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Plasma fatty acids: Biomarkers of dietary intake?

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ABSTRACT

Objective: To our knowledge, there is currently no consensus in the literature on the association between dietary fatty acids and circulating levels in plasma. The aim of this study was to assess the association of the intake of fatty acids with their relative plasma concentrations.
Methods: We conducted a study with 300 adults from the population-based health survey in São Paulo city (ISA-Capital 2008). We assessed demographic, lifestyle and anthropometric data, biochemical measurements, and two 24-h dietary recalls collected on non-consecutive days. Intake distribution was adjusted for intrapersonal variance to give usual dietary intake using the multiple source method (MSM). Percentage of fatty acids in plasma were analyzed by gas chromatography. The κ statistic, Spearman's correlation, and multiple linear regression (adjusted for confounders) and ratio limits of agreement were employed to determine the relationship between plasma and dietary measurements.
Results: Low correlation and agreement were found between dietary and plasma fatty acids. Docosahexaenoic acid ($\beta = 0.25$; $P < 0.001$) and saturated ($\beta = 0.19$; $P = 0.048$) fatty acids exhibited an association for means of intake adjusted by the MSM and for confounding variables. A large mean difference, with a large variation of "ratio limits," were observed between the measurements.
Conclusion: Plasma and dietary polyunsaturated and saturated fatty acids exhibited low correlation and agreement, as well as weak association between each other. No association between intake and plasma concentrations of monounsaturated fat was found. Plasma fatty acids are not good biomarkers of food intake.

Introduction

Dietary polyunsaturated fatty acids (PUFAs) have been increasingly linked with health benefits and lower risk for cardiovascular diseases (CVDs). The potential cardioprotective mechanisms of these nutrients stem from their ability to reduce plasma triacylglycerol (TG) levels, lower blood pressure, inhibit or block proinflammatory and proatherosclerotic cellular pathways, and enhance platelet aggregation [1–4].

In contrast, high energy-dense diets that are rich in saturated fats constitute cardiovascular risk factors [5,6]. Moreover, saturated fats derived from meat have been associated with greater risk for CVDs [7], although these associations remain controversial [8]. These conclusions are largely based on food intake questionnaires that use self-reporting methods susceptible to numerous measurement errors that can mask associations with disease risk [9]. The use of biomarkers of dietary intake as an objective measure of food consumption has been discussed in the literature as an additional or alternative measure for dietary intake and nutritional status [9].

Concentration biomarkers (i.e., those exhibiting correlation with food intake) include plasma concentrations of lipids or vitamins [10]. However, the concentrations of these biomarkers can be subject to the influence of physiological aspects, interference of sex and age, and interaction with the environment, such as exposure to smoking, as well as endogenous synthesis, biochemical complex, and genetic factors. Thus, individuals can have similar dietary intake yet differ for absorption, transport and metabolism in target tissue [9,11]. Currently, no single recommended biomarker exists

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for assessing total fat intake. However, concentrations in serum or plasma, erythrocyte membrane, and adipose tissue are commonly used [9,11].

Therefore, the objective of the present study was to assess the association and agreement of dietary fatty acids (FAs) obtained using two 24-h dietary recall (24 hRs) measurements with their respective plasma concentrations determined by gas chromatography, considering factors influencing this relationship, in adults from São Paulo city.

Material and methods

Study population and sampling

The study data were drawn from the São Paulo Health Survey, a cross-sectional, population-based household health survey. The study population comprised residents of private households in the urban area of São Paulo city. The sample was obtained by complex probabilistic two-cluster sampling as follows: census sectors and households (data taken from the 2005 National Survey by Household Samples, IBGE). In all, 2691 individuals from different age groups (adolescents: 12–19 y, adults: 20–59 y, and elderly: ≥60 y) answered a questionnaire collecting socioeconomic, demographic, and lifestyle information. Additional information on diet was collected from 1662 participants.

Further dietary and sociodemographic information, as well as blood samples drawn at participants' homes, were collected from 750 participants. Study participant losses occurred because of refusal to participate, change in address/telephone, not being at home on multiple visits at different times during the week and on weekends, and repeated unanswered telephone contact. Of note, the loss of study participants was randomized among census tracts and sociodemographic features. In the present sample, only data from adults (N = 300) who had results of plasma FA tests were used (Fig. 1). A sensitivity analysis was performed and no differences were observed between the original sample and the final sample in the present study.

The study was approved by the Research Ethics Committee of the School of Public Health of the University of São Paulo and all participants signed an informed consent form.

Data collection and processing

Demographic, socioeconomic, and lifestyle information were collected at households using a structured questionnaire applied by previously trained interviewers. Collection of the biological material and anthropometric measurements were also carried out at participants' homes by a licensed practical nurse. Food intake was assessed by two 24 hRs, the first applied at the household and the second by telephone.

Anthropometric assessment

Weight and height measurements were taken according to the procedures outlined by the World Health Organization (WHO) [12] and by the anthropometric techniques manual: Guide for Performing Anthropometric Measurements of the Laboratory for Nutritional Assessment of Populations [13]. The anthropometric data were used to calculate body mass index (BMI) according to the cutoff points defined by the WHO [14].

The physical activity included energy expenditure in leisure time by reporting type and duration of activity according to the predetermined questionnaire items of the long version of the International Physical Activity Questionnaire, validated in Brazil [15,16]. In the present study, physical activity level was categorized as active (practice of physical activity for ≥30 min/d, 5 d/wk, of moderate intensity, or ≥20 min/d, 3 d/wk, of vigorous intensity) or inactive.

Dietary data

Dietary intake was assessed using two nonconsecutive 24 hRs collected on different weekdays and weekends, and in different seasons—the first at the participant's home and the second by telephone, using the multiple-pass and automated multiple-pass methods, respectively [17,18]. Mean time interval between the first and second measurement was about 6 mo.

The multiple source method (MSM) statistical technique was used for estimating usual intake of foods and nutrients. This procedure used the measurements collected by first and second 24 hRs to estimate usual intake adjusted for intrapersonal variance [19,20].

Data collection was standardized by training interviewers to use a standard form for applying the 24 hRs and an explanatory manual for its completion [21]. All 24 hRs were reviewed before keying in the food intake data to identify errors in form completion and in the quantification of foods reported by the individuals.

The standardization and quantification of foods for conversion into household measures, and preparations into units of weight or volume, were performed according to Fisberg and Villar [22] and Pinheiro et al. [23]. The reported dietary intake information was converted into nutrient and energy values using the Nutrition Data System for Research (NDSR) software version 2005 (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN, USA), whose main database is the North American food composition tables of the U.S. Department of Agriculture [24].

The software uses a North American database and therefore a list, together with respective translations (English to Portuguese), was produced containing >700 foods, beverages, preparations, and methods of cooking preparations used by the São Paulo residents. In addition, the energy value and macronutrients of the foods from the NDSR were set against those of national tables, ensuring that the foods used had an 80% to 120% match between the food composition tables for energy and macronutrients.

Blood collection

After the second 24 hR, blood samples were collected with a mean time interval of 7 d. After 12 h of fasting, an aliquot of blood (20 mL) was collected by venous puncture into Vacutainer tubes containing EDTA (1 mg/mL), stored on ice, and shielded from light until plasma obtention (1500 g (rpm), 10 min, 4°C).

Determination of FA profile in plasma

The modified method used for determining FAs in plasma entailed preparing the sample and extracting the FAs. A total of 1 mL of chromatographic methanol/chloroform (2:1 v/v) was added to the material, which was then centrifuged for 20 min at 20 000g. The esterification stage was than performed and the FAs were converted to methylic esters by reacting the sample with sodium methoxide (0.5 M) [25].

The FA profile was determined on a Shimadzu, CG-2010 gas chromatograph equipped with a DB-FFAP capillary column (15 m × 0.100 mm × 0.10 um; J&W Scientific, Agilent Technologies, Folsom, CA, USA). Hydrogen was used as the carrier gas with a flow rate of 0.27 mL/min, linear velocity of 35 cm/s, and pressure of 187.8 kPa. Flow rates of synthetic air, nitrogen, and hydrogen were 300, 30, 30 mL/min, respectively. The standard employed consisted of a mixture of 37 fatty acid methyl esters (FAME 37, code 47885, Sigma Chemical Co, St. Louis, MO, USA). The FAs were identified by comparing retention times of the external standard against the samples. An internal standard of methyl tricosanoate (C23:0, T9900, Sigma Chemical Co., St Louis, Mo., USA) was also used. Results were expressed as total percentage FAs present in the sample [26].

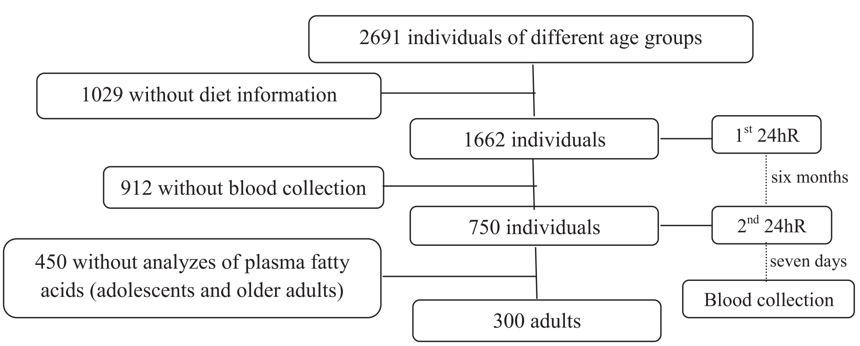

Fig. 1. Description of the study sample selection process. São Paulo, Brazil, 2008.

Statistical analysis

Eleven FAs in plasma and 24-h recalls were identified (myristic – C14:0, palmitic – C16:0, palmitoleic – C16:1 ω-9, stearic – C18:0, oleic – C18:1 ω-9, linoleic - C18:2 ω-6, α-linolenic - C18:3 ω-3, arachidonic – C20:4 ω-6, eicosapentanoic [EPA] – C20:5 ω-3, docosapentaenoic [DPA] – C22:5 ω-3, and docosahexaenoic [DHA] – C22:6 ω-3) and summarized under three groups as follows: saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), and PUFAs.

Normality of variables was tested using the Kolmogorov–Smirnov test. Spearman's correlation coefficient was used to assess the correlation between FA measurements (diet and plasma). Differences between plasma and dietary FAs among sex were performed by Mann–Whitney test.

A hierarchical model was used in the association analysis between plasma and dietary FAs. Multiple regression models were estimated and adjusted for sex, age, self-declared race, physical activity, BMI, smoking, and total energy intake. All explanatory variables were treated as if belonging to the hierarchical level. The standardized coefficients and *P*-value were estimated. In level 1 the following variables were inserted: sex, age, self-declared, and race. At level 2 the following were added: physical activity, BMI, smoking, and total energy intake.

The plasma and dietary FA measurements were distributed into tertiles and assessed for their agreement using the weighted κ test.

For limits of agreement analysis, the variables were converted to the logarithmic scale (ln), and then the mean differences between the measures and the ratio limits of agreement were calculated, as described previously by Nevill and Atkinson [27].

For all analyses, Stata statistical software package version 12 (StataCorp LLC, College Station, TX, USA) was used and $P < 0.05$ was considered statistically significant.

Results

Study participants had a mean age of 39 y (12.4 y), were predominantly self-declared race of white (58.3%), were overweight (31%), and were mostly non-smokers (58.7%) and physically inactive (78.3%; Table 1). Statistical differences were not observed in the demographic, anthropometric, and lifestyle variables among sex ($P > 0.05$).

Table 2 shows that the plasmatic FAs, such as myristic, oleic, DPA, DHA, MUFA, and PUFA, were significantly different between men and women. In the diet, only EPA and DHA showed no differences between sex.

A low correlation was observed between dietary and circulating FAs (Table 3). The plasma FAs that exhibited significant correlations with diet were DHA and linoleic FAs, when considering mean intakes of the two 24 hRs and mean MSM-adjusted intakes. After adjusting for confounding factors, DHA, when considering mean intakes of the two 24 hRs and adjusting for intrapersonal variance, and SFAs, when considering the mean MSM-adjusted intakes, exhibited a significant correlation. Palmitoleic FA had an inverse correlation with diet. The correlation between plasma FAs and daily ingestion in the 24 hRs are presented in Supplementary Table 1.

Low agreement was observed between dietary and plasma FAs (Table 4). For mean intake of the two 24 hRs, myristic, linoleic, linolenic acids, and DHA showed statistically significant agreement. Myristic and linoleic acids showed significant agreement for mean MSM-adjusted intake. Significant percentage agreement of 39.3% ($P = 0.014$) and 39.3% ($P = 0.014$) for linoleic, and of 38.3% ($P = 0.033$) and 38% ($P = 0.043$) for linolenic acids, was observed for adjusted and simple mean measurements, respectively.

Table 5 shows a high mean difference and SD between FA measurements, and the ratio of agreement limits. There was also a possible bias between the correlation among the difference and the mean of the measures, which demonstrate low concordance and a possible heteroscedastic error between the measurements.

Table 1
Demographic, anthropometric, and lifestyle indicators of individuals from São Paulo, Brazil, 2008

	Male (n = 110)		Female (n = 190)			Total	
Variables	n	%	n	%	*P*-value*	n	%
Age (y)					0.410		
<45	63	57.3	118	62.1		181	60.3
≥45	47	42.7	72	37.9		119	39.7
Self-declared race					0.082		
White	57	51.8	118	62.1		175	58.3
Non-white	53	48.2	72	37.9		125	41.7
BMI (kg/m^2)					0.886		
Underweight	4	3.6	6	3.2		10	3.4
Normal	51	46.4	79	42.4		130	43.8
Overweight	33	30	59	31.6		92	31
Obese	22	20	43	23		65	21.9
Smoking					0.158		
Non-smoker	57	51.8	119	62.6		176	58.7
Former smoker	19	17.3	29	15.3		48	16
Active smoker	34	30.9	42	22.1		76	25.3
Physical activity					0.369		
Inactive	83	75.7	151	79.9		234	78.3
Active	27	24.5	38	20.1		65	21.7

*χ^2 test.

Table 2
Differences of plasma and dietary fatty acids between sex: São Paulo, Brazil, 2008

Plasma, %	Mean (SD)	Mean (SD)	*P*-value*	Mean (SD)
Myristic	1 (0.5)	0.8 (0.4)	**0.048**	0.9 (0.5)
Palmitic	27.9 (2.7)	27.7 (2.7)	0.304	27.7 (2.7)
Palmitoleic	1.9 (1)	1.9 (0.7)	0.470	1.9 (0.8)
Stearic	10.9 (1.5)	11.1 (1.6)	0.119	11.1 (1.6)
Oleic	19.1 (4.1)	17.5 (3.3)	**0.001**	18.1 (3.7)
Linoleic	24.3 (4.3)	25.3 (3.6)	0.050	25.0 (3.9)
α-Linolenic	0.7 (0.3)	0.6 (0.2)	0.122	0.6 (0.2)
AA	8.6 (2.3)	8.9 (2)	0.124	8.8 (2.1)
EPA	0.6 (0.4)	0.5 (0.3)	0.342	0.5 (0.4)
DPA	0.9 (0.2)	0.8 (0.2)	**0.018**	0.8 (0.2)
DHA	1.9 (0.8)	2.2 (0.7)	<**0.001**	2.1 (0.8)
SFA	39.7 (2.9)	39.6 (2.6)	0.818	39.7 (2.7)
MUFA	21 (4.8)	19.4 (3.8)	**0.007**	20 (4.3)
PUFA	39.3 (5.6)	41 (4.2)	**0.018**	40.4 (4.8)
Dietary, g/d				
Myristic	1.9 (0.9)	1.6 (0.8)	**0.002**	1.7 (0.8)
Palmitic	13.4 (4.9)	10.6 (3.8)	<**0.001**	11.6 (4.5)
Palmitoleic	1.3 (0.6)	1.0 (0.5)	<**0.001**	1.1 (0.5)
Stearic	6.3 (2.4)	5.0 (2)	<**0.001**	5.5 (2.2)
Oleic	24.2 (8.5)	19.4 (7)	<**0.001**	21.1 (7.9)
Linoleic	14.3 (4.2)	12.1 (4)	<**0.001**	12.9 (4.2)
α-Linolenic	1.8 (0.5)	1.5 (0.5)	<**0.001**	1.6 (0.5)
AA	0.1 (0.05)	0.1 (0.04)	<**0.001**	0.12 (0.05)
EPA	0.03 (0.02)	0.02 (0.02)	0.449	0.03 (0.02)
DPA	0.02 (0.004)	0.02 (0.004)	**0.032**	0.02 (0.004)
DHA	0.05 (0.04)	0.05 (0.03)	0.135	0.05 (0.03)
SFA	23.7 (9.2)	19.0 (7.4)	<**0.001**	20.7 (8.4)
MUFA	26.0 (9.2)	20.7 (7.5)	<**0.001**	22.7 (8.5)
PUFA	16.5 (4.8)	13.9 (4.5)	<**0.001**	14.8 (4.8)

AA, arachidonic fatty acid; DHA, docosahexaenoic; DPA, docosapentaenoic; EPA, eicosapentanoic; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid.
Values in bold indicate significant P-values.
*Mann–Whitney.

Discussion

The present study assessed the association and agreement between FAs estimated from the diet and their relative levels in plasma. Plasma concentrations of PUFAs and SFAs exhibited weak association and agreement with FA values estimated by dietary questionnaires, as observed for myristic, linoleic, and DHA FAs.

In previous studies based on the same biological fluid used in the present study, the FAs that showed strongest correlations with diet (mostly estimated using FFQ) were DHA (coefficient of correlation 0.25–0.59) and other polyunsaturated fats, such as linoleic FA

Table 3
Correlation between dietary and circulating fatty acids in plasma of individuals from São Paulo, 2008

	Raw mean		MSM-adjusted mean	
Fatty acids	*r* (*P*-value)*	Standardized coefficients (*P*-value)†	*r* (*P*-value)*	Standardized coefficients (*P*-value)†
Myristic	0.095 (0.101)	0.09 (0.201)	0.079 (0.171)	0.11 (0.107)
Palmitic	0.030 (0.606)	0.14 (0.201)	0.032 (0.582)	0.17 (0.094)
Palmitoleic	−0.075 (0.198)	**−0.15 (0.040)**	−0.096 (0.097)	**−0.15 (0.041)**
Stearic	−0.018 (0.756)	−0.03 (0.757)	−0.011 (0.851)	−0.03 (0.792)
Oleic	0.006 (0.922)	0.19 (0.071)	−0.005 (0.937)	0.13 (0.233)
Linoleic	**0.114 (0.048)**	0.12 (0.122)	**0.115 (0.046)**	0.11 (0.167)
α-Linolenic	0.080 (0.164)	0.13 (0.153)	0.081 (0.662)	0.15 (0.106)
AA	−0.073 (0.210)	−0.06 (0.353)	−0.079 (0.175)	−0.02 (0.707)
EPA	0.046 (0.427)	0.06 (0.263)	−0.010 (0.859)	0.01 (0.925)
DPA	0.038 (0.515)	−0.01 (0.860)	0.068 (0.240)	0.02 (0.764)
DHA	**0.167 (0.004)**	**0.22 (<0.001)**	**0.116 (0.044)**	**0.25 (<0.001)**
SFA	0.020 (0.736)	0.14 (0.139)	0.030 (0.610)	**0.19 (0.048)**
MUFA	−0.002 (0.968)	0.18 (0.090)	−0.016 (0.785)	0.12 (0.251)
PUFA	0.076 (0.189)	0.03 (0.682)	0.078 (0.178)	0.04 (0.663)

AA, arachidonic fatty acid; DHA, docosahexaenoic; DPA, docosapentaenoic; EPA, eicosapentanoic; MSM, multiple source method; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid.
Values considered significant for $P < 0.05$. Values in boldface were considered significant *Spearman's correlation coefficient.
†Standardized coefficient of regression adjusted for sex, age, self-declared race, physical activity, body mass index, smoking and total energy intake.

Table 4
Agreement between dietary and circulating fatty acids in plasma of individuals from São Paulo, 2008

				Raw mean†	MSM-adjusted mean†
Fatty acids	Tertile	Dietary*, g/d	Plasma, %	κ (*P*-value)‡	κ (*P* value)‡
Myristic	T1	<1.24	<0.63	**010 (0.042)**	**0.12 (0.023)**
	T2	1.24–1.88	0.63–0.97		
	T3	>1.88	>0.97		
Palmitic	T1	<9.35	<26.76	0.04 (0.272)	0.04 (0.244)
	T2	9.35–12.87	26.76–28.54		
	T3	>12.87	>28.54		
Palmitoleic	T1	<0.82	<1.49	0.05 (0.782)	0.03 (0.668)
	T2	0.82–1.22	1.49–2.15		
	T3	>1.22	>2.15		
Stearic	T1	<4.24	<10.32	0.02 (0.603)	0.02 (0.398)
	T2	4.24–6.22	10.32–11.70		
	T3	>6.22	>11.70		
Oleic	T1	<17.09	<16.27	0.02 (0.398)	0.03 (0.302)
	T2	17.09–23.32	16.27–19.02		
	T3	>23.32	>19.02		
Linoleic	T1	<10.47	<23.34	**0.14 (0.008)**	**0.14 (0.008)**
	T2	10.47–14.14	23.34–26.81		
	T3	>14.14	>26.81		
α-Linolenic	T1	<1.31	<0.53	**0.10 (0.042)**	0.10 (0.050)
	T2	1.31–1.79	0.53–0.72		
	T3	>1.79	>0.72		
AA	T1	<0.10	<7.89	0.07 (0.901)	0.11 (0.971)
	T2	0.10–0.14	7.89–9.57		
	T3	>0.14	>9.57		
EPA	T1	<0.01	<0.39	0.04 (0.238)	0.010 (0.534)
	T2	0.01–0.02	0.39–0.58		
	T3	>0.02	>0.58		
DPA	T1	<0.01	<0.71	0.01 (0.465)	0.04 (0.219)
	T2	0.01–0.02	0.71–0.89		
	T3	>0.02	>0.89		
DHA	T1	<0.03	<1.82	**0.11 (0.032)**	0.04 (0.271)
	T2	0.03–0.05	1.82–2.30		
	T3	>0.05	>2.30		
SFA	T1	<16.36	<38.54	0.04 (0.244)	0.04 (0.272)
	T2	16.36–23.20	38.54–40.69		
	T3	>23.20	>40.69		
MUFA	T1	<18.46	<17.96	0.01 (0.569)	0.02 (0.603)
	T2	18.46–25.38	17.96–21.11		
	T3	>25.38	>21.11		
PUFA	T1	<12.01	<39.07	0.09 (0.070)	0.08 (0.083)
	T2	12.01–16.39	39.07–42.78		
	T3	>16.39	>42.78		

AA, arachidonic fatty acid; DHA, docosahexaenoic; DPA, docosapentaenoic; EPA, eicosapentanoic; MSM, multiple source method; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid.
Values in boldface were considered significant.
*Mean adjusted or intrapersonal variance calculated using the MSM online platform.
†Arithmetical mean of two 24-h recalls.
‡Weighted κ test.

Table 5
The log transformed (ln) measurement means and differences, the ratio limits of agreement, together with the correlation between the absolute differences and the mean (log transformed): São Paulo, Brazil, 2008

	Log transformed measurements			Plasma vs Raw mean			Plasma vs adjusted mean		
Fatty acids	Dietary mean*	Dietary mean†	Plasma mean	Difference (SD)	Ratio limits	Correlation (abs [diff] vs mean)	Difference (SD)	Ratio limits	Correlation (abs [diff] vs mean)
Myristic	0.26	0.40	−0.27	−0.53 (0.96)	0.59 (6.56)	−0.44	−0.67 (0.70)	0.51 (3.94)	0.05
Palmitic	2.35	2.38	3.32	0.97 (0.55)	2.64 (2.94)	−0.93	0.94 (0.40)	2.56 (2.19)	−0.87
Palmitoleic	−0.12	−0.02	0.57	0.69 (0.89)	1.99 (5.72)	−0.51	0.60 (0.68)	1.82 (3.79)	−0.13
Stearic	1.57	1.62	2.39	0.82 (0.62)	2.27 (3.37)	−0.88	0.77 (0.44)	2.16 (2.37)	−0.78
Oleic	2.95	2.98	2.87	−0.07 (0.59)	0.93 (3.18)	−0.77	−0.11 (0.42)	0.90 (2.28)	−0.56
Linoleic	2.47	2.51	3.20	0.74 (0.53)	2.10 (2.83)	−0.82	0.70 (0.35)	2.01 (1.99)	−0.59
α-Linolenic	0.40	0.43	−0.50	−0.90 (0.59)	0.41 (3.18)	−0.29	−0.93 (0.46)	0.39 (2.46)	0.17
AA	−0.16	−2.17	2.15	3.71 (0.48)	40.85 (2.56)	−0.43	4.31 (0.50)	74.44 (2.66)	−0.49
EPA	−2.11	−3.92	−0.73	1.38 (0.60)	3.97 (3.24)	0.15	3.18 (0.79)	24.05 (4.70)	−0.27
DPA	−2.15	−4.19	−0.25	1.92 (0.35)‡	6.82 (7.10)	0.36	3.94 (0.36)	51.42 (2.03)	0.21
DHA	−1.96	−3.11	0.70	2.66 (0.54)‡	14.30 (2.88)	−0.36	3.81 (0.52)	45.15 (2.77)	−0.31
SFA	2.91	2.95	3.68	0.76 (0.58)	2.14 (3.12)	−0.97	0.73 (0.41)	2.08 (2.23)	−0.94
MUFA	3.02	3.05	2.97	−0.04 (0.59)	0.96 (3.18)	−0.75	−0.08 (0.43)	0.92 (2.32)	−0.53
PUFA	2.61	2.65	3.69	1.08 (0.51)	2.94 (2.72)	−0.88	1.04 (0.34)	2.83 (1.95)	−0.73

AA, arachidonic fatty acid; DHA, docosahexaenoic; DPA, docosapentaenoic; EPA, eicosapentanoic; MSM, multiple source method; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; SFA, saturated fatty acid.
*Arithmetical mean of two 24-h recalls.
†Mean adjusted for intrapersonal variance calculated using the MSM online platform.
‡Differences not normally distributed ($P < 0.05$); using the Kolmogorov–Smirnov normality test.

(coefficient of correlation 0.22–0.41) [28–32]. In the present data, DHA showed a significant correlation and agreement ($r = 0.17$ and $\kappa = 0.11$), where the same profile was observed for linoleic acid ($r = 0.14$) and SFAs ($\beta = 0.06$). This can be explained by the fact that FAs that are not synthesized endogenously and therefore have stronger associations with diet [33]. Despite the low conversion of EPA and DHA by α-linolenic acid [34], DHA proved to be the best FA in this analysis. DHA has a very limited storage capacity owing to its low concentration in adipose tissue, which implies the need of a constant dietary supply. Furthermore, plasma concentrations of DHA are highly sensitive to dietary intake of ≤2 g/d [35]. DHA is mainly transported in phospholipids, a more stable lipid fraction in plasma, and its concentrations can rapidly raise with daily consumption [36,37], reaching balance within 1 mo [35].

One way of assessing the reliability of information acquired from different dietary questionnaires is by comparing them to objective measurements, such as the use of biological markers of nutrient intake. To serve as good markers of intake, the biomarkers measured should represent components of the diet and reflect the absorption of nutrients, and also should not be synthesized endogenously [38].

The biomarkers of concentration used in the present study correlate with dietary intake, but are affected by metabolism, environment, or lifestyle, where these variables must be controlled to determine association with intake [10]. The present results were adjusted for the main confounding factors; namely, sex, age, self-declared race, physical activity, BMI, smoking, and total energy intake. The main factors associated with FA concentrations were age and smoking, where younger individuals and non-smokers had the highest PUFA concentrations (data not shown).

Approximately 98% of fat in foods is in the form of TG, formed by one glycerol molecule esterified to three FAs, albeit saturated, monounsaturated, polyunsaturated, or trans [5]. The laboratory method employed in this study used total plasma FAs, predominantly extracted from TGs, and to a lesser degree from phospholipids and cholesteryl ester [33]. The composition of FAs measured in plasma TGs appears to be inappropriate for estimating dietary intake of these FA in the long term [33]. According to Katan et al. [39], the composition of FAs and of serum lipids is related, in the short and medium term, with dietary intake. Thus, the determination of lipid concentration in erythrocyte membrane or in adipose tissue is more indicated as a marker of dietary intake over the long term, and can have better correlation with usual diet than the plasma markers used in the present study [33]. These results may be influenced by the nature of the metabolism of FAs [40] as competition between metabolic pathways may lead to changes in FA composition not directly related to the diet. In summary, plasma FAs are not good biomarkers of food intake, but this lipid fraction may be useful in providing indices of hepatic enzyme activity such as stearoyl-coenzyme A desaturase [33].

In an effort to reduce the effect of errors associated with the dietary data collection instrument, the multiple-pass and automated multiple-pass methods were used. These approaches provide a more accurate estimate of dietary intake by requiring the individual to record the foods and beverages consumed on the previous day and to report them in a more detailed fashion, thereby reducing collection errors [18,19]. The MSM was employed to attenuate intrapersonal variance, estimating usual intake and reducing the effect of day-to-day dietary variation. Some differences in agreement and correlation findings were observed after adjusting for intrapersonal variance relative to simple mean intake. However, in this study, the best form of estimating mean intake obtained by 24 hR could not be determined. The best coefficients of correlation were obtained for the MSM-adjusted means, although the κ values showing best results were derived from raw means.

Based on the results obtained and on elements found in the literature, the findings reported in the present study suggest possible limitations in extrapolating dietary data from corresponding plasma levels. These limitations were evidenced by the fact that, when significant, association and agreement between these measures were weak.

Conclusion

Weak association and agreement were found between dietary and plasma FAs. Thus, the present results do not support the use of plasma FAs as biomarkers of dietary FAs. Therefore, further studies elucidating the present results are warranted, given that lipid components such as FAs are associated with chronic conditions, particularly CVDs.

Supplementary materials

Supplementary data related to this article can be found at doi:10.1016/j.nut.2018.08.008.

References

[1] Deckelbaum RJ. n-6 and n-3 Fatty acids and atherosclerosis ratios or amounts? Arterioscler Thromb Vasc Biol 2010;30(12):2325–6.
[2] Farvid MS, Ding M, Pan A, Sun Q, Chiuve SE, Steffen LM, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. Circulation 2014;130(18):1568.
[3] DiNicolantonio JJ, Niazi AK, McCarty MF, O'Keefe JH, Meier P, Lavie CJ. Omega-3s and cardiovascular health. Ochsner J 2014;14:399–412.
[4] McEwen Bradley J, et al. The effect of omega-3 polyunsaturated fatty acids on fibrin and thrombin generation in healthy subjects and subjects with cardiovascular disease. Semin Thromb Hemost 2015;41:315–22.
[5] Lottenberg AMP. Importance of dietary fat in the presention and control of metabolic disorders and cardiovascular disease. Arq Braz Endocrinol Metab 2009;53:595–607.
[6] Johns DJ, Lindroos AK, Jebb SA, Sjöström L, Carlsson L, Ambrosini GL. Dietary patterns, cardiometabolic risk factors, and the incidence of cardiovascular disease in severe obesity. Obesity 2015;23:1063–70.
[7] de Oliveira Otto MC, Mozaffarian D, Kromhout D, Bertoni AG, Sibley CT, Jacobs DR, et al. Dietary intake of saturated fat by food source and incident cardiovascular disease: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr 2012;96:397–404.
[8] Szajewska H, Szajewski T. Saturated fat controversy: importance of systematic reviews and meta-analyses. Crit Rev Food Sci Nutr 2016;56:1947–51.
[9] Jenab M, Slimani N, Bictash M, Ferrari P, Bingham SA. Biomarkers in nutritional epidemiology: applications, needs and new horizons. Hum Genet 2009;125:507–25.
[10] Corella D, Ordovás JM. Biomarkers: background, classification and guidelines for applications in nutritional epidemiology. Nutr Hosp 2015;31:177–88.
[11] Arab L, Akbar J. Biomarkers and the measurement of fatty acids. Public Health Nutr 2002;5:865–71.
[12] World Health Organization. Physical status: the use and interpretation of anthropometry. Geneva, Switzerland: Author; 1995.
[13] University of São Paulo. Manual of anthropometric techniques: guide to anthropometric measurements. São Paulo: Lanpop; 2010. Available at: www.fsp.usp.br/lanpop/index_arquivos/Page571.htm Accessed September 11, 2018.
[14] World Health Organization. Obesity: preventing and managing the global epidemic. Geneva, Switzerland: Author; 2000 No. 894.
[15] Matsudo S, Araújo T, Marsudo V, Andrade D, Andrade E, Oliveira LC, et al. International Physical Activity Questionnaire (IPAQ): a study of validity and reproducibilty in Brazil. Rev Bras Activ Fis Health 2001;6:5–18.
[16] Craig C, Marshall A, Sjöström M, Bauman A, Booth M, Ainsworth B. the IPAQ Consensus Group and the IPAQ Reliability and Validity Study Group. International Physical Activity Questionnaire (IPAQ): 12-country reliability and validity. Med Sci Sports Exerc 2003;35:81–95.
[17] Raper N, Perloff B, Ingwersen L, Steinfeldt L, Anand J. An overview of USDA's dietary intake data system. J Food Compost Anal 2004;17:545–55.
[18] Moshfegh AJ, Rhodes DG, Baer DJ, Murayi T, Clemens JC, Rumpler WV, et al. The US Department of Agriculture automated multiple-pass method reduces bias in the collection of energy intakes. Am J Clin Nutr 2008;88:324–32.
[19] Haubrock J, Nöthlings U, Volatier J-L, Dekkers A, Ocké M, Harttig U, et al. Estimating usual food intake distributions by using the multiple source method in the EPIC-Potsdam Calibration Study. J Nutr 2011;141:914–20.
[20] Harttig U, Haubrock J, Knüppel S, Boeing H. The MSM program: web-based statistics package for estimating usual dietary intake using the multiple source method. Eur J Clin Nutr 2011;65:S87–91.
[21] Fisberg R, Marchioni D. Manual for food, nutrition and health population studies: the experience of the health survey in São Paulo (ISA). Food COnsumption Assessment Group (GAC). São Paulo: FSP/USP; 2012.
[22] Fisberg RM, Villar BS. Manual of recipes and homemade measures for the calculation of food surveys: a manual developed toa id the processing of food survey data. São Paulo: Signus; 2002.
[23] Pinheiro ABV, Lacerda EMdA, Benzecry EH, Gomes MCdS, Costa VMd. Table for evaluation of food consumption in domestic measures. 5th ed São Paulo: Atheneu; 2008.
[24] Nutrition Data System for Research. Nutrition Data System for Research. Minneapolis, MN: University of Minnesota; 2005 Version.
[25] Huang Z, Wang B, Crenshaw AA. A simple method for the analysis of trans fatty acid with GC–MS and AT^{TM}-Silar-90 capillary column. Food Chem 2006;98:593–8.
[26] Oki E, Norde MM, Carioca AAF, Ikeda RE, Souza JMP, Castro IA, et al. Interaction of SNP in the CRP gene and plasma fatty acid profile in inflammatory pattern: a cross-sectional population-based study. Nutrition 2016;32:88–94.
[27] Nevill AN, Atkinson G. Assessing agreement between measurements recorded on a ratio scale in sports medicine and sports science. Br J Sports Med 1997;31:314–8.
[28] Andersen LF, Solvoll K, Johansson LR, Salminen I, Aro A, Drevon CA. Evaluation of a food frequency questionnaire with weighed records, fatty acids, and alpha-tocopherol in adipose tissue and serum. Am J Epidemiol 1999;150:75–87.
[29] KurKuriki K, Nagaya T, Tokudome Y, Imaeda N, Fujiwara N, Sato J, et al. Plasma concentrations of (n-3) highly unsaturated fatty acids are good biomarkers of relative dietary fatty acid intakes: a cross-sectional study. J Nutr 2003;133:3643–50.
[30] SuSun Q, Ma J, Campos H, Hankinson SE, Hu FB. Comparison between plasma and erythrocyte fatty acid content as biomarkers of fatty acid intake in US women. Am J Clin Nutr 2007;86:74–81.
[31] Baylin A, Kim MK, Donovan-Palmer A, Siles X, Dougherty L, Tocco P, et al. Fasting whole blood as a biomarker of essential fatty acid intake in epidemiologic studies: comparison with adipose tissue and plasma. Am J Epidemiol 2005;162:373–81.
[32] Astorg P, Bertrais S, Laporte F, Arnault N, Estaquio C, Galan P, et al. Plasma n-6 and n-3 polyunsaturated fatty acids as biomarkers of their dietary intakes: a cross-sectional study within a cohort of middle-aged French men and women. Eur J Clin Nutr 2008;62:1155–61.
[33] Hodson L, Skeaff CM, Fielding BA. Fatty acid composition of adipose tissue and blood in humans and its use as a biomarker of dietary intake. Prog Lipid Res 2008;47:348–80.
[34] Emken EA, Adlof RO, Gulley RM. Dietary linoleic acid influences desaturation and acylation of deuterium-labeled linoleic and linolenic acids in young adult males. Biochim Biophys Acta Lipid Lipid Metab 1994;1213:277–88.
[35] Arterburn LM, Hall EB, Oken H. Distribution, interconversion, and dose response of n-3 fatty acids in humans. Am J Clin Nutr 2006;83:1467..S–76 S.
[36] Subbaiah PV, Kaufman D, Bagdade JD. Incorporation of dietary n-3 fatty acids into molecular species of phosphatidyl choline and cholesteryl ester in normal human plasma. Am J Clin Nutr 1993;58:360–8.
[37] Zuijdgeest-van Leeuwen SD, Dagnelie PC, Rietveld T, van den Berg JW, Wilson JH. Incorporation and washout of orally administered n-3 fatty acid ethyl esters in different plasma lipid fractions. Br J Nutr 1999;82:481–8.
[38] Vaz JdS, Deboni F, Azevedo MJd, Gross JL, Zelmanovitz T. Fatty acids as biological markers of fat intake. J Nutr 2006;19:489–500.
[39] Katan MB, Deslypere J, Van Birgelen A, Penders M, Zegwaard M. Kinetics of the incorporation of dietary fatty acids into serum cholesteryl esters, erythrocyte membranes, and adipose tissue: an 18-month controlled study. J Lipid Res 1997;38:2012–22.
[40] Appolinário PP, Derogis PBMC, Yamaguti TH, Miyamoto S. Metabolism, oxidation and biological implications of docosahexaenoic acid in neurodegenerative diseases. New Chem 2011;34:1409–16.