# Effect of two dietary oils from : *Ricinodendron heutelotii* (Bail.) and *Tetracarpidium Conophorum* müll. nuts on some biochemical blood parameters of rats

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*Abstract*
The aim of this study was to compare the effects of two dietary vegetable oils on the growth, blood lipids, lipid peroxidation and oxidative stress in male rats. Male Sprague Dawley rats (age : 1 month, n = 18) were fed with a standard diet (AS group) ; a diet containing *Tetracarpidium conophorum* oil (TCO group) or *Ricinodendron heudelotii* oil (RHO group). After 60 days of feeding with different diets, serum were collected and used for total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, glycerol, creatinine, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, bilirubine determinations. Plasma were used for selenium and malondialdehyde determinations. Plasma fatty acids were determined by using Gaz Liquid Chromatography. Glutathione peroxydase and superoxide dismutase were also measured in red blood cells using appropriated kits. AS group presents the highest value of triglycerides, total cholesterol, LDL-cholesterol, HDL-cholesterol and alanine aminotransferase compared to the RHO and TCO groups. The AS group has the lowest glutathione peroxydase activity. The TCO and RHO groups present the lowest atherogenicity index. HDL-cholesterol, creatinine, alaninine aminotransferase, alkaline phosphatase, total bilirubine, malondialdehyde and selenium levels were not significantly differents ($p < 0.05$) for rats fed with RHO and TCO diets. The atherogenicity index is higher for AS group than RHO and TCO groups, then it could be concluded that oils experimented decrease the risk factor of cardiovascular disease to 5.90 % for RHO and 22.72 % for TCO groups. *Tetracarpidium conophorum* oil can acts by reducing the cholesterol level, arachidonic acid proportion and consequently the risk of atherogenicy and inflammatory.

*Key-Words* : *Tetracarpidium conophorum*, *Ricinodendron heudelotii*, oils, blood biochemistry, lipid peroxidation, oxidative stress.

# 1 Introduction

Central Africa forests contain a wide range of wild oil bearing seeds which are underused with respect to their potential. Among these are *Ricinodendron heudelotii* (Bail.) Pierre ex Pax and *Tetracarpidium conophorum* Müll. Arg. Hutch. and Dalz., which belong to the Euphorbiaceae family. *T. conophorum* kernels are often eaten as nibbles and *R. heudelotii* kernels are used as spices in many dishes in Cameroon [1, 2]. They have high lipids contents (more than 42 %).[3] Therefore, they are considered as hidden fats. Their valorisation could make them as an alternative source of dietary fats. In their general dietary habits, the population uses fats from some conventional oil seeds (palm kernels, groundnut seeds, cotton seeds, maize seeds). However, they don't always cover their requirement in fats. The fatty acids composition of these oils indicates a high content of oleic acid (*R. heudelotii* 26.88 %) ; linolenic acid (*T. conophorum*, 72.24 %) ; elaeostearic acid (*R. heudelotii* 42.40 %) [3]. The fatty acid composition of dietary fats constitues a main argument to define their nutritional value. Hypercholesterolemia and hypertriglyceridemia are two of the important risk factors for cardiovascular disease [4]. Many studies have shown a correlation between dietary fatty acids and their beneficial effects on health. Those studies indicated that the type of lipid in the diet influences the metabolism and the level of circulating lipids, and consequently the risk of development of cardiovascular disease and atheroclerosis [5-10]. Hypercholesterolemia can be linked to atherosclerosis via oxidative stress [11,12]. As far as our knowledge is concerned, the consequence of the consumption of diets containing *T. Conophorum* and *R. heudelotii* oils on circulating lipids and oxidative stress has not been studied. It is therefore important to determine the beneficial effect of the consumption of these oils on metabolic blood parameters and risk factors of cardiovascular diseases since it is well established that coronary diseases are the most common factor for the death of men and women [13]. The aim of this investigation was to compare the effect of the consumption of *T. conophorum* and *R. heudelotii* oils on growth, serum lipids, lipid peroxidation and oxidative stress of rats.

# 2 Material and Methods

## 2.1 Oils

Oils from the kernels of both fruits were obtained from the Laboratory of Food Technology and Biochemistry (National Advanced School of Agro-Industrial Sciences, University of Ngaoundere - Cameroon) [3].

## 2.2 Animals and Diets

1 month old weaned male albinos Sprague Dawley rats (Harlan, France) weighing 140 to 151 g were individually housed in polycarbonate cages in a controlled environment with a temperature of 23°C, 12-hours light-and-dark cycle (light at 7 : 00 AM) [9, 14, 15]. During an adaptation period of 1 week, the rats received tap water and a commercial rat diet (Special Diets Services , Paris - France) *ad libitu* [7, 16]. At the end of this period, the rats were weighed and randomly assigned to one of the three groups (n = 6 / group) according to diet composition. For 60 days, each group was fed a diet containing one of the following : *R. heudelotii* oil (RHO group), *T. conophorum* (TCO group). Oil represented 5 % of the composition of the diet as prescribed by American Institute of Nutrition [17]. The diet was reconstituted by using an alipidic diet (moisture content 8.53 %, proteins 21.48 %, dextrose 32.00 %, starch 26.42 %, cellulose 6.35 %, mineral mix 4.58 %, vitamins mix 0.64 %). One group was fed a commercial standard M20 diet (Special Diets Services , Paris - France) (Proteins 17.93 %, Carbohydrates 57.46 %, Crude fiber 3.94 %, Mineral mix 6.84 %, oil 3.15 %). The fatty acid composition of these diets was analysed by gas chromatography [18]. Animals had free access to water and food.[11] Food was given each week and water twice per week.

## 2.3 Experimental procedure

Food consumption and body weight were determined twice a week on the same day at the same time. Feed – to – gain ratio were calculated by dividing the total feed intake per rat by total weight gain. At the end of the feeding period (60 days), the rats fasted overnight (12 hours), then were weighed, anesthezed 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 separated 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, malondialdehyde and selenium determinations. Oxidative stress was performed by measuring glutathione peroxydase (GPx) and superoxide dismutase (SOD) activities in red blood cells. The protocol was approuved by the Experimental Research Committee of the Faculty of Medicine of Nancy (France).

#### 2.3.1 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 performed using an enzymatic kit test at 37°C (Cat. No. GY 105, Randox-France). Protein content was determined according to the method of Gornall *et al*. [19]. Creatinine was analyzed according to the method of Jaffe (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 using Olympus kit (Cat. No OSR6104). The total bilirubine level was performed according the Winkelman's *et al.* method by using bilirubine total reagent (IR701 – T, Synermed®, Synermed International Inc. - USA) [20].

#### 2.3.2 Plasmatic fatty acids

The plasmatic fatty acids concentration was analysed by a direct transesterification method using tricosanoic acid (Sigma Aldrich Co, Saint Louis - USA) as internal standard [21,22].

#### 2.3.3 Malondialdehyde level (MDA)

Plasmatic malondialdehyde was determined using Sheu *et al*. methods [23].

0.5 ml of plasma were introduice in tubes containing 1.5 ml of phosphoric acid solution (9 % w/v using 85 % analytical grade phosphoric acid). The blank reagent was prepared by pipetting 0.5 ml of ethanol into a tube containing 1.5 ml of phosphoric acid (9 % v/v) solution. 0.5 ml of each standard solution (2.5, 5, 10, and 20 µmol/l) (1,1,3,3-tetraethoxypropan, Sigma - France) were pipetted into tubes containing 1.5 ml of phosphoric acid (9 % p/v) solution. 0.5 ml of thiobarbituric acid (30 mM) solution was added into each tube, and the contents were mixed vigourously. The tubes were closed and placed in the boiling water bath for 30 min. After cooling the tubes at 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 rpm, 25°C). The absorbance of each butanol extract was read at 534 nm.

#### 2.3.4 Parameters of oxidative stress

Oxidative stress was evaluated through 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 oxidised glutathione (GSSG) formed was measured by evaluating the number of oxidized NADPH per minute and per gram of haemoglobin [24,25]. Superoxide dismutase activity was determined using RANSOD kit (Cat. No. SD 125, Randox France). Selenium plasma was analyzed by the atomic absorption technic at 196 nm using Chappuis' method [26].

### 2.4 Statistical Analysis

Results were expressed as means ± standard deviation. For each group, the result obtained was 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. Differences between means were considered to be significant at $p < 0.05$.

## 3 Results

### 3.1 Fatty acids composition of diets

The diets experimented were rich in polyunsaturated fatty acids (Table 1). Diet RHO have the highest proportions of α-eleaostearic acid, however, TCO diet is rich in α-linolenic acid. The standard diet has an important proportion of linoleic acid.

### 3.2 Rat growth

Body weight gain was (Table 2) greater for the AS group (241,90 g) compared to the RHO (193,68 g) and TCO (196,45 g) groups. The feed – to gain ratio (ratio of total diet consumed on body weight gain) did not significantly vary ($p < 0.05$) for the RHO, TCO and AS groups (5.51 ± 0.35 ; 5.38 ± 0.23 and 5.41 ± 0.40 respectively).

### 3.3 Serum metabolites

Table 3 summarises the biochemichal parameters in serum. Serum total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, alanine aminotransferase, alkaline phosphatase, total bilirubine levels for rats of TCO and RHO groups were not significantly different ($p > 0.05$). Compared to AS group, values obtained were lower. TCO, RHO and AS diet have no significant

effect on glycerol, creatinine and proteins level of the rats experimented.

CT/HDL ratio (atherogenic index) helps as good marker for identifying cardiovascular diseases risks [27]. The increase of CT/HDL ratio increases the risk of cardiovascular diseases [28]. TCO and RHO have the lowest CT/HDL ratio (1.76 and 2.07 respectively) compare to AS group. For this reason, TCO and RHO contribute to the decrease of the cardiovascular risk compared to AS group.

### 3.4 Plasmatic fatty acids

Table 4 summarises the plasmatic concentration of fatty acids of rats (µg/ml of plasma). TCO group has the lowest concentration of total fatty acids (859.00 ± 93.54 µg/ml) compared to RHO group (1408.17 ± 35.11µg/ml) and AS group (1943.50 ± 61.60 µg/ml).

The percentage of each fatty acid was calculated according to the total fatty acids. For the saturated fatty acids, the proportion of palmitic acid (C16 : 0) is lower for TCO group (19.78 % of total fatty acids) compared to the RHO group (23.06 % of total fatty acids) and the AS group (24.22 % of total fatty acids). Stearic acid (C18 : 0) content is similar for TCO and RHO groups (10.16 and 10.40 % of total fatty acids respectively), but more than in the AS group (7.2 % of total fatty acids).

In the case of monoinsaturated fatty acids, in addition to palmitoleic (C16 : 1) and oleic (C18 : 1 ω – 9) acids, the presence of trans vaccenic (C18 : 1, ω - 7) was noticed in the plasma of all groups. This fatty acid is more accumulated in AS group (4.2 % of fatty acids) compared to TCO and RHO (2.10 and 2.90 % of total fatty acids respectively).

For polyunsaturated fatty acids, the proportion of linoleic acid (C 18 : 2, ω - 6), is higher for AS group (30.30 % of total fatty acids) than TCO group (20.40 % of total fatty acids) and RHO (16.90 % of total fatty acids). In RHO group (which diet is riched in α-eleaostearic acid), no peak correspondind to α-eleaostearic acid was detected. However, a new fatty acid was found. Using the Gaz Chromatograpgy – Mass spectrophotometer (GC- MS), this fatty acid was identified as a conjugated linoleic fatty acid (4.02 % of total fatty acids). Only TCO group presents eicosapentaenoic acid (EPA) (C20 : 5 ω - 3) and docosapentaenoic acid (DPA) (C22 : 5 ω - 3). Arachidonic fatty acids (C 20 : ω – 6) was lower in the TCO groups (8.10 % of total fatty acids) compared to AS and RHO (19.91 % and 24.90 % of total fatty acids respectively).

### 3.5 Malondialdehyde (MDA)

MDA constitutes a biological marker of lipid peroxidation. The Plasma MDA of the AS group was lower than that of the TCO and RHO groups. The Duncan test shows that there is no significant difference ($p < 0.05$) between the value of plasma MDA for TCO and RHO (Table 5).

### 3.6 Superoxide dismutase (SOD), Glutathion peroxidase (GPx) and selenium

Dietary lipids significantly affected the activity of SOD in red blood cells ($p < 0.05$). The SOD activity of RHO group (Table 5) was lower than that of the TCO group.

GPx activity of AS was lower than that of TCO and RHO. The Duncan test shows that there is no difference ($p < 0.05$) between GPx levels for rats fed with TCO and RHO diets.

Plasma selenium level for TCO and RHO did not differ significantly ($p < 0.05$), but was lower than that of AS group. The variation of the glutathion peroxidase level was correlated to the variation of the selenium level ($r = 0.84$, $p = 0.01$).

## 4 Discussion

Fatty acids in dietary lipids have been implicated in the serum lipids and lipid peroxidation response [29]. This study examined the fact that, oils extracted from commonly consumed oil bearing seeds, differing on their degree of unsaturation and fatty acids type, vary in their effects on serum lipids, plasma fatty acids, lipid peroxidation and oxidative stress. Oils used in this study had different characteristics. AS, RHO and TCO are all rich in polyunsatured fatty acids (PUFA) : RHO contain 32.53 % of a conjugated fatty acid (α-elaestearic acid c9, t11, t13) ; TCO contains 62.00 % of linolenic acid ; AS contains 54.61 % of linoleic acid. Feeding rats with diets containing the above oils resulted in differential effects on body weight and lipid profile. In the present study, feeding rats with TCO and RHO diets lead to the reduction of triglycerides, total cholesterol, HDL-cholesterol and LDL-cholesterol levels of the serum when compared to AS diet. The decrease in total cholesterol level can probably be associated with the reduction in cholesterol absorption and its low incorporation in chylomicrons, VLDL and IDL cholesterols.[30] Many enzymes and transport proteins participated to cholesterol absorption. Plasma cholesterol level depends on several parameters, including endogenous synthesis, secretion, and catabolism of various plasma lipoproteins [4]. Previous studies clearly demonstrated that saturated fatty acids (lauric, myristic and palmitic) increased the serum total cholesterol and LDL-cholesterol levels ; inversely, polyunsaturated fatty acids (particularly linoleic acid) decreased the serum cholesterol level [5,31,32].

HDL-cholesterol is essential in the transport of cholesterol from cells and artery to liver where it is catabolised [33]. In this case, HDL-cholesterol

constitutes a protective factor against cardiovascular disease. TCO and RHO have the lowest CT/HDL ratio. Some epidemiological studies defined a relation between the HDL cholesterol level and cardiovascular risk : an increase in the HDL-cholesterol level of 1mg /dl reduces the cardiovascular risk by 2 - 3 % [34,35]. Apolipoprotein A-I (Apo A-I), the major protein in HDL, activates the mobilization of cholesterol ester stored in macrophages, leading to the reduction of the cholesterol content in this major cell type in atherosclerosis [36].

LDL-cholesterol is responsible for the accumulation of lipids in the arterial wall, therefore it constitutes a risk factor for coronary heart disease.

Triglycerides are defined as an independent risk factor for coronary heart [27]. The differences between triglyceride levels can be explained by the difference in the metabolism levels of various lipids according to their fatty acids content. An increase in ω-3 fatty acids mainly decreases serum triglycerides and increases HDL-cholesterol [37,38]. The high serum triglyceride concentrations increase the risk of coronary heart disease even when all other known risk factors are taken into account [6]. One could concluded that α-eleaostearic acid, a conjugated linolenic fatty acid contribute to the decrease of triglycerides level as linolenic acid (ω – 3 fatty acid).

Dietary lipid consumption can affect kidneys and liver functions. 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 [28]. Creatinine level does not vary to one group to another. Oils experimented did not affect creatinine level.

Some tests were carried out to assess the state of the liver function. Alanine aminotransferase and aspartate aminotransferase are the most common markers used to appreciate the hepatocyte injuries. Alanine aminotransferase is more abundant in the liver, whereas aspartate aminotransferase are found in the heart, the liver, the skeletal muscle and the kidney. According to Hohnadel [39], the high serum levels of alanine aminotransferase are caused by an increase of membrane permeability of the hepatocyte and the release of enzymes from liver cells to serum. Generally, in myocardial injuries, aspartate aminotransferase activities increases rapidly, while alanine aminotransferase increases slightly. Liver alterations induce considerable increases of both enzymes, predominantly on aminotransferase [15].

The plasma of TCO group was concentrated in eicosapentaenoic acid. Wiensenfeld *et al.* [40]. mentioned the effect of the consumption of lin oil (rich in α-linolenic : 34 – 58 % of total fatty acid) by pregnants rats. These authors, clearly established that lin oil contributes to decrease serum arachidonic acid concentration and increase serum eicosapentaenoic acid concentration contrary to soy oil (rich in linoleic acid : 55 % of total fatty acid) present in control diet. Arachidonic acid have pro-inflammatory properties and eicosapentaenoic acid have anti-inflammatory properties [41]. Our result can be justified by the fact that TCO diet is rich in linolenic acid (62.00 % of total fatty acids), an essential fatty acid for the synthesis of EPA in the presence of $\triangle^6$ desaturase, elongase and $\triangle^5$ desaturase) [41]. The lowest proportion of arachidonic acid in TCO group can be justified by the inhibition effect of EPA on $\triangle^5$ desaturase [42]. The presence of EPA and the low concentration of arachidonic acid in TCO group influence the synthesis of eicosanoids. Indeed, arachidonic acid is the precussor of thromboxane of type 2 which has a strong agglutination effect of plaquettes and an vasoconstriction effect ; type 4 leucotriens which have pro-inflammatory properties. EPA is the precussor of prostagladine $PGl_3$ which have anti-agglutination effect [41].

α-linolenic fatty acid is the precussor of EPA and docosahexaenoic acid synthesis. Being given that *T. conophorum* oil contains high percentage of linolenic acid, this oil can be used as an important source of ω – 3 fatty acids and for the substitution of long chain ω – 3 fatty acids found in marines products.

Lipid oxidation, a process mediated by free radicals, is considered to be important in the development of atherosclerosis [6]. The estimation of free radical activity is performed through the determination of malondialdehyde which is a by-product of lipid peroxidation [43,44]. Halliwel & Chirico [45] demonstrated the higher stability of saturated and monounsaturated oils in lipid peroxidation than polyunsaturated fatty acids. Tocopherols concentrations of oils are important in the lipid peroxidation. A high concentration of tocopherols in oils reduces the plasma MDA level [46,47]. This is verify with the AS group. The tocopherol levels of oils experimented were : 0.39 (mg/g) ; 1.34 (mg/g) and 1.33 (mg/g) respectively for *R. heudelotii* oil, *T. conophorum* oil and maize oil [3].

Superoxyde dismutase (SOD) and glutathione peroxidase (GPx) are two important enzymes of antioxidant defense system which participate in regulating the lipid peroxidation. A decrease in the activities or expression of these enzymes may predispose tissues to free radical damage. Dietary lipid composition infuences the level of these enzymes [48]. GPx is very important in the elimination of peroxide hydroxide in cells. Rats of TCO group have the most important activity of SOD.

Selenium is an indispensable trace element, acting as a GPx cofactor. Selenium activates GPx, GPx therefore block the formation of hydroxyl radicals. For TCO and RHO groups, the selenium level does not vary.

# 5 Conclusion

*T. conophorum* and *R. heudelotii* oils constituted hypocholesterolemic treatments compare to maize oil contains in the standard diet. Diets experimented did not affected the glycerol, creatinine and proteins levels of serum. Standard diet experimented in this study is the most atherogenic. The rats the TCO groups were the only ones that showed the presence of eicosapentaenoic acid on plasmatic fatty acids. Morever, this group has the lowest arachidonic acid, which presented pro-inflammatory properties. α-eleaostearic acid consumme in diet RHO was absent in plasmatic fatty acids. However, a conjugated linoleic fatty acid was found. *T. conophorum* and *R. heudelotii* oils are interesting in the reduction of cardiovascular diseases riks. *T. conophorum* oil is the best in the reduction inflammation.

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**Table 1 : Fatty acid composition of the studied diets (% of total fatty acids)**

| Fatty acids | RHO | TCO | AS |
|---|---|---|---|
| C 8 | / | 0.26 | / |
| C10 | / | / | 0.03 |
| C12 | / | / | 0.03 |
| C14 | / | 0.1 | 0.20 |
| C16 | 9.35 | 2.80 | 17.04 |
| C16 : 1 (ω – 7) | 0.81 | / | / |
| C18 | 10.59 | 3.45 | 2.56 |
| C18 : 1 (ω - 9) | 10.30 | 16.00 | 18.64 |
| C18 : 2 (ω - 6) | 36.04 | 15.37 | 54.61 |
| C18 : 3 (ω - 3) | / | 62 | 6.40 |
| C18 : 3 (ω - 5) | 32.53 | / | / |
| 20 : 1 (ω -9) | 0.86 | / | 0.45 |
| Total satured (SFA) | 19.94 | 6.61 | 19.86 |
| Total monounsatured (MUFA) | 11.92 | 16.00 | 19.09 |
| Total polyunsaturated (PUFA) | 68.57 | 77.37 | 61.01 |
| PUFA/SFA | 3.44 | 11.70 | 3.07 |
| ω – 6 / ω - 3 | / | 0.24 | 8.53 |

TCO : Diet containing *T. conophorum* oil ; RHO : Diet containing *R. heudelotii* oil ; AS : Standard diet.

**Table 2 : Effects of diets on body weight, body gain and feed - to - gain ratio**

| | TCO | RHO | AS |
|---|---|---|---|
| Body final weight (g) | $345.40 \pm 16.96^{a}$ | $345.36 \pm 13.44^{a}$ | $382.50 \pm 9.58^{b}$ |
| Body gain (g) | $193.68 \pm 10.10^{a}$ | $196.45 \pm 8.17^{a}$ | $241.90 \pm 5.03^{b}$ |
| Feed - to – gain ratio | $5.38 \pm 0.23^{a}$ | $5,51 \pm 0.35^{a}$ | $5.41 \pm 0.40^{a}$ |

**Table 3 : Biochemical parameters of serum**

| | TCO | RHO | AS |
|---|---|---|---|
| Total cholesterol (mg/dL) | 70.16 ± 9.80 [a] | 75.50 ± 9.70 [a] | 101.25 ± 3.00 [b] |
| HDL-cholesterol (mg/dL) | 39.66 ± 4.67 [a,b] | 36.33 ± 5.08 [a] | 46.00 ± 2.94 [b] |
| LDL-cholesterol (mg/dL) | 9.00 ± 1.90 [a] | 8.16 ± 1.60 [a] | 13.25 ± 1.25 [b] |
| Triglycerides (mg/dL) | 46.83 ± 10.43 [a] | 45.10 ± 16.73 [a] | 100.75 ± 5.73 [b] |
| Glycerol (mg/dL) | 9.50 ± 2.81 [a] | 7.33 ± 1.86 [a] | 6.75 ± 0.95 [a] |
| Creatinine (mg/dL) | 1199.54 ± 110.47 [a] | 1225.96 ± 89.56 [a] | 1079.17 ± 68.14 [a] |
| Alanine aminotransferase (UI/l) | 51.33 ± 6.30 [a] | 47.33 ± 3.01 [a] | 74.25 ± 2.62 [b] |
| Aspartate aminotransferase (UI/l) | 139.16 ± 20.33 [a,b] | 113.40 ± 21.75 [a] | 162.25 ± 21.92 [b] |
| Alkaline phosphatase (UI/l) | 135.33 ± 23.03 [a] | 119.00 ± 24.56 [a] | nd* |
| Total bilirubine (mg/dL) | 100.00 ± 0.00 [a] | 100.00 ± 0.00 [a] | nd |
| Proteins (mg/dL) | 6650.00 ± 39.74 [a] | 6516.66 ± 306.05 [a] | 6563.00 ± 405.08 [a] |
| CT/HDL (atherogenic index) | 1.76 | 2.07 | 2.20 |

* nd = not determined

TCO : Diet containing *T. conophorum* oil ; RHO : Diet containing *R. heudelotii* oil ; AS : Standard diet.
*Values on the same line with different superscripts are significantly different at p < 0,05 (Duncan's test).*

**Table 4 : Plasmatic fatty acid concentrations (µg/ml) with respect to the diets after 60 days***

| | TCO | RHO | AS |
|---|---|---|---|
| C 14 : 0 | / | / | / |
| C 16 : 0 | 169.90 ± 14.60 | 326.40 ± 54.00 | 470.90 ± 16.80 |
| C 16 : 1 ω – 7 | 17.70 ± 0.60 | 44.80 ± 15.60 | 41.30 ± 2.70 |
| C 18 : 0 | 87.30 ± 10.20 | 147.30 ± 0.40 | 140.00 ± 2.63 |
| C18 : 1 ω – 9 | 80.00 ± 3.00 | 161.90 ± 5.40 | 174.80 ± 4.40 |
| C 18 : 1 ω – 7 | 18.00 ± 0.90 | 41.20 ± 7.80 | 81.70 ± 2.10 |
| C 18 : 2 ω – 6 | 175.30 ± 14.70 | 239.30 ± 20.20 | 589.00 ± 14.60 |
| C 18 : 2 (conjugated) | / | 56.90 ± 6.40 | / |
| C18 : 3 ω – 3 | 109.70 ± 21.20 | / | 24.01 ± 1.40 |
| C 20 : 1 ω – 9 | / | 28.90 ± 2.10 | / |
| C 20 : 4 ω – 6 | 69.60 ± 7.00 | 352.30 ± 73.60 | 387.00 ± 9.10 |
| C 20 : 5 ω – 3 | 99.40 ± 14.90 | / | / |
| C22 : 5 ω – 3 | 16.10 ± 2.40 | / | / |
| C 22 : 6 ω – 3 | 16.30 ± 4.30 | 9.30 ± 0.90 | 34.80 ± 8.00 |
| Total fatty acids | 859.00 ± 93.54 | 1408.17 ± 35.11 | 1943.50 ± 61.60 |

* TCO : Diet containing *T. conophorum* oil ; RHO : Diet containing *R. heudelotii* oil ; AS : Standard diet.

**Table 5 : Malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx) and selenium in rats plasma with respect to diet***

| | TCO | RHO | AS |
|---|---|---|---|
| MDA (µmol/l) | 1.91 ± 0.70 [b] | 2.09 ± 0.66 [b] | 1.00 ± 0.18 [a] |
| SOD (U/ml) | 645.00 ± 92.17 [b] | 537.00 ± 73.05 [a] | 617.25 ± 30.76 [a,b] |
| GPx (mmoles/l/min) | 71.44 ± 4.05 [b] | 73.00 ± 2.40 [b] | 59.16 ± 0.79 [a] |
| Selenium (µg/l) | 636.33 ± 52.47 [a] | 638.66 ± 40.70 [a] | 755.50 ± 29.55 [b] |

* TCO : Diet containing *T. conophorum* oil ; RHO : Diet containing *R. heudelotii* oil ; AS : Standard diet.
*Values on the same line with different superscript letters are significantly different at p < 0,05 (Duncan's test).*