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Effect of Palm Oil Consumption on Cardiac Biomarkers of a Population Living in Jacqueville (Côte d'Ivoire)

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Abstract

Background and purpose: The use of palm oil in human food is widely debated. Some believe that this oil is unfit for consumption because it would promote the occurrence of cardiovascular diseases. For other authors on the other hand, palm oil is rich in compounds which give it enormous beneficial properties for the consumer. It is in this context of debate on palm oil that this study takes place. And it was conducted to examine the cardiovascular health of a population traditionally consuming palm oil by measuring serum values of cardiac biomarkers.

Methods: 83 people aged 18 to 50 of both sexes who had consumed exclusively palm oil at least during the 6 months preceding the present study were divided into 2 groups: The 30PoC group or persons who used 30 g of palm oil per day and the 67PoC group or persons who consumed 67 g/day. These persons have consumed palm oil either in its traditional form (red oil) or in its industrial form (refined oil), not both. Blood samples were taken and cardiac biomarkers were subsequently assayed.

Results: CRP, CK, CK-MB, LDH, AST, ALT and ALP were the cardiac biomarkers studied. No statistical difference was observed in the biomarkers CK, CK-MB, LDH, ALT and ALP regardless of sex, age range of subjects and the form in which palm oil is consumed. Statistical variations were recorded with AST and CRP. A significant difference (P=0.03) was observed by comparing the mean value of the AST of the 2 groups of subjects with 13,63 ± 4,91 IU/L (67PoC) and 16,45 ± 7,20 IU/L (30PoC). Considering sex, a very significant

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difference (*P*=0.006) in the mean value of AST between men (20.19 ± 70 IU/L) and women (13.96 ± 6.29 IU/L) in the 30PoC group and a significant difference (*P*=0.02) between men (16.23 ± 4.95 IU/L) and women (12.50 ± 4.52 IU/L) in the 67PoC group were been observed. A significant difference (*P*=0.02) was observed in the AST for the age group of 18 to 35 years with 16.92 ± 7.07 IU/L (30PoC) against 12.56 ± 4.30 IU/L (67PoC).CRP and AST of 30PoC subjects who consumed refined palm oil were statistically different to those of 67PoC subjects who consumed refined palm oil.

Conclusion: Consumption of palm oil did not adversely affect cardiac biomarkers. The recorded values are within the recommended physiological intervals. This study population would therefore not be exposed to the risk of cardiovascular disease attributable to palm oil.

Introduction

The use of palm oil in human food is the subject of much debate and controversy. According to some studies, the consumption of palm oil is bad for cardiovascular health. The main claim against palm oil is that it is a highly saturated fat and its consumption raises blood cholesterol levels. This increase in blood cholesterol levels would strongly promote the occurrence of coronary heart disease and the associated risks [1-3]. During the last decades, several other works including those of Shimizu and Desrochers [4] and Annamaria et al. [5] have also led to the same conclusion. For these authors, palm oil because of its high content of palmitic acid would not be beneficial for the health of the consumer. This could be explained by the structure of palmitic acid which is a Saturated Fatty Acid (SFA) with a strong hypercholesterolemic potential.

In opposition to this theory, Siri-Tarino et al [6]. published a meta-analysis of prospective studies that showed no relationship between SFA intake and the risk of coronary heart disease, cardiovascular disease and strokes. Better yet, recent studies have found protective effects of SFA consumption. For example, that of Praagman et al [7]. which has not established a relationship between the SFA intake and the occurrence of ischemic heart diseases. This study, carried out over a period of 12 years, rather underlined a protective effect of SFA against the onset of ischemic heart disease. For many authors, this beneficial effect of palm oil would be linked to chemical compounds it contains. Beyond its richness in palmitic acid, many other compounds are found in red palm oil. The non-glyceridic or non-saponifiable fraction of this oil contains many so-called minor compounds such as tocotrienols and tocopherols (partially preserved by refining), carotenoids, free and esterified phytosterols. The soluble fraction is rich in phenolic compounds (phenolic acids and flavonoids) [8]. It is the richest oil in tocotrienols [9,10] and beta-carotene which is the precursor of vitamin A [11]. Tocotrienols help to reduce plasma cholesterol by inhibiting HMG-CoA reductase with the direct consequence the decrease of endogenous cholesterol synthesis. Carotenoids and Tocotrienols exert antioxidant effects [12,13]. Palm oil is also an important source of phenolic compounds, including phenolic acids. The antioxidant effect of phenolic acids is well known [14]. It contains almost no trans fatty acids (TFA or *Trans* Fats), which is one of the reasons for the increase in its use in recent years [15].

Bataï et al. [16] evaluated the lipid profile and blood pressure in people consuming palm oil in the commune of Jacqueville (Côte d'Ivoire). These people use this oil exclusively for their meals every day for many years. They have a preference for palm oil for economic and cultural reasons. Palm oil has been part of West African culinary traditions for generations [17,18]. Its low cost, ease of conservation, resistance to oxidation and its melting point above 35°C explain its predominance in the cuisine of many people [15]. The results obtained are twofold. On the one hand, these results did not show no influence of the consumption of palm oil on the lipid profile and blood pressure which are risk factors for cardiovascular disease. And on the other hand, these authors also found that people investigated with an average BMI of 25.58 ± 3.76 kg/m^2 were overweight. However, various studies have established a relationship between being overweight and the risk of developing cardiovascular disease. These two recorded results would therefore seem contradictory with regard to certain literature. Also, the present study which is a logical continuation of the work of Bataï et al. [16] was carried out in order to better assess the cardiovascular health of these people investigated. For this, the effect of the consumption of palm oil on cardiac biomarkers was studied in these users of this oil.

Materials and methods

Ethics approval and consent to participate

This study was approved by the Ministry of Health and Public Hygiene in the Republic of Côte d'Ivoire. And the Ethical approval for this study was obtained from the Life Sciences and Health Ethics Committee of Côte d'Ivoire (CNESVS, reference: N / Ref: 043-20 / MSHP / CNESVS-kp).

Individual verbal consent to participate in the study was obtained prior to implementing the investigation. The objectives of the study were explained to each participant. They were assured that their information was anonymous and that it was only for research purposes. Only individuals that consented to participate in the study were considered.

Location and study population

The study took place in Jacqueville, a commune in the south of Côte d'Ivoire. Based on criteria defined by Bataï et al. [16], 83 people out of an initial staff of 363 investigated for a dietary survey were selected for the present study. They were adults of both sexes and aged 18 to 50 years. In addition to individual consent, the exclusive use of palm oil (refined, unrefined or both) for all meals during the six months preceding the survey, the preservation of anonymity, absence of chronic diseases, pregnancy and permanent presence in Jacqueville for a long time, other criteria prevailed for the selection of the 83 subjects. These 83 apparently healthy persons were chosen because they had the same eating habits. They do not combine the two types of palm oil. They use either refined palm oil or red (unrefined) palm oil, not both.

Depending on the daily quantity of palm oil consumed, the 83 persons were divided into 2 groups. Group 1 (30PoC) consisted of 40 persons consuming on average 2 tablespoons of 15 ml of palm oil. This quantity is estimated at 30 g of oil supplied to each person and corresponds to 270 Kcal/day or 10.8% of the total daily energy. Group 2 (67PoC) was composed of 43 persons consuming an average of 4.5 tablespoons 15 ml of palm oil corresponding to 67 g of oil. This oil quantity provides 603 Kcal corresponding to 24.12% of the total daily energy of each subject in this group. Anthropometric characteristics of the study subjects are shown in Table 1.

Blood collection and Biochemical essays

The blood samples from subjects with a 12-hour fast was carried out in the laboratory of the General Hospital of Jacqueville in the morning between 7 and 9 hours. On each person, 5 ml of blood was collected into Vacutainer tubes by venipuncture at the crease of the elbow. Blood samples were centrifuged at 4000 rpm for 5 min. Serum was collected. Each serum aliquot was duplicated for the assessment of cardiac biomarkers using Chemistry Analyzer (Cobas C311 HITACHI auto-analyser). Cardiac biomarkers measured were C-reactive protein (CRP), Creatine kinase (CK), Creatine kinase-MB isoforms (CK-MB), Lactate deshydrogénase (LDH), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT) and Alkaline phosphatase (ALP).

Data analysis

All the data were expressed as Mean ± SD (standard deviation). Statistical analyses were performed by one way analysis of variance ANOVA and differences between means were determined by Student's test t using SPSS 20 software. A value of $P < 0.05$ was considered significant.

Results

Cardiac biomarkers of the two study groups

The mean values of the cardiac biomarkers of the study groups are reported in Table 2. Analysis of this table shows that the average values obtained in the 2 groups (30PoC and 67PoC) were in the range of values universally accepted or recommended. Comparison of the mean values of CRP, CK, CK-MB, LDH, ALT and ALP of the 2 groups showed that these mean values did not change significantly ($P > 0.05$). For against, a significant difference (P=0.03) was observed by comparing the AST mean value of the 2 groups of subjects. The mean value of AST of 67PoC group was estimated to be 13.63 ± 4.91 IU/L. A higher value (16.45 ± 7.20 IU/L) was recorded with the 30PoC group consuming this oil at the rate of 30 g/day.

Effect of palm oil consumption on cardiac biomarkers based to the sex of the subjects

The comparison of mean values of cardiac biomarkers (CRP, CK, CK-MB, LDH, ALT and ALP) in subjects female and male of the same study group showed no significant difference ($P > 0.05$). With AST, a very significant difference (P=0.006) between men (20.19 ± 70 IU/L) and women (13.96 ± 6.29 IU/L) of 30PoC group and a significant difference (P=0.02) between men (16.23 ± 4.95 IU/L) and women (12.50 ± 4.52 IU/L) in 67PoC group were recorded. The comparison of the mean values of the cardiac biomarkers of the men of the 30PoC group with those of the men of the 67PoC group on the one hand, and on the other hand the mean values of the cardiac biomarkers of the women of the 30PoC group with those of the women of the 67PoC group revealed that the mean values varied not significantly ($P > 0.05$) (Table 3).

Effect of palm oil consumption on cardiac biomarkers based to the age of the subjects

Regarding to the age of the subjects (Table 4), the comparison between the mean values of cardiac biomarkers of subjects aged 18 to 35 years and those of subjects aged 36 to 50 years in the same study group did not did not show a significant difference ($P > 0.05$). Similarly, comparison of mean values of cardiac biomarkers (CRP, CK, CK-MB, LDH) of 30PoC subjects aged 18 to 35 and those of 67PoC subjects 18 to 35 years revealed no statistically significant difference ($P > 0.05$). There was also no significant difference between subjects aged 36 to 50 of the study groups ($P > 0.05$). On the contrary, a significant difference (P = 0.02) was observed concerning AST for the age fraction of 18 to 35 years. The AST mean value of 30PoC subjects was estimated to be 16.92 ± 7.07 IU/L. This ASAT value is greater than that of 67PoC subjects (12.56 ± 4.30 IU/L).

Incidence of palm oil form on consumer cardiac biomarkers

Regarding the consumption of the 2 different forms of palm oil (Table 5), a significant difference (P=0.04) was observed with the cardiac biomarkers CRP and LDH of the 30PoC subjects. CRP and LDH values of 30PoC subjects who consumed red palm oil were estimated at 8.87 ± 8.23 mg/L and 300.07 ± 54.55 IU/L respectively against 5.36 ± 1.82 mg/L (CRP) and 267.24 ± 46.06 IU/L for 30PoC subjects who consumed refined palm oil. In 67PoC group, no significant difference ($P > 0.05$) was observed between subjects who consumed red palm oil and those who consumed refined palm oil regardless of the cardiac biomarker studied. Comparison of the mean cardiac biomarker values of 30PoC subjects who consumed red palm oil with those of 67PoC subjects who also consumed palm oil did not reveal a significant difference ($P > 0.05$). CRP and AST values of 30PoC subjects who consumed refined palm oil were statistically different (P=0.02) to those of 67PoC subjects who consumed refined palm oil. The CRP of 30PoC subjects was estimated to be 5.36 ± 1.82 mg/L. This CRP value was lower than that of 67PoC subjects (7.57 ± 4.30 mg/L). With 5.36 ± 17.64 IU / L, 30PoC subjects consuming refined palm oil had a higher value of AST than that of 67PoC subjects (13.22 ± 4.83 IU/L).

Table 1: Anthropometric data of the study groups.

Anthropometric parameters	30PoC Group			67PoC Group			P_3-value	P_4-value
	Men (n = 16)	Women (n = 24)	P_1-value	Men (n = 13)	Women (n = 30)	P_2-value		
Weight (kg)	73.25 ± 12.00	69.28 ± 11.00	0.29	70.42 ± 10.00	71.71 ± 11.00	0.72	0.50	0.43
Age (year)	33.00 ± 9.00	32.00 ± 8.00	0.71	35.00 ± 10.00	36.00 ± 7.00	0.42	0.71	0.03
Height (m)	1.75 ± 0.07	1.61 ± 0.06	0.00	1.75 ± 0.07	1.63 ± 0.07	0.00	0.96	0.24
BMI (kg/m^2)	24.14 ± 4.00	26.53 ± 3.00	0.05	22.88 ± 3.00	26.76 ± 3.00	0.001	0.36	0.80

Values were expressed as Mean ± Standard Deviation. 30PoC: people consuming 30 g/day of palm oil; 67PoC: people consuming 67 g/day of palm oil; BMI: Body Mass Index ; P_1: p-value between men and women in the 30PoC group; P_2: p-value between men and women in the 67PoC group ; P_3 : p-value between men in the 30PoC group and those in the 67PoC group ; P_4: p-value between women in the 30PoC group and those in the 67PoC group. Bataï et al. [16].

Table 2: Mean values of cardiac biomarkers of the study population groups.

Cardiac biomarkers	Participants		*P*-value
	30PoC (n = 40)	67PoC (n = 43)	
CRP (mg/L)	6.68 ± 5.42	7.07 ± 4.04	0.70
CK (IU/L)	163.25 ± 42.10	174.79 ± 30.70	0.77
CK-MB (IU/L)	18.88 ± 3.32	18.51 ± 4.14	0.66
LDH (IU/L)	279.55 ± 51.31	282.67 ± 46.22	0.15
AST (IU/L)	16.45 ± 7.20	13.63 ± 4.91	0.03
ALT (IU/L)	9.50 ± 4.50	8.37 ± 3.43	0.20
ALP (IU/L)	252.63 ± 47.53	249.28 ± 48.95	0.75

Values were expressed as Mean ± Standard Deviation. 30PoC: people consuming 30 g/day of palm oil; 67PoC: people consuming 67 g/day of palm oil; CRP: C-reactive protein; CK: Creatine kinase; CK-MB: Creatine kinase-MB isoforms; LDH: Lactate deshydrogénase; AST: Aspartate aminotransferase; ALT: Alanine Aminotransferase, ALP: Alkaline Phosphatase.

Table 3: Mean values of cardiac biomarkers according to the sex of the respondents.

Cardiac biomarkers	30PoC Group			67PoC Group			P_3-value	P_4-value
	Men (n = 16)	Women (n = 24)	P_1-value	Men (n = 13)	Women (n = 30)	P_2-value		
CRP (mg/L)	5.25 ± 1.61	7.63 ± 6.76	0.17	6.69 ± 2.56	7.23 ± 4.56	0.69	0.07	0.80
CK (IU/L)	175.88 ± 18.25	154.83 ± 30.85	0.12	165.77 ± 28.49	178.70 ± 31.24	0.20	0.54	0.007
CK-MB (IU/L)	18.25 ± 2.69	19.29 ± 3.67	0.33	17.77 ± 4.47	18.83 ± 4.02	0.44	0.72	0.66
LDH (IU/L)	277.75 ± 49.23	280.75 ± 53.67	0.85	284.46 ± 53.28	281.90 ± 43.78	0.87	0.72	0.93
AST (IU/L)	20.19 ± 7.00	13.96 ± 6.29	0.006	16.23 ± 4.95	12.50 ± 4.52	0.02	0.09	0.32
ALT (IU/L)	11.06 ± 5.20	8.46 ± 3.73	0.07	8.15 ± 2.30	8.47 ± 3.85	0.78	0.07	0.99
ALP (IU/L)	245.56 ± 38.44	257.33 ± 53.00	0.45	233.23 ± 35.06	256.23 ± 52.88	0.16	0.38	0.94

Values were expressed as Mean ± Standard Deviation. 30PoC: people consuming 30 g/day of palm oil; 67PoC: people consuming 67 g/day of palm oil; CRP: C-reactive protein; CK: Creatine kinase; CK-MB: Creatine kinase-MB isoforms; LDH: Lactate Déshydrogénase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline Phosphatase; P_1 : *p*-value between men and women in the 30PoC group; P_2 : *p*-value between men and women in the 67PoC group; P_3: *p*-value between men in the 30PoC group and those in the 67PoC group ; P_4: *p*-value between women in the 30PoC group and those in the 67PoC group.

Table 4: Mean values of cardiac biomarkers based on the age of respondents.

Cardiac biomarkers	30PoC Group			67PoC Group			P_3-value	P_4-value
	18-35 years (n = 26)	36-50 years (n = 14)	P_1-value	18-35 years (n = 18)	36-50 years (n = 25)	P_2-value		
CRP (mg/L)	7.50 ± 6.51	5.14 ± 1.61	0.17	7.28 ± 3.65	6.92 ± 4.36	0.77	0.89	0.15
CK (IU/L)	158.92 ± 39.84	171.29 ± 46.45	0.38	165.50 ± 32.35	181.48 ± 28.21	0.09	0.56	0.39
CK-MB (IU/L)	18.62 ± 3.64	19.36 ± 2.67	0.50	18.33 ± 4.45	18.64 ± 3.98	0.81	0.81	0.55
LDH (IU/L)	271.92 ± 40.83	293.71 ± 66.03	0.20	285.89 ± 46.26	280.36 ± 47.00	0.70	0.29	0.46
AST (IU/L)	16.92 ± 7.07	15.57 ± 7.60	0.57	12.56 ± 4.30	14.40 ± 5.26	0.22	0.02	0.57
ALT (IU/L)	9.58 ± 4.88	9.36 ± 3.87	0.88	8.06 ± 3.78	8.60 ± 3.22	0.61	0.27	0.51
ALP (IU/L)	262.00 ± 45.94	235.21 ± 47.07	0.08	245.67 ± 41.45	251.88 ± 54.40	0.68	0.23	0.34

Values were expressed as Mean ± Standard Deviation. 30PoC: people consuming 30 g/day of palm oil; 67PoC: people consuming 67 g/day of palm oil; CRP: C-reactive protein; CK: Creatine Kinase; CK-MB: Creatine Kinase-MB isoforms; LDH: Lactate Déshydrogénase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase, ALP: Alkaline phosphatase; P_1: p-value between the two age group of 30PoC P_2: p-value between the two age range of 67PoC; P_3: p-value between the 18-35 age range of 30PoC subjects and that of 67PoC subjects; P_4: p-value between the 36-50 age group of 30PoC subjects and that of 67PoC subjects.

Table 5: Mean values of cardiac biomarkers based on the type of palm oil consumed.

Cardiac biomarkers	30PoC Group			67PoC			P_3-value	P_4-value
	Huile rouge (n = 15)	Huile raffinée (n = 25)	P_1-value	Huile rouge (n = 20)	Huile raffinée (n = 23)	P_2-value		
CRP (mg/L)	8.87 ± 8.23	5.36 ± 1.82	0.04	6.50 ± 3.74	7.57 ± 4.30	0.39	0.26	0.02
CK (IU/L)	153.07 ± 37.97	169.36 ± 43.99	0.24	173.60 ± 25.16	175.83 ± 35.34	0.81	0.06	0.57
CK-MB (IU/L)	17.67 ± 3.77	19.60 ± 2.85	0.07	17.45 ± 4.41	19.43 ± 3.74	0.11	0.88	0.86
LDH (IU/L)	300.07 ± 54.55	267.24 ± 46.06	0.04	288.05 ± 56.88	278.00 ± 35.16	0.48	0.53	0.37
AST (IU/L)	14.47 ± 5.31	17.64 ± 7.98	0.18	14.10 ± 5.08	13.22 ± 4.83	0.56	0.83	0.02
ALT (IU/L)	8.00 ± 3.16	10.40 ± 4.99	0.10	8.75 ± 4.10	8.04 ± 2.78	0.50	0.56	0.05
ALP (IU/L)	262.73 ± 48.92	246.56 ± 46.61	0.30	247.30 ± 46.82	251.00 ± 51.72	0.80	0.35	0.75

Values were expressed as Mean ± Standard Deviation. 30PoC: people consuming 30g/day of palm oil; 67PoC: people consuming 67g/day of palm oil; CRP: C-reactive protein ; CK: Creatine Kinase; CK-MB: Creatine kinase-MB isoforms ; LDH: Lactate Déshydrogénase; AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP : Alkaline phosphatase; P_1: p-value between consumers of red palm oil and those consuming refined palm oil in the 30PoC group; P_2: p-value between consumers of red palm oil and those consuming refined palm oil in the 67PoC group; P_3: p-value between consumers of red palm oil in the 30PoC group and those in the 67PoC group; P_4: p-value between consumers of refined palm oil in the 30PoC group and those in the 67PoC group.

Discussion

The biological evaluation of the cardiovascular health of this population was carried out from measurements of serum concentrations of cardiac biomarkers such as C-reactive protein (CRP), Creatine Kinase (CK), Creatine kinase-MB isoforms (CK-MB), Lactate Dehydrogenase (LDH), Aspartate Aminotransferase (AST) and Alkaline Phosphatase (ALP).

The mean CRP values of the 30PoC and 67PoC subjects were 6.68 ± 3.32 and 7.07 ± 4.04 mg/L, respectively. These are statistically equal values ($P > 0.05$) and below the critical threshold value of 10 mg/L. Studies suggest that despite intra-individual changes in CRP levels related to acute inflammatory episodes, it is currently recognized that a moderate and chronic increase in CRP represents a risk factor for cardiovascular disease in a general population [19,20] and more specifically in patients with chronic kidney disease [21,22]. Results obtained in the laboratory on a cohort of 186 dialysis patients show that on univariate analysis the increase in CRP is a risk factor for atherosclerosis with symptomatic manifestations. Several arguments support the hypothesis that CRP would not only witness a "micro-inflammation" present in atheromatous plaque but could be a direct actor in atherogenesis. Thus, it appears that the CRP produced locally in the plaque [23] increases increase oxidant production by inflammatory cells infiltrate subendothelial [24]. Indeed, by its binding to phosphorylcholine groups, CRP could facilitate capture of LDL by macrophages [25] and promote the expression of adhesion molecules [26,27]. Finally, according Chenillot et al. [28], elevation of CRP values above the normal value of this parameter occurs during acute inflammatory conditions. This shows that the study population was not prone to these inflammatory affections.

Regarding the total LDH, the results showed no significant difference ($P > 0.05$) between plasma average values of 30PoC subjects (279.55 ± 51.31 IU/L) and those of 67PoC subjects (282.67 ± 46.22 IU/L). The relationship between the plasma level of LDH and obesity is not established formally. Opinions are divergent. From a study conducted in a Saudian population of 240 subjects (136 obese vs 104 non-obese) and aged 39.78 ± 12.77 years, Cyrus et al. [29] concluded that the elevation of plasma LDH levels is significantly correlated with obesity (respectively 187.60 ± 50.30 versus 164.07 ± 26.97; $P < 0.05$). This is contrary to the results of Choi et al. [30]. Their work focused on a representative sample of the North Korean population (732 healthy adults aged 27 to 69) and showed that there is no statistically significant association between obesity and the plasma level of LDH ($P > 0.05$).

A significant change (P=0.03) in the mean value of AST in 30PoC subjects (16.45 ± 7.20 IU/L) compared to that of 67PoC subjects (13.63 ± 4.91 IU/L) was observed. With ALT, no significant variation ($P > 0.05$) in the mean value of this cardiac biomarker was recorded between 30PoC persons (9.50 ± 4.50 IU/L) and 67PoC persons (8 , 37 ± 3.43 IU / L). Similar results were reported by Das et al. [31] who worked on a population of 156 subjects (72 normal weight, 39 overweight and 45 obese). However, the results of this study are inconsistent with those of other studies which have shown a positive relationship between overweight and / or obesity and the increase of transaminases level, particularly ALT plasma elevation.

A study including 800 obese subjects in Italy showed that an increase in the mean values of AST and ALT was proportional to the BMI values in 21 % and 10 % of cases respectively [32]. These elevated transaminase levels have been found in the absence of any symptoms, signs or history of liver disease. In the Third National Health and Nutrition Survey (NHANES III), conducted from 1988 to 1994 in the United States, the prevalence of elevated transaminases was estimated at 7.9% in 15,676 adults aged 17 years and more. In 69% of these cases, hypertransaminasemia was significantly associated with overweight and obesity [33].

In another study in 5724 participants, ALT concentration increase was observed in 2.8% of subjects. 65% of these cases were attributed to overweight or obesity [34]. Jamali et al. [35] showed that plasma concentrations of ALT were significantly higher in obese than in normal-weight subjects. According to the data in the literature, obesity may be responsible for an in-

crease in transaminases, in particular that of ALT which could be linked to Non-Alcoholic Fatty Liver Disease (NAFLD) [36]. In addition, recent studies show that NAFLD is associated with cardiovascular diseases such as heart attacks and strokes. In fact, the biological mechanisms that explain the relationship between NAFLD and heart disease did not clearly established. A first hypothesis is the stimulator role of atherosclerosis played by hepatic steatosis. The second hypothesis involves oxidative stress and chronic inflammatory state, two phenomena encountered in cases of atherosclerosis and NAFLD. The third hypothesis incriminates adiponectin, a cytokine that has antiatherogenic properties. It was observed low adiponectin levels in patients with hepatic steatosis [37].

Although within the range of physiological values (18-215 IU/L), the mean CK level was not significantly higher in the 67PoC subjects (174.79 ± 30.70 IU / L) than in 30PoC subjects (163.25 ± 42.10 IU / L) with $P > 0.05$. In the same vein, CK-MB values were in the range of physiological values [0-23 IU/L]. It was valued at 18.88 ± 3.32 IU / L in 30PoC subjects against 18.51 ± 4.14 IU / L in the people of 67PoC group. Comparison of the CK-MB values did not show a statistical difference ($P > 0.05$) between the 30PoC subjects consuming moderately palm oil according to the literature and those of the 67PoC group who consumed large amounts of palm oil.

CK is a muscle necrosis indicator that increases with its magnitude. A study of CK in sports medicine provides insight into condition of muscle [38,39]. Thus, the monitoring of CK and the characterization of its isoenzymes are widely used in the diagnosis of myopathies, cardiomyopathies and encephalopathies [40,41]. CK and in particular its isoenzyme MB, is a reliable marker of myocardial necrosis, offering great sensitivity to detect infarct extension to predict the worst prognosis [42,43]. High levels of serum CK in apparently healthy individuals can be correlated with the physical training status. However, if these levels persist while at rest, it may be a sign of subclinical muscle disease, which training loads may indicate by the onset of symptoms such as deep fatigue [44]. Because primary skeletal muscle disorders are manifested by pain, fatigue, weakness and elevated serum CK [45]**.** Serum CK activity is also markedly increased in the preclinical stages of certain muscle diseases [46].

Other causes of serum CK elevation may be intramuscular injection. And the magnitude of serum CK elevation is proportional to the volume of injection [47] and the drug injected [48]. An elevation of serum CK levels may also occur in damaged muscles consecutive to a surgery. In these cases, CK levels are significantly higher in major surgeries than in minor surgeries [49]. Finally, intense exercise that damages the structure of skeletal muscle cells in the sarcolemma level [50] and Z-discs leads to an increase in the total CK [51, 52]. The origin of any increase in serum CK levels, whatever the cause, is believed to be due to defects in glycogen storage affecting the glycolytic or glycogenolytic pathway, causing myopathy with hyperCKemia [53]. A persistent asymptomatic form of hyperCKemia may be due to minor abnormalities. It has been reported in patients with only mild neuromuscular abnormalities [54].

The serum CK level assay in this study is interesting because constantly increased serum CK levels are sometimes seen in healthy persons. Often, subjects show no clinical signs of a neuromuscular disorder or any condition known to be associated with increased serum CK levels. Galassi et al. [55], studying subjects with high CK levels at rest, observed that, years later, the subjects developed weakness. They suggest that early myopathy may be asymptomatic [56]. Other authors have shown that in most of these patients, hyperCKemia probably does not involve disease [57]. Patients without skeletal muscle abnormalities on muscle biopsy may have idiopathic hyperCKemia [58]. In this study, a CK value of men in the 30PoC group greater than that of women in the same group was recorded. This result is similar to that of Fu et al. [59]. They showed the existence of real gender differences in resting serum CK levels. On the other hand, this result is contrary to that obtained in the 67PoC group. In this group, the value of CK for men was lower than that of women, probably because of the numbers of men and women. After muscle exercise, gender-related differences are still present. Estrogens could be an important factor in maintaining the stability of the membrane after exercise, thus limiting CK leakage from damaged muscle [60,61]. So the persistent elevation of CK should be thoroughly investigated [62] and could be important to assess the serum CK activity at rest and after exercise to identify silent myopathies.

A review of the results of this study shows that the measured cardiac biomarker values are within the intervals of physiological required for cardiovascular health. Though overweight [16], the study population would not present a risk of cardiovascular disease. Analysis of cardiac biomarkers showed that the mean values obtained in the 30PoC subjects and those of the 67PoC subjects were within the intervals of normal physiological values of the cardiac markers. Regardless of the quantity consumed, palm oil did not cause negative effects on consumers' cardiac markers in comparison to defined standards. Indeed, it is believed that the negative action of oils on the health of consumers would depend on the quality and quantity consumed. Thus, certain oils are considered good for human consumption and other qualified oils bad for human health. These bad oils are unfit for human consumption. Palm oil qualified as poor quality by several studies did not affect the levels of the biological parameters measured, with the exception of AST, both in 30PoC subjects consuming the required quantity of oil and in 67PoC subjects consuming an important quantity of this oil. The 30PoC people had mean values of cardiac markers within the recommended value ranges. This finding shows that in terms of quantity, palm oil did not impact cardiac biomarkers. The quantity of palm oil did not negatively influence these biological parameters either. The values obtained in subjects consuming a high amount of palm oil (67PoC) were not statistically different from those of 30PoC subjects who consumed an amount of palm oil corresponding to the amount of oil recommended for human consumption. These results are in agreement with those of previous studies which argue in favor of the use of palm oil in human food because of the various properties that this oil possesses. These properties are often overshadowed by other considerations. Contrary to the ideas conveyed by some research, palm oil does not contain bad cholesterol. It is the oil best suited to frying. It is an excellent source of vitamin A and E, essential for the mother-child health and the fight against rickets and childhood blindness [19]. It contains virtually no trans fatty acids, which is one of the reasons why its use has increased in recent years [15].

Conclusion

The study population despite the high BMI value both in 30PoC subjects moderately consuming palm oil and in 67PoC subjects consuming large amounts of palm oil did not present a risk of developing cardiovascular diseases. Consumption of palm oil did not adversely affect cardiac biomarkers (CRP, CK,

CK-MB, LDH, AST and ALT). The recorded values are within the recommended intervals.

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Competing interests

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

Authors' contribution

NFB proposed the research idea, collected the data from the respondents, organized the data in computer and did the analysis and interpretation wrote the manuscript. MLAB proposed the research idea, collected the data from the respondents and organized the data in computer. BAK did the analysis, interpretation and wrote the manuscript. AFF revised the manuscript for scientific content and did the language check. All authors gave final approval for its submission to the journal for consideration of publication.

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References

1. Mensah GA. Ischaemic heart disease in Africa. Heart. 2008; 94: 836-843.
2. Corley RHV, Tinker PBH. The oil palm. Blackwell Science: Oxford Malden MA. 2003; 4.
3. De Souza RJ, Mente A, Maroleanu A, et al. Intake of saturated and trans unsaturated fatty acids and risk of all-cause mortality, cardiovascular disease, and type 2 diabetes: Systematic review and meta-analysis of observational studies. BMJ. 2015; 351: h3978.
4. Shimizu H, Desrochers P. L'huile de palme: avantages sanitaires, environnementaux et économiques. Note économique. 2012.
5. Mancini A, Imperlini E, Nigro E, et al. Biological and nutritional proper-ties of palm oil and palmitic acid: Effects on health. Molecules. 2015; 20: 17339-17361.
6. Siri-Tarino PW, Sun Q, Hu FB, et al. Saturated fat, carbohydrate, and cardiovascular disease. Am J Clin Nutr. 2010; 91: 502-509.
7. Praagman J, Beulens JW, Alssema M, et al. The association between dietary saturated fatty acids and ischemic heart disease depends on the type and source of fatty acid in the European Prospective Investigation into cancer and Nutrition-Netherlands cohort. Am J Clin Nutr. 2016; 103: 356-365.
8. Cottrell RC. Introduction: Nutritional aspects of palm oil. Am J Clin Nutr. 1991; 53: 989S-1009S.
9. Sen CK, Rink C, Khanna S. Palm oil-derived natural vitamin E alpha-tocotrienol in brain health and disease. J Am Coll Nutr. 2010; 29: 314S-323S.
10. Sundram K, Sambanthamurthi R, Tan YA. Palm fruit chemistry and nutrition. Asia Pac J Clin Nutr. 2003; 12: 355-362.
11. Rice AL, Burns JB. Moving from efficacy to effectiveness: red palm oil's role in preventing vitamin a deficiency. J Am Coll Nutr. 2010; 29: 302S-313S.
12. Vasanthi HR, Parameswari RP, Das DK. Multifaceted role of tocotriénols in cardioprotection supports their structure: function relation. Genes Nutr. 2012; 7: 19-28.
13. Wong RSY, Radhakrishnan AK. Tocotrienol research: past into present. Nutr Rev. 2012; 70: 483-490.
14. Neo YP, Ariffin A, Tan CP, et al. Phenolic acid analysis and antioxidant activity assessment of oil palm (E. guineensis) fruits extracts. Food Chemistry. 2010; 122: 353-359.
15. CNUCED. Huile de palme. 2016. 21p. https://unctad.org/fr/PublicationsLibrary/INFOCOMM_cp08_PalmOil_fr.pdf. Accessed August 10, 2020.
16. Bataï NF, Ahui-Bitty ML, Fossou AF, et al. Lipid profil of a population of Jacqueville, consumer of palm oil in southern Côte d'Ivoire. Eur J Nutr Food Saf. 2020; 12: 1-10.
17. Haxaire C. Le palmier à huile chez les Gouro de Côte d'Ivoire. Journal des Africanistes. 1992; 62: 55-77.
18. Djè A. Afrique: L'huile de palme - Enjeux mondiaux pour une denrée alimentaire. Le Hub Rural. http://www.hubrural.org/Afrique-L-huile-de-palme-Enjeux.html. Accessed August 10, 2020.
19. Blake GJ, Ridker PM. Novel clinical markers of vascular wall inflammation. Circ Res. 2001; 89: 763-771.
20. Blake GJ, Ridker PM. C-reactive protein, subclinical atherosclerosis, and risk of cardiovascular events. Arterioscler Thromb Vasc Biol. 2002; 22: 1512-1513.
21. Stenvinkel P, Heimbürger O, Paultre F, et al. Strong association between malnutrition, inflammation, and atherosclerosis in chronic renal failure. Kidney Int. 1999; 55: 1899-1911.
22. Park CW, Shin YS, Kim CM, et al. Increased C-reactive protein following hemodialysis predicts cardiac hypertrophy in chronic hemodialysis patients. Am J Kidney Dis. 2002; 40: 1230-1239.
23. Yasojima K, Schwab C, McGeer EG, et al. Generation of C-reactive protein an complement components in atherosclerotic plaques. Am J Pathol. 2001; 158: 1039-1051.
24. Zeller JM, Landay AL, Lint TF, et al. Enhancement of human peripheral blood monocyte respiratory burst activity by aggregated C-reactive protein. J Leukoc Biol. 1986; 40: 769-783.
25. Zwaka TP, Hombach V, Torzewski J. C-reactive protein-mediated low density lipoprotein uptake by macrophages. Implications for atherosclerosis. Circulation. 2001; 103: 1194-1197.
26. Pasceri V, Cheng JS, Willerson JT, et al. Modulation of C-reactive protein-mediated monocyte chemoattractant protein-1 induction in human endothelial cells by anti- atherosclerosis drugs. Circulation. 2001; 103: 2531-2534.
27. Pasceri V, Willerson JT, Yeh ET. Direct proinflammatory effect of C-reactive protein on human endothelial cells. Circulation. 2000; 102: 2165-2168.
28. Chenillot O, Henny J, Steinmetz J, et al. High sensitivity C-reactive protein: biological variations and reference limits. Clin Chem Lab Med. 2000; 10: 1003-1011.
29. Cyrus C, Ismail MH, Chathoth S, et al. Analysis of the impact of common polymorphisms of the FTO and MC4R genes with the risk of severe obesity in saudi arabian population. Genet Test Mol Biomarkers. 2018; 22: 170-177.
30. Choi JW. Association between elevated serum hepatic enzyme activity and total body fat in obese humans. Ann Clin Lab Sci. 2003; 33: 257-264.
31. Das AK, Chandra P, Gupta A, et al. Obesity and the levels of liver

enzymes (ALT, AST & GGT) in East Medinipur, India. Asian J Med Sci. 2014; 6: 40-42.

32. Marchesini G, Avagnina S, Barantani EG, et al. Aminotransferases and gamma-glutamyltranspeptidase levels in obesity are associated with insulin resistance and the metabolic syndrome. J Endocrinol Invest. 2005; 28: 333-339.

33. Clark JM, Brancati FL, Diehl AM. The prevalence and etiology of elevated aminotransferase levels in the United States. Am J Gastroenterol. 2003; 98: 960-967.

34. Ruhl CE, Everhart JE. Determinants of the association of overweight with elevated serum alanine aminotransferase activity in the United States. Gastroenterology. 2003; 124: 71-79.

35. Jamali R, Pourshams A, Amini S, et al. The upper normal limit of serum alanine aminotransferase in Golestan Province, northeast Iran. Arch Iran Med. 2008; 11: 602-607.

36. Durand DV, Lega JC, Fassier T, et al. Unexplained, subclinical chronically elevated transaminases. Rev Med Interne. 2013; 34: 472-478.

37. Sdiri W, Romdhane H, Mbarack D, et al. La stéatose hépatique non alcoolique: un nouveau facteur de risque cardiovasculaire? Tunis Med. 2013; 91: 171-174.

38. Ponraj D, Gopalakrishnakone P. Establishment of an animal model for myoglobinuria by use of a myotoxin from pseudechis australis (king brown snake) venom in mice. Lab Anim Sci. 1996; 46: 393-398.

39. Lopes-Ferreira M, Núñez J, Rucavado A, et al. Skeletal muscle necrosis and regeneration after injection of Thalassophryne nattereri (niquim) fish venom in mice. Int J Exp Pathol. 2001; 82: 55-64.

40. Hoffman EP, Clemens PR. HyperCKemic, proximal muscular dystrophies and the dystrophin membrane cytoskeleton, including dystrophinopathies, sarcoglycanopathies, and merosinopathies. Curr Opin Rheumatol. 1996; 8: 528-538.

41. Hina K, Kusachi S, Iwasaki K, et al. Use of serum creatine kinase MM isoforms for predicting the progression of left ventricular dilatation in patients with hypertrophic cardiomyopathy. Jpn Circ J. 1997; 61: 315-322.

42. Rajappa M, Sharma A. Biomarkers of cardiac injury: An update. Angiology. 2005; 56: 677-691.

43. Galla JM, Mahaffey KW, Sapp SK, et al. Elevated creatine kinase-MB with normal creatine kinase predicts worse outcomes in patients with acute coronary syndromes: results from 4 large clinical trials. Am Heart J. 2006; 151: 16-24.

44. Angelini C. Limb-girdle muscular dystrophies: heterogeneity of clinical phenotypes and pathogenetic mechanisms. Acta Myol. 2004; 23: 130-136.

45. Engel AG, Orawa E. Chapter 34: Dystrophinopathies. In Engel AG, Franzini-Armstrong C, Myology: Basic and clinical, 3rd ed., New York, McGraw-Hill. 2004; 978p.

46. Miyoshi K, Kawai H, Iwasa M, et al. Autosomal recessive distal muscular dystrophy as a new type of progressive muscular dystrophy. Seventeen cases in eight families including an autopsied case. Brain. 1986; 109: 31-54.

47. Diness V. Local tissue damage after intramuscular injections in rabbits and pigs: quantitation by determination of creatine kinase activity at injection sites. Acta Pharmacol Toxicol (Copenh). 1985; 56: 410-415.

48. Montero Pérez FJ, Muñoz Avila J, Berlango Jiménez A, et al. Increase of blood levels of creative kinase following intramuscular injection. Med Clin (Barc). 1996; 107: 649-654.

49. Yousef MA, Vaida S, Somri M, et al. Changes in creatine phosphokinase (CK) concentrations after minor and major surgeries in children. Br J Anaesth. 2006; 96: 786-789.

50. Hornemann T, Stolz M, Wallimann T. Isoenzyme-specific interaction of muscle-type creatine kinase with the sarcomeric M-line is mediated by NH2–terminal lysine charge-clamps. J Cell Biol. 2000; 149: 1225-1234.

51. Noakes TD. Effect of exercise on serum enzyme activities in humans. Sports Med. 1987; 4: 245-267.

52. Epstein Y. Clinical significance of serum creatine phosphokinase activity levels following exercise. Isr J Med Sci. 1995; 31: 698-699.

53. Tsujino S, Nonaka I, DiMauro S. Glycogen storage myopathies. Neurol Clin. 2000; 18: 125-150.

54. Prelle A, Rigoletto C, Moggio M, et al. Asymptomatic familial hyperCKemia associated with desmin accumulation in skeletal muscle. J Neurol Sci. 1996; 140: 132-136.

55. Galassi G, Rowland LP, Hays AP, et al. High serum levels of creatine kinase: asymptomatic prelude to distal myopathy. Muscle Nerve. 1987; 10: 346-350.

56. Joy JL, Oh SJ. Asymptomatic hyper-CK-emia: An electrophysiologic and histopathologic study. Muscle Nerve. 1989; 12: 206-209.

57. Wokke JH. Raised creatine-kinase serum activity: not necessarily a sign of disease. Ned Tijdschr Geneeskd. 2003; 147: 1998-2000.

58. Prelle A, Tancredi L, Sciacco M, et al. Retrospective study of a large population of patients with asymptomatic or minimally symptomatic raised serum greatine kinase levels. J Neurol. 2002; 249: 305-311.

59. Fu FH, You CY, Kong ZW. Acute changes in selected serum enzyme and metabolite concentrations in 12-yr to 14 yr old athletes after an all-out 100 m swimming sprint. Percept Motor Skills. 2002. 95: 1171-1178.

60. Amelink GJ, Koot RW, Erich WB, et al. Sex-linked variation in creatine kinase release, and its dependence on oestradiol, can be demonstrated in an in-vitro rat skeletal muscle preparation. Acta Physiol Scand. 1990; 138: 115-124.

61. Tiidus PM. Estrogen and gender effects on muscle damage, inflammation and oxidative stress. Can J Appl Physiol. 2000; 25: 274-287.

62. Finsterer J, Neuhuber W, Mittendorfer B. Reconsidering idiopathic CK-elevation. Int J Neurosci. 2004; 114: 1333-1342.