Effect of *Momordica charantia* on Lipid Profile and Oral Glucose Tolerance in Diabetic Rats

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In this study, the methanol extract of *Momordica charantia* fruit extract was administered to diabetic rats to assess the long-term effect of the extract on the lipid profile and the oral glucose tolerance test. Treatment for 30 days showed a significant decrease in triglyceride, low density lipoprotein and a significant increase in high density lipoprotein level. A significant effect on oral glucose tolerance was also noted. Chronic administration showed an improvement in the oral glucose tolerance curve. The effect was more pronounced when the test was done in rats fed the extract on the day of the test compared with tests done in rats which were not fed the extract on the same day. Copyright © 2004 John Wiley & Sons, Ltd.

**Keywords:** *Momordica charantia*; oral glucose tolerance; triglyceride; cholesterol; high density lipoprotein; low density lipoprotein.

INTRODUCTION

In diabetes mellitus, due to the lower availability or reduced effects of insulin, there is less transport of glucose in the body cells and therefore lipids are used much more as fuel to generate ATP. Lipids are made available from the fat depots as LDL (low density lipoprotein) and from the liver as VLDL (very low density lipoprotein) and from the intestine as chylomicrons. This results in a high level of circulating lipids in the blood and their deposition in the wall of the artery as fatty plaques (Wisse, 1991) which leads to atherosclerosis and other cardiovascular problems. HDL (high density lipoprotein) removes these deposited fats and transports them to the liver for elimination (Balgade and Subbanna, 1989). Fat oxidation during diabetes also causes the production of ketone bodies, high levels of which can not be buffered completely, which can result in acidosis and even death.

There are many botanicals reported for the management of the disease (Chaturvedi et al., 1993; Chaturvedi, 1996). *Momordica charantia* being one. The hypoglycemic and hyperglycemic effects of the plant have already been reported by many workers (Dag et al., 1990; Chaturvedi, 2001). Some antidiabetic compounds regulate the metabolic pathways during experimental diabetes (Baquer et al., 1998). The thyrogenic response of *M. charantia* has recently been reported from our laboratory (Chaturvedi and Akali, 2005). *M. charantia* is widely used as a food and as a medicine. The seeds of *M. charantia* contain peptides having antilipolytic and lipogenic activities (Ng et al., 1987). The present study aimed to further assess the effect of *M. charantia*

extract on lipid profiles and oral glucose tolerance in diabetic rats.

MATERIALS AND METHODS

**Preparation of extract.** The fruits of *M. charantia* were collected from local farmers and their authenticity was confirmed from the Herbarium Section of the Botany Department, University of Nairobi. Fruits were dried and extracted with 70% methanol. The yield was 5% (w/w) total dry weight of the fruit.

**Experimental animals.** Male albino rats of Horts Men strain of weighing approximately 250 g were used for all the experiments. They were kept on standard diet bought from Unna company, Kenya, and water *ad libitum* in the animal house of the Zoology Department. Diabetes in the rats was produced by injections of alloxan monohydrate (60 mg/kg body weight) dissolved in normal saline, for 2 consecutive days.

**Biochemical estimations.** Parameters including blood glucose, triglyceride, LDL, HDL and cholesterol were estimated by using kits bought from Human Gesellschaft Company, Germany.

**Experimental design.** Ten albino rats were used to induce diabetes mellitus and divided into two groups, each with five rats. These two groups were diabetic control (DC) and diabetic experimental (DE). Another group of five rats served as the normal control (NC). After the induction of diabetes, all rats to be used for experiment were kept in the laboratory on a normal diet for 15 days so that they could be acclimatized well with the laboratory conditions. After 15 days, extract administration began and every day DC rats and NC rats were administered distilled water orally while DE rats were
Table 1. Effect of chronic trial of *Momordica charantia* fruit extract on lipid profile

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Initial phase 0 day</th>
<th>Middle phase 15 days</th>
<th>Final phase 30 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglyceride</td>
<td>NC</td>
<td>95.23 ± 4.03</td>
<td>97.51 ± 5.11</td>
<td>96.20 ± 6.23</td>
</tr>
<tr>
<td>(mg/100 mL)</td>
<td>DC</td>
<td>170.43 ± 3.98</td>
<td>227.09 ± 6.20</td>
<td>235.31 ± 7.96</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>DE</td>
<td>163.75 ± 7.60</td>
<td>106.14 ± 2.55*</td>
<td>104.30 ± 9.30*</td>
</tr>
<tr>
<td>HDL (mg/100 mL)</td>
<td>NC</td>
<td>47.65 ± 6.21</td>
<td>44.50 ± 4.11</td>
<td>46.31 ± 5.10</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>DC</td>
<td>24.92 ± 2.98</td>
<td>21.70 ± 6.73</td>
<td>16.80 ± 6.05</td>
</tr>
<tr>
<td>LDL (mg/100 mL)</td>
<td>NC</td>
<td>16.98 ± 5.60</td>
<td>15.48 ± 4.90</td>
<td>17.86 ± 5.75</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>DC</td>
<td>34.17 ± 5.16</td>
<td>37.33 ± 5.20</td>
<td>36.12 ± 6.83</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>NC</td>
<td>79.80 ± 4.51</td>
<td>76.73 ± 7.80</td>
<td>79.82 ± 5.43</td>
</tr>
<tr>
<td>(mg/100 mL)</td>
<td>DC</td>
<td>88.91 ± 4.10</td>
<td>89.35 ± 4.40</td>
<td>92.67 ± 5.36</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>DE</td>
<td>80.41 ± 4.86</td>
<td>80.31 ± 3.11</td>
<td>81.34 ± 4.62</td>
</tr>
</tbody>
</table>

* p < 0.0001 when compared with diabetic control.
NC, normal control rats, administered 1 mL of distilled water.
DC, diabetic control rats, administered 1 mL of distilled water.
DE, diabetic rats, administered the extract (140 mg/kg body weight).

n = 5 in each group.

Figure 1. Effect of methanol extract of *M. charantia* on oral glucose tolerance test in diabetic rats (fed on extract on the day of experiment). • normal control; ■ diabetic control; ▲ diabetic group.

Administered extract (140 mg/kg body weight) dissolved in distilled water. All the rats were bled at intervals of 15 days for 30 days. Oral glucose tolerance tests were performed twice, one without feeding the extract and the other after feeding the extract at the end of the experiment. To perform OGT, the rats were fasted overnight and the next day bled at 0 h to estimate the fasting blood glucose level. After bleeding, they were fed on glucose (2 g/kg body weight) and bled at intervals of 1 h for 5 h and blood glucose levels were estimated.

RESULTS AND DISCUSSION

Effect on lipid profile

A significant reduction (p < 0.001) was observed in the level of LDL and triglyceride levels in DE rats compared with DC rats (Table 1). The level was found to approach the normal value as in NC rats. The HDL value was found to be elevated in DE rats (p < 0.001) compared with DC rats that suffered a continuous lowering in the level of HDL. A slight increment in the total cholesterol (12%) level was noticed in DC rats throughout the experiment while the values in DE rats matched the values in NC rats.

Effect on OGT

Oral glucose tolerance showed a drug dependent response (Figs 1 and 2) when performed at the end of the experiment. OGT when performed with the extract showed more improvement compared with that without the extract in terms of its height.

It is evident from these results that *M. charantia* helps the clearance of glucose, triglyceride and LDL from
cells and also potentiates lipoprotein catabolism (Arner et al., 1984). The enhanced transportation of glucose due to potentiation by insulin brings the elevated glucose level back to normal. Once the glucose is available to the cell for ATP production, mobilization of triglyceride to body cells for energy production is reduced and this might have resulted in the lowering of blood triglyceride level in DE rats compared with the level in DC rats. A high level of HDL might have also caused the lowering of triglyceride and LDL level as it has been reported to pick up LDL from the circulation and deliver them to the liver for elimination (Wissler, 1991). An increase in HDL level might be due to the high thyroxine level.

The improved OGTT curve after prolonged treatment with M. charantia might also be due to potentiation of insulin action by thyroid hormones. Further improvement in the OGTT curve was noticed when the test was performed after administration of the extract. This suggests the possibility of stimulation of T3 and T4 release by the extract.

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REFERENCES