Effects of high-fat diets on inflammation and antioxidant status in rats: comparison between palm olein and olive oil


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Effects of high-fat diets on inflammation and antioxidant status in rats: Comparison between palm olein and olive oil


Palm olein (PO) and olive oil (OO) are widely consumed in the world. PO is considered harmful to health, whereas OO is considered healthy. This study aims to discover the effects of consumption of these oils on antioxidant status and inflammation that can be beneficial for consumer. This was an experimental study in male wistar rats fed a diet containing 30% of each oil. Rats had free access to food and water. After being fed for 12 weeks, animals were sacrificed and liver and aortic blood were collected. Plasma was used for the determination of interleukin-6 and oxidative stress parameters (Superoxide dismutase, Glutathione peroxidase, Thiobarbituric acid reactive substances, THiol groups and isoprostane). The inflammation and oxidative stress status as well as the expression of several genes/proteins were also analyzed in liver homogenate. No significant differences were observed between PO and OO in plasma and liver levels of the studied inflammation and oxidative stress parameters. We noted that the consumption of PO does not promote inflammation and oxidative stress. This study will help the researchers to uncover the critical areas of antioxidant and anti-inflammatory effects of olive oil and palm olein consumption.

Key words: palm olein, olive oil, oxidative stress, inflammation, high fat diet

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Abbreviations: AU, arbitrary Unit; CD68, Cluster of differentiation 68; GCLC, Glutamate-cysteine ligase catalytic subunit; Gpx, Glutathione peroxidase; GSH, Reduced glutathione; GSSG, Oxidized glutathione; HFD, High fat diet; HO-1, Heme oxygenase-1; IkB-α, Inhibitor of kappa B-alpha; IL-1β, Interleukin-1β; IL-6, Interleukin-6; NF-κB, Nuclear factor-kappa B Nrf2 (Nfe2l2), Nuclear factor-erythroid 2 related factor 2; qPCR, Quantitative-Polymerase chain reaction; PO, Palm olein; PUFA, Polyunsaturated fatty acids; SOD, Superoxide dismutase; TBARS, Thiobarbituric acid reactive substances; ww, Wet weight

INTRODUCTION

Palm oil is the most produced (USAD, 2021) and consumed vegetable oil in the world (USAD, 2021). Although crude palm oil is known for its nutritional benefits (Edem, 2002; Ong & Goh, 2002; Sen et al., 2007), industry prefers deodorised and decoloured palm oil for which refining is mandatory. This refining can be based on chemical methods (treatment with alcalis or acids) (Cmolik & Pokorny, 2000; Dunford, 2012) or physical methods (steam refining, inert gas stripping, molecular distillation, etc.) (Dunford, 2012). This is followed by bleaching and decodistillation steps as well as other specific treatments depending on the first refining process applied. After refining, the product obtained will undergo fractionation to give derived fractions: palm olein or super-olein (a colourless, bland and stable oil, rich in oleic acid) and palm stearin (a fat, rich in saturated fatty acids) (Dunford, 2012; Lecerf, 2013). Refining results in a significant loss of carotenoids and a moderate loss of vitamin E (Tarmizi & Lin, 2008).

Palm olein is widely used in African, South American and Asian cuisines. Instead of partially hydrogenated oils that contain trans fatty acids, palm stearin is widely used by the food industry in the manufacture of many products such as sweets, cakes, cheese analogues, crisps, chocolates, confectionary fats, biscuits, doughnuts, frozen meals and products (pancakes, pies, pizzas, potatoes, etc.), instant meals, etc. (Mancini et al., 2015; Mba et al., 2015).

Olive oil (OO) is a vegetable oil known to be rich in polyphenols (Owen et al., 2000). Numerous studies report the nutritional benefits of OO polyphenols (Perez-Jimenez et al., 2005; Cavas et al., 2006; Assy et al., 2009). PO, although naturally rich in phytoneutrients (vitamin E, Coenzyme Q, etc.) considered beneficial for human health especially for their antioxidant properties (Ng et al., 2012; Tiahou et al., 2004; Rooyen et al., 2008), is due to its high content of saturated fatty acids (50%) especially palmitic acid, accused of being potentially harmful to health (Fattore et al., 2014; Odia et al., 2015). Given this, it seemed appropriate to undertake this study to compare the effects of consuming diets rich in PO and OO on inflammation and antioxidant status in rats.

MATERIALS AND METHODS

Animals and diets

A total of twenty-four young male Wistar rats (Charles River, L’Arbresle, France) aged 6 weeks were used in the

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present study. The rats were housed, two per cage, under conditions of constant temperature (20–22°C), humidity (45–50%), and a standard dark cycle (20.00–08.00 hours). The rats were randomised into four groups of eight animals and fed for 12 weeks with one of the following semi-purified diets: (1) control diet (Control), containing 5% lipid as soybean oil (11% energy from fat) this is sham control group, (2) high-fat diet (HFD) (55% energy from fat) rich in PO with 2.5% soybean oil and 30% PO or (3) HFD rich in OO with 2.5% soybean oil and 30% OO. PO was supplied by SANIA company (Côte d’Ivoire), and OO (virgin) was bought in a supermarket (these oils were chosen for their large current consumption). The detailed composition of these experimental diets is shown in Table 1. Rats were given free access to water and food during the whole experiment and body growth was determined weekly. Our institution guidelines for the care and use of laboratory animals were followed, and all the experimental procedures were approved by the local ethical committee in Montpellier, France (Reference CEEA-LR-12002).

Rat sacrifice and sampling

Blood was obtained from 16 h fasted rats anaesthetised with pentobarbital (Ceva Sante Animale, Libourne, France) by puncturing the abdominal vein with a heparinised syringe (Sodium heparinate, Panpharma SA Fougeres, France). Blood was then distributed into a dry tube (3–4 ml) and a heparinised tube (5–6 ml), centrifuged at 1000 x g for 10 min at 4°C, and serum and plasma were collected and stored at −80°C until analysis. The liver was perfused with 10 ml of 0.9% NaCl solution, quickly removed, weighed, and cut into different parts. One part was immediately frozen in liquid nitrogen and then kept at −80°C until analysis. Another part was fixed in 10% neutral buffered formalin and embedded in paraffin for histological analysis.

Inflammation and oxidative stress parameters in blood

Plasma interleukin-6 (IL-6) levels were quantified with ELISA kits (Fisher Scientific, France). The activity of antioxidant enzymes has been determined by spectrophotometric methods. Glutathione peroxidase (GPx) activity and total superoxide dismutase (SOD) were measured in blood according to the method of Flohe & Gunzler (1984) and Marklund (1976), respectively. Thiobarbituric acid-reactive substances (TBARS), was measured according to the method of Sunderman et al., (1985). Protein oxidation was assessed by measurement of sulphydryl groups (Faure & Lafond, 1995) in plasma.

Plasma 15-F_{2}-isoproteans, the more specific lipid per-oxidation parameter, was also measured by mass spectrometry as described by Mas and others (Mas et al., 2008). Briefly, aliquots of plasma samples were added with 15-F_{2}-isoprostane D4 as an internal standard before extraction using an Agilent Bond Elut Certify II cartridges. Washes were performed with methanol 50% and ethyl acetate/hexane (1/3 v/v) and elution was performed with ethyl acetate/methanol (9/1 v/v). After esterification, samples were analyzed on a ThermoFinnigan Trace DSQ II interfaced with a Trace GC Ultra 2000 gas chromatograph, equipped with an AS 3000 automatic sampler (ThermoFinnigan).

Liver macrophage identification

Liver samples were fixed in a neutral 10% formalin buffer and then embedded in paraffin. Sections of 5 μm were made with a microtome (Leica RM 2145, Microsystems Nussloch GmbH, Germany). After staining with haematoxylin, macrophage infiltrations were detected by immuno-labelling with an anti - cluster of differentiation 68 (anti-CD68) antibody (Bio-Rad, France). Antibody distribution was visualized by a Vectastain® ABC kit and an ImmPACT AEC substrate kit (Clinisciences, France). For CD68 determination, 10–20 fields per sample were analyzed and results were expressed as the average percentage of surface with positive staining to total surface of the field.

Table 1. Diet composition (g/kg) on the basis of the AIN-93M diet formulation.

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>Casein</th>
<th>Cornstarch</th>
<th>Maltodextrine</th>
<th>Sucrose</th>
<th>Soybean oil</th>
<th>Palm olein</th>
<th>Olive oil</th>
<th>Mineral mix (AIN-93M)</th>
<th>Vitamin mix (AIN-93M)</th>
<th>L-Cystine</th>
<th>Choline chloride</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>165</td>
<td>434</td>
<td>144</td>
<td>100</td>
<td>50</td>
<td>0</td>
<td>0</td>
<td>35</td>
<td>10</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>Palm Olein</td>
<td>200</td>
<td>234</td>
<td>80</td>
<td>53</td>
<td>25</td>
<td>300</td>
<td>0</td>
<td>42</td>
<td>12</td>
<td>2.4</td>
<td>1.8</td>
</tr>
<tr>
<td>Olive Oil</td>
<td>200</td>
<td>234</td>
<td>80</td>
<td>53</td>
<td>25</td>
<td>0</td>
<td>0</td>
<td>42</td>
<td>12</td>
<td>2.4</td>
<td>1.8</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td>1000</td>
<td></td>
</tr>
</tbody>
</table>

Liver mRNA expression

Real-time quantitative PCR was used to measure the mRNA expression of the target genes in the tissues and was performed as described previously (Djohan et al., 2019). The primer sequences used for real-time PCR are shown in the Supplemental Table (at https://ojs.pt-bioch.edu.pl/index.php/abp/). Results were normalized to RPLP0 gene and were expressed as a percentage of the control. Liver genes analyzed include GCLC, HO-1, IKB-a, IL-1β, NF-κB, NQO-1, Nrf2.

Statistical analysis

Results were expressed as mean and standard deviations, n=7–8 animals per group. Statistical analysis was based on one-way ANOVA followed by a Tukey Kram-
er multiple comparisons test. When statistical variances were unequal, a Welch test was performed. The limit of statistical significance was set at \( p<0.05 \). The group mean values with different letters (a, b, c) are significantly different. Statistical analyses were performed using the StatView program (SAS Institute, Cary, NC, USA).

### RESULTS AND DISCUSSION

#### Effects of diet on inflammation

With regard to the inflammatory parameters studied (plasma IL-6, liver IL-1\(\beta\), NF-\(\kappa\)B and I\(\kappa\)B-\(\alpha\) genes), no diet promoted inflammation (Table 2). In liver, PO diet promoted a significant increase \( (\pm 0.027) \) in the gene expression of I\(\kappa\)B-\(\alpha\) \( (+78\%) \), compared to the control diet. OO diet has favoured a non-significant increase in the gene expression of I\(\kappa\)B-\(\alpha\) \( (+33\%) \) compared to the control diet. The gene expression of NF-\(\kappa\)B was decreased by 4% with PO diet and increased by 7% with OO diet compared to the control diet. On the other hand, OO diet induced a significant increase \( (p<0.0001) \) of macrophage density \( (+31\%) \) at least in rat liver compared to PO and control diets. PO diet induced a significant decrease \( (p<0.0001) \) in macrophage density \( (-36\%) \) at least in rat liver compared to OO and control diets (Table 2).

NF-\(\kappa\)B is the linchpin of phagocytic cells because it enables them to be activated. Consequently, an increase in NF-\(\kappa\)B and/or its activators is observed in acute or chronic inflammation (Monaco et al., 2004; Song et al., 2009; Hajishengallis & Chavakis, 2013). I\(\kappa\)B-\(\alpha\) inhibits NF-\(\kappa\)B by masking the nuclear localisation signals of NF-\(\kappa\)B proteins and sequestering them in an inactive state in the cytoplasm (Jacobs & Harrison, 1998; Hinz, 2012). In addition, I\(\kappa\)B-\(\alpha\) blocks the ability of NF-\(\kappa\)B transcription factors to bind to deoxyribonucleic acid, which is necessary for the proper functioning of NF-\(\kappa\)B (Verma et al., 1995; Huang, 2000; Birbach, 2002).

The significant increase in the gene expression of I\(\kappa\)B-\(\alpha\) by PO diet and the non-significant decrease in the gene expression of NF-\(\kappa\)B show that PO diet protects the liver better against inflammation. The actions of PO on NF-\(\kappa\)B and I\(\kappa\)B-\(\alpha\) genes could be explained by its high tocotrienol content (Sambanthamurthi et al., 2000; Lecerf, 2013). Indeed, tocotrienols have anti-inflammatory properties (Reiter et al., 2007; Yam et al., 2009) due to their involvement in inhibiting NF-\(\kappa\)B activation pathway (Ahn et al., 2007; Ng & Ko, 2012). In-vitro studies with palm oil tocotrienols have shown its anti-inflammatory effects (Wu et al., 2008) and its ability to reduce cancer cell proliferation by inhibiting NF-\(\kappa\)B activation pathway (Yap et al., 2008; Ji et al., 2015).

Studies in humans have shown that diets based on PO and OO do not promote inflammation at the plasma level (Teng et al., 2011; Tielstru et al., 2011). The search for macrophage infiltrations in liver with CD68 labeling (Fig. 1) showed a significant increase \( (p<0.0001) \) in macrophage density with the OO diet compared to other diets. The significant increase of macrophage density in liver of the rats that consumed OO suggests that OO promoted inflammation in liver. This action of OO on liver could be explained by its high content of \( \omega-6 \) polyunsaturated fatty acids (PUFA). Indeed, according to many authors (Raphael & Sordillo, 2013; Marion-Letellier et al., 2015) \( \omega-6 \) PUFA promote inflammation.

Despite its richness in saturated fatty acids, particularly palmitic acid, which is considered pro-inflammatory because it activates the NF-\(\kappa\)B pathway (Ajuwon & Tse, 2013; Marion-Letellier et al., 2015). The significant decrease in the gene expression of I\(\kappa\)B-\(\alpha\) in the OO diet indicates its anti-inflammatory properties. This is in agreement with the findings of Yang et al. (2011) that tocotrienols downregulate the expression of I\(\kappa\)B-\(\alpha\) in RAW264.7 macrophages. This study suggests that tocotrienols could be used as a potential anti-inflammatory agent.

#### Table 2. Blood and liver inflammation parameters

Rats were fed their respective diet for 12 weeks. Results were expressed as mean values ± S.D., \( n=7–8 \) animals per group. Statistical analysis was based on one-way ANOVA followed by a Tukey Kramer multiple comparisons test. The limit of statistical significance was set at \( p<0.05 \). The group mean values with different letters (a, b, c) are significantly different.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>PO</th>
<th>OO</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plasma</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>14.67±2.7</td>
<td>13.98±2.6</td>
<td>14.48±4.1</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Liver</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NF-(\kappa)B (qPCR)</td>
<td>1.00±0.07</td>
<td>0.96±0.04</td>
<td>1.07±0.02</td>
<td>NS</td>
</tr>
<tr>
<td>I(\kappa)B-(\alpha) (qPCR)</td>
<td>1.00±0.12(^a)</td>
<td>1.78±0.23(^b)</td>
<td>1.33±0.15(^c)</td>
<td>0.027</td>
</tr>
<tr>
<td>IL-1(\beta) (qPCR)</td>
<td>1.00±0.12(^a)</td>
<td>0.92±0.07</td>
<td>1.00±0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

\(^a\) inhibitor of kappa-alpha; \(^b\) Interleukin-1 beta; \(^c\) Interleukin-6; NF-kappa-beta; qPCR, Quantitative-Polymerase chain reaction FF; OO, Olive oil; PO, palm olein.
Spurlock, 2005; Laine et al., 2007), the results of this study and data from the literature show that PO has anti-inflammatory properties due to its high tocotrienol content.

Effects of diets on antioxidant status

In blood, SOD was significantly reduced \( (p=0.0108) \) with PO diet compared to other diets. None of the diets resulted in a significant increase in oxidation products (Table 3). Concerning oxidation products, compared to the control diet, OO diet has favoured a decrease of 1% of thiol groups, a decrease of 7% of TBARS and a significant decrease \( (p=0.0167) \) of 15-F2t-isoprostane of 31% (Table 3).

In liver, no significant differences were observed between diets with regard to their effects on oxidative stress parameters (Table 4). Concerning oxidation products, the level of thiol groups showed a non-significant tendency to increase \( (p=0.0779) \) with PO (+11%) and OO (+5%) compared to the control diet (Table 4). The study of the expression of genes involved in the antioxidant system showed a non-significant tendency to increase \( (p=0.0696) \) Nrf2 gene with OO diet (+35%) and PO diet (+9%) compared to the control diet. The gene expression of NQO-1, HO-1 and GCLC was not modified in any diet (Table 4).

Thiols play a very important «buffer» role in the body. In the event of severe oxidative stress, thiols restore the «redox» balance (oxidation/reduction balance) by eliminating free radicals (Ferrer-Sueta et al., 2011). PO diet effectively protects liver against free radicals because it promotes an increase in plasma and liver thiols. Moreover, no diet has led to a significant decrease in the level of plasma and liver thiols groups. This suggests that no diet has favoured oxidative stress, as only the collapsed thiol levels objectivise old and/or chronic oxidative stress (Musaogullari & Chai, 2020).

Lipoperoxidation was assessed by the determination of blood and liver TBARS and plasma 15-F2t-isoprostane. No diet resulted in a significant increase in these parameters compared to the control diet. Since only an

<table>
<thead>
<tr>
<th>Table 3. Blood oxidative stress parameters</th>
</tr>
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<tbody>
<tr>
<td>Rats were fed their respective diet for 12 weeks. Results were expressed as mean values ± S.D., n=7–8 animals per group. Statistical analysis was based on one-way ANOVA followed by a Tukey Kramer multiple comparisons test. The limit of statistical significance was set at ( p&lt;0.05. ) The group mean values with different letters (a, b, c) are significantly different.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antioxidant system</th>
<th>Control</th>
<th>PO</th>
<th>OO</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOD (IU/mL)</td>
<td>324±15( ^a )</td>
<td>296±23( ^a )</td>
<td>326±18( ^a )</td>
<td>0.0108</td>
</tr>
<tr>
<td>GPx (IU/mL)</td>
<td>14.4±3.3</td>
<td>15.9±4.4</td>
<td>13.9±1.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

| Oxidation products | | | |
|--------------------| | | |
| Thiol groups (µM)  | 112±52 | 124±49 | 111±44 | NS |
| TBARS (µM)         | 5.12±0.86 | 5.26±1.04 | 4.79±0.88 | NS |
| 15-F2t-isoprostane | 53±16\( ^a \) | 44±06\( ^a \) | 37±06 | 0.0167 |

AU, arbitrary Unit; GPx, Glutathione peroxidase; PO, Palm olein; SOD, Superoxide dismutase; TBARS, Thioabarbituric acid reactive substances.

<table>
<thead>
<tr>
<th>Table 4. Liver oxidative stress parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rats were fed their respective diet for 12 weeks. Results were expressed as mean values ± S.D., n=7–8 animals per group. Statistical analysis was based on one-way ANOVA followed by a Tukey Kramer multiple comparisons test. The limit of statistical significance was set at ( p&lt;0.05. ) The group mean values with different letters (a, b, c) are significantly different.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antioxidant system</th>
<th>Control</th>
<th>PO</th>
<th>OO</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOD (U/mg protein)</td>
<td>45.7±4.3</td>
<td>43.9±3.3</td>
<td>43.9±4.9</td>
<td>NS</td>
</tr>
<tr>
<td>GPx (U/mg protein)</td>
<td>6.69±0.51</td>
<td>6.96±0.75</td>
<td>6.52±0.72</td>
<td>NS</td>
</tr>
<tr>
<td>Catalase (U/mg protein)</td>
<td>725±57</td>
<td>791±125</td>
<td>787±53</td>
<td>NS</td>
</tr>
<tr>
<td>GSH (nmol/g ww)</td>
<td>126±16</td>
<td>140±16</td>
<td>122±18</td>
<td>NS</td>
</tr>
<tr>
<td>GSH/GSSG</td>
<td>4.91±0.34</td>
<td>5.02±0.18</td>
<td>4.96±0.46</td>
<td>NS</td>
</tr>
<tr>
<td>Nrf2 (qPCR)</td>
<td>1.00±0.27</td>
<td>1.09±0.21</td>
<td>1.35±0.30</td>
<td>0.0065</td>
</tr>
<tr>
<td>NQO-1 (qPCR)</td>
<td>1.00±0.57</td>
<td>0.87±0.40</td>
<td>1.14±0.60</td>
<td>NS</td>
</tr>
<tr>
<td>HO-1 (qPCR)</td>
<td>1.00±0.62</td>
<td>0.91±0.25</td>
<td>0.81±0.25</td>
<td>NS</td>
</tr>
<tr>
<td>GCLC (qPCR)</td>
<td>1.00±0.42</td>
<td>1.26±0.30</td>
<td>1.44±0.42</td>
<td>NS</td>
</tr>
</tbody>
</table>

| Oxidation products | | | |
|--------------------| | | |
| Thiol groups (nmol/mg protein) | 110±10\( ^a \) | 123±9\( ^a \) | 116±11\( ^a \) | 0.0779 |
| TBARS (nmol/mg protein) | 99±20 | 90±18 | 83±12 | NS |

GCLC, Glutamate-cysteine ligase catalytic subunit; GPx, Glutathione peroxidase; GSH, Reduced glutathione; GSSG, Oxidized glutathione; HO-1, Heme oxygenase-1; Nrf2 (Nfe2l2), Nuclear factor-erythroid 2 related factor 2; NQO-1, NAD(P)H quinone dehydrogenase 1; qPCR, Quantitative-Polymerase chain reaction; PO, Palm olein; SOD, Superoxide dismutase; TBARS, Thioabarbituric acid reactive substances; ww, Wet weight.
increase in these parameters indicates lipid oxidation by free radicals, we can say that PO and OO diets did not favour lipoperoxidation.

These results indicate that despite the significant decrease in SOD activity by PO diet compared to the control diet, PO diet does not favour oxidative stress compared to OO diet. This antioxidant power could be partly explained by the increase in the amount of thiols in the PO diet.

In addition, numerous studies argue in favour of the antioxidant power of palm oil. Selenium deficiency favours the reduction of GPx activity with the consequent occurrence of a significant oxidative stress, the source of many pathologies (Navarro-Alarcon & Lopez-Martinez, 2000; Rayman, 2000). A study carried out in Côte d'Ivoire showed that subjects deficient in selenium and GPx, regular consumers of palm oil (crude or olein) had a good antioxidant status and did not present oxidative stress (Tiahou et al., 2004). In another study conducted on four varieties of palm oil from Côte d'Ivoire, Mondé and others (Mondé et al., 2011) showed that antioxidants in these different variants reduce LDL oxidation in vitro.

Palm oil owes its nutritional benefits, linked to its antioxidant power, to its «minor» components. In animals, numerous studies (Suarna et al., 1993; Azlina et al., 2005; Suzana et al., 2005) have highlighted the antioxidant effects of palm oil tocotrienols. Coenzyme Q10 (ubiquinone), a natural coenzyme of palm oil, is a powerful free radical scavenger (Niklowitz et al., 2007) with ten times the antioxidant power of carotenoids and vitamin E (Ng et al., 2006). Palm oil is the vegetable oil richest in tocotrienols (Sundram et al., 2003; Sen et al., 2010). Tocotrienols are powerful antiradical agents with a proven cardio-protective role (Rooyen et al., 2008; Vasanthi et al., 2012; Wong & Radathrakshin, 2012).

Despite the loss of carotenoids (Lecerf, 2013) and micro-constituents such as flavonoids and phenolic acids during refining (Tan et al., 2001), PO still retains its antioxidant properties, as the results of this study show. This may be due to the fact that during refining, PO is enriched with tocotrienols (Sambanthamurthi et al., 2000; Lecerf, 2013), which are powerful free radical scavengers.

**CONCLUSION**

This study showed that PO decreases SOD activity compared to OO while OO increases macrophagic inflammation in the liver. PO consumption does not promote inflammation and oxidative stress.

**Acknowledgements**

We thank Dr Jonas Laget for his help during the dissection of the rats. We thank SANIA company for providing us with palm oil.

**Conflicts of Interest**

The authors declare that there is no conflict of interest regarding publication of this article.

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