranes may better reflect longer-term dietary fatty acids intake because fatty acid turnover may be slower in cell membranes than in plasma.

VI. CONCLUSION

Saturated and ω-3fatty acids of dietary origin (reflected in and inferred from plasma fatty acid concentration) are associated with serum adiponectin concentrations in healthy industrial (Rig) workers. The proportion of C20:1 ω-9 also appears to be positively associated with serum adiponectin. Adequate knowledge of how these interactions occur will be helpful in the planning of dietary measures aimed at the modulation of inflammatory activity.

Table 1: Anthropometric and Biochemical Characteristics of the subjects.

Parameter	**Men**	**Women**	**P-value**
Number	**45**	**25**	
Age, years	39.2(10.1)*	35.4(8.9)	NS
Weight, Kg	77.1(9)	59.8(10.5)	0.0001
BMI, Kg/m^2	24.7(2.8)	22.9(3.9)	0.01
WHR	0.95(0.03)	0.86(0.05)	0.0001
SBP, mmHg	125.8(12)	118.4(14)	0.006
DBP, mmHg	75.1(10)	67.1(7.3)	0.001
FPG, mmol/L	4.6(0.4)	4.2(0.5)	0.02
FSI, mIU/L	7.0(4.0)	6.9(4.1)	NS
HOMA Score	1.45(0.7)	1.30(0.7)	NS
Cholesterol, mmol/L	5.5(1.1)	5.1(1.0)	0.03
Triglycerides, mmol/L	1.3(0.7)	0.7(0.3)	0.0001
Adiponectin, mg/L	15.2(3.8)	18.5(4.3)	0.005
Fatty acids, % of total fatty acids:			
14:0	0.43(0.25)	0.50(0.35)	NS
16:0	21.0(2.1)	20.5(4.0)	NS
16:1 (ω-9)	0.36(0.8)	0.36(0.9)	NS
18:0	7.70(0.70)	7.40(1.0)	NS
18:1 (ω-9)	19.7(2.6)	21.8(8.0)	0.07
18:2 (ω-6)	30.2(4.7)	29.5(6.0)	NS
18:3 (ω-6)	0.43(0.13)	0.42(0.15)	NS
18:3(ω-3)	0.25(0.1)	0.31(0.4)	NS
20:0	0.28(0.06)	0.26(0.07)	NS

Table 2: Plasma fatty acid components in relation to smoking status of subjects

Fatty acid*	Relative concentration, %		
	Non-smokers (n=48)	**Smokers (n=22)**	**P-value**
14:0	0.42 (0.27)	0.47 (0.35)	NS
16:0	19.8 (2.1)	19.6 (3.0)	NS
16:1 (ω-9)	0.31 (0.09)	0.31 (0.08)	NS
18:0	7.80 (0.61)	7.52 (1.0)	NS
18:1 (ω-9)	20.6 (3.5)	21.8 (7.0)	0.06
18:2 (ω-6)	30.2 (4.1)	29.8 (6.0)	NS
18:3 (ω-6)	0.41 (0.12)	0.40 (0.14)	
NS			
18:3 (ω-3)	0.25 (0.09)	0.31 (0.34)	NS
20:0	0.28 (0.05)	0.26 (0.06)	NS
20:1 (ω-9)	0.10 (0.07)	0.21 (0.15)	0.013
20:3 (ω-6)	1.36 (0.3)	1.50 (0.31)	NS
20:4 (ω-6)	7.16 (1.27)	7.01 (1.29)	NS
20:5 (ω-3)	0.54 (0.40)	0.53 (0.39)	NS
22:0	0.82 (0.16)	0.80 (0.14)	NS
22:6 (ω-3)	2.00 (0.58)	1.92 (0.54)	NS
24:0	1.02 (0.14)	1.00 (0.18)	NS
24:1 (ω-9)	1.21 (0.31)	1.20 (0.28)	NS
Essential fatty acids	44.1 (4.9)	42.9 (6.2)	NS
Saturated fatty acids	31.7 (1.8)	29.9 (3.5)	0.09
Monounsaturated	23.3 (3.5)	23.0 (3.2)	NS
Polyunsaturated, ω-3	2.67 (0.7)	2.60 (0.8)	NS
Polyunsaturated, ω-6	37.6 (4.8)	36.9 (5.7)	NS

Table 3: Showing correlation between plasma fatty acid components and Adiponectin

Fatty acids	All subjects (n=70)		Men (n=45)		Women (n=25)		Non-smokers (n=48)		Smokers (n=22)	
	r	**P**	**r**	**P**	**r**	**P**	**r**	**P**	**r**	**P**
14:0	-0.10	0.3	-0.01	0.7	**-0.40**	**0.01**	**-0.26**	**0.02**	0.05	0.6
16:0	**-0.30**	**0.001**	-0.09	0.4	**-0.37**	**0.01**	**-0.32**	**0.005**	-0.20	0.1
16:1 (ω-9)	0.04	0.6	-0.05	0.5	-0.03	0.7	0.05	0.4	0.001	0.9
18:0	0.07	0.3	0.15	0.1	0.12	0.3	0.02	0.6	0.20	0.1
18:1 (ω-9)	0.07	0.3	0.08	0.4	0.01	0.8	0.03	0.7	0.23	0.3
18:2 (ω-6)	-0.01	0.8	-0.10	0.3	0.16	0.25	0.04	0.5	-0.20	0.1
18:3 (ω-3)	0.01	0.7	-0.05	0.6	-0.002	0.9	0.04	0.7	-0.10	0.4
20:0	.0.03	0.6	0.03	0.7	0.20	0.1	0.01	0.7	0.08	0.2
20:1 (ω-9)	**0.22**	**0.01**	0.13	0.1	0.15	0.3	0.5	0.6	**0.42**	**0.006**
20:3 (ω-6)	0.08	0.30	0.10	0.3	0.21	0.1	0.05	0.6	0.18	0.1
20:4 (ω-6)	0.01	0.68	0.02	0.7	0.10	0.5	0.10	0.3	-0.1	0.4
20:5 (ω-3)	0.07	0.3	0.07	0.3	0.10	0.3	0.06	0.3	0.11	0.2
22:0	0.10	0.1	0.11	0.3	0.14	0.10	0.13	0.2	0.05	0.1
22:6 (ω-3)	0.05	0.3	0.06	0.3	0.08	0.4	0.11	0.3	-0.01	0.5
24:0	0.05	0.3	0.08	0.4	0.11	0.3	0.06	0.4	0.03	0.4
24:1 (ω-9)	0.06	0.5	-0.001	0.8	0.23	0.1	0.07	0.4	-0.01	0.4
EFA	0.01	0.8	-0.06	0.5	0.21	0.1	0.10	0.3	-0.20	0.7
SFA	**-0.23**	**0.01**	-0.04	0.6	-0.30	0.04	-0.20	0.07	-0.1	0.3
MUSFA (ω-9)	0.07	0.2	0.08	0.4	0.01	0.8	0.02	0.8	**0.34**	**0.001**
PUSFA (ω-3)	0.07	0.2	0.08	0.4	0.10	0.5	0.20	0.06	0.001	0.8
PUSFA (ω-6)	0.001	0.7	-0.07	0.4	0.19	0.2	0.06	0.3	-0.21	0.1

EFA=Essential fatty acid, SFA=Saturated FA, MUSFA= Monounsaturated FA, PUSFA=Polyunsaturated FA

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