Protective Effect of Natural Honey, Urtica dioica and Their Mixture against Oxidative Stress Caused by Chronic Ethanol Consumption.

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Abstract

**Background:** There is increasing implicating oxidative stress in the pathogenesis of chronic pancreatitis. The aim of this study is to investigate affect alcohol addiction and role of some protecting agent.

**Material and methods:** Forty eight rats (*Rattus norvigicus*) were divided into 8 groups. Honey (2.5 g /kg b.w), *Urtica dioica* (250 mg/kg) and Alcohol orally administered at dose (20% exceeds by 2.5% weekly).

**Results:** Ethanol feeding results in increasing serum glucose, total lipids, cholesterol, Low Density Lipoprotein (LDL), triglycerides, urea, liver Glucose-6-Phosphatase (G6Pase), pancreas and liver Malondialdehyde (MDA), Protein Carbonyl (PC). While a decrease were noted in serum insulin, High Density Lipoprotein (HDL), total Protein, Na, K, Ca, Mg, Cu, liver glycogen, pancreas and liver Glucose-6-Phosphate Dehydrogenase (G6PD), Glutathione-S-Transferase (GST), Reduced Glutathione (GSH), Catalase (CAT), Superoxide Dismutase (SOD).

**Conclusion:** Administration of honey, urtica or both with alcohol prevent to great extent the lesions caused by only chronic alcohol administration. Consequently, honey and urtica administration are useful to minimize the hazardous effects resulting from ethanol abuse.

**Key Words:** Alcohol- Antioxidant- Honey- Urtica- Oxidative stress.

Introduction

It is well known that, chronic ethanol administration results in oxidative stress. Generally, the conditions affecting cardio-vascular, gastro-intestinal and the central nervous systems follow the severity of more chronic heavy alcohol use related to more significant morbidity (Meera *et al.*, 2001).

The physiological functionality of foods has received much attention, due to the increasing interest in human health. Besides carbohydrates which are the major constituents (70–80%), honey contains, in low amounts, various substances such as organic acids, proteins, amino acids, vitamins, enzymes, minerals and different other molecules [pigments, flavonoids, antibacterial factors, etc.] (White, 1979).

*Urtica dioica* herbs are used against liver insufficiency (Yesilada *et al.*, 1993) and are used to treat stomachache in Turkish folk medicine (Yesilada *et al.*, 2001).

The aim of the present work is to evaluate the role of either honey, Urtica or both on the oxidative stress caused by ethanol abuse.

**Material And Methods**

**Male Albino Rats** (*Rattus norvigicus*) (48 rats) weighing about 100-120 g were used in these experiments.

**Animals were divided into 8 groups:**
1- Rats served as control
2- Rats treated with honey (2.5 g /kg b.w, orally)
3- Rats treated with *Urtica dioica* (250 mg/kg, orally)
4- Rats treated with honey and *Urtica dioica*
5- Rats treated with alcohol (20% exceeds by 2.5% weekly, orally)
6- Rats treated with alcohol and honey
7- Rats treated with alcohol and Urtica dioica
8- Rats treated with alcohol, honey and Urtica dioica

Experimental period extended to 8 weeks after that animal were sacrificed. Serum, part of pancreas & liver were collected for the following estimation.

Serum glucose (Trinder, 1969), insulin (Yallow and Berson, 1959), total lipid (Frings et al., 1972), total cholesterol (Young, 2001), HDL$_C$ (Grove et al., 1979), LDL$_C$, triglycerides (Fossati and Prencipe, 1982), urea (Palton and Crouch 1977) and total protein (Henry 1964), Na, K, Ca (ion selective electrode method using ISE AVL 988-3), Mg (Bohoun, 1962), Cu (Abe et al., 1989), liver glycogen content (Nicholas et al., 1956), liver G6Pase (Rossetti et al., 1993), as well as pancreas & liver G6PD (Chan et al., 1965), MDA (Ohkawa et al., 1982), PC (Smith et al., 1991), GST (Habig et al., 1974), GSH (Prins and Loose, 1969), CAT (Bock et al., 1980) and SOD (Niskikimi et al., 1972).

Statistical analysis was done using SPSS.

Results

As regarding serum glucose and glucose-6-phosphatase, rats treated with alcohol alone or all other treatments showed highly significant increase (p<0.001) in comparison with control rat [except rats treated with alcohol, honey & Urtica they recorded non significant change in liver G6Pase] (table 1). Meanwhile, positive control rats treated with honey and Urtica & honey showed significant decrease (p<0.05 & p<0.001) in liver G6Pase respectively. On the other hand, positive control group showed significant increase in serum insulin, liver glycogen, pancreas & liver G6PD. While, a dramatically drop was recorded in these parameters in alcohol group or alcohol group treated with honey, Urtica or both when compared with control group.

As regarding serum total lipids, cholesterol, LDL, triglycerides and urea, rats treated with alcohol alone or other treatments, showed highly significant increase (p<0.001) in comparison with control rat [except rats treated with alcohol, honey & Urtica, they recorded non significant change in total lipids, cholesterol and triglycerides] (table 2). On the other hand, positive control group treated with honey and Urtica & honey showed significant increase in serum HDL and total protein. While, in these parameters reduced in alcohol group or alcohol group treated with honey, Urtica when compared with control group.

Table (3) represent pancreas & liver MDA and PC, It is clear that rats treated with alcohol alone or with other treatments showed highly significant increase (p<0.001) in comparison with control rat (except rats treated with alcohol, honey & Urtica that recorded non significant change in liver PC). Meanwhile, positive control group treated with honey and Urtica & honey showed significant decrease in this parameter. On the other hand, positive control group treated with honey and Urtica & honey showed significant increase in pancreas & liver GST, GSH, CAT and SOD. While, a dramatically drop was recorded in these parameters in alcohol group or alcohol group treated with Urtica or honey when compared with control group.

As regarding serum Na, K, Ca, Mg and Cu, It seems that rats treated only with alcohol or other treatments showed significant decrease (p<0.001) in comparison with control rat [except rats treated with alcohol, honey & Urtica that recorded non significant change in Na and Cu] (table 4). On the other hand, positive control group showed significant increase in K, Ca, Mg and Cu when compared with control group.
Table (1) Some physiological parameters affecting carbohydrate in control and treated rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Urtica</th>
<th>Honey</th>
<th>Urtica &amp; Honey</th>
<th>Alcohol</th>
<th>Alcohol &amp; Urtica</th>
<th>Alcohol &amp; Honey</th>
<th>Alcohol, Urtica &amp; Honey</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose X ± SE mg/dL</td>
<td>92.05±2.6</td>
<td>86.7±2.4</td>
<td>81.7±2.6</td>
<td>76.6±1.9</td>
<td>276.7±6***</td>
<td>193.2±5***</td>
<td>157.1±3.2***</td>
<td>126.6±3.8***</td>
</tr>
<tr>
<td>Insulin X ± SE µIU/mL</td>
<td>0.99±0.01</td>
<td>1.11±0.04**</td>
<td>1.34±0.01***</td>
<td>1.45±0.02***</td>
<td>0.31±0.02***</td>
<td>0.55±0.03***</td>
<td>0.75±0.02***</td>
<td>0.86±0.02***</td>
</tr>
<tr>
<td>Liver Glycogen X ± SE mg/100g of tissue</td>
<td>18.5±0.2</td>
<td>20.7±0.5**</td>
<td>23.3±0.3***</td>
<td>25.5±0.4***</td>
<td>6.6±0.2***</td>
<td>10.7±0.3***</td>
<td>13.6±0.24***</td>
<td>16.2±0.34***</td>
</tr>
<tr>
<td>Liver G6Pase X ± SE µmol P/min/g wet tissue</td>
<td>0.39±0.01</td>
<td>0.38±0.01</td>
<td>0.28±0.01*</td>
<td>0.16±0.006***</td>
<td>1.2±0.05***</td>
<td>0.73±0.03***</td>
<td>0.54±0.02***</td>
<td>0.47±0.005</td>
</tr>
<tr>
<td>Pancreas G6PD X ± SE Unit/g wet tissue</td>
<td>32.9±0.5</td>
<td>38.1±0.5**</td>
<td>46.5±0.7***</td>
<td>58.7±1.7***</td>
<td>8.1±0.5***</td>
<td>12.8±0.7***</td>
<td>18.8±0.8***</td>
<td>26.2±0.9***</td>
</tr>
<tr>
<td>Liver G6PD X ± SE Unit/g wet tissue</td>
<td>31.6±0.5</td>
<td>37.5±0.5**</td>
<td>46.2±1.1***</td>
<td>59.1±2.1***</td>
<td>8.07±0.5***</td>
<td>14.3±0.8***</td>
<td>19.7±0.8***</td>
<td>25.8±1.1**</td>
</tr>
</tbody>
</table>

* = significant at P<0.05. ** = highly significant at P<0.01. *** = very highly significant at P<0.001. Significant relative to Control.
Table (2) Serum lipid profile and kidney function related parameter in control and treated rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Urtica</th>
<th>Honey</th>
<th>Ur&amp;Ho</th>
<th>Alcohol</th>
<th>Al&amp;Ur</th>
<th>Al&amp;Ho</th>
<th>Al,Ur&amp;Ho</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Lipids (mg/dL)</td>
<td>363.6±5</td>
<td>356.7±5</td>
<td>343.3±4.3</td>
<td>335±4.6</td>
<td>476±7.4</td>
<td>434.4±5.6</td>
<td>406±6.5</td>
<td>384.4±3.4</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>90.4±1.4</td>
<td>86.7±1.5</td>
<td>83.1±1.4</td>
<td>80.6±1.6</td>
<td>132±1.7</td>
<td>112.6±1.7</td>
<td>103.8±1.6</td>
<td>96.4±1.4</td>
</tr>
<tr>
<td>HDL (mg/dL)</td>
<td>38.1±0.5</td>
<td>38.7±0.6</td>
<td>41.3±0.7</td>
<td>44.5±0.7</td>
<td>24.2±0.5</td>
<td>29.5±0.7</td>
<td>33.4±0.9</td>
<td>36.2±0.8</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>32.5±0.6</td>
<td>28.8±0.7</td>
<td>23.3±0.6</td>
<td>18±0.4</td>
<td>79.4±1.2</td>
<td>58.7±0.9</td>
<td>47.8±0.7</td>
<td>39.1±0.7</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>99.1±1.5</td>
<td>96.1±1.4</td>
<td>92.5±1.3</td>
<td>90±1.3</td>
<td>141.2±2.5</td>
<td>121.4±1.9</td>
<td>112±1.6</td>
<td>105.2±1.5</td>
</tr>
<tr>
<td>Total Protein (g/dL)</td>
<td>7.1±0.1</td>
<td>7.3±0.08</td>
<td>7.8±0.13</td>
<td>8.3±0.2</td>
<td>3.2±0.05</td>
<td>6.3±0.07</td>
<td>6.6±0.06</td>
<td>6.8±0.08</td>
</tr>
<tr>
<td>Urea (mg/dL)</td>
<td>37.2±0.6</td>
<td>36.4±0.5</td>
<td>33.3±0.4</td>
<td>30.1±0.4</td>
<td>70.3±1.3</td>
<td>49.5±0.8</td>
<td>44.7±0.6</td>
<td>40.5±0.6</td>
</tr>
</tbody>
</table>

* = significant at P<0.05. ** = highly significant at P<0.01. *** = very highly significant at P<0.001. Significant relative to Control.
Table (3) Lipid peroxidation index, non enzymatic and enzymatic antioxidant in control and treated rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Urtica</th>
<th>Honey</th>
<th>Ur&amp;Ho</th>
<th>Alcohol</th>
<th>Al&amp;Ur</th>
<th>Al&amp;Ho</th>
<th>Al,Ur&amp;Ho</th>
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<tbody>
<tr>
<td>Pancreas MDA</td>
<td>155.7±3</td>
<td>143.1±2.3</td>
<td>119±2.1***</td>
<td>99.7±2***</td>
<td>385.5±6.8**</td>
<td>293.4±4.3***</td>
<td>239.4±4.3***</td>
<td>197.2±3.6***</td>
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<td></td>
<td>nmol/mg wet tissue</td>
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<tr>
<td>Liver MDA</td>
<td>166.1±3.5</td>
<td>156.3±3</td>
<td>142.7±2.7**</td>
<td>127±2***</td>
<td>396.7±7.7***</td>
<td>309.9±5.9***</td>
<td>247.9±4.6***</td>
<td>205.1±3.8***</td>
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<td></td>
<td>nmol/mg wet tissue</td>
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<tr>
<td>Pancreas PC</td>
<td>0.36±0.005</td>
<td>0.32±0.006</td>
<td>0.29±0.003***</td>
<td>0.21±0.003***</td>
<td>0.8±0.02***</td>
<td>0.57±0.01***</td>
<td>0.5±0.009***</td>
<td>0.43±0.007***</td>
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<td>µmol/mg wet tissue</td>
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<tr>
<td>Liver PC</td>
<td>0.37±0.009</td>
<td>0.33±0.02</td>
<td>0.3±0.004**</td>
<td>0.21±0.007***</td>
<td>0.8±0.02***</td>
<td>0.6±0.01***</td>
<td>0.51±0.02***</td>
<td>0.42±0.009</td>
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<td>µmol/mg wet tissue</td>
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<tr>
<td>Pancreas GST</td>
<td>4.1±0.1</td>
<td>4.5±0.09</td>
<td>5.4±0.1***</td>
<td>6.02±0.17***</td>
<td>1.3±0.05***</td>
<td>2.3±0.06***</td>
<td>3.2±0.07***</td>
<td>3.8±0.09</td>
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<td>µmol/g wet tissue</td>
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<tr>
<td>Liver GST</td>
<td>4.9±0.07</td>
<td>5.4±0.07†</td>
<td>6.2±0.1***</td>
<td>6.8±0.17***</td>
<td>1.6±0.03***</td>
<td>2.8±0.05***</td>
<td>3.9±0.07***</td>
<td>4.6±0.06</td>
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<td>µmol/g wet tissue</td>
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<tr>
<td>Pancreas GSH</td>
<td>1.3±0.03</td>
<td>1.45±0.02</td>
<td>1.6±0.03***</td>
<td>1.7±0.02***</td>
<td>0.44±0.03***</td>
<td>0.76±0.04***</td>
<td>0.98±0.06***</td>
<td>1.1±0.04**</td>
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<td>mg /g wet tissue</td>
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<tr>
<td>Liver GSH</td>
<td>1.25±0.03</td>
<td>1.34±0.03</td>
<td>1.5±0.02**</td>
<td>1.6±0.02***</td>
<td>0.41±0.03***</td>
<td>0.69±0.04***</td>
<td>0.88±0.06***</td>
<td>1.02±0.05**</td>
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<td>mg /g wet tissue</td>
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<tr>
<td>Pancreas CAT</td>
<td>32.9±1.5</td>
<td>38.3±1.1†</td>
<td>43.1±1.3***</td>
<td>48.1±0.7***</td>
<td>9.8±1***</td>
<td>18.3±1.3***</td>
<td>23.8±0.9***</td>
<td>28.1±1</td>
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<tr>
<td></td>
<td>µmol H2O2/Sec /g wet ti</td>
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<tr>
<td>Liver CAT</td>
<td>34.6±1.3</td>
<td>39.5±0.7†</td>
<td>45.8±0.8***</td>
<td>50.7±1.2***</td>
<td>10.1±0.6***</td>
<td>19.2±1.2***</td>
<td>26.5±1.7***</td>
<td>30.1±0.8</td>
</tr>
<tr>
<td></td>
<td>µmol H2O2/Sec /g wet ti</td>
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<tr>
<td>Pancreas SOD</td>
<td>172.2±3</td>
<td>177.2±3.4</td>
<td>182.2±4</td>
<td>188.1±4.5***</td>
<td>112.3±3***</td>
<td>146.2±2.7***</td>
<td>157.5±2***</td>
<td>166.1±3</td>
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<tr>
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<td>Unit/g wet tissue</td>
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</tr>
<tr>
<td>Liver SOD</td>
<td>169.6±0.8</td>
<td>174.9±1.1</td>
<td>180.3±0.9***</td>
<td>187.2±0.7***</td>
<td>113.2±4.5***</td>
<td>146.2±1***</td>
<td>157.8±0.7***</td>
<td>163.8±0.6</td>
</tr>
<tr>
<td></td>
<td>Unit/g wet tissue</td>
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</tr>
</tbody>
</table>

* = significant at P<0.05. ** = highly significant at P<0.01. *** = very highly significant at P<0.001. Significant relative to Control.
Table (4) Serum mineral concentration in control and treated rats.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Urtica</th>
<th>Honey</th>
<th>Ur&amp;Ho</th>
<th>Alcohol</th>
<th>Al&amp;Ur</th>
<th>Al&amp;Ho</th>
<th>Al,Ur&amp;Ho</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>144.4±3</td>
<td>147.9±3</td>
<td>151.4±3</td>
<td>154.9±3</td>
<td>96.5±2.6***</td>
<td>118.6±2***</td>
<td>128.1±1.9**</td>
<td>136.8±2.7</td>
</tr>
<tr>
<td>Mg</td>
<td>96.5±2.6***</td>
<td>118.6±2***</td>
<td>128.1±1.9**</td>
<td>136.8±2.7</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Ca</td>
<td>7±0.07</td>
<td>7.5±0.07***</td>
<td>8.4±0.04***</td>
<td>9±0.08***</td>
<td>3±0.05***</td>
<td>5±0.06***</td>
<td>5.7±0.06***</td>
<td>6±0.05***</td>
</tr>
<tr>
<td>Mg</td>
<td>1.9±0.04</td>
<td>2.1±0.03***</td>
<td>2.3±0.03***</td>
<td>2.5±0.04***</td>
<td>0.7±0.02***</td>
<td>1.2±0.01***</td>
<td>1.5±0.02***</td>
<td>1.7±0.02***</td>
</tr>
<tr>
<td>Cu</td>
<td>1.09±0.03</td>
<td>1.25±0.02***</td>
<td>1.5±0.035***</td>
<td>1.6±0.04***</td>
<td>0.3±0.01***</td>
<td>0.65±0.01***</td>
<td>0.85±0.02***</td>
<td>0.99±0.03***</td>
</tr>
</tbody>
</table>

* = significant at P<0.05. ** = highly significant at P<0.01. *** = very highly significant at P<0.001. Significant relative to Control.

Discussion

An immediate product of ethanol metabolism is acetaldehyde which is involved in a number of non-enzymatic modifications of proteins, including formation of semialdehydes, mercapto-semialdehydes and Schiff bases with consequent protein modification and inhibition of their biological functions (Morgan et al., 2002).

In the present study, alcohol administration leads to hyperglycemia in concomitant with decline in serum insulin level and decrease liver glycogen content Siler et al. (1998) found that, alcohol intake increased glycogenolysis accompanied by reduced gluconeogenesis.

These results may be attributed to decrease insulin level by chronic alcohol intake as reported by Lazarus et al. (1997).

Chronic ethanol consumption decreased the total glycogen synthesis activity which correlated closely with a loss in glycogen synthase protein (Van Horn et al., 2001) as well as depressed rate of synthesis or an increase rate of breakdown.

In addition, it is predictable that, prolonged exposure to ethanol would associated with the over production of reactive oxygen species (ROS) induce oxidative stress and hence lysis of liver glycogen (Petersen et al., 2005).

As honey rich in K⁺ content, obtained amelioration in alcohol treated group received honey, may be attributed to K⁺ content that exceed pancreatic insulin secretion where Hiatt et al. (1972) reported that, the increase of K⁺ within the physiological range increases the secretion of pancreatic insulin.

The amelioration caused in case of urtica is in accordance with previous study of (Bnouham et al., 2003) who reported that, aquous extract of Urtica dioica had a significant antihyperglycemic effect; this result may be caused in part by the reduction of intestinal glucose absorption. G-6-Pase may participate in the phosphorylation of glucose by using phosphate donor carbamoyl phosphate when the sugar concentration is elevated as in diabetes (Nordlie, 1964). Obtained hyper-glycemia indicate that G-6-Pase may have physiological role in glucose production (Bontemps et al., 1978).

The decreased G6PD activities in liver and pancreas during hyperglycemia can be explained by a reduced insulin secretion and action, or inhibition of this
Protective Effect of Natural Honey, Urtica dioica and ............

enzyme due to a phosphorylation or oxidative modification (Diaz-Flores, et al., 2006).

The increments in serum total lipid and cholesterol in the present data may be resulted from excessive lipolysis from fat depot due to catecholamine secretion and defective removal at lipid from blood by ethanol which mediated decreased lipoprotein lipase activity (Nakamura, 1994). Where, adrenergic hyperactivity is a common finding with chronic ethanol intake (Diamond, 1996), that is mainly ascribed to acetaldehyde, the intermeiated metabolite at ethanol that directly causes the release of catecholamines from the tissues (Sano et al., 1992).

Also, these results may be due to decrease the hepatic mitochondrial oxidation of fatty acids as a result of decrease NAD and high NADH, where Enomoto et al. (2002) reported that, the metabolism of ethanol into acetaldehyde by alcohol dehydrogenase and the metabolism of acetaldehyde into acetate by acetaldehyde dehydrogenase both transform NAD into NADH.

The hypercholesterolaemia obtained in this study reflect perturbation of lipid transport system by alcohol consumption affecting lipid metabolism in both hepatic and extra hepatic tissues as well as its marked toxic effect on liver function (Duhamel et al., 1984).

The obtained decline in HDLc level noted in alcohol treated group, may be due to reduced Apo B level as mentioned by Taskinen et al. (1987).

Schroder et al. (2006) Reported that, increased alcohol consumption was associated with an increment in ox-LDLc plasma concentration.

The obtained elevation of triglyc-eride in alcohol treated group agrees with Little et al. (1986) who reported that alcohol can elevate triglycerides.

The protection of honey may be due to the release of protective agents and the involvement of SH group in the protective mechanism of honey (Ali, 1995).

The enhanced protection caused by urtica may be due to their polyphenol content as reported by McDonald et al. (1998) who stated that polyphenols exert a large array of biological actions such as free radical scavenging, metal chelation and enzyme modulation which are responsible for the antioxidant activity.

The noticed lower serum total protein in alcohol treated group may be explained by nutrition insufficiency or liver damage commonly occur from alcohol, where Diamond, (1996) showed that, alcohol supplies some calories needed by the abuser, depresses appetite, cause liver damage and in hence defective protein synthesis. Also, the toxic effect of ethanol on hepatocytes may depress protein synthesis by the liver (Preedy et al., 1994).

The present results showed high serum urea level in alcohol treated group. A result which may be due to direct damage to the kidney, (Heidland et al., 1985) or indirectly affect renal function by elevating blood pressure, (Marmat et al., 1994) inducing electrolyte imbalance (Vamvakas et al., 1998) and inducing hyperuricaemia, (Yamanaka, 1996).

Obtained increase in MDA and PC may be attributed to lipid peroxidation by alcohol where chronic ethanol consumption increased hepatic lipid peroxidation (Roig et al., 2000). These results perhaps due to the chronic oxidative stress in alcoholism that can lead to oxidative modification of protein (Patel et al., 2005), such modification occur principally by formation of protein carbonyl.

These results may be due to increase formation of ROS by CYP2E1 through ethanol consumption, where Letteron et al. (1996) reported that ethanol consum-ption increases the formation of ROS by CYP2E1 which triggers lipid peroxidation in mice.

In addition, the decline in antioxidant defense system may lead to these results as it was noted by Jurczuk et al. (2004) who stated that after ethanol ingestion the antioxidant defense system in the liver is insufficient to give complete protection and thus the process of lipid peroxidation escalated.

Excess iron release from haemol-lyzed RBCs by alcohol may lead to these lipid peroxidation, where iron can stimulate lipid peroxidation by the fenton reaction, and

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also accelerates peroxidation by decomposing lipid hydroperoxides into peroxyl and alkoxyl radicals that can themselves abstract hydrogen and perpetuate the chain reaction of lipid peroxidation (Chang et al., 2002).

Amelioration caused by honey may be attributed to the involvement of SH in the protective mechanism of honey as shown by Szaba et al., (1981) and Ali (1995) who showed that SH may play a role in these ameliorative processes. The obtained amelioration by urtica or honey administration is corroborated by reduced levels of MDA and protein carboxyl level in liver and pancreas of animals received alcohol with urtica or honey or both, these results may be attributed to elimination of ROS by SOD and CAT enzymes that protect lipid peroxidation (Jurczuk et al., 2004). Glutathione has been recognized as a primary component in protecting hemoglobin, enzymes and red blood cell membranes from oxidative damage (Wintrobe et al., 1975).

The significant reduction of glutathione levels in the alcohol treated group may be explained through either increased peroxidation or increased loss from the liver, or impairment in its synthesis (Speisky et al., 1985). The data presented in Table (3) reveal that alcohol intake greatly affect GST, GSH, CAT and SOD. These results coincide with, Ali et al. (1997) who reported that, ethanol decreased level of GSH. These results may be attributed to excess generation of ROS as a consequence of induction of cytochrome P-450 II E1 by ethanol consumption that plays a major role in ethanol-induced oxidative stress (Lecomte et al., 1994). Where, oxidative stress in the cells or tissues refers to enhanced generation of ROS and/or depletion in antioxidant defense system.

Honey is known to prevent ethanol-induced depletion of GSH (Ali, 1991). Hyperosmotic solutions of honey lead to an increase of prostacyclin formation in both antral and fundic mucosa of the rat. The antioxidant capacity of Urtica may be attributed to the antioxidant actions of polyphenols (Rodrigo and Rivera, 2002). Obtained decline in serum Na & K concentrations of alcohol treated group is in accordance with Epstein, (1997) who stated that chronic alcohol consumption may experience low blood concentration of key electrolytes, impaired sodium, and fluid handling as well as it can disrupt hormonal control mechanisms that control kidney function. Also it may be due to increase sodium loss through excretion with urine as a result of exceed blood pressure as mentioned by Parekh et al. (2001).

Ethanol abuse induced magnesium deficiency; as shown in table (4) Magnesium is an obligatory factor in the enzyme reactions of GSH synthesis (Mills et al., 1986); this decline is corroborated by low GSH level in alcohol treated group. This mineral was also restored it normal level to some extent in honey or Urtica treated group.

The decreased in Cu concentration in animals receiving alcohol is in accordance with Brzoska et al. (2002). The protective effects of honey may indicate that it possess antioxidant properties (Ali et al., 1997).

In conclusion, chronic ethanol use moderately increases the susceptibility of body systems abnormalities where honey or Urtica may chair in the protection of these alterations.

References


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زيادة في مستوى الجلوكوز والدهون الكلية والكوليسترول والدهون منخفضة الكثافة و
الجليسريدات الثلاثية والبروتيا في المصل بالإضافة إلى وجود زيادة نشاط إنزيم جلوكوز-6-
فوسفاتي بالكبد وزيادة دلالات الدهون فوق المؤكسدة في الكبد والبنكرياس.

إضافة إلى حدوث نقص في مستوى الإنسولين والدهون عالية الكثافة والبروتين الكلي و
العناصر المقيسة في المصل وكذلك انخفاض معدل الجليكوجين في الكبد وانخفاض نشاط إنزيم
جلوكوز-6-فوسفات ديهيدروجينيز ومضادات الأكسدة الأنزيمي وغير الأنزيمي في الكبد والبنكرياس.

وكان تأثير تناول العسل أو البورتىكا أو كلاهما واقع ولا يغير من المعدلات المقيسة اما تناول
الcohol مع هذه المواد فأخذت تأثير محسن لهذه النتائج.

الخلاص:

أظهرت هذه الدراسة أن التناول السبئي للكحول يؤدي إلى اختلالات عديدة في اجهزة الجسم.
وان تناول عسل النحل أو البورتىكا قد يؤدي إلى خفض كثير من هذه الاختلالات بدرجة ملحوظة.
ومن ثم نوصي بتناول عسل النحل والبورتىكا لخفض الآثار الضارة الناجمة من تعاطى الكحول.