RESEARCH ARTICLE

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THE EFFECT OF DIFFERENT DIETARY FATS ON LIPID PROFILE, GLUCOSE, T₃, T₄, AND IRON LEVELS IN PLASMA OF MICE

ABSTRACT:
The present study was conducted to define the relative effects of changes in the type of dietary fat on a number of key parameters involved in the regulation of cholesterol and lipoproteins levels. The present data indicated that intake of butter and cholesterol diet caused significant elevations in body weight, total lipids, triglycerides, total cholesterol, LDL, VLDL, glucose and iron levels in mice during the experimental period. On the other hand, this atherogenic diet caused low levels of HDL, T₃, and T₄ as compared to control. It was obvious that olive oil could improve these changes completely. While, sunflower oil improved these changes partially. It was evident that olive oil is more effective than sunflower oil, this may be due to its high content of oleic acid and its antioxidant phenolic substances.

KEY WORDS:
Cholesterol, Olive oil, Sunflower oil, Mice.

INTRODUCTION:
Despite the use of cholesterol lowering drugs, atherosclerotic vascular diseases will likely continue to be the main cause of death allover the world. The interest in preventive approaches complementary or alternative to cholesterol reduction should be one of the main objectives.

Dietary cholesterol exerts specific effect on plasma cholesterol concentrations although the extent of the effects are highly variable due to individual metabolic heterogenicity, different degrees of response among various animal species, and whether physiological or pharmacological amounts of cholesterol are used as the dietary challenge (Grundy and Denk, 1990).

The dietary cholesterol may play a role in atherosclerosis and the link between dietary cholesterol and atherosclerotic lesions is thought to be primarily related to its effect on plasma low density lipoprotein; LDL (Horton et al., 1995). Diets that contain large amount of cholesterol partially inhibit endogenous cholesterol synthesis but result in a net increase in serum cholesterol level because of suppression of synthesis of low density lipoprotein receptors (Robert, 2004). Replacing saturated fatty acids (SFA) with monounsaturated (MUSF) or polyunsaturated fatty acids (PUSF) would be an appropriate way to meet the nutrients recommendations and to lower serum total cholesterol (Hu et al., 2001). Sola et al. (1997) reported that dietary MUSF prevent the oxidative modification of lipoproteins. A randomized trial known as the optimal macronutrient intake trial for heart health showed that replacing a carbohydrate-rich diet with one rich in unsaturated fat, predominantly monounsaturated fats, lowers blood pressure, improves the lipid levels and reduces the estimated cardiovascular risk (Appel et al., 2005). Ewers et al. (2009) stated that the supplementation of unsaturated fat reduces systemic inflammation as assessed by serum c-reactive protein concentrations. Also, the authors reported that adding unsaturated fat to the diet seems to be a safe and effective in malnutrition in hemodialysis patients.

Several data suggested that vegetable oils may have hypocholesterolemic effect. Rape seed oil (MUSF) can replace oils and...
fats in a lipid–lowering diet (Valsta et al., 1992; Gustafsson et al., 1994). Garlic oil capsules mostly decrease cardiovascular heart disease (CVD) (Zhang et al., 2001). Intake of corn oil (PUSF) results in lowering plasma total LDL-cholesterol concentrations (Fernandez et al., 1992). Animals fed rice bran oil in combination with defatted rice bran diet had significantly lower total liver cholesterol content (Sugano and Tsuji, 1997). Using of Soyabean oil (PUSF) in reduced-fat diets resulted in clinically relevant plasma cholesterol - lowering (Insull et al., 1994).

Virgin olive oil contains a number of glycerides of oleic acid 83.5%, palmitic acid 9.4%, linoleic acid 4.0%, stearic acid 2.0% and arachidonic acid 0.9%. Oleic acid has shown to have an activity in cancer prevention (Waterman and Lockwood, 2007). Oleic acid diminishes the risk of cell damage by scavenging free radicals (Owen et al., 2000 and 2004). The flavonoids polyphenols in olive oil have a beneficial effect in lowering cholesterol level, blood pressure and risk of coronary heart disease (Visioli and Galli, 2007 and Waterman and Lockwood, 2007).

Sunflower oil is considered as premium edible oil because linolenic acid is totally absent and it has very high content of linoleic acid(72%), this acid seems to play an important role in cardiac ailments (Quiles et al., 2004). The British pharmacopoeia lists (2005) the following profile of sunflower oil: palmitic acid (4-9%), stearic acid (1-7%), oleic acid (14-42%), linoleic acid (48-72%). Linoleic acid is a member of the group of essential fatty acids called omega-6 fatty acids, so called because they are essential dietary requirements for all mammals. Linoleic acid is essential nutrient required by the body for the synthesis of arachidonic acid, the major precursor of prostaglandins. Deficiency of linoleic acid results in dermatitis, hair loss and impaired wound healing (Ruthing and Meckling-Gill, 1999). Polyunsaturated fatty acids (PUFA) of the omega-6 series (Linoleic acid) are essential for normal growth and development. The health effects of these fatty acids include reduction of cardiovascular risk due to antiarrhythmic, anti-inflammatory, anti-thrombotic and lipid lowering actions.

So, the present study was designed to define the relative effects of changing the type of dietary fat on a number of key parameters involved in regulation of cholesterol and lipoproteins metabolism in mice.

MATERIAL AND METHODS:

Material:
- Cholesterol (Sigma Chemical Co.st Louis, MO, USA).
- Commercial Virgin Olive oil and Sunflower oil (one of the supermarkets Cairo, Egypt).

Male mice, each weighing 25 ± 2g, were used throughout the experiment. They were obtained from the animal house of the High Institute of Public Health, University of Alexandria. The animals were housed in stainless steel cages in a room maintained at 22°C with a 12-h dark-light cycle. They were provided with water and diet ad libitum and they were acclimatized in the laboratory for one week.

Study design:

Thirty animals were divided into two main groups:

Group A: Control group; The animals of this group (10) were fed on a diet as shown in table 1.

Group B: The animals of this group (20) were fed on butter and cholesterol diet (Table 1).

After one month from the beginning of the experiment, five animals were randomly selected from each group and were anaesthetized using sodium pentobarbitone. Blood samples were collected by cardiac puncture and placed into EDTA-containing tubes for plasma preparations.

The remaining five mice of group A continued their feeding on the same diet for another one month, while those of group B were subdivided into the following three subgroups (five mice each):

Subgroup (1):
- Mice were fed on diet containing butter and cholesterol for another month.

Subgroup (2):
- Mice were fed on diet containing virgin olive oil for one month.

Subgroup (3):
- Mice were fed on diet containing sunflower oil for one month as shown in table 1.

At the end of the experiment all animals were anaesthetized and blood samples were collected. Plasma was separated for determination of total cholesterol by the method of Allain et al. (1974), low density lipoprotein (LDL), very low density lipoprotein (VLDL) and high density lipoprotein (HDL) by the method of Burstein et al. (1970). Triglycerides were determined according to Wahlefeld and Bergmeyer (1974). Glucose level was measured by the method of Trinder (1969). Triiodothyronine (T3) and thyroxine (T4) were estimated by using the method of Tietz (1995). Spectrophotometry was used for determination of iron (Fe²⁺) using Perkin Elmer-2380 atomic absorption spectrophotometer.

Weight gains of the control, as well as the experimental animals were determined after the 1st and 2nd month of the start of the experiment.
and T4 levels as compared to control group. A diet caused a significant decrease in HDL, T3, T4 levels as compared to control group. Also, this diet caused partial improvement in these parameters as compared to control and olive oil diets. However, sunflower oil diet caused complete improvement in these parameters as compared to control and olive oil diets. While, it induced non-significant differences when compared with butter and cholesterol diet.

RESULTS:

The present data revealed that animals fed on butter and cholesterol diet had a significant increase in the body weight as compared to control group as shown in table 2. Olive oil diet caused a highly significant decrease in body weight since there were significant differences between this group and the other experimental groups. The animals fed on sunflower diet showed significant decrease in body weight as compared to butter and cholesterol group. On the other hand, no significant difference was observed in body weight between control and sunflower groups. However, sunflower oil diet caused significant differences between this group and the other experimental groups. It was followed by least-significant difference (LSD) test at the level of P< 0.05 by using SPSS program (version 16).

Table 1. Composition of the experimental diets

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>g/kg</th>
<th>Control group</th>
<th>Butter &amp; Cholesterol subgroup</th>
<th>Olive oil subgroup</th>
<th>Sunflower oil subgroup</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein</td>
<td>250</td>
<td>250</td>
<td>250</td>
<td>250</td>
<td></td>
</tr>
<tr>
<td>Corn Starch</td>
<td>400</td>
<td>400</td>
<td>400</td>
<td>400</td>
<td></td>
</tr>
<tr>
<td>Butter</td>
<td>-</td>
<td>245</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Olive Oil</td>
<td>-</td>
<td>-</td>
<td>250</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Sunflower Oil</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>250</td>
<td></td>
</tr>
<tr>
<td>Mineral mixture</td>
<td>40</td>
<td>40</td>
<td>40</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Cellulose</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Vitamin mixture</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

Statistical analysis:

One way analysis of variance (ANOVA) was performed to test for differences between experimental groups. It was followed by least-significant difference (LSD) test at the level of P< 0.05 by using SPSS program (version 16).

Table 3. Body weight of animals fed on control, butter and cholesterol, olive oil and sunflower oil diets during the experimental period

<table>
<thead>
<tr>
<th>Time</th>
<th>Control group</th>
<th>Butter and cholesterol group</th>
<th>Virgin Olive oil</th>
<th>Sunflower oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st month</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2nd month</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>3rd month</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>4th month</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One month after fat replacement</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Weight</td>
<td>25.60 ± 0.600</td>
<td>26.80 ± 1.200</td>
<td>31.00 ± 1.158 a</td>
<td>38.40 ± 1.428 a</td>
</tr>
<tr>
<td></td>
<td>20.00 ± 1.158</td>
<td>1.200 ± 1.066</td>
<td>14.28 ± 1.304 ab</td>
<td>8.00 ± 0.583</td>
</tr>
<tr>
<td></td>
<td>25.20 ± 1.378</td>
<td>1.518 ± 1.095</td>
<td>8.60 ± 1.581 b</td>
<td>1.789 ± 0.374</td>
</tr>
</tbody>
</table>

- Each value represents the mean of five experiments ± standard error (g)
- a, b, c, d, e statistically significant (p<0.05) when compared the mean values of all experimental groups.

Table 4. Glucose, triiodothyronine (T3) and thyroxine (T4) levels and iron concentrations in animals from study

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group</th>
<th>Butter &amp; cholesterol group</th>
<th>Virgin Olive oil</th>
<th>Sunflower oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st month</td>
<td></td>
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<tr>
<td>2nd month</td>
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<tr>
<td>3rd month</td>
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<tr>
<td>One month after fat replacement</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>81.00 ± 1.147</td>
<td>82.00 ± 1.087</td>
<td>92.00 ± 0.862</td>
<td>82.00 ± 1.107</td>
</tr>
<tr>
<td></td>
<td>± 1.703 ± 1.170</td>
<td>± 1.500 ± 1.107</td>
<td>± 1.180 ± 1.673</td>
<td>± 1.107 ± 1.095</td>
</tr>
<tr>
<td></td>
<td>± 1.800 ± 1.703</td>
<td>± 1.540 ± 1.500</td>
<td>± 1.594 ± 1.800</td>
<td>± 1.540 ± 1.500</td>
</tr>
<tr>
<td></td>
<td>± 1.900 ± 1.800</td>
<td>± 1.594 ± 1.673</td>
<td>± 2.898 ± 1.900</td>
<td>± 2.898 ± 1.900</td>
</tr>
<tr>
<td></td>
<td>± 1.897 ± 1.594</td>
<td>± 2.898 ± 1.900</td>
<td>± 2.097 ± 1.897</td>
<td>± 2.097 ± 1.897</td>
</tr>
<tr>
<td></td>
<td>± 1.844 ± 1.789</td>
<td>± 2.097 ± 1.897</td>
<td>± 1.789 ± 1.844</td>
<td>± 1.789 ± 1.844</td>
</tr>
<tr>
<td>T3 (ng/ml)</td>
<td>129.00 ± 1.099</td>
<td>130.00 ± 1.060</td>
<td>94.30 ± 0.725</td>
<td>130.00 ± 1.060</td>
</tr>
<tr>
<td></td>
<td>± 0.837 ± 0.400</td>
<td>± 0.847 ± 0.400</td>
<td>± 0.962 ± 0.725</td>
<td>± 0.962 ± 0.725</td>
</tr>
<tr>
<td></td>
<td>± 1.208 ± 1.740</td>
<td>± 0.962 ± 0.725</td>
<td>± 2.000 ± 1.208</td>
<td>± 2.000 ± 1.208</td>
</tr>
<tr>
<td></td>
<td>± 1.173 ± 1.800</td>
<td>± 2.000 ± 1.208</td>
<td>± 1.800 ± 1.900</td>
<td>± 1.800 ± 1.900</td>
</tr>
<tr>
<td></td>
<td>± 1.789 ± 1.844</td>
<td>± 2.898 ± 1.900</td>
<td>± 2.097 ± 1.897</td>
<td>± 2.097 ± 1.897</td>
</tr>
<tr>
<td>T4 (ng/ml)</td>
<td>6.60 ± 1.109</td>
<td>6.50 ± 0.758</td>
<td>3.60 ± 0.600</td>
<td>3.60 ± 0.600</td>
</tr>
<tr>
<td></td>
<td>± 6.00 ± 0.400</td>
<td>± 0.400 ± 0.300</td>
<td>± 3.20 ± 0.300</td>
<td>± 3.20 ± 0.300</td>
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<tr>
<td></td>
<td>± 3.00 ± 0.300</td>
<td>± 3.20 ± 0.300</td>
<td>± 5.70 ± 0.500</td>
<td>± 5.70 ± 0.500</td>
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<tr>
<td></td>
<td>± 3.80 ± 0.500</td>
<td>± 5.70 ± 0.500</td>
<td>± 10.00 ± 2.000</td>
<td>± 10.00 ± 2.000</td>
</tr>
<tr>
<td>Iron (ppm)</td>
<td>97.00 ± 1.517</td>
<td>97.00 ± 1.581</td>
<td>138.00 ± 1.500</td>
<td>138.00 ± 1.500</td>
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<td>± 1.500 ± 1.000</td>
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</table>

- Each value represents the mean of five experiments ± standard error.
- a, b, c, d statistically significant (p<0.05) when compared the mean values of all experimental groups.
DISCUSSION:

Obesity represents a major threat to health and quality of life. The present data indicated that butter and cholesterol rich-diet induced body weight enhancement through the experimental duration in mice. These findings agree with Hill et al. (2000) who stated that supplementation of high fat diets has been suggested to promote obesity by increasing energy intake, thus increasing the probability of positive energy balance and weight gain. Field et al. (2007) reported that animals fed on the high-fat diet showed more body weight, higher fat deposition and total liver weight, and increased energy intake. The increased body weight by dietary saturated fats, that induces overconsumption and weight gain, may be due to its low satiety properties and high caloric density.

On the basis of the available data, the current public health recommendations to lower dietary fat intake appear to be appropriate. However, the present observation of decreased body weight with different dietary oils (virgin olive oil and sunflower oil) confirms their beneficial effects on the body weight enhancement caused by butter and cholesterol diet. Body weight reduction via improvement of carbohydrate and lipid metabolism would be very beneficial for obese hypertensive persons (Pederson, 1989). The results of the present work were in agreement with findings indicated by Nydahl et al. (1994) who estimated a significant decrease in mean body weight during the treatment periods with monounsaturated and polyunsaturated enriched diets. Bes-Rastrollo et al. (2006) reported that a high amount of olive oil consumption is not associated with higher weight gain or a significantly higher risk of developing over-weight or obesity in the context of the Mediterranean food pattern. The Mediterranean-type diet rich in monounsaturated fat from olive oil prevents centripetal fat collection. It was found that olive oil causes an increase in the breakdown of fats in fat cells (Paniagua et al., 2007). A 3-years follow up of a Mediterranean diet rich in virgin olive oil is associated with high plasma antioxidant capacity of reduced body weight gain (Razquin et al., 2009). Susan et al. (2009) suggested that replacement of dietary SFA with MUFA could prevent adipose tissue inflammation and may reduce the risk of inflammation – related diseases such as metabolic syndrome. Ferrini et al. (2008) found that feeding of polyunsaturated fatty acids to broiler chickens, in comparison to dietary saturated fatty acids, reduced the amount of both abdominal fat pad and subcutaneous fat by approximately 30 and 9%, respectively. Leigh et al. (2009) stated that supplementation with conjugated linoleic acid (CLA) reduced body mass index (BMI) and total adipose mass.

Suppression of body weight may be due to the presence of the oleic acid in both virgin olive and sunflower oils. Oleic acid has been identified as an appetite suppressor because of its ability to produce oleylethanolamide (OEA) which sends a message to brain that the stomach is full (Campolongo et al., 2009). In addition to appetite regulation, OEA may regulate body weight altered peripheral lipid metabolism including increased lipolysis in adipocytes and enhanced fatty acid intake in enterocytes (Yang et al., 2007; Thabuis et al., 2008). OEA mediates fat – induced satiety by engaging type – alpha peroxisome proliferator – activated receptor (PPAR-alpha) in the gut and recruiting local afferents of the vagus nerve (Lo verme et al., 2005). Also, the effect of olive oil against increased body weight may be due to antioxidant effect of its phenolic substances.

The present investigation indicates that butter and cholesterol rich diet caused significant increases in total lipid, triglycerides, total cholesterol, LDL, VLDL levels. The elevated levels of lipids and lipoproteins may be attributed to the elevation of plasma free fatty acids that will lead to increased VLDL secretion by the liver involving extra acylglycerol and cholesterol output into the circulation (Murray et al., 1996). In addition, high cholesterol diets lead to the formation of cholesteryl ester enriched VLDL that are readily converted to LDL and enhance the effect of cholesterol in suppressing receptor mediated hepatic uptake of LDL and increasing plasma LDL levels. Therefore the rates of VLDL production and clearance of triacylglycerol may be altered as a result of the amount and the type of fat in the diet (Ruiz-Gutierrez et al., 1998). Meals enriched with different fats lead to the formation of VLDL particles with varying resistance to oxidation (Nielsen et al., 2000).

Several studies have consistently demonstrated that dietary saturated fat increases plasma and hepatic lipid profile. Triscari et al. (1978) demonstrated that the prolonged feeding of saturated fat increased hepatic cholesterol synthesis in rat. Trevisan et al. (1990) found that the increased consumption of butter was associated with significantly higher cholesterol. Fremont and Gozzelino (1996) reported that cholesterol, lard diets (rich in saturated fatty acids) increased serum cholesterol. Mangiapane et al. (1999) stated that feeding of hamster on a diet containing butter and cholesterol resulted in dramatic increases in LDL cholesterol. Fernandez et al. (1999) have shown that Guinea pigs exhibit higher plasma LDL cholesterol concentrations as the amount of dietary cholesterol increased. Diniz et al. (2004) found that the SFA group had higher triacylglycerol, cholesterol, low-density lipoprotein cholesterol, and atherogenic index (ratio of cholesterol to high-density lipoprotein cholesterol) relative to the olive oil group.
lipoprotein). Lin et al. (2009) stated that the abdominal fat content and abdominal fat index as well as liver lipid index, total cholesterol, LDL-C and FFA levels were significantly increased, HDL-C significantly decreased while the levels of triglycerides had no significant changes in model group (hypercholesterolemia) compared with control group.

Measurements of triglycerides are used in screening of the lipid status to detect atherosclerotic risks and in monitoring of lipid lowering measures. Elevated triglycerides levels combined with increased low density lipoprotein concentrations constitute an especially high risk for coronary heart disease. Trautwein et al. (1993) and Woollett et al. (1992) stated that saturated fat and cholesterol produce severe hypertriglyceridemia in hamster. Mangiapane et al. (1999) and Ali et al. (2000) found that cholesterol and lard diets increased triglycerides.

The observed data indicated that the butter and cholesterol rich diets resulted in decreasing high density lipoprotein levels (HDL) in the plasma of mice. These results are in agreement with Alexaki et al. (2004) who suggested that dietary cholesterol and saturated fatty acids could have an effect on atherosclerosis not only beyond their role in affecting plasma lipoproteins but also through increased production of inflammatory cytokines in the arterial wall. It was found that low levels of high density lipoprotein were associated with increased cardiovascular diseases risk, while high levels appear to confirm protection (Kannel et al., 1971). HDL particles are made in the liver and intestine and appear to facilitate the transfer of apoprotein among lipoproteins. The actual function of HDL is to remove cholesterol from peripheral tissues through the action of the plasma enzyme, lecithin: cholesterol acyl transferase (LCAT), and transport it centripetally for hepatic excretion. This process is thought to be antiatherogenic (Robert et al., 2004; Waterman et al., 2007).

The obtained data indicated that the substitution of saturated fat and cholesterol with monounsaturated fatty acids (virgin olive oil) is effective in reducing the alterations in lipid profile.

The antiatherogenic effect of virgin olive oil was predicted from previous investigations. People in the Mediterranean area of the world, who consume monounsaturated fatty acids as a primary fat source, suffer neither ill nor increase heart disease (Garg et al., 1988). Mangiapane et al. (1999) reported that although saturated fatty acids tend in general to raise plasma cholesterol concentrations, monounsaturated fatty acids from olive oil have a more or less equivalent hypocholesterolemic activity with some increase in HDL cholesterol. A decrease in hepatic cholesterol synthesis has been observed in hamster (Spady and Dietschy, 1988) and guinea pigs (Fernandez et al., 1990) fed diets high in monounsaturated fat olive oil. Reaven (1995) observed that monounsaturated fatty acid diets reduce low density lipoprotein (LDL) oxidative susceptibility in vitro. Also, Lin et al. (1992) stated that olive oil–fed animals have no significant increase in plasma cholesterol levels. Trautwein et al. (1993) demonstrated that MUFA-rich dietary fats; e.g. olive oil has hypocholesterolemic effect in hamsters when the dietary cholesterol intake is moderate. Mangas–cruz et al. (2001) and Quiles et al. (2004) found that animals fed on virgin olive oil had lower triglycerol and cholesterol values, suggesting that the possible use of that edible oil to provide a healthier aging. Dela cruz et al. (2000) found that rabbits fed on saturated fatty acids – enriched with 15% olive oil showed an improved lipid profile with decreased morphological lesions of the endothelium and vascular wall. In addition, Hargrove et al. (2001) suggested that different high MUFA sources varying in the ratio of MUFA to PUFA can be incorporated into a high MUFA diet without increasing the susceptibility of LDL to oxidation. Replacing saturated fatty acids with monounsaturated fatty acids does not appear to reduce serum HDL-cholesterol concentrations thus the best way to compensate for reduction in saturated fatty acids intake may be a replacement with monounsaturated fatty acids (Perona et al., 2009). A moderate substitution of saturated fatty acids with monounsaturated fatty acids has beneficial effects on lipid metabolism in healthy individuals (Rivellese et al., 2003). Ashton et al. (2001) found that HDL cholesterol was significantly higher on the monounsaturated rich sunflower oil diet which was expected to be associated with a decrease in CHD risk.

It was demonstrated that olive oil phenolics are powerful antioxidant both in vitro and in vivo and exert additional potent biological activities that could partially account for the observed cardio-protective effect of the Mediterranean diet (Visioli and Galli, 2007). Wiseman et al. (1996 & 2002) and Corona et al. (2009) stated that antioxidant possibly phenolic compounds which are present only in virgin olive oil, improve the antioxidant defense system in plasma by sparing the consumption of vitamin E under normal physiological circumstances. Also, olive oil may contribute to the prevention of atherosclerosis through a modulation of gene expression for endothelial leukocyte adhesion molecules (Massaro et al., 1999). Olive oil was effective in normalizing the activity of a membrane – bound enzyme responsible for sodium transport and the distribution and transbilayer membrane of erythrocyte membrane cholesterol (Muriana et al., 1997a&b). When a phenolic olive oil
extract has been incubated in vitro, a correlation between the inhibition of LDL oxidation and an increase in the concentration of tyrosol is observed (Covas et al., 2000) that together with hydroxyl tyrosol is one of the main phenolic compounds present in virgin olive oil. An antioxidant, tyrosol can protect cells against injury due to oxidation (Giovannini et al., 1999). Although tyrosol is not as potent as other antioxidants present in olive oil, its higher concentration and good bioavailability indicate that it may have an important overall effect (Miro-Casas et al., 2003). This effect may contribute significantly to the health benefits of olive oil.

The hypolipidemic effect with replacement of butter and cholesterol with sunflower oil (PUFA) has been cited in numerous studies. A shift from dietary saturated fat to polyunsaturated fat resulted in decrease of plasma LDL in normal human male (Grundy and Denk, 1990) by increasing hepatic uptake of plasma LDL. The hypocholesterolemic effect of polyunsaturated fat has been shown in hamster (Spady and Dietschy, 1985), rhesus monkey (Chang et al., 1987), in guinea pigs (Lin et al., 1994), African monkeys (Rudel et al., 1995) and rats (Jeffery et al., 1996). Wardlaw and Snook (1990) studied the effect of blending different vegetable oils on serum cholesterol levels of healthy men and they found that sunflower oil has hypocholesterolemic effect even when five eggs were consumed daily for 7 days. Insull et al. (1994) reported that VLDL, LDL total cholesterol, triglycerides were lower during sunflower oil diet treatment compared with the cholesterol and fat diet. Fremont and Gozzelino (1996) found that sunflower (rich in oleic acid) reduced VLDL cholesterol and triglycerides concentrations suggesting that oleic acid might limit the triglycerides production in sunflower fed rats. Quiles et al. (2003) stated that fatty acids profile showed a higher omega-6 polyunsaturated fatty acids proportion (linoleic acid) in sunflower oil fed animals. Nicolosi et al. (2004) stated that diets higher in polyunsaturated fat are believed to lower blood cholesterol concentrations and thus reduce atherosclerosis, greater than diets containing high amounts of saturated fats. Samaha (2005) reported that unsaturated fats have the combined benefits of lowering serum cholesterol and raising high-density lipoprotein, as well as, favourable effects on insulin resistance and inflammation. They also lower cardiovascular events in high-risk patients.

The reason for the cholesterol – lowering effect of polyunsaturated fatty acids includes the stimulation of cholesterol excretion into the intestine and the stimulation of oxidation of cholesterol to bile acids. There is other evidence that the effect is largely due to a shift in distribution of cholesterol from the plasma into the tissues because of increased catabolic rate of LDL due to up – regulation of the LDL-receptor by polyunsaturated fatty acids and down regulation by saturated acids (apo B/E receptor path way) or may be due to the presence of the series omega-6 linoleic acid in sunflower oil that may alleviate exogenous hypercholesterolemia by activating the process involved in the hepatic uptake and biliary excretion of serum cholesterol (Murray et al., 1996).

Glucose metabolism is defective in two very common metabolic diseases obesity and diabetes, which contribute to the development of a number of major medical problems including atherosclerosis , hypertension , small vessel disease , kidney disease , blindness and abnormalities in transport of lipid in blood (Delvin, 2002). The present investigation showed that butter and cholesterol diet caused hyperglycemia in rats. This hyperglycemic effect of atherogenic diet was well documented. Trevisan et al. (1990) found that consumption of butter and cholesterol was associated with higher glucose levels in men. High fat intake leading to obesity contributes to development of non-insulin-dependent diabetes mellitus; NIDDM,type 2 (Ikemoto et al.,1995; Adamopouls et al., 1996). Ikemoto et al. (1996) stated that high fat diet induced hyperglycemia and obesity in mice. Mann (2002) found that a high intake of saturated fat is an important risk factor for type 2 diabetes. In obese animals, a significant hyperglycemia was developed (Canò et al., 2008). Brøns et al. (2009) reported that high-fat over feeding increases fasting glucose levels due to increased hepatic glucose production. The increasing rates of obesity are probably the most important explanation for the increase in diabetes,also marked elevations of LDL or triglycerides can cause pancreatitis which may lead to disturbances in insulin secretion (Ritter et al., 1999). Rosa et al. (2008) stated that increased dietary fat intake generally and saturated fat specifically, will lead to impairment of insulin action and to development of metabolic syndrome.

The present data indicated that olive oil abolished the hyperglycaemic effect of the atherogenic diet, while sunflower oil incompletely restored the normal level. Several studies have been conducted to illustrate the effect of both olive oil and sunflower oil on the hyperglycaemic effect of saturated fats. Ricardo et al. (2003) found that amylase out put was significantly enhanced by sunflower oil feeding rats. However, olive oil group values showed a prolonged plateau elevation above the base line that was maintained for at least the infusion time. A diet rich in monounsaturated fat has beneficial effects on blood pressure and glucose metabolism (Anette et al., 2008). Covas et al. (2006) stated that olive oil lowers
blood glucose levels and decreases insulin requirements in persons with type 2 diabetes. Insulin resistance was significantly less in people who used olive oil (Soriguer et al., 2004). Ingestion of virgin olive oil based breakfast decreased post prandial glucose and improved sensitivity to insulin (Paniagua et al., 2007). An increase in unsaturation of the muscle membrane fatty acids is associated with improved insulin sensitivity (Landmark and Alm, 2006).

There is a close relationship between alterations of thyroid hormones status and cholesterol metabolism. To compare the levels of serum cholesterol with thyroid function, T3 and T4 were estimated. There are at least three factors in serum capable of regulating thyroid cell metabolism that is controlled by the type of fat in the diet. The data of this investigation showed that butter and cholesterol decreased the levels of thyroid hormones in mice. Bai et al. (1991) concluded that hypothyroidism is relatively common in apparently healthy people with raised plasma cholesterol concentrations. Takeuchi et al. (1995) found that the serum triiodothyronine level and the activity of Na+, K (+) -ATPase in the liver and skeletal muscle were significantly lower in the lard diet rat group than in the other diet groups. Also, the authors suggested that the intake of lard, compared with the intake of the vegetable oils, may decrease Na+, K (+) -ATPase activity in the liver and skeletal muscle by lowering serum triiodothyronine level, resulting in the promotion of body fat accumulation. Additionally, high-fat fed rats showed significantly lower total values of plasma TSH. Cano et al. (2008) stated that obesity has a circadian organization of hormone secretion. Siddhanti et al. (1990) reported that the type of dietary fat affected the production of cyclic adenosine monophosphate (cAMP) by cultured thyroid cells incubated with mouse and rat sera. The present data indicated that olive oil diets intake improved the thyroid hormones level. Sunflower oil partially improved these changes. Deshpande and Hultbert (1995) observed a significant body weight loss in hyperthyroid mice fed on the PUFA diet but not in those fed on the SFA diet.

There is an increasing evidence that the amount and type of dietary fat may modify the utilization of the trace element iron (Fe³⁺). The present data indicated that the plasma iron levels were enhanced in animals fed on butter and cholesterol diet as compared to control level. Fields and Lewis (1999) stated that beef tallow (BT) increased hepatic iron levels, which in turn was associated with increased plasma cholesterol. In contrast, when hepatic iron retention was not increased, such as by feeding a diet containing corn oil, plasma cholesterol was not elevated. Based on these data, it was suggested that nutrients that have the ability to increase hepatic iron have the potentiality to increase plasma cholesterol. Pabón and Lönnerdal (2001) suggested that saturated fats may increase iron absorption which may be achieved by changes in the fatty acid composition of the intestinal mucosa.

The data reported here showed also that olive oil diets improved the plasma iron levels, while sunflower oil diet partially improved the change in this parameter. Similar results were also recorded by other authors. Boesch-Saadatmandi et al. (2007) stated that the BT diets resulted in significantly higher iron concentrations in the liver compared to both the sunflower and control diets. Miret et al. (2003) stated that the olive oil-rich diet did not increase oxidative stress and did not alter iron metabolism. Shotton and Droke (2004) reported that diets with a higher proportion of polyunsaturated fatty acids (e.g. linoleic acid) have decreased iron absorption as compared with diets containing a higher proportion of the saturated fatty acid.

CONCLUSION:

The action of both olive oil and sunflower oil may be due to the presence of phenolic compounds, oleic acid and linoleic acid, respectively. According to the present data it can be concluded that the antioxidative action of olive oil may be attributed to the antioxidant properties of oleic acid and phenolic compounds that related to their abilities to donate electrons and to act as free radical scavengers.

Dietary factors including the substitution in the diet of monounsaturated or polyunsaturated fatty acids are most beneficial in lowering blood cholesterol. In the present investigation, a diet rich in monounsaturated fatty acids (olive oil) is more efficient than a polyunsaturated diet (sunflower oil) in relation to total cholesterol levels because of the antiatherogenic effect of HDL which is increased with this type of fat.

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