Preventive action of food seasoning spices mixture on fructose-induced lipid abnormalities

Ramu Suganthi MSc, Shanmuganathan Rajamani MSc, Mambakkam Katchapeswaran Ravichandran MSc, MPhil, PhD and Carani Venkatraman Anuradha MSc, MPhil, PhD

Department of Biochemistry, Faculty of Science, Annamalai University, Annamalai Nagar, Tamil Nadu, India
Department of Statistics, Faculty of Science, Annamalai University, Annamalai Nagar, Tamil Nadu, India

Key Words: fructose, spices, cinnamon, cloves, fenugreek, cumin, cardamom, mace, garam masala, black pepper, ginger, nutmeg, insulin resistance, glucose, lipids

Introduction
Rats fed high dosage of fructose in diet (60g/100g diet) form a useful model of the multi-metabolic syndrome or syndrome X, a clinical condition which involves a cluster of abnormalities such as insulin resistance, hyperinsulinemia, glucose intolerance, dyslipidemia and hypertension. The insulin resistance in fructose-fed rats is associated with the defects in insulin signalling pathways. The sites of fructose-induced insulin resistance are documented to be the liver, skeletal muscle and adipose tissue. The dyslipidemia observed in high fructose-fed rats include elevated triglycerides (TG), free fatty acids (FFA) and lipoprotein abnormalities. These alterations are secondary to the development of insulin resistance. Spices mixture (SM) is a food-seasoning item commonly used in Indian homes and in several oriental countries. Spices have been used traditionally as flavouring agents. Most of the spices are reported to possess antioxidant, anti-inflammatory and antitumour properties.

Recent studies have shown that the active constituents of some of these spices, in particular, cinnamon, bay leaf, clove and fenugreek seeds have insulin potentiating effects. Agents that improve insulin action could also have beneficial effects on lipid metabolism. Based on the foregoing observations, the present study was undertaken to investigate the hypolipidemic effect of food seasoning SM in high fructose-fed rats, which form an acquired model of insulin resistance. The results obtained are compared with those obtained from untreated fructose-fed rats.

Materials and methods
Materials
The spices were purchased from the local market at Chidambaram. The authenticity of the samples was identified by Dr B Vembu, Professor, Department of Botany, Annamalai University. In our formulation we have chosen to use the SM instead of individual spices owing to the wide spread use of SM commonly known as “Garam masala” in food preparation.

Materials and methods
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Correspondence address: Dr.C.V. Anuradha Reader, Department of Biochemistry, Faculty of Science, Annamalai University, Annamalai Nagar-608 002, Tamil Nadu, India
Fax: +91 04144 238343
Email: cvaradha@hotmail.com

High fructose feeding in rats induces insulin resistance, hyperinsulinemia, hyperglycemia and dyslipidemia. The present study was undertaken to determine the hypolipidemic effect of food seasoning spices mixture on fructose-fed insulin resistant rats. Male Wistar rats received a daily diet containing either 60% fructose or 60% starch. They were administered with the spices mixture at three different doses (10mg, 30mg or 50mg/day/rat) orally 15 days later. At the end of 45 days of the experimental period fructose-fed rats displayed elevated plasma glucose and insulin levels and restoration of lipid levels in plasma and tissues. The insulin potentiating action of the active principles in these spices may contribute to the hypolipidemic effect of spices mixture in high fructose-fed rats.

Original Article


Ravichandran, Shanmuganathan Rajamani, Mambakkam Katchapeswaran, Ramu Suganthi, MSc

Department of Biochemistry, Faculty of Science, Annamalai University, Annamalai Nagar, Tamil Nadu, India

Ramu Suganthi MSc, Shanmuganathan Rajamani MSc, Mambakkam Katchapeswaran Ravichandran MSc, MPhil, PhD

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Recent studies have shown that the active constituents of some of these spices, in particular, cinnamon, bay leaf, clove and fenugreek seeds have insulin potentiating effects. Agents that improve insulin action could also have beneficial effects on lipid metabolism. Based on the foregoing observations, the present study was undertaken to investigate the hypolipidemic effect of food seasoning SM in high fructose-fed rats, which form an acquired model of insulin resistance. The results obtained are compared with those obtained from untreated fructose-fed rats.
powder in an electric grinder. A suspension was prepared in tap water. Preparations were made just before administration. Animals received these preparations through oral gavage.

**Animals and experimental diet**

Adult male Wistar albino rats weighing 150-170g were obtained from the Central Animal House, Rajah Muthiah Medical College, Annamalai University, Annamalai Nagar, India. They were housed in the animal room under controlled conditions on a 12h light - 12h dark cycle. They received a standard pellet diet (Karnataka State Agro Corporation, Agro Feeds Division, Bangalore, India) and water ad libitum. The animals used in the study were cared for according to the principles and guidelines of the Institutional Animal Ethics Committee and all procedures were cleared by the committee.

After a week of acclimatization, the animals were divided into two batches. One batch received a control diet containing starch as the source of carbohydrate while the other was fed with fructose-enriched diet for 15 days. The composition of the control and fructose diets is given in Table 2. The diets were prepared fresh everyday. Treatment with SM was initiated on the 16th day and continued for the next 30 days. The following six experimental groups consisting of six rats each were maintained for a total experimental period of 45 days.

**Experimental groups**

**Group – 1 (CON)**
Control animals received control diet and tap water ad libitum for 45 days.

**Group – 2 (FRU)**
Fructose-fed rats received high fructose diet and tap water ad libitum for 45 days.

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>Control diet</th>
<th>High fructose diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn starch</td>
<td>60</td>
<td>0</td>
</tr>
<tr>
<td>Fructose</td>
<td></td>
<td>60</td>
</tr>
<tr>
<td>Casein</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Methionine</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Groundnut oil</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>10.6</td>
<td>10.6</td>
</tr>
<tr>
<td>Salt mixture†</td>
<td>3.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Vitamin mixture‡</td>
<td>0.2</td>
<td>0.2</td>
</tr>
</tbody>
</table>

The composition of mineral mix (g/kg)- MgSO₄·7H₂O, 30.5; NaCl, 65.2; KCl, 105.7; KH₂PO₄, 200.2; MgCO₃, 3.65; Mg(OH)₂, 3H₂O, 38.8; FeC₆H₅O₇·5H₂O, 40.0; CaCO₃, 512.4; KI, 0.8; NaF, 0.9; CuSO₄·5H₂O, 1.4; MnSO₄·4H₂O, 0.4 and CONH₃, 0.05. One kg of vitamin mix contained: thiamine mononitrate, 3g; riboflavin, 3g; pyridoxine HCl, 3.5g; nicotinamide, 15g; d-calcium pantothenate, 8g; folic acid, 1g; d-biotin, 0.1g; cyanocobalamin, 5mg; vitamin A acetate, 0.6g; α-tocopherol acetate, 25g and choline chloride, 10g.

Table 1. The active constituents of spices used in the preparation of spices mixture

<table>
<thead>
<tr>
<th>Spices</th>
<th>Botanical name</th>
<th>Principle active components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry ginger (rhizome)</td>
<td>Zingiber officinale</td>
<td>d-Camphene, β-phellandrene, cineole, zingiberene, citral, gingerol, shogaol, zingerone</td>
</tr>
<tr>
<td>Cardamom (seeds)</td>
<td>Elettaria cardamomum</td>
<td>1,8-Cineol, α-terpinyl acetate, limonene, γ-terpineol, α-pinene, β-pinene, geraniol</td>
</tr>
<tr>
<td>Cinnamon (bark)</td>
<td>Cinnamomum zeylanicum</td>
<td>Cinnamic aldehyde, phellandrene, pinene, linalool, carvophyllene, eugenol</td>
</tr>
<tr>
<td>Clove (buds)</td>
<td>Eugenia caryophyllatae</td>
<td>Eugenol, esters, ketones, sesquiterpenes, alcohols</td>
</tr>
<tr>
<td>Cumin (seeds)</td>
<td>Cuminum cyminum</td>
<td>Cuminaldehyde, p-cymene, limonene, cuminal ester</td>
</tr>
<tr>
<td>Black pepper (fruit)</td>
<td>Piper nigrum</td>
<td>Pipernol, chavicine, piperidine, pipertine, limonene</td>
</tr>
<tr>
<td>Bay leaf (leaf)</td>
<td>Cinnamomum tamala</td>
<td>d-α-Phellandrene, eugenol</td>
</tr>
<tr>
<td>Nutmeg (fruit)</td>
<td>Myristica fragrans</td>
<td>Myristicin, eugenol, d-pinene, d-camphene, isoeugenol, α-pinene, geraniol, safrole, myristic acid, dipentene, p-cymene</td>
</tr>
<tr>
<td>Fenugreek (seeds)</td>
<td>Trigonella foenum graecum</td>
<td>Vitexin, tricin, naringenin, quercetin &amp; tricin-7-0-beta-D-glucopyranoside, trigonelline &amp; coumarine</td>
</tr>
<tr>
<td>Mace (aril)</td>
<td>Myristica fragrans</td>
<td>Myristicin, eugenol, isoeugenol, d-camphene, d-pinene, dipentene, p-cymene, myristic acid, geraniol, safrole</td>
</tr>
</tbody>
</table>
Table 3. Initial and final body weights of control and experimental animals

<table>
<thead>
<tr>
<th>Parameters</th>
<th>CON</th>
<th>FRU</th>
<th>FRU + SM1</th>
<th>FRU + SM2</th>
<th>FRU + SM3</th>
<th>CON + SM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial weight (g)</td>
<td>153.33 ± 7.60</td>
<td>153.83 ± 3.92</td>
<td>156.50 ± 5.47</td>
<td>154.50 ± 3.61</td>
<td>152.66 ± 2.80</td>
<td>155.50 ± 6.22</td>
</tr>
<tr>
<td>Final weight (g)</td>
<td>198.50 ± 5.20</td>
<td>202.83 ± 7.49</td>
<td>202.83 ± 5.11</td>
<td>198.67 ± 7.92</td>
<td>195.00 ± 8.94</td>
<td>197.50 ± 5.24</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD, N = 6

**Group – 3 (FRU+SM1)**
Fructose-fed rats received high fructose diet and tap water ad libitum for 45 days. SM (10mg/day/rat) was administered from the 16th day of fructose feeding.

**Group – 4 (FRU+SM2)**
Fructose-fed rats received fructose diet and water ad libitum for 45 days. SM treatment (30mg/day/rat) was started from the 16th day of the experimental period.

**Group – 5 (FRU+SM3)**
Fructose-fed rats received fructose diet and water ad libitum for 45 days. SM (50mg/day/rat) was given from the 16th day of fructose feeding.

**Group – 6 (CON+SM3)**
Control animals received control diet and water ad libitum for 45 days. SM (50mg/day/rat) was initiated from the 16th day of the experimental period.

**Biochemical Analysis**
At the end of 45 days the animals were sacrificed by cervical decapitation. Fasting blood samples were collected in heparinised tubes. Plasma was separated by centrifugation at 1000xg for 10 minutes. Glucose was estimated by the formula: [insulin (µU/ml) x glucose (mmol/L)/22.5].

**Statistical Analysis**
Values are expressed as mean ± SD. Data within the groups were analysed using one-way analysis of variance (ANOVA) followed by Duncan’s Multiple Range Test (DMRT). A value of P < 0.05 was considered statistically significant.

**Results**

**Effect of SM on body weight**
The initial and final body weights of the rats during the experimental period of 45 days are given in Table 3. The body weights of the animals increased progressively during the experimental period. There was a trend for the fructose animals to gain more weight than other rats. However the weight gain was not significantly different as compared with those of normal rats. Administration of SM did not significantly alter the final body weights of animals.

**Effect of SM on plasma insulin and glucose levels**
Fructose-fed rats showed significantly higher glucose and insulin levels in plasma and HOMA values as compared to control rats. On administration of SM the levels were decreased and were near-normal (Fig. 1-3).
The concentration of plasma lipids in control and experimental animals are given in Figures 4, 5, 6 and 7. Fructose-fed rats had elevated levels of cholesterol in plasma as compared to control rats. Triglycerides are significantly elevated (P<0.05) in the FRU rats as compared to CON rats while FRU+SM rats showed a significant reduction (P<0.05) in cholesterol and TG levels as compared to FRU rats. FFA concentration was also elevated in FRU as compared to CON and was near normal after SM treatment. Phospholipid concentrations were elevated in the fructose groups as compared to control rats. SM supplementation significantly reduced the phospholipid level in fructose-fed rats.

Effect of SM on cholesterol in lipoprotein fractions
Concentration of plasma total cholesterol and that in lipoprotein fractions is given in Table 4. Significant increases in VLDL-C and LDL-C concentrations and a

**Effect of SM on plasma lipids**

The concentration of plasma lipids in control and experimental animals are given in Figures 4, 5, 6 and 7. Fructose-fed rats had elevated levels of cholesterol in plasma as compared to control rats. Triglycerides are significantly elevated (P<0.05) in the FRU rats as compared to CON rats while FRU+SM rats showed a significant reduction (P<0.05) in cholesterol and TG levels as compared to FRU rats. FFA concentration was also elevated in FRU as compared to CON and was near normal after SM treatment. Phospholipid concentrations were elevated in the fructose groups as compared to control rats. SM supplementation significantly reduced the phospholipid level in fructose-fed rats.
Table 4. Distribution of cholesterol in the lipoprotein fractions of control and experimental animals (values expressed as mg/dl)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>CON</th>
<th>FRU</th>
<th>FRU + SM1</th>
<th>FRU + SM2</th>
<th>FRU + SM3</th>
<th>CON + SM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>73.06±2.25&lt;sup&gt;a&lt;/sup&gt;</td>
<td>76.63±2.48&lt;sup&gt;b&lt;/sup&gt;</td>
<td>73.86±1.37&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.79±2.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.64±1.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>73.59±2.57&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>35.53±2.44&lt;sup&gt;a&lt;/sup&gt;</td>
<td>30.55±1.54&lt;sup&gt;b&lt;/sup&gt;</td>
<td>32.54±1.87&lt;sup&gt;c&lt;/sup&gt;</td>
<td>35.22±1.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>35.47±1.87&lt;sup&gt;a&lt;/sup&gt;</td>
<td>35.78±1.77&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>27.24±2.48&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>33.81±1.42&lt;sup&gt;b&lt;/sup&gt;</td>
<td>28.98±1.61&lt;sup&gt;c&lt;/sup&gt;</td>
<td>27.71±2.28&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>27.52±0.62&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>25.45±2.18&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>VLDL-cholesterol</td>
<td>11.28±0.75&lt;sup&gt;a&lt;/sup&gt;</td>
<td>13.19±0.38&lt;sup&gt;b&lt;/sup&gt;</td>
<td>12.38±0.25&lt;sup&gt;c&lt;/sup&gt;</td>
<td>11.68±0.75&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.15±0.61&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.95±0.55&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD, n = 6 Values not sharing common superscript letter differ significantly from each other at P<0.05 (DMRT)

Table 5. Concentrations of lipids in liver of control and experimental animals. Values are expressed as mg/g wet

<table>
<thead>
<tr>
<th>Parameters</th>
<th>CON</th>
<th>FRU</th>
<th>FRU + SM1</th>
<th>FRU + SM2</th>
<th>FRU + SM3</th>
<th>CON + SM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>4.73±0.14&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.66±0.41&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.83±0.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.78±0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.75±0.15&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.73±0.28&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>5.13±0.22&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>5.92±0.35&lt;sup&gt;b&lt;/sup&gt;</td>
<td>5.43±0.08&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.23±0.17&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>5.16±0.06&lt;sup&gt;ac&lt;/sup&gt;</td>
<td>5.13±0.34&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Free fatty acids</td>
<td>4.33±0.25&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.44±0.71&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.61±0.27&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.55±0.43&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.41±0.20&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.35±0.19&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Phospholipid</td>
<td>18.47±1.49&lt;sup&gt;a&lt;/sup&gt;</td>
<td>15.13±1.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>17.42±0.75&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.85±0.84&lt;sup&gt;a&lt;/sup&gt;</td>
<td>18.03±0.60&lt;sup&gt;a&lt;/sup&gt;</td>
<td>18.30±1.77&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD, n = 6 Values not sharing common superscript letter differ significantly from each other at P<0.05 (DMRT)

decrease in HDL-C were observed in fructose-fed rats. In SM supplemented fructose-fed rats LDL-C and VLDL-C were lower while HDL-C was higher as compared to fructose-fed rats.

**Effect of SM on liver lipids**

Concentration of lipids in liver of control and experimental animals are given in Table 5. Cholesterol concentrations were elevated in FRU group as compared to CON rats. TG and FFA concentrations were significantly increased (P<0.05) in fructose-fed rats as compared to normal rats. SM supplementation to the fructose-fed rats resulted in significant decrease (P<0.05) in TG, cholesterol and FFA as compared to fructose-fed rats. The concentrations were restored to near normal values. The phospholipid concentration was significantly decreased (P<0.05) in fructose-fed rats as compared to that of control rats. SM administration at all three doses to fructose-fed rats brought the concentrations of lipid constituents in liver to near normal. The levels remained unaltered in control rats treated with SM.

SM was effective in controlling hyperglycemia and hyperlipidemia at all three doses tested. A dose-dependent effect was observed in parameters namely glucose, insulin, phospholipids and TG in plasma and cholesterol, FFA and TG in liver. In rest of the parameters the dose-dependent effect was not observed.

**Discussion**

Many studies have reported that fructose administration can have profound the effects on plasma and tissue lipids levels. Michaelis et al.,<sup>15</sup> described an increase in total liver lipids in rats when glucose was isocalorically substituted by either sucrose or fructose. This effect was attributed to the induction of various lipogenic enzymes in liver by fructose.

Fructose feeding may lead to hypertriglyceridemia by increasing the formation of glycerol-3-phosphate, a precursor of lipid synthesis. Hypertriglyceridemia may also arise due to defect in removal of VLDL from plasma or increased secretion of VLDL in the liver. Lipoprotein lipase is an important enzyme responsible for the hydrolysis of TG from chylomicrons and LDL. Plasma lipoprotein lipase activity was reported to be lower in high fructose-fed rats.<sup>17</sup> The elevated circulating TG concentration may be associated with impaired insulin action. Bieger et al.,<sup>18</sup> have shown that an increase in blood TG concentration can reduce the number of insulin receptors thereby reducing insulin sensitivity. A causative link between elevated circulating TG and impaired insulin action was observed in fructose-fed rats by Thorburn et al.<sup>16</sup>

Insulin has a regulatory effect of on FFA metabolism. A defect in the ability of insulin to regulate the FFA metabolism could contribute to increased FFA levels in fructose-fed rats. Elevated concentration of plasma FFA may play a key role in the pathogenesis of type 2 diabetes, by impairing peripheral glucose utilization and by promoting hepatic glucose overproduction.<sup>19</sup>

Cellular and membrane phospholipids are the major targets of damaging free radicals and therefore depletion of phospholipids in liver of high fructose-fed rats could attributed to oxidative stress.<sup>20</sup> Studies from our laboratory have shown that fructose facilitates oxidative damage in tissues.<sup>21</sup> Further a positive correlation between the levels of lipid peroxidation products and insulin resistance has been documented.<sup>22</sup>

Botanical products that show antihyperglycemic effect have a positive influence on lipid metabolism. This is due to the inter-relationship between metabolism of glucose and lipid and to the regulatory influences of insulin on both the metabolic pathways. Some spices especially fenugreek seeds,<sup>23,24</sup> clove,<sup>25</sup> cumin seeds,<sup>26</sup> cinnamon<sup>27</sup> and bay leaf<sup>2</sup> have been found to have both hypolipidemic and hypoglycemic effects.

Increase in blood glucose level associated with hyperinsulinemia in fructose-fed rats suggests impaired insulin action. This is supported by high HOMA values. Administration of SM lowered blood glucose and insulin levels and also reduced HOMA values. Administering SM to insulin resistant rats normalized the levels of lipids in
plasma and liver in the present study. The active principles may act through the insulin potentiating action.

Some of the spices like cinnamon, clove and bay leaf have been reported to have insulin-potentiating effect in vitro. Cinnamon potentiates the action of insulin on carbohydrate metabolism more than three fold. In the last decade, in vitro studies related that the cinnamon extract mimics the effect of insulin, which potentiates insulin action in isolated adipocytes. Cinnamon extract (CE) administration to high fructose diet-fed rats prevented the development of insulin resistance possibly by enhancing insulin signaling pathway in skeletal muscle. The active principle in cinnamon was identified to be methyl hydroxy chalcone polymers (MHCP). Further, Jarvill-Taylor et al., proposed that MHCP is an effective insulin mimetic which activates the pathways leading to glucose utilization in cells.

The hypoglycemic effect of fenugreek is thought to be largely due to its high content of soluble fiber, which acts to decrease the rate of gastric emptying by delaying the absorption of glucose from small intestine. Also, fiber in general (except for cellulose), enhance fecal excretion of bile acid and cholesterol, which could explain in part the hypcholesterolemic properties of fenugreek seeds. Hydroxy isocoumarin which represent 80% of the free amino acid in fenugreek seeds may posses insulin stimulating properties. Administration of fenugreek seeds to alloxan-induced diabetic rats produced significant fall in various serum lipids, especially total cholesterol in diabetic rats.

Cinnamon significantly reduced the fasting blood glucose, TG, cholesterol and LDL-C, but did not alter the mean fasting HDL-C level in type 2 diabetic individuals. The hypolipidemic effect of nutmeg in rabbits and of cumin seeds in alloxan-induced diabetic rats have been reported. Treatment of ginger juice to diabetic rats significantly reduced the serum cholesterol, TG and blood pressure in streptozotocin diabetic rats and also significantly reduced the insulin level in blood. Plate et al., reported that the SM containing piperine, ginger and cumin favourably enhanced the activity of pancreatic lipase and stimulated secretory rate of bile acids. Hypolipidemic effect of clove in rats fed high fat diet has been reported. These effects of spices could be responsible for the observed positive influence of SM on lipid metabolism.

Our findings indicate that administration of SM attenuates hyperglycemia and hyperinsulinemia and improves plasma and liver lipid concentrations in insulin resistant rats. Insulin resistance is a contributing factor for type 2 diabetes and is also shown to be a precursor for endothelial dysfunction, hypertension and dyslipidemia ultimately leading to cardiovascular disease. Increased plasma lipid concentration represents a risk factor for CHD. In fact high fructose feeding is reported to induce atherosclerotic changes in the aorta and hypertension in animals. Lowering of plasma lipid levels through diet or drug therapy is associated with a decrease in the risk of vascular disease.

The results of our study are significant considering the high prevalence of insulin resistance in the general population and the increased fructose intake as high fructose corn syrup. Three doses of spices (10, 30, 50mg/ day/rat) were administered. A clear dose response was not observed in the study. The lack of dose response indicates that the spices are effective at the low dose itself. The lower dose (10mg/day/rat) used in the study corresponds to the human consumption of 4-5g spices mixture/day. It is likely that life style changes especially modern food habits involving high refined sugar low starch and high fat content with low intake of traditional herbs, spices and other plant products could be implicated in the increased incidence of insulin resistance in India. Increasing the consumption of spices could serve as an effective support therapy in the prevention and management of insulin resistance. More research on the effect of spices on the other components of syndrome X and on the insulin-sensitizing effects of active components are certainly needed.

References


Preventive action of food seasoning spices mixture on fructose-induced lipid abnormalities
食品调料混合香辛料对果糖诱变脂质异常的预防作用

Ramu Suganathi MSc, Shanmuganathan Rajamani MSc, Mambakkam Katcheswaran Ravichandran MSc, MPhil, PhD and Carani Venkatraman Anuradha MSc, MPhil, PhD

1. Department of Biochemistry, Faculty of Science, Annamalai University, Annamalai Nagar, Tamil Nadu, India
2. Department of Statistics, Faculty of Science, Annamalai University, Annamalai Nagar, Tamil Nadu, India

给老鼠喂食大量的果糖将导致胰岛素抵抗、高胰岛素血症、高血糖症和血脂代谢紊乱。本实验用62861只果糖喂食胰岛素抵抗的大鼠来测定食品调料混合香辛料对其的降血脂作用。雄的Wistar每天喂食含60%果糖或60%淀粉, 15天后, 用三种不同剂量的混合香辛料通过嘴喂食这些大鼠（每天每只大鼠10mg, 30mg 或50mg）。实验时间45天后, 用果糖喂食的大鼠的血浆葡萄糖浓度升高, 糖尿病血脂异常甘油三酯及自由脂肪酸浓度均升高, 高密度脂蛋白胆固醇降低, 极低密度脂蛋白胆固醇升高。同样也可发现组织脂质的变化。同时用混合香辛料处理和果糖饮食一起的大鼠血浆葡萄糖和胰岛素水平都正常, 血浆和组织中的脂质水平也恢复正常。这些香辛料的胰岛素加强作用的积极原理将有助于这些混合香辛料对高剂量果糖喂食大鼠的降血脂作用。

关键词：果糖饮食 香辛料 胰岛素抵抗 葡萄糖 脂质