Influence of Diets Containing Olive Oil, Sunflower Oil or Hydrogenated Coconut Oil on the Immune Response of Mice

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Summary The effect of diets containing olive oil, sunflower oil or hydrogenated coconut oil on immune cell function was investigated. Lipid administration in assays in vitro as well as in the diet has been demonstrated that affect to lymphocyte proliferation, cytokine synthesis, natural killer (NK) cell activity and antigen presentation. Therefore, unsaturated fatty acids may be used in the treatment of inflammatory diseases due to their immunomodulatory properties. Three groups of Balb/c mice were fed three diets at various times for 5, 15, 30, 60, or 90 days. Diets were different for each group, as each contained 15 wt% olive oil, sunflower oil or hydrogenated coconut oil. The number of splenic cells showed significant differences with respect to the type of dietary lipid and the time of dietary administration. At the end of dietary lipid supplementation mitogen-stimulated lymphocyte proliferation from the spleens of mice fed diets that contained olive oil or hydrogenated coconut oil was significantly higher than that of mice fed diet that contained sunflower oil in which mitogen-stimulated proliferation of lymphocytes decreased lightly. Interleukin-2 (IL-2) production was significantly higher in mice fed the olive oil- or sunflower oil-enriched diets for 90 days when compared with that of mice fed the diet containing hydrogenated coconut oil. Dietary lipid manipulation also modified NK cell activity, since the results obtained showed a significant difference between the NK cell activity of mice fed the diet that contained olive oil and that observed in the mice fed the diet containing hydrogenated coconut oil. The results demonstrate that the addition to the diet of unsaturated fatty acids for different times may play an important role as a modulatory agent of immune cell function.

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Previous studies have reported that diets containing oils or fatty acids added to the culture medium have strong effects on lymphocyte functions, in both animals and humans [1-4]. Polyunsaturated fatty acids (PUFA) such as (n-3) PUFA suppressed mitogen-stimulated proliferation of lymphocytes isolated from lymph nodes [1] and spleen of rats [5], as well as from spleen of mice [5] and peripheral blood mononuclear cells from humans [4].

Therefore, the supply of oils in the diet, as well as the addition of fatty acids in assays in vitro, may affect the immune response. In this way, Yaqoob et al. [6] reported that diets containing unsaturated fatty acids found in olive oil, evening primrose oil and menhaden oil inhibited the mitogenic response of lymphocytes from rats to a greater extent than diets containing saturated fatty acids found in hydrogenated coconut oil.

Activation of lymphocytes involves synthesis and secretion of a large number of cytokines and of their receptors. Interleukin (IL)-2 is a cytokine responsible for T-cell growth and its production is also modified by dietary lipids [3]. Many studies have investigated the effects of fatty acids on IL-2 production and have reported that a wide range of unsaturated fatty acids suppress the production of this cytokine but that they do not affect the expression of the low-affinity subunit of the IL-2 receptor [7-9]. However, unsaturated fatty acids can inhibit the expression of the transferrin receptor by Concanavalin A (Con A)-stimulated rat lymphocytes [7].

Dietary lipid manipulation can also modify natural killer (NK) cell activity, both in humans and animals [10, 11]. Barone et al. [10] demonstrated that a reduced fat intake in human volunteers leads to a significant increase in NK cell activity, and Yamashita et al. [12] evidenced that intravenous injection of triacylglycerols containing eicosapentaenoic acid or docosahexaenoic acid into human volunteers suppressed NK cell activity. Yaqoob et al. [11] confirmed these results in weanling rats fed diets containing oils and found that a fish oil-enriched diet seems to be the most suppressive oil, followed by the olive oil and evening primrose oil diets.

The effect of diets containing oils on immune system regulation depends on the composition and amount of fatty acids in the diet, the duration of dietary supplementation, the species and sex of experimental animals, etc. However, changes in the eicosanoid levels may trigger the mechanisms involved in the modification of the immune response by lipids, since prostaglandin E₂ inhibits lymphocyte proliferation [13], IL-2 production [14], cytotoxicity [15] and NK cell activity [16]. In contrast, leukotriene B₄ increases the production of IL-2 [17] and NK cell activity [18].

Finally, the results from clinical studies have also demonstrated the beneficial
effects of fish oil on autoimmune diseases such as rheumatoid arthritis [19], psoriasis [20] and ulcerative colitis [21]. Therefore, it is possible that an olive oil-supplemented diet may be used in the treatment of inflammatory diseases as are fish oil-enriched diets. Our current study shows how a diet containing unsaturated fatty acids affects immune cell function. The protocol has been designed to establish the effects of a diet containing olive oil on lymphocyte proliferation, IL-2 production and NK cell activity at different time-points from 5 to 90 days of dietary lipid supplementation and to compare these results with the effects of diets containing sunflower oil or hydrogenated coconut oil.

MATERIALS AND METHODS

Animals and experimental diets. Balb/c mice (obtained from the animal breeding colony of University of Jaén, Spain), 10 weeks of age and approximately 25 g of weight were randomly housed in cages at a temperature of 24°C with a 12-h light-dark cycle and fed a given diet for 5, 15, 30, 60, or 90 days. During these periods the mice had free access to water and to the experimental diets. The animals were divided into three dietary groups according to the type of diet, five animals per cage. Diets contained either olive oil (15 wt%), sunflower oil (15 wt%) or hydrogenated coconut oil (15 wt%), and were protected from light and kept at 4°C to prevent the growth of microorganisms. The composition of the diets is shown in Table 1.

Isolation of mouse splenic cells. Mice were sacrificed by cervical dislocation, and their spleens were collected aseptically and homogenized. Mononuclear cells were isolated by a discontinuous gradient centrifugation on Ficoll-Histopaque®-1083 (Sigma, St. Louis, MO) in order to discard the polymorphonuclear cell subset. Mononuclear cells were washed three times by adding Hanks’ balanced salt solution (HBSS) (Sigma) by centrifugation at 400 × g for 10 min at 4°C. Finally, the cells were resuspended in HEPES-buffered RPMI 1640 supplemented with 10% fetal calf serum (FCS) (Sigma), antibiotics (Sigma), and 2 mM glutamine (Sigma).

Table 1. Composition of the experimental diets.

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>wt%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein</td>
<td>20</td>
</tr>
<tr>
<td>D.L-Methionine</td>
<td>0.3</td>
</tr>
<tr>
<td>Cornstarch</td>
<td>31.5</td>
</tr>
<tr>
<td>Sucrose</td>
<td>20.5</td>
</tr>
<tr>
<td>Cellulose fiber</td>
<td>8</td>
</tr>
<tr>
<td>Fat*</td>
<td>15</td>
</tr>
<tr>
<td>Mineral mixture</td>
<td>3.5</td>
</tr>
<tr>
<td>Vitamin mixture</td>
<td>1</td>
</tr>
<tr>
<td>Choline bitartrate</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*Fat supplement was olive oil, sunflower oil or hydrogenated coconut oil.
Cell number was adjusted to $7 \times 10^6$ cells/ml in HEPES-buffered RPMI 1640 with 10% FCS. Cell viability, assessed by trypan blue exclusion, was greater than 95%.

**Cellular proliferation assay.** Mononuclear cells were adjusted to $7 \times 10^6$ cells/ml in HEPES-buffered medium supplemented with 10% FCS. Then, 100 μl of the cell suspension was added to each well of a 96-well culture plate (Corning, Corning, NY). Subsequently, 100 μl of the mitogen suspension in HEPES-buffered medium supplemented with 10% FCS were also added to each well. Mitogens used in this study were the following: lipopolysaccharide (LPS) from *Escherichia coli* serotype 026:B6 (Sigma) at a concentration of 25 μg/ml, pokeweed mitogen (PWM) (Sigma) at a concentration of 15 μg/ml, Con A (Sigma) at a concentration of 10 μg/ml, and phytohemagglutinin (PHA) (Sigma) at a concentration of 15 μg/ml. Finally, the plates were cultured for 72 h at 37°C in a 5% CO2 atmosphere with 98% humidity.

**Measurement of lymphocyte proliferation.** Lymphocyte proliferation was measured by an ELISA method as previously described by Mosmann [22]. Briefly, 50 μl of (3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide) (MTT, Sigma) in PBS at a concentration of $5 \times 10^5$ mg/liter (stock solution) and sterilized by filtration (Millipore 0.22 μm) was added to each well of a 96-well culture plate (Corning). The plate was incubated for 4 h at 37°C in a 5% CO2 atmosphere with 98% humidity. Then, 100 μl of HCl in isopropanol (0.04 M) was added to each well, and the plate was shaken vigorously to dissolve the dark blue crystals. Finally, the samples were measured with a microplate reader (Whittaker Microplate Reader 2001, Salzburg, Austria) using a test wavelength of 550 nm and a reference wavelength of 620 nm. The values were expressed in optical density units.

**Production and determination of IL-2.** The measurement of IL-2 production was performed by an ELISA method. Splenic cells were adjusted to $1.2 \times 10^6$ cells/ml in HEPES-buffered RPMI 1640 supplemented with 10% FCS. One hundred microliters of this cellular suspension and 100 μl of Con A at a concentration of 8 μg/ml were added to each well of a 96-well plate (Corning). The cells were then incubated for 96 h at 37°C in a 5% CO2 atmosphere with 98% humidity. Subsequently, aliquots of the supernatants were frozen and stored at $-80^\circ$C until analysis of IL-2 could be performed by an ELISA method. The assay for IL-2 production was carried out by use of a commercially available ELISA kit (Endogen, Cambridge, MA) in accord with the manufacturer's instructions. The absorbance values were obtained with a microplate reader (Whittaker Microplate Reader 2001) using a test wavelength of 450 nm and a reference wavelength of 550 nm. The results were expressed in optical density units.

**Preparation of cells and measurement of NK cell activity.** Spleen cells (effector cells) were prepared as described above. Then, mononuclear cells were resuspended and adjusted to a final concentration of $6 \times 10^5$ cells/ml in HEPES-buffered RPMI 1640 with 10% FCS.

The NK-susceptible K-562 cell line (target cells) was cultured in HEPES-buffered RPMI 1640 supplemented with 10% FCS, glutamine, and a solution of

antibiotics, at 37°C in a 5% CO₂ atmosphere with 98% humidity. Before starting the assay, we checked the cells for viability by trypan blue exclusion and adjusted them to a concentration of 1 × 10⁵ cells/ml in HEPES-buffered RPMI 1640 supplemented with 10% FCS. For the measurement of NK cytotoxic activity, we employed the method described by Dinota et al. [23]. Briefly, 200 µl of medium containing the target cells and 200 µl of medium containing the effector cells were mixed in a tube and cultured for 4 h at 37°C in a 5% CO₂ atmosphere with 98% humidity. Control experiments were carried out with K-562 cells alone. After the incubation, 20 µl of the cellular mixture (effector cells/target cells) was added to a 6-well culture plate (Corning). Then, 1 ml of plasma clot medium, consisting of 25% FCS and 75% complete RPMI 1640 was added to each well. Then, 40 µl of CaCl₂ at a concentration of 55 mg/ml was added to each well in order to obtain the plasma clot. Finally, the plate was incubated for 96 h at 37°C in a 5% CO₂ atmosphere with 98% humidity.

NK cell activity was calculated as percentage cytolysis and it was obtained by use of an inverted microscope to count clones having more than eight cells.

Statistical analysis. Results are shown as means ± SEM. Statistical analysis was performed by two-way factorial ANOVA contained in Statgraphics statistical package version 5.0 (STSC, Inc., Rockville, MD). Comparisons between dietary groups at different time points were made by use of the LSD test. The level of statistical significance was determined at p < 0.05.

RESULTS

Splenic cell counts and mouse body weight

Cell numbers from the spleens of mice consuming the diet containing sunflower oil were significantly different (p < 0.05) from those from the spleen of olive oil- or hydrogenated coconut oil-fed mice at the 30th day of dietary supplementation. We also found differences between cell numbers of sunflower oil- and hydrogenated coconut oil- or olive oil-fed mice at the 5th day and between sunflower oil- and olive oil-fed mice at the 90th day of dietary supplementation (Fig. 1).

There was no difference in the body weights of mice consuming the different diets over the 90-day feeding period.

Effect of dietary lipid on lymphocyte proliferation

Splenic cells were stimulated with specific mitogens, and lymphocyte proliferation was comparatively determined for each of the dietary groups.

The LPS-stimulated lymphocyte proliferation (Table 2) showed a reduction at the 5th and at the 90th day of administration of the diet containing olive oil compared with the proliferation of LPS-stimulated lymphocytes from mice fed diets containing sunflower oil or hydrogenated coconut oil (p < 0.05). In the sunflower oil-fed mice there were no great differences in the lymphocyte prolifera-
tion, except at the 5th day of administration of this diet. By contrast, proliferation of lymphocytes from the hydrogenated coconut oil-fed mice showed higher values than that in other groups at the 5th, at the 60th, and at the 90th days of dietary lipid supplementation.

Proliferation of Con A- or PHA-stimulated lymphocytes from olive oil-fed mice was significantly lower than that of cells from the other groups at the 5th and at the 90th days of dietary lipid administration. By contrast, in hydrogenated coconut oil-fed mice we obtained the highest response with both Con A and PHA mitogens in the 90th day of dietary administration in comparison with lymphocyte proliferation observed in olive oil- or hydrogenated coconut oil-enriched diets (Tables 3 and 4).

Fig. 1. Effect of diets containing olive oil (OO), sunflower oil (SO) or hydrogenated coconut oil (HCO) in the number of splenic cells from Balb/c mice. Results are means ± SEM of cell preparations from groups of 5 animals. Bars with different letters for a single time period are significantly different at p < 0.05.

Table 2. Effects of dietary lipids on LPS-stimulated proliferation of lymphocytes from Balb/c mice fed diets containing olive oil, sunflower oil or hydrogenated coconut oil.

<table>
<thead>
<tr>
<th>Time (d)</th>
<th>Olive oil</th>
<th>Sunflower oil</th>
<th>Coconut oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>0.13 ± 0.04&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.27 ± 0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.26 ± 0.06&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>15</td>
<td>0.14 ± 0.08&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.17 ± 0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.11 ± 0.06&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>30</td>
<td>0.24 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.19 ± 0.02&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.17 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>60</td>
<td>0.24 ± 0.05&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.19 ± 0.02&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.26 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>90</td>
<td>0.13 ± 0.05&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.18 ± 0.07&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.47 ± 0.07&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Each value is the mean ± SEM of 5 animals measured as optical density units. Values in a row with different superscripts are significantly different at p < 0.05 as determined by two-way factorial ANOVA.
Finally, PWM-stimulated lymphocyte proliferation (Table 5) showed the greatest reduction at the 5th and 15th day of administration of the diet containing olive oil compared with that of mice fed diets containing sunflower oil or hydrogenated coconut oil. The highest values were obtained with lymphocytes from hydrogenated coconut oil-fed mice where we found significant differences in comparison with values for sunflower oil- or olive oil-fed mice (p < 0.05), at 60th and 90th days.

**Determination and production of IL-2**

IL-2 production by splenic mononuclear cells from mice fed diets containing olive oil or sunflower oil was higher at the 5th day of dietary administration compared with the production of IL-2 by cells from mice fed the diet containing hydrogenated coconut oil (Fig. 2). At the end of dietary administration we observed the highest values for the olive oil-fed mice whereas in the groups fed diets that contained sunflower oil or hydrogenated coconut oil we could observed a significant reduction in comparison to the olive oil-fed mice.

**Influence of dietary lipids on NK cell activity**

NK cell activity was measured as percent inhibition of K-562 cell growth. The
The influence of dietary lipid manipulation on NK activity is shown in Fig. 3. Cells from olive oil-fed mice showed the lowest percent inhibition, and the values for cells of mice fed the diet containing hydrogenated coconut oil were higher than those for cells obtained from mice fed the other two diets. We found statistically significant differences between NK cell activity of mice fed the diet containing olive oil and that of mice fed the diet containing hydrogenated coconut oil from the 15th to the 90th days of dietary lipid supplementation (p < 0.05). However at the beginning of dietary lipid supplementation we did not find statistical significant changes in the NK activity in all of the three groups assayed.

**Table 5. Effects of dietary lipids on PWM-stimulated proliferation of lymphocytes from Balb/c mice fed diets containing olive oil, sunflower oil or hydrogenated coconut oil.**

<table>
<thead>
<tr>
<th>Time (d)</th>
<th>Olive oil</th>
<th>Sunflower oil</th>
<th>Coconut oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>0.11±0.04a</td>
<td>0.19±0.04a</td>
<td>0.21±0.03a</td>
</tr>
<tr>
<td>15</td>
<td>0.10±0.06c</td>
<td>0.19±0.02a</td>
<td>0.13±0.08b</td>
</tr>
<tr>
<td>30</td>
<td>0.26±0.02a</td>
<td>0.15±0.03c</td>
<td>0.19±0.01b</td>
</tr>
<tr>
<td>60</td>
<td>0.23±0.12b</td>
<td>0.23±0.02b</td>
<td>0.30±0.02a</td>
</tr>
<tr>
<td>90</td>
<td>0.17±0.02b</td>
<td>0.20±0.02b</td>
<td>0.45±0.06a</td>
</tr>
</tbody>
</table>

Each value is the mean ± SEM of 5 animals measured as optical density units. Values in a row with different superscripts are significantly different at p < 0.05 as determined by two-way factorial ANOVA.

**Fig. 2.** Effects of dietary lipids on IL-2 production from splenic cells of Balb/c mice fed diets containing olive oil (OO), sunflower oil (SO) or hydrogenated coconut oil (HCO). Results are means ± SEM of cell preparations from groups of 5 animals. Bars with different letters for a single time period are significantly different at p < 0.05.
DISCUSSION

These results show that dietary lipid manipulation modifies the immune cell function and that this change depends on the type of oil supplied in the diet and on the duration of dietary lipid supplementation.

Previous studies have shown that fatty acids are involved in the modification of lymphocyte proliferation in both humans and animals [2, 6, 24], IL-2 synthesis [25, 26], NK cell activity [11] and antigen presentation [27] both in vitro and in vivo and that these changes depend on the type of dietary lipid and the age of the experimental animals [28]. The results presented in this paper show that the supply of diets containing olive oil may increase the lymphocyte proliferation, although at the end of dietary supplementation the values decreased and these were very similar to those obtained at the beginning of the dietary supplementation, whereas IL-2 production was reduced except at the end of dietary administration where we have observed a significant increase with regard to the values obtained from the other groups. However, percentage cytolysis of NK cell is significantly reduced at the 15th day of dietary lipid supplementation. On the contrary in mice fed sunflower oil, lymphocyte proliferation is lightly reduced, except in PWM-stimulated proliferation of lymphocytes as well as NK cell activity of mononuclear cells from spleens of mice. By contrast, the supply of diet containing hydrogenated coconut oil enhances both lymphocyte proliferation and NK cell activity by comparison with those observed in mice fed diets containing olive oil or sunflower oil.

Fig. 3. Influence of dietary lipids on NK cell activity of Balb/c mice fed diets containing olive oil (OO), sunflower oil (SO) or hydrogenated coconut oil (HCO). Results are means±SEM of cell preparations from groups of 5 animals. Bars with different letters for a single time period are significantly different at p<0.05.
In this study the splenic cell number showed significant differences between dietary lipid manipulation and duration of dietary lipid supplementation, although previous studies that used cells from spleen found no significant effect of dietary lipid manipulation on lymphocyte subsets [6]. These authors also reported that lymphocyte counts from different tissues respond differently to dietary lipid manipulation.

Our results show that dietary lipid supplementation leads to an increase in lymphocyte proliferation, since the proliferative response of splenic lymphocytes was lightly increased in the group fed the diet containing olive oil, when the cells were stimulated by either T cell (Con A and PHA)- or B cell-specific mitogens (LPS and PWM). Several studies have reported that feeding experimental animals or humans a diet containing (n-3) PUFA inhibits lymphocyte proliferation [3, 29]. Our results are partially consistent with such findings, since we observed an increase in lymphocyte proliferation, when we have provided diets containing olive oil or hydrogenated coconut oil compared with such inhibition in mice fed the diet containing sunflower oil, where we observed a light reduction in lymphocyte proliferation. However, there are different methodologic factors that affect to this response, it has been described that this effect depends on both the tissue origin and the cell culture conditions, such as the type of serum used. In fact, Yaqoob et al. [6] observed that the response of lymphocytes to mitogen was greater when cells were cultured in autologous serum than when cultured in FCS. These authors also showed that some dietary lipids containing olive oil, evening primrose oil, and fish oil possess immunosuppressive properties.

The mechanism involved in these processes is unknown yet, but it seems to be that these effects are caused by cell culture conditions that modify the phospholipid fatty acid composition and, thereby alter the plasma membrane fluidity of these cells [30]. In our study FCS has been used in the cultures of cells from mice fed the three diets, so that the results obtained would reflect the effect of this factor. However, other studies have shown that lymphocyte reduction by dietary lipid manipulation is also caused by regulation of eicosanoid metabolites, such as prostaglandin E$_2$ and leukotriene B$_4$ [13], by production of lipid peroxides that are toxic to certain cells [31], and by direct interaction with cellular activation mechanisms [32]. However, it is not clear how dietary lipid manipulation modifies these factors to promote a reduction in the lymphocyte proliferation and alter the immune response. Many studies are in accord with our results about the enhanced proliferation of lymphocytes from mice fed a diet containing hydrogenated coconut oil, because such a diet is much less immunosuppressive than an olive oil- or sunflower oil-enriched one. In fact, several authors reported that saturated fatty acids are not as inhibitory as unsaturated fatty acids in vitro or in vivo [4, 6, 33].

Previous studies have also shown that diets containing PUFA reduce cytokine production [3, 9] whereas diets containing saturated fatty acids have no effect on the production of the cytokines. In fact, the levels of other cytokines as IL-1 and tumor necrosis factor (TNF) did not change in mice fed a hydrogenated coconut oil.
oil diet [34]. In our research, IL-2 production showed significant differences between dietary lipids; but they were not statistically significant in all the cases, and the results within each group differed greatly. In olive oil- or sunflower oil-fed mice, production of IL-2 showed the highest values at almost all the points that have been assayed, whereas we found the smallest values in the hydrogenated coconut oil-fed group. Therefore, these observations are in accord with Yaqoob and Calder [35] who showed that IL-2 production was enhanced by diets that contained olive oil or sunflower oil, whereas it was reduced in animals fed diets containing hydrogenated coconut oil. These effects appeared when autologous serum was employed in the culture medium. However, the levels of IL-2 production were reduced when FCS was used in the culture medium and both olive oil- or hydrogenated coconut oil-fed mice showed the same values. These data suggest that the increase in the lymphocyte proliferation of mice fed diets containing olive oil or hydrogenated coconut oil and the partial reduction showed in mice fed sunflower oil diet did not occur because of a direct alteration in the production of IL-2. Instead, this modulation could be due to an increase in the production of a different cytokine. In this way, Calder and Newsholme [7] suggested that fatty acids would regulate the immune system by acting at more than one stage in the proliferative cycle.

This study has also evaluated NK cell activity from splenic cells, since it is regulated by dietary lipids in both humans and animals [10, 11]. A reduction in the total fat content in the diet of humans leads to an increase in NK cell activity [10]. We observed that NK cell activity measured as percentage cytolysis for olive oil- or sunflower oil-fed mice showed a greater reduction by comparison with that from hydrogenated coconut oil-fed mice. Our results are consistent with the recently published data on the inhibitory effect of dietary lipids on NK activity of splenic cells isolated from rats [11], since we found a lower percentage cytolysis of NK cell activity from mice fed diets containing olive oil or sunflower oil. The mechanism involved in the reduction of NK cell activity is not known yet, although a possible explanation for these effects could be regulation of eicosanoid levels.

Our results suggest that in olive oil- or the sunflower oil-fed mice a light increase in lymphocyte proliferation and a reduction of NK cell activity occurs, whereas IL-2 production is enhanced in comparison with that observed in hydrogenated coconut oil-fed mice. However, further studies will need in order to discover the usefulness of fatty acids as modulators of the immune system and their possible application in the treatment of patients suffering from autoimmune diseases.

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REFERENCES


