**SUMMARY**

The present study investigated the effects of *Momordica* (M.) *charantia* on physical changes and on histomorphometry of the aorta, pulmonary trunk and left ventricle in streptozotocin-induced diabetic Wistar rats. Physical changes and body weight were monitored throughout the experimental period. At the end of the experiment, stained sections were obtained for histomorphometry. Results showed a significant ($p<0.05$) difference in the mean body weight of the control and treated groups when compared with diabetic group. Also, there was a significant increase ($p<0.05$) in myocardial thickness and tunics of the aorta and pulmonary trunk in diabetic animals compared to other groups and control. In conclusion, *M. charantia* exhibit cardiovascular protective potential possibly via its anti-atherogenic properties.

**INTRODUCTION**

Diabetes mellitus (DM) is a serious metabolic disorder with micro- and macrovascular complications that result in a significant morbidity and mortality. The increasing proportion of the aging population, consumption of calorie rich diet, obesity and sedentary lifestyle have led to a tremendous increase in the number of diabetics worldwide (1). Diabetes mellitus is one of the most important world health problems, especially in developing countries where the prevalence and incidence rates are highest. Diabetic patients are particularly prone to cardiovascular diseases including hypertension, atherosclerosis, diabetic cardiomyopathy, congestive heart failure and cardiac autonomic neuropathy (2). Coronary atherosclerosis and cardiomyopathy occur as a result of the metabolic abnormalities associated with diabetes (3).

*Momordica* (M.) *charantia* (Linn Family: Cucurbitaceae) is one of the popular herbs that grows in different regions of Nigeria. It is commonly called bitter melon, bittergourd, balsam pear. Bittergourd is known in some tribes of Nigeria as Ejirin wewe (Yoruba) Okban, Ndeme (Igbo) and Garafun (Hausa). It is a slender, climbing annual vine with long-stalked leaves and yellow, solitary male and female flowers.
borne in the leaf axils. The fruit looks like a warty gourd, usually oblong and resembling a small cucumber. The young fruit is emerald green, turning to orange-yellow when ripe. At maturity, the fruit splits into three irregular valves that curl backwards and release numerous reddish-brown or white seeds encased in scarlet arils. The Latin name *Momordica* means “to bite,” referring to the jagged edges of the leaves, which appear as if they have been bitten. All parts of the plant, including the fruit, taste very bitter (4).

Various parts of *M. charantia* such as the seed, fruit and even the whole plant have been reported to have beneficial effects in the prevention and treatment of many diseases in folkloric medicine, especially in the treatment of DM in individuals with non-insulin dependent diabetes (5,6). It has hypoglycemic properties as it significantly suppressed the rise in blood glucose concentrations in albino rats (5,7). The first clinical study into the influence of the fresh juice of bittergourd on the management of DM was performed by Akhtar in 1981. These findings suggested the intervention would effectively treat all symptoms of diabetes including polyuria, polydipsia, and polyphagia. Sarkar *et al.* (1996) and Miura *et al.* (2001) indicated that the fresh bittergourd juice caused a significant reduction in plasma glucose concentration and an improvement in the response to oral glucose load. Bitter melon contains an array of biologically active plant chemicals including triterpenes, proteins and steroids. In addition, a protein found in bitter melon, momordin, has clinically demonstrated anticancerous activity against Hodgkin’s lymphoma in animals. Other proteins in the plant, alpha- and beta-momorcharin and cucurbitacin B, have been tested for possible anticancerous effects (11). In some studies, at least three different groups of constituents found in all parts of bitter melon have clinically demonstrated hypoglycemic (blood sugar lowering) properties or other actions of potential benefit against DM (12). These chemicals that lower blood sugar include a mixture of steroidal saponins known as charantins, insulin-like peptides, and alkaloids. The hypoglycemic effect is more pronounced in the fruit of bitter melon where these chemicals are found in greater abundance. The present study investigated the effects of *M. charantia* on physical changes observed in rat models of diabetes and on the histomorphometry of the aorta, pulmonary trunk and left ventricle of the heart in streptozotocin (STZ)-induced diabetic Wistar rats and compared the effects with those of glimepiride, an oral blood glucose-lowering drug of the sulfonylurea class (13).

**MATERIALS AND METHODS**

**Animal care**

Forty healthy adult Wistar rats of both sexes, mean weight 134.4 g, were used for the experiment. The rats were bred in the animal holding of College of Health Sciences, Obafemi Awolowo University, Ile-Ife. They were maintained on standard rat pellet (Capsfeed, Ibadan, Nigeria) and water was provided *ad libitum.*

The animals were randomly assigned into five groups A, B, C, D and E of eight rats each.

- group A, control (normal rats)
- group B, experimentally induced diabetic rats administered 10% Tween 80
- group C, experimentally induced diabetic rats treated with methanolic extracts of *Momordica charantia* dissolved in10% Tween 80 for two weeks (withdrawal group)
- group D, experimentally induced diabetic rats treated with methanolic extracts of *Momordica charantia* dissolved in 10% Tween 80 for four weeks
- group E, diabetic rats treated with a standard diabetic drug (2 mg/kg of glimepiride) dissolved in 10% Tween 80 for four weeks

Physical changes were observed throughout the experimental period and body weight was also monitored. The animals received humane treatment as outlined in the Care and Management of Laboratory Animals published by the National Institute of Health (NIH, 1985).
Plant material

Mature leaves of *M. charantia* (Cucurbitaceae) were collected during the raining season from suburban villages of Ile-Ife metropolis in Osun State of Nigeria. The leaves were taken to the Herbarium at the Department of Botany, Obafemi Awolowo University, Ile-Ife to confirm identification and a voucher specimen (No. UHI 16510) was placed in the Herbarium.

Preparation of methanolic extract of *Momordica charantia*

Leaves of *M. charantia* were air dried and powdered in a waring blender. Then, 765 g of the powdered leaves were extracted in 1950 mL of absolute methanol for 72 hours with intermittent shaking and filtered. The filtrate was concentrated *in vacuo* at 35 °C using a vacuum rotary evaporator (Büchi Rotavapor R110, Switzerland). The extract was partitioned between water and dichloromethane, the dichloromethane fraction (5.94%) was oven-dried at 37 °C and stored until it was ready to be used. The aqueous portion obtained was very little. Aliquot portions of the extract were weighed and dissolved in 10% Tween 80 for use on each day of the experiment.

Induction of diabetes

Diabetes mellitus was experimentally induced in groups B, C, D and E by a single intraperitoneal injection of 65 mg/kg body weight of streptozotocin (Tocris Bioscience, UK) dissolved in 0.1M sodium citrate buffer (pH 6.3). Diabetes was confirmed in animals 48 hours after induction by determining fasting blood glucose level using a digital glucometer (Accu-chek® Advantage, Roche Diagnostic, Germany) consisting of a digital meter and test strips using blood samples obtained from the tail vein of the rats. Group A animals were given equal volume of citrate buffer used on dissolving STZ intraperitoneally.

Administration of extract and anti-diabetic drug

Methanolic extracts of the leaves of *M. charantia* (100 mg/kg) were dissolved in 10% Tween 80 and administered daily (orally) by gastric intubation to the rats in groups C and D for 2 and 4 weeks, respectively. The standard antidiabetic drug (glimepiride 2 mg/kg) was administered to group E rats for four weeks (15), while those in group B were left untreated.

Sacrifice of the animals

At the end of the experimental period, all animals were physically observed and anesthetized by chloroform inhalation. A midline incision was performed at the thoracic region. The organs were dissected out, weighed and fixed in 10% formol saline processed histologically for routine hematoxylin and eosin staining.

Histomorphometry

All histomorphometric studies were carried out on an Olympus research microscope (Leica Galen III, Germany) with a linear scale-ocular micrometer inserted into the eyepiece. The ocular micrometer was calibrated with 1-mm stage micrometer (Graticles Tonbridge, Kent, England). Histologically stained sections were used for morphometric analysis of the aorta, pulmonary trunk and left ventricle of the control and diabetic groups to estimate the thickness of each of the tunica intima, media, adventitia and myocardium.

RESULTS

Physical observation

The control group appeared presumably healthy as evident in their physical agility, which was characterized by healthy fur and pinkish eyeball without any evidence of alopecia. The feeding and fluid intakes were also appreciably normal. In the diabetic group, there was alopecia on the head, neck and some other parts of the body. Other observable physical changes included polyuria, which was
characterized by wetness of the ventral and lateral body surfaces of the animals. The animals were less active as compared with the controls, which suggested a sign of ill health. Animals that were treated with *M. charantia* therapy and glimepiride, i.e. group D and E, respectively, presented a milder physical abnormality as compared with the diabetic group. Withdrawal of the extract in group C did not cause much change in the physical appearance when compared with the group that completed the extract treatment for four weeks (Fig. 1).

**Figure 1.** One animal from each group (groups A, B, C, D and E) is shown.

Prior to the commencement of the research work (day 0), body weight of all animals in both control and experimental groups showed no significant difference ($p>0.05$). By the end of day 7, after DM induction, body weight increased significantly in animals from groups A, D and E, and decreased significantly in animals from groups B and C. By day 14 of the experiment, body weight of the rats in all groups appeared to be constant. By day 21 of the experiment, there was a significant ($p<0.05$) increase in body weight of the animals. By day 28 of the experiment (commencement of extract administration), there was a significant increase in the mean body weight of the animals in groups A, D and E compared to animals in groups B and C, which showed a decrease in the mean body weight. By the end of day 35 of the experiment, the animals in groups A, C and D significantly differed in the mean body weight when compared with the mean body weight of group B rats. The animals in group E showed a relatively constant body weight, as shown in Table 1. By day 42 of the experiment, there was a significant increase in body weight of the rats in
groups A, C, D and E, while body weight of the rats in group B reduced significantly. By day 49 of the experiment, the animals in groups A, D and E maintained a nonsignificant difference in the mean body weight, while the mean body weight in group B and C rats decreased significantly by 1.97% and 2.54%, respectively. At the end of the experimental period (day 56), there was a significant (p<0.05) decrease in the mean body weight of diabetic group (121.25±6.11) as compared with control (172.50±7.79). Withdrawal of the extract from group C rats showed a nonsignificant difference (p>0.05) in the mean body weight when compared with diabetic group. The rats in groups D and E presented a significant increase (p< 0.05) in the mean body weight when compared with the rats in groups A, B and C (Table 1).

Relative organ weight

There was a significant (p<0.05) increase in the relative heart weight of diabetic group (0.44±0.10) when compared with control group (0.31±0.08) and a nonsignificant (p>0.05) decrease in the relative pancreas weight of diabetic group (0.29±0.08) as compared with control group (0.34±0.09). However, there was a significant (p<0.05) decrease in the relative weights of the heart and pancreas in the animals that received *M. charantia* extract therapy for four weeks (group D) when compared with diabetic group (group B). The relative heart weight in group C (withdrawal group) increased significantly (p<0.05), while the relative weight of the pancreas decreased nonsignificantly (p>0.05) when compared with group E. The rats in group E treated with antidiabetic drug (glimepiride) showed a significant decrease in the relative weights of the heart and pancreas when compared with diabetic rats (Table 2).

### Table 1. The effect of *Momordica charantia* on body weight (g)

<table>
<thead>
<tr>
<th>Day</th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
<th>Group D</th>
<th>Group E</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>110.13±2.90&lt;sup&gt;a&lt;/sup&gt;</td>
<td>148.00±16.78&lt;sup&gt;b&lt;/sup&gt;</td>
<td>155.00±7.79&lt;sup&gt;b&lt;/sup&gt;</td>
<td>130.63±4.67&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>128.25±4.06&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>7</td>
<td>126.88±4.52&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>128.13±12.50&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>105.00±5.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>137.86±9.00&lt;sup&gt;b&lt;/sup&gt;</td>
<td>139.00±9.54&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>14</td>
<td>126.88±4.53&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>128.75±12.24&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>105.00±5.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>137.86±9.00&lt;sup&gt;b&lt;/sup&gt;</td>
<td>139.00±9.54&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>21</td>
<td>145.00±4.33&lt;sup&gt;a&lt;/sup&gt;</td>
<td>135.63±6.91&lt;sup&gt;a&lt;/sup&gt;</td>
<td>135.00±7.91&lt;sup&gt;a&lt;/sup&gt;</td>
<td>146.67±7.60&lt;sup&gt;b&lt;/sup&gt;</td>
<td>170.00±10.21&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>28</td>
<td>149.38±5.30&lt;sup&gt;a&lt;/sup&gt;</td>
<td>132.50±7.50&lt;sup&gt;a&lt;/sup&gt;</td>
<td>128.00±9.17&lt;sup&gt;a&lt;/sup&gt;</td>
<td>180.00±5.48&lt;sup&gt;b&lt;/sup&gt;</td>
<td>176.67±3.33&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>35</td>
<td>158.13±6.47&lt;sup&gt;a&lt;/sup&gt;</td>
<td>127.50±7.20&lt;sup&gt;a&lt;/sup&gt;</td>
<td>130.00±5.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>183.33±6.28&lt;sup&gt;c&lt;/sup&gt;</td>
<td>176.67±1.67&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>42</td>
<td>162.50±7.13&lt;sup&gt;b&lt;/sup&gt;</td>
<td>126.88±6.68&lt;sup&gt;b&lt;/sup&gt;</td>
<td>131.67±7.27&lt;sup&gt;a&lt;/sup&gt;</td>
<td>188.33±6.91&lt;sup&gt;c&lt;/sup&gt;</td>
<td>181.67±1.67&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>49</td>
<td>166.25±7.30&lt;sup&gt;c&lt;/sup&gt;</td>
<td>124.38±6.58&lt;sup&gt;a&lt;/sup&gt;</td>
<td>128.33±6.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>191.67±6.41&lt;sup&gt;c&lt;/sup&gt;</td>
<td>185.00±2.89&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>56</td>
<td>172.50±7.79&lt;sup&gt;b&lt;/sup&gt;</td>
<td>121.25±6.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>130.00±5.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>204.17±3.27&lt;sup&gt;c&lt;/sup&gt;</td>
<td>190.00±5.00&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM for body weight on days coded as 0, 7, 14, 21, 28, 35, 42, 49 and 56 in each group; a, b, c, d, ab, bc, cd, abc within column signify that the means with different letters differ significantly at p<0.05, while the means with the same letters do not differ significantly at p>0.05 (one-way ANOVA with Duncan multiple range test).

### Table 2. The effect of *Momordica charantia* on the relative organ weight (g) in streptozotocin-induced diabetic rats

<table>
<thead>
<tr>
<th></th>
<th>RWH</th>
<th>RWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>0.31±0.08&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.34±0.09&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group B</td>
<td>0.44±0.10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.29±0.08&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group C</td>
<td>0.35±0.13&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.10±0.07&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group D</td>
<td>0.21±0.08&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.16±0.06&lt;sup&gt;abc&lt;/sup&gt;</td>
</tr>
<tr>
<td>Group E</td>
<td>0.12±0.08&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.10±0.06&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM for relative weight of heart and relative weight of pancreas coded RWH and RWP, respectively, in each group; a, b, ab, abc within column signify that the means with different letters differ significantly at p<0.05, while the means with the same letters do not differ significantly at p>0.05 (one-way ANOVA with Duncan multiple range test).

Morphometry

**Aorta**

Tunica intima of the diabetic group increased significantly (p<0.05) when compared with groups A (control), C (withdrawal group), D (treated with *M. charantia* for four weeks) and E (treated with glimepiride for four weeks). However, tunica media of groups B, C, D and E showed a significant decrease (p<0.05) when compared with control group (group...
A). Tunica adventitia in the diabetic group showed a nonsignificant decrease (p>0.05) when compared with control group. There was a significant reduction in groups C, D and E as compared with diabetic and control group (Table 3).

### Pulmonary trunk

The three tunics (intima, media and adventitia) of the diabetic group increased significantly (p<0.05) when compared with groups A (control), C (withdrawal group), D (treated with *M. charantia* for four weeks) and E (treated with glimepiride for four weeks). However, the three tunics of groups A, C, D and E showed a nonsignificant difference (p>0.05) when compared (Table 4).

### Myocardium

The thickness of the myocardium in diabetic group increased significantly (p<0.05) when compared with groups A (control), C (withdrawal group), D (treated with *M. charantia* for four weeks) and E (treated with glimepiride for four weeks). However, the thickness of the myocardium in groups C, D and E showed a nonsignificant difference (p>0.05) when compared (Table 5).

### DISCUSSION

Diabetes has been reported to be associated with profound alterations in glucose transport function and microanatomy of visceral organs, leading to an increased risk of coronary heart disease (16-19). The reduced polyuria and alopecia noticed in the *M. charantia* treated group in this investigation buttressed the nontoxic and promising effects of *M. charantia*, which is in correlation with other investigators (8,20).

As the extract treatment commenced, *M. charantia* was observed to have significantly increased the mean body weight of animals in group D as compared with animals in groups B and C, which showed a decrease in their mean body weight. This observation supports previous studies such as that carried out by Shetty et al. (21) who report a weight gain in diabetic rats following treatment with *M. charantia*. Garau et al. (22) also report an improved weight after diabetic rats were treated with *M. charantia*. Other investigations on the benefit of *M. charantia* on body weight are documented (23,24).

The relative weight of the heart in group D (diabetic rats treated with *M. charantia* for four weeks) and group E (diabetic rats treated with glimepiride for four weeks) was observed not to differ significantly as

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**Table 3. The effect of *Momordica charantia* on the tunics of the aorta**

<table>
<thead>
<tr>
<th>Group</th>
<th>Tunica intima (µm)</th>
<th>Tunica media (µm)</th>
<th>Tunica adventitia (µm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.38±0.22&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.63±0.16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>B</td>
<td>0.53±0.11&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.13±0.24&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.81±0.15&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>C</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.63±0.13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.88±0.07&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>D</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.31±0.60&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.75±0.14&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>E</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.50±0.10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.56±0.06&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM for each morphometric parameter, i.e. tunica intima, media and adventitia in each group; a, b, within column signify that the means with different letters differ significantly at p<0.05, while the means with the same letters do not differ significantly at p>0.05 (one-way ANOVA with Duncan multiple range test).

**Table 4. The effect of *Momordica charantia* on the tunics of pulmonary trunk**

<table>
<thead>
<tr>
<th>Group</th>
<th>Tunica intima (µm)</th>
<th>Tunica media (µm)</th>
<th>Tunica adventitia (µm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.25±0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.88±0.07&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>B</td>
<td>0.56±0.06&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.38±0.41&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.00±0.57&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>C</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.94±0.33&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.88±0.66&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>D</td>
<td>0.31±0.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.13±0.22&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.13±0.16&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>E</td>
<td>0.25±0.00&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.94±0.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.69±0.11&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM for each parameter: tunica intima, media and adventitia in each group; a, b within column signify that the means with different letters differ significantly at p<0.05, while the means with the same letters do not differ significantly at p>0.05 (one-way ANOVA with Duncan multiple range test).

**Table 5. The effect of *Momordica charantia* on the thickness of the myocardium in the left ventricle**

<table>
<thead>
<tr>
<th>Group</th>
<th>Myocardium (µm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>176.25±12.14&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>B</td>
<td>368.75±23.11&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>C</td>
<td>221.87±10.67&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>D</td>
<td>220.62±3.87&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>E</td>
<td>230.00±6.77&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM for myocardium in each group; a, b, c, within column signify that the means with different letters differ significantly at p<0.05, while the means with the same letters do not differ significantly at p>0.05 (one-way ANOVA with Duncan multiple range test) (n=10).
compared with the control, whereas the relative heart weight increased significantly (p<0.05) in diabetic group (group B). The reason for this may be due to an increase in cholesterol deposit, as diabetes is known to be associated with hyperlipidemia (25). Following the withdrawal of the extract treatment in group C animals, the relative heart weight increased by 66.67%. This may be due to systemic depletion of *M. charantia*, thus creating an avenue for cholesterol deposit. The relative weight of the pancreas, which decreased significantly in diabetic group, may be due to a reduction in β cell mass by STZ induction. Selective destruction of beta cells can be obtained by injecting STZ or alloxan (26). Streptozotocin is a DNA-alkylating agent and alloxan is a generator of oxygen free radicals, both causing extensive DNA damage (26,27). Their selectivity is thought to be due to better uptake by beta cells. Bouwens and Rooman (26) state that intravenous administration of 100 mg/kg STZ on the day of birth in rats reduces total beta-cell mass by ~90% in 48 h. Twenty days later, <40% of the normal beta-cell mass is restored (26,28). These animals can maintain normoglycemia up to a certain body weight, but at the age of ~6 weeks they become glucose intolerant (26).

Morphometric findings in this study showed that the three tunics of the aorta and pulmonary trunk were significantly thicker in diabetic rats when compared with control group. The thickness of the tunica intima may be due to lipid build up within these vessels to form atherosclerotic plaques, which may prevent the free flow of blood. This observation is consistent with previous studies (29-31). These effects were abrogated with the administration of *M. charantia* extract and glimepiride for four weeks. The reason for the significant increase of tunica media and adventitia in diabetic group is yet to be understood. Further investigation is, however, ongoing to ascertain the cause. The thickness of the myocardium in diabetic rat may be due to blood viscosity associated with hyperglycemia, thus leading to left ventricular hypertrophy. This finding corroborates a previous study (32).

This suggests that *M. charantia* exhibits a cardiovascular protective potential, possibly *via* its antiatherogenic properties.

**REFERENCES**


