Beneficial effects of a polar fraction of garlic (*Allium sativum* Linn) oil in rats fed with two different high fat diets*

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Feeding a diet containing 20% of sesame oil (SO) or coconut oil (CNO) along with 2% cholesterol to rats for two months showed differences in their serum and tissue lipid profile and certain enzyme activities. Hyperlipidemia and related oxidative effects were more pronounced in coconut oil fed rats than those fed sesame oil. Feeding a combination of the oils (10% CNO+10% SO) lowered significantly the hyperlipidemia and certain other deleterious effects of CNO. Feeding a polar fraction of garlic oil (PFGO) prepared in the same way as for ajoene and administered at a dosage of 100 mg/kg along with each of the above oil containing diets counteracted significantly the hyperlipidemic, oxidant and also most of the other deleterious effects of the oils like raised lipid levels in serum and tissues, raised serum levels of AST and tissue levels of HMGCoA reductase and the lowered serum and tissue levels of glutathione reductase. The results support the claims that ajoene, the major polar compound of garlic oil, has very good biological action, which warrants further study.

**Keywords**: Cholesterol, Garlic oil, PFGO, SO, CNO, Hypcholesterolemia, Hypolipidemic.

The polar fractions of garlic1 and onion oils2 have a far better hypoglycemic action than the non-polar fractions. This was further confirmed by using a synthetic preparation of a sulfoxide compound3 viz; dipropyl disulphide oxide, prepared from a non-polar unreactive dipropyl disulphide. Both of them are present as a mixture in onion oil3. In garlic oil, two polar compounds, allicin (C<sub>3</sub>H<sub>5</sub>S (O)SC<sub>3</sub>H<sub>5</sub>) and its polymer form ajoene (C<sub>3</sub>H<sub>5</sub>-S(O)-CH<sub>r</sub>CH=CH-S-S-C<sub>3</sub>H<sub>5</sub>) and various non-polar polysulphides (C<sub>3</sub>H<sub>r</sub>-S<sub>n</sub>-C<sub>3</sub>H<sub>5</sub>) are present and beneficial effects of these have been reported4. Although the polar fraction of garlic oil (PFGO) was prepared according to the method of Singh et al.5 for the preparation of ajoene from garlic, it was not identified as there was no standard sample of ajoene available. The present study has been undertaken to find out whether this PFGO has any antiatherogenic effect in rats fed with two types of high fat diets i.e. sesame oil and coconut oil, both separately, and combined with cholesterol containing diets. In addition it is also attempted to find out whether by mixing the above two edible oils in equal proportions with cholesterol containing diet could show a better effect.

*Sesame oil*—Sesame (*Sesamum indicum* Linn) a traditional health food of medicinal value is known for its role in the prevention of regressive diseases such as atherosclerosis, hypertension and ageing6. The saturated fatty acids present in sesame oil are palmitic acid (8%), stearic acid (4%), arachidic acid (0.4-1%), and traces of myristic and lignoceric acids. Unsaturated fatty acids in sesame oil are oleic acid (45%), α-linolenic acid (14.2), linoleic acid (27%), and certain highly unsaturated fatty acids (1.2%). Presence of phytosterol, sesamin and sesamolin (a condensed polyphenol) and a potential antioxidant lignan have also been reported in sesame oil7. Hirose et al.8 have shown that sesamin lowered both serum and liver cholesterol level by inhibiting absorption and synthesis of cholesterol simultaneously.

*Coconut oil*—Excessive use of coconut oil is considered to be atherogenic which is due to the presence of high percentage of saturated fatty acids viz, 15% short chain fatty acids, 64% medium chain fatty acids and 12% long chain fatty acids, even though it contained unsaturated fatty acids like oleic acid (6.8%) and linoleic acid (1.8%). Short chain fatty acids have been reported9 to have cholesterol lowering action. However long chain fatty acids are partly oxidized and partly converted to cholesterol, triacylglycerol and other lipids10. According to Van Heck and Zilversmit11 plasma total cholesterol was increased more than the basal levels.
in coconut oil-cholesterol fed rabbits. Aortic cholesterol was increased and triacylglycerol (TAG) was eight times greater in liver. Therefore coconut oil has been included in the high fat cholesterol diet in order to study the counteracting effects of PFGO. As per Biju et al.13. 2% cholesterol diet increased the lipid parameters in rats and garlic oil counteracted the same. In the present study in place of garlic oil its polar fraction is being tested.

In the present experiments comparative biochemical effects of PFGO on serum and tissues of rats fed with a diet containing cholesterol and one of the above oils have been studied. In addition, it is also tested whether a combination of the above oils in the diet could ameliorate the bad effects of either of them. The work on rats was conducted after getting the ethical clearance of a committee of the institution.

Materials and Methods
Adult albino rats (Sprague Dawley strain), 6-12 months old and weighing 150-200 g were selected from the stock colonies of the animal house maintained by the Department of Biochemistry of the institute. The rats were divided into 7 groups of 6 rats each.

Preparation of PFG05—Fresh garlic (Allium sativum Linn) was cleaned, sliced and ground into a pulp in a mortar. The pulp was soaked in enough methanol in a conical flask and left overnight. Next day the mixture was filtered through an ordinary filter paper. The residue left behind was further extracted with methanol and the procedure was repeated. Both the extracts were combined. Methanol was distilled off and the oil left behind was collected. This oil was extracted with diethyl ether repeatedly (2-3 times) and only the ether soluble fraction was collected. Ether was evaporated off on a water bath and the oil left over was kept in aqueous methanol for 4 days at 25°C. It was extracted with hexane and methylene chloride (CH₂Cl₂) successively. The CH₂Cl₂ solution contained the polar fraction ajoene and the solution was evaporated off to obtain the same (yield = 1 g/kg garlic). A stock solution of it was prepared in glycerol so that 1 ml corresponds to 100 mg ajoene. This was used for feeding the rats at the required dose, (i.e. 100 mg/kg).

Diet preparation—In a pilot study it was found that 10 g feed was required per rat per day. For each group 60 g diet was prepared by mixing appropriate amounts of various constituents as shown below and made into small balls and fed daily. Feed for groups 2-7 contained 2 g cholesterol/100 g feed. Both sesame oil (SO) and coconut oil (CNO) were purchased from an oil mill at Thiruvalla, Kerala.

Group 1: normal control, 60g normal feed (Gold Mohur rat feed supplied by Brook Bond, Lipton, India Ltd).
Group 2 (CD): 2% cholesterol mixed in normal feed (2g/100g) 
Group 3 (SOD): 2% cholesterol + 78% normal feed + 20% sesame oil 
Group 4 (CNOD): do + 78% normal feed + 20% coconut oil
Group 5 (SCNOD): do + 78% normal feed + 10% SO + 10% CNO 
Group 6 (SOD+PFGO): do + 78% normal feed + 20% SO + PFGO (dose 100 mg/kg in 0.1 ml glycerol was orally fed)
Group 7 (CNOD+PFGO): do + 78% normal feed + 20% CNO + PFGO as above.

Glycerol (0.1 ml) was orally fed daily to the rats of groups 1-5 as this was the solvent required for PFGO in the remaining groups. All groups were given drinking water ad libitum. After two months feeding of the above diets the rats were sacrificed by decapitation after an overnight fasting. Their blood, liver and heart tissues were collected for analysis of various biochemical parameters. The blood was collected in clean dry centrifuge tubes by cutting the jugular vein. After proper clotting of the blood, serum was separated by centrifugation. Clear serum was used for the estimation of lipid parameters and enzyme activities.

Estimation of lipid parameters—Serum and tissue cholesterol were determined by a modified method of Zlatkis14. HDL cholesterol in serum was determined by the above method after removal of other lipoprotein fractions from serum15. Serum LDL+VLDL cholesterol was calculated by subtracting HDL cholesterol from total serum cholesterol. Triacylglycerol (TAG)16 and total lipid17 in serum and tissues were also estimated by standard methods. Whole serum and chloroform-methanol (1:1) extracts of tissues were used for this purpose.

Estimation of enzyme activities—Alanine transaminase (ALT), aspartate transaminase (AST), and the liver and heart specific enzymes in serum were determined by the methods of Horder and Rej18 and Reitman and Frankel19 respectively. Thiobarbituric acid reacting substances (TBARS) in

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serum which is a measure of lipid peroxidation, was estimated by the method of Niehaus and Samuelson. The antioxidant enzyme glutathione reductase in serum was assayed spectrophotometrically according to the method of David and Richard.

HMGCoA reductase in liver was determined by a modification of the method of Rao and Ramakrishnan. Statistical analysis was performed using one way ANOVA followed by calculation of significance at 5% level using t values for separating out significant treatment effects. The results are expressed as mean ± SD of 6 rats in each group and P<0.05 was considered statistically significant.

Results and Discussion

Serum lipids—The results on serum lipid levels are given in Table 1. There were significant differences between groups (P<0.05) for values of serum total cholesterol and LDL cholesterol. These results reflect on the comparatively lesser hypercholesterolemic effect of sesame oil than coconut oil and the beneficial effect of mixing both the oils in the diet or treatment of each oil diet fed group with PFGO.

Groups 5, 6 and 7 had significantly lower values from groups 3 or 4 as the case may be.

With respect to serum HDL cholesterol value no significant differences between the groups. The results show that coconut oil was 1.5 times more hyperlipidemic than sesame oil and on mixing both the oils the harm was lessened. Treatment with PFGO reduced the harmful effects of both the oils. The TAG values also reflect on the comparatively less hyperlipidemic effect of sesame oil than coconut oil and also on the beneficial effects of mixing these oils in diet or treatment with PFGO on single oil diet fed groups. (see Tables 1 and 2 and Figs 1 and 2 for significant differences between the groups).

Serum enzymes, AST, ALT and glutathione reductase—The activities of the above serum enzymes are represented in Table 1. There were significant differences for their activities among the groups. The results reflect that cholesterol and coconut oil in the diet significantly raised AST

Table 1—Serum lipids and certain enzyme levels in normal and high fat diet fed rats with and without treatment with polar fraction of garlic oil (PFGO).

<table>
<thead>
<tr>
<th>Groups &amp; diets</th>
<th>Gr.1 (Normal)</th>
<th>Gr.2 (CD)</th>
<th>Gr.3 (SOD)</th>
<th>Gr.4 (CNOD)</th>
<th>Gr.5 (SCNOD)</th>
<th>Gr.6 (SOD+PFGO)</th>
<th>Gr.7 (CNOD+PFGO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Total Chol.</td>
<td>80.2±3.11</td>
<td>96.1±4.02</td>
<td>144.3±8.52</td>
<td>300.22±10.50</td>
<td>240.31±9.5</td>
<td>103.22±7.5a</td>
<td>245.31±10.2</td>
</tr>
<tr>
<td>2. LDL Chol.</td>
<td>27.21±1.32</td>
<td>70.21±5.02</td>
<td>82.05±6.1</td>
<td>246.1±10.50</td>
<td>184.2±9.00</td>
<td>37.00±1.70</td>
<td>183.10±8.70</td>
</tr>
<tr>
<td>3. HDL Chol.</td>
<td>38.8±0.98</td>
<td>54.00±1.5</td>
<td>45.4±1.97</td>
<td>36.8±1.27</td>
<td>39.16±2.01b</td>
<td>55.0±4.32a</td>
<td>48.16±2.98</td>
</tr>
<tr>
<td>4. Total lipids</td>
<td>164.04±9.36</td>
<td>240.2±10.5</td>
<td>246.8±11.50</td>
<td>387.71±12.96</td>
<td>297.62±10.52</td>
<td>206.80±14.15</td>
<td>270.22±12.56</td>
</tr>
<tr>
<td>5. TAG</td>
<td>57.64±3.35</td>
<td>68.25±3.10</td>
<td>66.2±2.98a</td>
<td>88.24±4.02</td>
<td>71.02±3.98e</td>
<td>60.62±4.11a</td>
<td>72.4±3.76</td>
</tr>
<tr>
<td>Serum enzymes</td>
<td>AST (IU/L)</td>
<td>30.0±2.02</td>
<td>37.5±2.8</td>
<td>39.2±3.08</td>
<td>50.73±3.07</td>
<td>45.64±3.04a</td>
<td>33.53±2.0b</td>
</tr>
<tr>
<td></td>
<td>ALT (µ/L)</td>
<td>30.18±2.26</td>
<td>33.01±2.5</td>
<td>33.47±1.8</td>
<td>36.5±1.72e</td>
<td>35.94±1.88b</td>
<td>33.04±2.64b</td>
</tr>
<tr>
<td></td>
<td>Glutathione</td>
<td>5.4±0.32</td>
<td>3.4±0.2</td>
<td>7.2±0.49</td>
<td>4.8±0.24</td>
<td>6.4±0.38</td>
<td>9.7±0.62</td>
</tr>
</tbody>
</table>

ANOVA followed by t-test. All the values except marked as a,b,c,d are significantly different from their controls.

Level of Significance is fixed at P<0.05.

* Non-significant from normal for C.D and for other groups non-significant from C.D. as control. ** Non-significant from CNOD. * Non-significant from SOD. * Non-significant from SCNOD when concerned groups are compared. The values with "b" may be noted as unbeneficial.
activity and the sesame oil in the diet had no such effect. Further it was observed that by mixing these oils in the diet or on treatment with PFGO the stimulatory effects of cholesterol and coconut oil on AST could be counteracted. With regards to ALT the results showed that its activity was raised only by cholesterol and coconut oil diet and it is least affected by sesame oil in the diet or by PFGO treatment.

With regards to the antioxidant enzyme glutathione reductase in serum, coconut oil in the diet had an inhibitory and sesame oil a beneficial effect. By treatment with PFGO or by mixing the oils in the diet the antioxidant enzyme activity was significantly enhanced i.e these processes counteracted the harmful effects of cholesterol and coconut oil.

Heart and liver tissue lipids—Results on heart tissue lipid levels are presented in Table 2. There were significant differences among the groups. Group 4 showed the significantly highest cholesterol level than all the rest. Therefore coconut oil diet was more hypercholesterolemic than sesame oil diet. The results showed that mixing the oils in the diet is as good as treatment with PFGO on coconut oil fed group.

With respect to TAG and total lipid levels Gr. 4 had the significantly highest value than the rest, i.e. coconut oil in the diet was more hyperlipidemic than sesame oil in it. By mixing these oils or by treatment with PFGO the harmful effects of cholesterol and oils in each diet were significantly reduced.

It may be noted particularly that for Gr. 6 (SOD+PFGO) the total lipid level was found to be near normal.

Results on liver tissue lipids are also given in Table 2. There were significant differences among the groups other than Gr. 5 and 7 for cholesterol level and Gr. 3 and 7 for TAG level. Group 2 showed the significantly highest cholesterol and TAG levels among all groups. These results show that the harmful effects of cholesterol in the diet were reduced significantly by all other diet preparations. Sesame oil diet was better than coconut oil diet and on treatment with PFGO or by mixing the two oils in the diet the harmful effects of coconut oil were significantly reduced.

With respect to total lipids there were significant differences among most of the groups. Here coconut oil diet produced the maximum and PFGO treated sesame oil diet showed the minimum hyperlipidemic effect among the test groups. The combined oil diet had lesser hyperlipidemic effect than CNO diet. These results also fall in line with the merits of each diet as discussed above.

Table 2—Tissue lipid components in normal and high fat diet fed rats with and without treatment with PFGO as in Table 1

<table>
<thead>
<tr>
<th>Group &amp; diet</th>
<th>Gr.1 (Normal)</th>
<th>Gr.2 (C.D.)</th>
<th>Gr.3 (S.O.D)</th>
<th>Gr.4 (CNO)</th>
<th>Gr.5 (SCNO)</th>
<th>Gr.6 (SOD+PFGO)</th>
<th>Gr.7 (CNO+PFGO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart tissue lipids Cholesterol (mg/100 g wet tissue)</td>
<td>153.5 ±6.93</td>
<td>208.3 ±7.91</td>
<td>235.3 ±6.28</td>
<td>301.4 ±9.13</td>
<td>272.1 ±9.38</td>
<td>190.3 ±9.35</td>
<td>279.6 ±7.23</td>
</tr>
<tr>
<td>TAG (mg/100 g wet tissue)</td>
<td>62.5 ±5.96</td>
<td>73.3 ±3.78</td>
<td>76.6 ±5.5</td>
<td>95.3 ±6.77</td>
<td>83.6 ±4.63</td>
<td>64.6 ±3.71</td>
<td>81.8 ±4.07</td>
</tr>
<tr>
<td>Total lipids (mg/g wet tissue)</td>
<td>5.3 ±0.34</td>
<td>7.1 ±0.39</td>
<td>12.3 ±1.97</td>
<td>16.4 ±2.44</td>
<td>12.9 ±2.48</td>
<td>5.5 ±0.35</td>
<td>12.7 ±1.89</td>
</tr>
<tr>
<td>Liver tissue lipids Cholesterol (mg/100 g wet tissue)</td>
<td>366.1 ±10.59</td>
<td>1063.3 ±115.18</td>
<td>537.8 ±15.54</td>
<td>660.3 ±11.40</td>
<td>587.8 ±9.38</td>
<td>482.5 ±9.35</td>
<td>584.8 ±7.23</td>
</tr>
<tr>
<td>TAG (mg/100 g wet tissue)</td>
<td>392.8 ±9.52</td>
<td>610.8 ±7.36</td>
<td>481.0 ±7.07</td>
<td>534.3 ±13.21</td>
<td>509.0 ±5.93</td>
<td>409.3 ±7.25</td>
<td>487.6 ±9.44</td>
</tr>
<tr>
<td>Total lipids (mg/g wet tissue)</td>
<td>14.3 ±0.94</td>
<td>16.3 ±0.94</td>
<td>18.1 ±0.74</td>
<td>24.4 ±1.16</td>
<td>21.9 ±1.59</td>
<td>15.1 ±0.74</td>
<td>19.4 ±1.39</td>
</tr>
</tbody>
</table>

ANOVA followed by t-test. All the values except marked as a,c,d are significant as represented in Table 1. Level of Significance is fixed at P<0.05.
**TBARS in serum and tissues**—Values of lipid peroxidation products viz., TBARS in serum, liver and heart tissues are represented in Fig. 1. There were significant differences between certain groups. Groups 2, 4, 5, 6 and 7 were significantly different in their serum TBARS levels with gr. 4 showing the maximum. These results show that cholesterol with or without coconut oil in the diets raises lipid peroxidation significantly. Sesame oil containing diet had only a non-significant effect on lipid peroxidation and on mixing both the oils in the diet or on treatment with PFGO such effects of the oils were significantly reduced. A more or less similar effects of the oils and PFGO were found on lipid peroxidation in liver and heart also. Cholesterol with or without coconut oil in the diet raised lipid peroxidation significantly in both the tissues. Mixing the diet with SO or on treatment with PFGO these effects were significantly reversed. These results also illustrate the protective effects of sesame oil and PFGO against lipid peroxidation, i.e. against the oxidative effects of cholesterol and coconut oil rich diets.

**Liver HMGCoA reductase and glutathione reductase activities** (Fig. 2)—There were significant differences among most of the groups. With respect to the former enzyme activity the significantly lowest value was for Gr. 2 and highest for Gr. 4 and the results illustrate that cholesterol diet inhibited this enzyme and coconut oil diet raised it to the maximum. Sesame oil present in the combined oil diet or treatment of groups 6 and 7 with PFGO significantly inhibited the enzyme activity.

With respect to liver glutathione reductase, groups 2 and 4 showed similar and also maximum inhibitory effects. Mixing the diets with SO or treatment with PFGO reversed these effects. These results illustrate that sesame oil containing diet or treatment with PFGO enhanced this antioxidant enzyme activity in liver and the coconut oil containing diet retarded the same. With respect to the activity of heart glutathione reductase there were significant differences among some groups. Gr. 4 had the significantly lowest value, and only Gr. 6 had obtained near normal level of the enzyme activity. These results also showed that coconut oil was inhibitory in action and that sesame oil has only a non-significant inhibition. Treatment with PFGO significantly enhanced the activity of the enzyme in both the oil rich diet groups.

A perusal of the results show that CNOD was more atherogenic than SOD. The garlic principle effectively ameliorated the derangements of lipids and enzyme levels brought about by both the oil diets. Mixing of the two edible oils in the diet also corrected to a significant extent the deleterious effects of one of them i.e. coconut oil. These results also established the facts that the PFGO which is prepared in the same way as for ajoene, \((C_3H_7-S(O)-CH=CH-S-S-C_3H_3)\) from garlic, was a sufficiently good hypolipidemic and antioxidant agent. It is quite surprising that when 50% of FAs in the coconut oil

![Fig. 1—TBARS values in normal and test groups.](image-url)
are of short chain group, they cannot counteract the atherogenic nature of the long chain FAs in the other half of this oil. This may be due to the high degree of saturation (91%) in that oil. An absence of hypocholesterolemic action of sesame oil is also equally unexpected as this oil is very rich in oleic acid (45%) and ω-linoleic acid (41%). It is useful to observe that when SO was mixed with CNO and cholest erol in the diet, the deleterious effect of the latter two were significantly lowered. This may be due to the fact that even though the unsaturated FAs in SO could not exhibit a hypolipidemic effect when it was used alone, the same may be effective to exhibit it against a highly saturated oil such as coconut oil.

Studies conducted by Gargouri et al.24 showed that ajoene which is also a polar fraction of garlic oil and its precursor allicin and related trisulphides bind rapidly to the –SH group enzyme called human gastric lipase (HGL), which is involved in the digestion and absorption of dietary fats. Garlic principles are therefore found to reduce serum lipids by decreasing fat digestion and absorption. HDL cholesterol in serum decreased significantly in all the oil rich diet groups as compared to the CD control group and on treatment with PFGO such deleterious effects of the oils could be nullified, i.e. HDL level was elevated over the control for each group very significantly and it highlights the beneficial effects of this fraction of garlic oil. The atherogenicity of each oil rich diet was in the order CNOD > SCNOD > SOD. Similar deleterious effects were also shown by butter fat and beef fat25 on serum lipid profile, lipid peroxidation and serum and tissue enzyme activities. By incorporating Alliums or amla (gooseberries) along with such oil rich diets favourable results were obtained25.

While the derangements of enzyme activities for AST and glutathione reductase were mostly caused by each high fat diet, ALT was affected adversely only by CNO and SCNO combination. PFGO showed some beneficial effects on glutathione reductase and AST but it had no such effect on ALT. As the oil rich diets, significantly increased the liver HMGCoA reductase activity on one hand, the PFGO decreased the same and also the activity of AST in serum on the other hand. Both the oil rich diets and PFGO increased the activity of glutathione reductase in serum, but they acted differently in the tissues, i.e. only SOD and PFGO showed a beneficial effect.

Rajamohan26 claimed that coconut oil is harmless if it is used only minimally or if it is supplemented with fiber rich coconut kernal. Our ancestors used mostly coconut kernal more than the oil and the present day generation does just the reverse. Tamilians call SO as Idayam Nallenna (oil good for heart). Its benefits are essentially due to an ideal proportion of unsaturated to saturated FAs in the oil. i.e. 87:13. Therefore if one uses both the above oils daily in minimal quantities,
the adverse effects of one of them i.e. CNO may be curtailed. There are many fractions of garlic oil such as polar and non-polar (with or without oxygen function), and all of them are antioxidant in nature and their reactions may be represented with allicin as a typical example \((\text{R} = \text{C}_3\text{H}_7)\)

\[
2\text{RSS} \cdot (\text{O}) + e^- \rightarrow \text{RSSR}^- + \text{RS} \cdot \text{S}(\text{O}_2) \cdot \text{R}
\]

Allicin Disulphide radical Thiosulfonate

\[
\text{RSSR}^- \rightarrow \text{RS}^- + \text{RS}^-
\]

Free radical Sulfide anion

\[
\text{RS}^- + \text{OH}^+ \rightarrow \text{RS}^+ + \text{OH}^- \text{ free radical repair}
\]

\[
\text{RSSR}^- + \text{OH}^+ \rightarrow \text{RSOH} + \text{RS}^-
\]

(Disulphides)

\[
\text{RS}^- + \text{RS}^+ \rightarrow \text{RSSR}^- \text{ (Termination)}
\]

Free radicals are responsible for ageing, diabetes, cancer, cataract and CHD. Ajoene is the most effective antioxidant and anticanicogenic principle. Allicin and ajoene are known to interact with -SH group enzymes/comounds like HMGCoA reductase and inactivate them. All the above said properties of garlic principles may account for their biologic activity. The present findings corroborate the beneficial effects of SO and PFGO (possibly ajoene) on lipid metabolism and certain enzymes as claimed by others. Further, these results demonstrate that coconut oil at 20% level of the diet is harmful and a combined diet with 10% CNO and 10% SO may be more beneficial than a whole saturated oil diet.

We strongly advocate the use of garlic/PFGO or aged garlic extracts (a good source of ajoene) in order to avoid the bad effects of high fat diets. At present W.H.O. promotes the use of fresh garlic.

References


AUGUSTI et al.: BENEFICIAL EFFECTS OF A POLAR FRACTION OF GARLIC


