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Effect of *Canarium Schweinfurthii* and *Dacryodes edulis* Oils on Blood Lipids, Lipid Peroxidation and Oxidative Stress in Rats

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Abstract: The aim of this study was to compare the effects of two vegetable oils on the growth, blood lipids, lipid peroxidation and oxidative stress in rats. One month old male rats Sprague Dawley were fed during 60 days with a diet containing *Canarium schweinfurthii* oil (CSO group), or *Dacryodes edulis* oil (DEO group), or palm oil (PO group). Palm oil was used as control. On 6 rats before the experimentation (baseline group) and on rats fed different diets, serum were used for total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, glycerol, creatinine, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, bilirubine determinations. Plasma were used for fatty acids, selenium and malondialdehyde determination. Glutathione peroxydase and superoxyde dismutase were measured in red blood cells. The daily food intake was different from one group to another.During the first month of the experiment, CSO diet was the most consummed. The gain in body weight was highest for the PO group. This group presents a high value of triglycerides, glycerol and asparatate aminotransferase compare to CSO and DEO groups. HDL-cholesterol, creatinine, alaninine aminotransferase, alkaline phosphatase, total bilirubine malondialdehyde and selenium level was not significantly different ($p<0.05$) for rats fed PO, CSO and DEO diets. Oils experimented contribute to the decrease the LDL-cholesterol level for over 50% in comparison of the baseline group. The atherogenicity index is highest for baseline group than PO, CSO and DEO groups. Oils experimented influenced glycerol, alanine aminotransferase level. We can conclude that oils experimented decrease the risk factor of cardiovascular disease to 43.91% for CSO and DEO groups and to 39.30% for PO group.

Key words: *Canarium schweinfurthii*, *dacryodes edulis*, oils, blood biochemistry, lipid peroxidation, oxidative stress

INTRODUCTION

Central Africa disposes of many oleaginous oil bearing seeds which are undervalued in relation to their potential. Among these are *Canarium schweinfurthii* Engl. and *Dacryodes edulis* (G. Don) H. J. Lam of the Burseraceae family. *C. Schweinfurthii* and *D. edulis* are often eaten as nibbles in Cameroon. They contain high of

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lipidscontents: between 39 and 59%[1]. They therefore constitute «hidden fats» yet their exploitation could make them an alternative source of dietary fats. In their general dietary habits, the population uses fats from some conventional oleaginous fruits (palm kernels, groundnut seeds, cotton seeds, maize seeds). However, they don't always cover their requirement in fats. Yet the fatty acids composition of these oils indicates a high amount of oleic acid (*C. schweinfurthii* 26.96%, *D. edulis*, 30.41%); linoleic acid (*D. edulis*, 24.55%, *C. schweinfurthii* 29.85%)[1]. Previous investigations have established that the type and proportion of fatty acids presents a particular effect on the lipoprotein profile[2-4]. The fatty acids composition of dietary fats constitutes a good argument to define their nutritional value. Many studies indicated a correlation between dietary fatty acids and their beneficial effects on health. Hypercholesterolemia and hypertriglyceridemia are one of the important risk factors for cardiovascular disease[5].

Many studies indicated that the type of lipid in the diet influences the metabolisme and level of circulate lipids and consequently the risk of development of cardiovascular disease and atheroclerosis[6-11]. According to Jaya and Pinnavaia and Balkan *et al.* works[12,13], the hypercholesterolemia can be linked to atherosclerosis via oxidative stress. Until now, the consequence of the consumption of diets containing *C. schweinfurthii* and *D. edulis* oils on blood lipids, circulate fatty acids, lipid peroxydation and oxidative stress have not yet been studied. It is therefore important to determine the beneficial effect of these vegetable oils with health and their consequence on risk factors of cardiovascular disease when it is well established that coronary diseases are the most common reason for the death of men and women[14]. The aim of this study was to compare the effect of consumption of *C. schweinfurthii* and *D. edulis* oils on growth, blood biochemistry, lipid peroxidation and oxidative stress of rats.

MATERIALS AND METHODS

Oils: Oils from the pulp of both fruits were obtained from Laboratory of Biochemistry and Food Technology (National Advanced School of Agro-Industrial Sciences, University of Ngaoundere-Cameroon)[1].

Animals and diets: 1 month old weaned male albinos Sprague Dawley rats (Harlan, France) weighing 142 to 157 g were individually housed in polycarbonate cages in a controlled environment with a temperature of 23°C, 12 h light-and-dark cycle light at 7:00 AM[15,10]. During an adaptation period of 1 week, the rats received tap water and a commercial rat diet *ad libitum*[16,8]. Animals had free access to water and food[8]. At the end of this period, 6 rats were used for general analysis (baseline group). The other rats were weighed and randomly assigned to one of the three groups (n = 6 rats/group) according to diet composition. For 60 days, each group was fed a diet containing one of the following: palm oil (PO group), *C. schweinfurthii*oil (CSO group), *D. edulis* oil (DEO group). Oil represents 5% of the composition of the diet as prescribed by American Institute of Nutrition[17]. The diet composition was as follow: Moisture content 8.53%, proteins 21.48%, dextrose 32.00%, starch 26.42%, cellulose 6.35%, mineral mix 4.58%, vitamines mix 0.64% and oil 5.00%. The fatty acids composition of these diets Table 1 was analysed by gas chromatography[18]. Palm oil was in this experiment as a control because it's an oil usually used and consumed in Cameroon. Food was given each week and water twice a week. Food consumption and body weight were determined twice a week the same day at the same time.

Table 1: Fatty acids composition of dietary fats (% of total fatty acids)

Fatty acids	PO	CSO	DEO
C 8	0.02	0.96	0.89
C10	0.06	0.53	0.29
C12	0.20	-	-
C14	0.85	0.44	0.30
C16	40.95	60.88	59.57
C16:1 (ω-7)	-	-	1.52
C18	5.70	2.46	4.47
C18:1 (ω-9)	41.35	30.70	29.31
C18:2 (ω-6)	9.70	3.05	3.18
C18:3 (ω-3)	0.37	0.93	0.42
C20	0.54	-	-
20:1 (ω-9)	0.20	-	-
Total satured (%)	48.32	65.27	65.52
Total monounsatured (%)	41.55	30.70	30.83
Total polyunsaturated (%)	10.07	3.98	3.60
AGPI/AGS	0.21	0.06	0.05
ω-6/ω-3	0.04	0.30	0.13

PO, the diet containing Palm oil; CSO, the diet containing *Canarium schweinfurthii* oil; DEO, the diet containing *Dacryodes edulis* oil

Experimental procedure: At the end of the feeding period (60 days), the rats fasted overnight at least 12 h, then were weighed, anesthetized with halothane. Blood samples were immediately collected from the heart in two tubes to obtain serum and plasma (heparin tubes). Serum was separated by centrifugation at 3000 rpm for 5 min (4°C) and plasma was obtained by centrifugation at 1500 rpm for 10 min (4°C). Serum was used for total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, glycerol, creatinine, alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT), alkaline phosphatase and bilirubine determinations. Plasma was used for fatty acids concentrations, for malondialdehyde and for selenium

determinations. Oxidative stress was was characterise by measuring Glutathione Peroxydase (GPx) and Superoxyde Dismutase (SOD) activities in red blood cells. The protocol was approved by the Experimental Research committee.

Serum metabolites: The serum triglycerides and the total cholesterol were analyzed using enzymatic colorimetric tests (Olympus, Cat. No. OSR6133 and OSR6516 respectively, Olympus Diagnostica GmbH, Ireland). HDL-cholesterol and LDL-cholesterol were analyzed using enzymatic kit BioMerieux (Cat. No. 61530 et 61534, respectively, BioMerieux, France). Glycerol was determined using an enzymatic kit test at 37°C (Cat. No. GY 105, Randox-France). Protein content was determined according to the method of Gornall[19]. Creatinine was analyzed according to the Jaffe method (Cat No. CR511, Olympus Diagnostica GmbH, Ireland). Alanine aminotransferase and Aspartate aminotransferase activities were determined by Olympus kits (Cat. No. OSR6209 and OSR6107, respectively, Olympus Diagnostica GmbH, Ireland) according to the recommandations of the International Federation of Clinical Chemistry. Alkaline phosphatase was analysed according to the recommandations of the International Federation of Clinical Chemistry by using Olympus kit (Cat. No. OSR6104). The activity of alkaline phosphatase is determine by the measuring the conversion level of p-nitro-phenyl-phosphate (pNPP) to p-nitro-phenol (pNP) in the presence of magnesium, zinc and 2-amino-2 methyl-1propanaol (AMP) as acceptor of phosphate at pH 10.4. The total bilirubine level was determined according the Winkelman *et al.* method[20] by using bilirubine total reactif (IR701-T, Synermed®, Synermed Internation Inc.-USA).

Plasmatic fatty acids: The plasmatic fatty acids concentration was analysed by a direct transesterification method[21,22]. Two hundred microliter of plasma were introduiced in glass tubes. Two milliliter of methanol/benzene 4:1 (v/v) containing 300 μg of tricosanoic acid (Sigma Aldrich Co, Saint Louis-USA) as internal standard w ere added to each tube. Two hundred microliter of acetyl chloride were slowly added over a period of 1 min. Tubes were tighty closed and subjected to methanolysis at 100°C for 1 h. After cooling the tubes in water, 5 mL of potassium carbonate 6% (w/v) was added to stop the reaction and neutralize the mixture. The tubes were then shaken for 5 min and centrifuged at 4500 rtm/min during 5 min. One microliter of aliquot of benzene upper phase was injected into the gas chromatography Peri 2000 (Perichrom; Saulx-le Chartreux, France) equipped with a flame ionization detector (300°C), utilizing a 50 m WCOT FUSED SILICA column with an external diameter of 0.39 mm and internal diameter of 0.35 mm. Nitrogen was used as carrier gas (100 mL min^{-1}) and hydrogen as conductor gas (3 to 5 mL min^{-1}). The injector port temperature was 270°C and the detector was 290°C. The identification of the peaks were established by comparison with methyl esters of PUFA-1 Marine source (Supelco, N° 4-7033, Bellefonte, PA-USA), of PUFA-2 Animal source (Supelco, N° 4-7015-U, Bellefonte, PA-USA).

Lipids peroxidation: Malondialdehyde (MDA): Plasmatic malondialdehyde was determined using the Sheu *et al.* method[23]. Zero point five milliliter of plasma were introduice in tubes containing 1.5 mL of phosphoric acid (9% p/v using 85% phosphoric acid) solution. The reagent blank was prepared by pipetting 0.5 mL of ethanol into tube containing 1.5 mL of phosphoric acid (9% p/v) solution. 0.5 mL of each standard solution (2.5, 5, 10 and 20 μmol L^{-1}) (1, 1, 3, 3-tetraethoxypropan, Sigma-France) were pipetting into tubes containing 1.5 mL of phosphoric acid (9% p/v) solution. 0.5 mL of thiobarbituric acid (30 mM) solution was added to each tube and the contents were mixed vigourously. The tubes were closed and placed in a boiling water bath for 30 min. After cooling tubes to room temperature with tap water, 2.5 mL of butanol were added to each tube. The contents were mixed for 20 s with a vortex mixer and centrifuged for 20 min (4500 rtm/min, 25°C). The absorbance of each butanol extract was readed at 534 nm.

Oxidative stress: Oxidative stress was evaluated by glutathione peroxydase, superoxyde dismutase and selenium. The determination of glutathion peroxydase activity is based on the measurement of the oxidation of the reduced glutathione by hydrogen peroxide. The quantity of GSSG formed was measured by evaluating the number of oxidized NADPH per minute and per gram of hemoglobine[24,25]. Superoxyde dismutase activity was determined using RANSOD kit (Cat. No. SD 125, Randox France). Selenium plasma was analyzed by atomic absorption technic at 196 nm using the Chappuis method[26].

Statiscal analysis: Results are presented as means±standard deviation. For each group, result obtained is the mean for 6 rats. All results were analysed using a one-way analysis of variance. Duncan's Multiple Range test was performed to evaluate differences between groups. $p<0.05$ was considered significant.

RESULTS AND DISCUSSION

Rat growth: Weight gain was greater for the PO group (199.67±8.38 g) than the CSO (134.67±3.42 g) and DEO

Table 2: Effects of diets on total cholesterol (TC), HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), triglycerides (TG), glycerol, creatinine, alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT), alkaline phosphatase (AP), total bilirubine and proteins levels in serum

Parameters	Baseline	PO	CSO	DEO
TC (g L^{-1})	1.42±0.12^{c}	0.92±0.06^{a,b}	0.9±0.1^{a}	0.88±0.13^{a}
HDL-C (g L^{-1})	0.55±0.02^{b}	0.41±0.05^{a}	0.43±0.06^{a}	0.41±0.05^{a}
LDL-C (g L^{-1})	0.23±0.03^{b}	0.10±0.01^{a}	0.10±0.01^{a}	0.10±0.02^{a}
TG (g L^{-1})	1.14±0.25^{d}	0.67±0.11^{b,c}	0.50±0.13^{a,b}	0.48±0.1^{a}
Glycerol(g L^{-1})	0.17±0.06^{a}	0.25±0.05^{b}	0.15±0.03^{a}	0.14±0.01^{a}
Creatinine (mg L^{-1})	11.97±0.77^{a}	10.70±0.80^{a}	11.92±0.74^{a}	11.09±0.77^{a}
ALAT (UI L^{-1})	74.91±4.71^{b}	59.66±13.44^{a,b}	54.16±9.15^{a}	48.66±4.92^{a}
ASAT (UI L^{-1})	137.33±8.28^{a,b}	210.00±54.85^{c}	149.75±14.17^{b}	159.5±31.8^{b}
AP (UI L^{-1})	nd	126.33±19.53^{a}	116.00±24.11^{a}	119.83±20.53^{a}
Bilirubine (mg L^{-1})	nd	1.16±0.40^{a}	1.16±0.40^{a}	1.00±0.00^{a}
Proteins (g L^{-1})	64.05±4.50^{a}	65.50±5.85^{a}	65.33±2.25^{a}	66.16±5.00^{a}
CT/HDL	2.56	2.26	2.09	2.13
LDL/HDL	0.41	0.23	0.23	0.25

* Values are means±standard error of the mean. PO, the diet containing palm oil; CSO, the diet containing *Canarium schweinfurthii* oil; DEO, the diet containing *Dacryodes edulis* oil, Values in the same row with different superscript letters are significantly different from one another at p<0,05 (Duncan's test) nd = No determined

Table 3: Plasmatic fatty acids concentration (µg mL^{-1}) in rats fed the study diets for 60 days*

	Group PO	Groupe CSO	Groupe DEO
C 14:0	7.64		
C 16:0	303.24±36.22	308.56±57.73	342.80±45.50
C 16:1 ω-7	34.90±1.60	56.44±20.16	42.30±2.30
C 18:0	157.00±22.00	149.69±30.03	175.00±25.00
C18:1 ω-9	272.50±9.00	195.77±52.56	223.00±24.00
C 18:1 ω-7	42.00±4.40	43.64±21.17	39.70±0.30
C 18:2 ω-6	124.70±5.00	185.84±43.14	171.00±1.30
C 20:4 ω-6	444.00±67.00	419.70±75.00	538.70±28.20
C 22:6 ω-3	17.90	22.50±7.60	20.60±1.10
Total	1386.50±125.30	1382.00±254.00	1552.80±118.60

* Values are means±standard error of the mean. PO, the diet containing Palm oil; CSO, the diet containing *Canarium schweinfurthii* oil; DEO, the diet containing *Dacryodes edulis* oil

(118.34±10.70 g) groups. The daily food intake was different from one group to another. The growth and feeding efficiency of the rats in the same group and for differents groups, varied during the experimental period.

Serum metabolites: Table 2 summarises the biochemichal parameters in serum samples. Total cholesterol varied from one group to another. The levels of serum of total cholesterol for the rats of PO, CSO and DEO groups are not significantly different (p<0.05). Compared to the Baseline, the PO, CSO and DEO HDL-cholesterol level in serum decreased. The decrease in the total cholesterol level can probably be associated with the decrease in cholesterol absorption and its low incorporation in chylomicrons, Very Low Density Lipoproteins and Intermediate Density Lipoproteins[27]. Many enzymes and proteins of transport participated in cholesterol absorption. Plasma cholesterol level is dependent on several parameters, including endogenous synthesis, secretion and catabolism of various plasma lipoproteins.

HDL-cholesterol is essential in the transport of cholesterol from cells and artery to liver where it is catabolised[28]. In this study, HDL-cholesterol constitutes a factor protecting against cardiovascular disease. No significant differences in HDL-cholesterol levels were observed between PO, CSO and DEO groups (p<0.05). The fact that HDL-cholesterol decreased with age (60 days) for PO, CSO and DEO groups is similar with results obtained by[29]. These authors showed that HDL-cholesterol level of men and women decrease with age. HDL-cholesterol have others fonctions that can contribute to reduice cardio-vascular risk: anti-inflammatory property (reduice cholesterol deposit in artery)[30].

At baseline, LDL-cholesterol level was higher (0.23±0.03 g L^{-1}) in rats compared to those fed PO, CSO and DEO diets. The levels of serum LDL-cholesterol of PO, CSO and DEO are not significatively different (p<0.05). LDL-cholesterol is responsible for the accumulation of lipids in the arterial wall, therefore it constitutes a risk factor for coronary heart disease. Oils experimented contribute to the decrease the LDL-cholesterol level for over 50% in comparison of the baseline group.

LDL/HDL ratio and CT/HDL (atherogenic index) are good markers for identifying and minimizing the risk of cardiovascular diseases. The increase of these ratios increase the risk of cardiovascular diseases[31,32]. Values obtained for CT/HDL or LDL/HDL ratio showed that this ratio is higher for baseline group than for DEO, CSO anf PO groups. We can conclude that oils expimented decrease the risk factor to 43.91% for CSO and DEO groups and to 39.30% for PO group.

The serum triacylglycerol levels in rats from the Baseline group were higher (1.14±0.25 g L^{-1}) than those of PO, CSO and DEO groups. The Duncan test showed that there was not a significant difference in the triglyceride levels of the CSO and DEO groups. Triglycerides are defined as an independent risk factor for coronary heart disease[31]. The differences between triglycerides levels can be explained by the fact of

difference in the metabolism of differents lipids according to their fatty acids content. The increase in ω-3 fatty acids mainly decreases serum triglycerides[33,34]. The high serum triglyceride concentrations increases the risk of coronary heart disease even when all other known risk factors are taken into account[7].

The type of diet influenced the serum glycerol level. PO group have the highest glycerol level.

Dietary lipid consumption can affect the functions of the kidneys or the liver. Determination of serum creatinine level is commonly used as a factor to evaluate the function of the kidneys because of its link to glomerular filtration rate. In general, a high serum creatinine level increases kidney failure[32]. Serum creatinine of Baseline, PO, CSO and DEO groups was not affected by diets.

Liver function tests are carried out to assess the state of liver function. Alanine aminotransferase (ALAT) and aspartate aminotransferase (ASAT) are the most commonly used markers of hepatocyte injuries. ASAT are found in the heart, the liver, the skeletal muscle and the kidney whereas ALAT is more abundant in the liver. According to Hohnadel[35], the high serum levels of ALAT are caused by the increased membrane permeability of the hepatocyte and the release of enzymes from liver cells into serum. Liver alterations produce considerable increases of both enzymes, predominantly ALAT[16]. The Baseline ALAT level was higher than that of DEO, CSO and PO groups. No significant difference in serum ALAT levels was observed between CSO, DEO and PO groups ($p<0.05$). Serum ASAT of PO rats were the highest compared to those of baseline, CSO and DEO.

The alkaline phosphatase level for differents groups is not significantly different ($p<0.05$). Oils experimented did not influenced the alkaline phosphatase level. The same conclusion is valid for the total bilirubine concentration and serum protein level.

Plasmatic fatty acids: Table 3 presentes the plasmatic concentration fatty acids of rats μg mL^{-1} de plasma). The plasmatic fatty acids concentration varied for 1382.00±254.00 μg mL^{-1} (CSO) to 1552.80±118.60 μg mL^{-1} (DEO). For the saturated fatty acids, the proportion of palmitic acid was similar in all groups (21.87; 21.33 and 22.07% of total fatty acid, respectively for PO, CSO and DEO groups). The remark was valid for stearic acid (11.31; 11.79 and 11.25% of total fatty acid respectively for PO, CSO and DEO groups).

In the case of mononsaturated fatty acids, addition to palmitoleic and oleic acids, trans vaccenic (C 18:1, ω-7) was identified in the plasma of rats of differents groups. This fatty acid was concentrated in groups PO (3.01% of total fatty acids) than of CSO (1.45% of total fatty acid, respectively) and DEO (2.55% of total fatty acid, respectively).

For polyunsaturated fatty acids, arachidonic acid (C 20: ω-6) was low in the PO group (32.02% of total fatty acids) compared to DEO group (34.69% of total fatty acids) and CSO group (37.86% of total fatty acids). The different plasmatic concentration of arachidonic acid could contribute in variation of the synthesis of eicosanoids. Indeed, arachidonic acid is the precussor of thromboxane of type 2 which have a strong agglutination effect of plaquettes and an vasoconstriction effect; leucotriens of type 4 which have pro-inflammatory properties[36]. Any of the three groups presented eicosapentaenoic acid (EPA) (C20: 5 ω-3) which have many physiological functions (protection of cardiovascular and nervous systems, protection from inflammatory). EPA is the precussor of prostagladine PGl_3 which has anti-agglutination effect[36]. This situation can be justified by the fact that PO, CSO and DEO diets were poor in linolenic acid (0.37; 0.93 and 0.42% of total fatty acids), an essential fatty acid for the synthesis of EPA in presence of $\triangle^6$ désaturase, $\triangle^5$ désaturase and an elongase. However, the presence of Docosahexaenoic Acid (DHA) (C22: 6 ω-3) was identified in all groups: 1.30% for PO group, 1.32 for DEO group and 2.38% for CSO group.

Malondialdehyde: Lipid oxidation, a process mediated by free radicals is considered to be important in the development of atherosclerosis[8]. The estimation of free radical activity was done through the determination of malondialdehyde (MDA) which is a by-product of lipid peroxidation[37]. The plasma malondialdehyde of the Baseline group was lower than that of the PO, DEO and CSO groups. The Duncan test showed that there was not a significant difference ($p<0.05$) between the value of plasma MDA for PO, CSO and DEO Table 4. Feeding PO, CSO and DEO diets increased plasma MDA concentrations compared to the baseline. Halliwel and Chirico[38] demonstrated the higher stability of saturated and monounsaturated oils in lipid peroxidation than polyunsaturated fatty acids. Tocopherols concentrations of oils are important in the preservation of lipid peroxidation. A high concentration of tocopherols in oils reduces the plasma MDA level[39]. The low plasma MDA of PO can be justified by the high concentration of palm oil in α-tocopherol (2.95 mg^{-1} d'huile) compared to CSO and DEO (0.53 and 1.63 mg g^{-1}, respectively)[1].

Superoxyde dismutase, glutathion peroxydase and selenium: Superoxyde dismutase (SOD) and glutathion

Table 4: Effects of dietary fat on malondialdehyde (MDA), superoxyde dismutase (SOD), glutathion peroxydase (GPx) and selenium levels in rats fed the study diets for 60 days*

Parameters	Baseline	PO	CSO	DEO
MDA (umol L^{-1})	0.56±0.17^{a}	2.08±0.92^{b}	2.43±0.78^{b}	2.19±0.71^{b}
SOD (U mL^{-1} GR)	739.80±34.50^{c}	621.16±115.85^{a,b}	654.00±49.47^{b,c}	54.00±92.17^{b,c}
GPx (mmoles/l/min)	23.46±0.15^{a}	69.21±0.77^{b}	75.00±6.24^{b}	70.54±9.40^{b}
Selenium (µg L^{-1})	308.67±27.30^{a}	652.00±35.83^{b}	671.33±71.77^{b}	629.66±38.43^{b}

* Values are means±standard error of the mean. PO, the diet containing Palm oil; CSO, the diet containing *Canarium schweinfurthii* oil; DEO, the diet containing *Dacryodes edulis* oil, Values in the same row with different superscript letters are significantly different from one another at p<0,05 (Duncan's test)

peroxydase (GPx) are enzymes of the antioxidant defense systems which participate in regulating the lipid peroxidation. A decrease in the activities or expression of these enzymes may predispose tissues to free radical damage. Selenium is an indispensable trace element, acting as a Gpx cofactor. Selenium activates GPx, Gpx therefore block the formation of hydroxyl radicals. Dietary lipids significantly affected the activity of SOD in red blood cells ($p<0.05$). The SOD Baseline activity (Table 4) was higher than that of the PO, CSO and DEO groups. The GPx Baseline activity and selenium level were lower than that of PO, CSO and DEO. The Duncan test showed that there was no difference ($p<0.05$) between GPx levels in rats fed CSO and DEO.

Plasma selenium Baseline level was lower than that of the DEO, PO and CSO groups (Table 4). The increase of the level of GPx was correlate with the increase of the selenium level ($r = 0.84$, $y = 6.54x+200.29$, where y is GPx level and x is the selenium level). The Duncan test showed that there was no significant difference ($p<0.05$) between plasma selenium levels of the PO, CSO and DEO groups.

CONCLUSION

HDL-cholesterol, LDL-cholesterol, creatinine, alaninine aminotransferase, alkaline phosphatase, total bilirubine, malondialdehyde, superoxyde dismutase, glutathion peroxydase and selenium level was not significantly different ($p<0.05$) for rats fed PO, CSO and DEO diets. The LDL/HDL ratio indicated that palm oil is most atherogenic than *C. schweinfurthii* and *D. edulis* oils. PO group has the highest ASAT concentration. Arachidonic acid was low in the PO group. The different plasmatic concentration of arachidonic acid could contribute in variation of the synthesis of eicosanoids. Any of the three groups presents eicosapentaenoic acid which have many physiological functions.

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