Anti-obesity Effect of PM-F2-OB, an Anti-obesity Herbal Formulation, on Rats Fed a High-Fat Diet

Moonkyu Kang, Jung-Wan Oh, Hee-Kyung Lee, Hwan-Sock Chung, Sang-Moon Lee, Changsook Kim, Hwa-Jin Lee, Dong-Won Yoon, Hyun Choi, Hongyeoul Kim, Minkyu Shin, Moochang Hong, and Hyunsu Bae*

PM-F2-OB is one of the most well-known traditional herbal medicines that are frequently used for the treatment of obesity in Korea. The anti-obesity effect of PM-F2-OB on rats fed a high-fat diet was investigated through analyses of changes in body weight, kidney fat weight, and blood biochemistry including cholesterol, free fatty acid, BUN, creatinine, HDL, LDL, phospholipids, SGOT, SGPT, total lipids, and triglycerides. The subjects in this study were divided into four groups: a normal group with a standard diet (N); a PM-F2-OB treatment group fed a standard diet (N+PM-F2-OB); a control group fed a high-fat diet (C); and a PM-F2-OB treatment group fed a high-fat diet (C+PM-F2-OB). There were no significant differences in body weight change between the N and N+PM-F2-OB treatments. Also, there was no significant difference in the amount of food intake between the C and C+PM-F2-OB treatments. These results suggest that PM-F2-OB has no significant toxicity and does not induce a dislike for that diet due to its smell or taste. Rats were administered a high-fat diet (20% w/w) for six weeks to induce obesity. The study shows that PM-F2-OB significantly prevented increases in body weight, cholesterol, LDL, and total lipids that resulted from the high-fat diet. PM-F2-OB also decreased kidney fat weight and free fatty acid, phospholipid, and triglyceride concentrations induced by the high-fat diet to level equals or below the normal diet group. It was concluded from the results that PM-F2-OB has a distinct anti-obesity effect.

Key words PM-F2-OB; traditional herbal medicine; anti-obesity effect; high-fat diet

Obesity is a medical condition involving an excess accumulation of body fat. It has become one of the fastest growing major disorders throughout the industrialized world.1—4) The main cause of obesity is an imbalance between intake and outflow of fat. Obesity leads to hypertension and diabetes, myocardial infarction, and peripheral vascular disease. Obesity therapies include reducing nutrient absorption and applying anorectic drugs, thermo-genic drugs or drugs that affect lipid mobilization and utilization. With the exception of Orlistat, a recently approved gastrointestinal lipase inhibitor, all drugs approved for the treatment of obesity are either catecholaminergic or serotonergic CNS-active (activating the sympathetic nervous system) anorectic agents. Since some of these drugs may lead to dependency, they are recommended for short-term use like amphetamine-like drugs. Upon termination of therapy with these drugs, weight is rapidly regained in many cases.5) Because of the adverse effects associated with these anti-obesity drugs, many trials have been recently conducted to find and develop new anti-obesity drugs through herbal medicines that would minimize the side effects. Numerous animal studies and clinical studies with various herbal medicines have been performed, and some studies reported significant improvements in controlling body weight without any noticeable adverse effects.5—10)

PM-F2-OB is composed of Lycii Fructus, Rehmanniae Radix, Coicis Semen, Carthami Flos, Hoelen, Angelicae Radix, Nelumbinis Semen, Radix Dioscorea and Aurantii Fructus. It is one of the most well-known traditional herbal medicines frequently used to treat obesity in Korea. Lycii Fructus is reported to have a lipolytic effect in adipocytes.13) Coicis Semen is known to have a diuretic action.14) Carthami Flos is reported to cure myocardial ischemia in dogs,15) cerebral ischemia in rats,16) while inhibiting platelet aggregation.17) Hoelen was found to have a diuretic action18) and an inhibitory effect on hemolysis.19) Angelicae Radix is known to help enhance blood circulation, decrease blood stasis20—22) and inhibit platelet aggregation.23) Nelumbinis Semen is reported to decrease blood lipids in rats induced by a high-fat diet24) and inhibit platelet aggregation.25) Radix Dioscorea is known to decrease damage in renal tubules, inflammation in the central vein, and necrosis in liver tissue.26) Aurantii Fructus is reported to reduce portal pressure in portal hypertensive rats.27) These specific functions of each component of PM-F2-OB suggest that PM-F2-OB can serve as an effective anti-obesity agent to improve bad blood circulation, adjust blood lipid profiles to normal, repair damaged of liver and kidney function, and remove the excessive accumulation of body fat induced by obesity. Based on the observations, the anti-obesity effect of PM-F2-OB in rats fed a high-fat diet was investigated through changes in body weight, kidney fat weight, and blood biochemistry.

MATERIALS AND METHODS

Preparation of PM-F2-OB All of the sprayed dried extracts of PM-F2-OB components used in this study were purchased from Sun-Ten Pharmaceutical Company (Taiwan). The prescription and ratio of each component of PM-F2-OB in the high-fat diet are indicated in Table 1.

Experimental Protocol Male Sprague–Dawley rats* To whom correspondence should be addressed. e-mail: hbae@khu.ac.kr © 2004 Pharmaceutical Society of Japan

* Purimed R&D Institute, Kyung-Hee University; b Department of Physiology, College of Oriental Medicine, Kyung-Hee University; and c Institute of Oriental Medicine, Kyung-Hee University; #1 Hoegi-Dong, Dongdaemun-Ku, Seoul 130–701, Korea. Received October 20, 2003; accepted March 17, 2004

Radix was found to have a lipolytic effect in adipocytes.13) Coicis Semen is known to have a diuretic action.14) Carthami Flos is reported to cure myocardial ischemia in dogs,15) cerebral ischemia in rats,16) while inhibiting platelet aggregation.17) Hoelen was found to have a diuretic action18) and an inhibitory effect on hemolysis.19) Angelicae Radix is known to help enhance blood circulation, decrease blood stasis20—22) and inhibit platelet aggregation.23) Nelumbinis Semen is reported to decrease blood lipids in rats induced by a high-fat diet24) and inhibit platelet aggregation.25) Radix Dioscorea is known to decrease damage in renal tubules, inflammation in the central vein, and necrosis in liver tissue.26) Aurantii Fructus is reported to reduce portal pressure in portal hypertensive rats.27) These specific functions of each component of PM-F2-OB suggest that PM-F2-OB can serve as an effective anti-obesity agent to improve bad blood circulation, adjust blood lipid profiles to normal, repair damaged of liver and kidney function, and remove the excessive accumulation of body fat induced by obesity. Based on the observations, the anti-obesity effect of PM-F2-OB in rats fed a high-fat diet was investigated through changes in body weight, kidney fat weight, and blood biochemistry.

MATERIALS AND METHODS

Preparation of PM-F2-OB All of the sprayed dried extracts of PM-F2-OB components used in this study were purchased from Sun-Ten Pharmaceutical Company (Taiwan). The prescription and ratio of each component of PM-F2-OB in the high-fat diet are indicated in Table 1.

Experimental Protocol Male Sprague–Dawley rats
(age, 85 to 95 d) weighing from 190 to 250 g were supplied by Taconic Korea (Taconic Korea, Seoul, Korea). The rats were housed and allowed free access to feed and tap water under strictly controlled and pathogen free conditions (room temperature: 23 ± 1 °C, relative humidity: 50 ± 10%, light cycle: 07:00—19:00). The rats were fed a standard rodent pellet chow and acclimatized to their environment for 2 weeks before commencement of the experiments. Next, the rats were randomly divided into 4 groups (n = 8); normal, control, and a PM-F2-OB treatment group for each group. The groups were fed a standard rodent pellet chow, a standard rodent pellet chow with PM-F2-OB, a high-fat diet (beef tallow fat 20% wt/wt, Purina Co. U.S.A.), or a high-fat diet supplemented with PM-F2-OB, respectively, for 6 weeks. Diets supplemented with PM-F2-OB were prepared by mixing the powdered normal chow or high-fat chow with each component of PM-F2-OB shown in Table 1. The four diets (normal diet plus PM-F2-OB, high-fat diet and high-fat diet plus PM-F2-OB) were prepared as follows. The powdered normal chow or high-fat chow or those supplemented with herbal medicines were mixed with water, formed into a shape similar to that of normal chow and dried for at least 24 h at 100 °C prior to feeding. The animals were weighed at the start of the experiment and then every week thereafter. Blood samples were collected by cardiac puncture at the time of inspection, and the resulting serum was stored at −20 °C until analysis was complete. The concentration of serum glucose was assessed using a hexokinase kit (Bayer, U.S.A.) from the serum of each treatment group.

**Statistical Analysis** The results are presented as the mean ± S.E.M. Statistical significance was compared between each treatment group and the control group by ANOVA followed by a post-hoc test (S-N-K test). Results with a p < 0.05 were considered statistically significant.

**RESULTS**

**Changes in Food Intake in High-Fat Diet and High-Fat Diet Plus PM-F2-OB**

Whether or not PM-F2-OB in the high-fat diet influenced food intake in rats compared to the high-fat diet only was investigated by the measurement of food intake per rat body weight per day, following assessment of that for last five consecutive days. This is very important because the addition of PM-F2-OB to a high-fat diet can cause decreased food intake, possibly induced by its smell or taste. In this case, the anti-weight gain effect of PM-F2-OB in the high-fat diet may be false because the effect can be attributed to reduced food intake induced by the smell or taste of the treatment. There was no difference in change of food intake between the high-fat diet and high-fat diet plus PM-F2-OB treatment (see Table 2). This result suggests that the anti-weight gain effect of PM-F2-OB in the high-fat diet was not caused by a refusal to ingest the feed.

**Inhibitory Effect of PM-F2-OM on Body Weight Gains and Kidney Fat Weight Gain Induced by the High-Fat Diet** A gain in body weight is a common index of obesity. High-fat feeding resulted in approximately a 9% increase in body weight for a period of 6 weeks (n = 8, p < 0.005, see Table 3). Significant changes in body weight from week 1 to week 6 were detected between the control and PM-F2-OB treatment groups (n = 8, p < 0.005, see Table 3). This result indicates that PM-F2-OB treatment significantly reduced the increase in body weight induced by the high-fat diet, a clear sign of an anti-obesity effect.

Gains in kidney fat weight can be also used as an index of obesity. As such, kidney fat weight was measured in the normal, control and PM-F2-OB treatment groups in this study. High-fat feeding resulted in an increase in kidney fat weight by approximately 26% (normal diet-fed rats (N), 1.913 ± 0.205 g, n = 8; high-fat diet-fed rats (C), 2.599 ± 0.278 g, n = 8).

---

**Table 1.** Percentages (%, w/w) and Amounts of Each Herbal Medicine in PM-F2-OB and PM-F2-OB in a 20% High-Fat Diet

<table>
<thead>
<tr>
<th>Herbal medicine</th>
<th>Percentage (%, w/w)</th>
<th>Percentage (%, w/w) of PM-F2-OB ingredient in high-fat diet chow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lycii Fructus</td>
<td>15%</td>
<td>0.33%</td>
</tr>
<tr>
<td>Rehmanniae Radix</td>
<td>15%</td>
<td>0.33%</td>
</tr>
<tr>
<td>Cicis Semen</td>
<td>22.7%</td>
<td>0.50%</td>
</tr>
<tr>
<td>Carthami Flos</td>
<td>11.8%</td>
<td>0.26%</td>
</tr>
<tr>
<td>Hoelen</td>
<td>5.9%</td>
<td>0.13%</td>
</tr>
<tr>
<td>Angelicae Radix</td>
<td>11.9%</td>
<td>0.26%</td>
</tr>
<tr>
<td>Nelumbinis Semen</td>
<td>5.9%</td>
<td>0.13%</td>
</tr>
<tr>
<td>Radix Dioscorea</td>
<td>5.9%</td>
<td>0.13%</td>
</tr>
<tr>
<td>Aurantri Fructus</td>
<td>5.9%</td>
<td>0.13%</td>
</tr>
<tr>
<td>Total</td>
<td>100%</td>
<td>2.2%/100% high-fat diet</td>
</tr>
</tbody>
</table>

**Table 2.** Changes in Food Intake between High-Fat Diet and High-Fat Diet Plus PM-F2-OB

<table>
<thead>
<tr>
<th>Diet</th>
<th>Food intake (g/kg/d)</th>
<th>High-fat diet (n=8)</th>
<th>High-fat diet plus PM-F2-OB (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 d</td>
<td>2 d</td>
</tr>
<tr>
<td></td>
<td></td>
<td>49.4 ± 1.072</td>
<td>51.4 ± 2.052</td>
</tr>
<tr>
<td></td>
<td></td>
<td>45 ± 3.453</td>
<td>52.2 ± 3.227</td>
</tr>
</tbody>
</table>

Rats were administered PM-F2-OB in the form of reconstituted high-fat chow for 6 weeks. Values indicate means ± S.E.M. of 8 rats.
The serum concentrations of total cholesterol (A), LDL-cholesterol (B) and HDL-cholesterol (C) were measured in the normal, control and PM-F2-OB treatment group rats using multifunctional biochemistry analyzers to detect an anti-obesity effect. Each bar presents the mean±S.E.M. from eight rats per group and three groups according to treatment: a normal group fed a standard rat diet (N), a control group fed a high-fat diet (C) and a PM-F2-OB treatment group fed a high-fat diet (PM-F2-OB). *Significantly different from control group (p<0.05), **significantly different from control group (p<0.01), ***significantly different from control group (p<0.001) based on ANOVA followed by a post-hoc test, the S-N-K test.

Table 3. Inhibitory Effect of PM-F2-OB on Body Weight Gain and Kidney Fat Weight Gain Induced by the High-Fat Diet

<table>
<thead>
<tr>
<th>Body weight (g)</th>
<th>Chow-fed diet (n=8)</th>
<th>High-fat diet (n=8)</th>
<th>High-fat diet plus PM-F2-OB treatment (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start (0 weeks)</td>
<td>289.67±2.965</td>
<td>297.2±2.455</td>
<td>288.2±2.489</td>
</tr>
<tr>
<td>1 week</td>
<td>316.9±3.19a</td>
<td>331.3±4.48</td>
<td>309.3±4.55a</td>
</tr>
<tr>
<td>2 weeks</td>
<td>358.4±4.27b</td>
<td>388.4±6.34</td>
<td>356.6±6.42b</td>
</tr>
<tr>
<td>3 weeks</td>
<td>382.1±4.35c</td>
<td>418.1±6.43</td>
<td>382.9±4.96c</td>
</tr>
<tr>
<td>4 weeks</td>
<td>406.4±4.71c</td>
<td>441.3±8.1</td>
<td>406.3±2.95c</td>
</tr>
<tr>
<td>5 weeks</td>
<td>415.3±4.64c</td>
<td>455±8.6</td>
<td>422.9±1.54c</td>
</tr>
<tr>
<td>6 weeks</td>
<td>434.3±5.22c</td>
<td>478±10.22</td>
<td>438.3±2.56c</td>
</tr>
<tr>
<td>Kidney fat weight (g)</td>
<td>1.913±0.205</td>
<td>2.599±0.278</td>
<td>1.875±0.170</td>
</tr>
</tbody>
</table>

Rats were administered PM-F2-OB in the form of reconstituted high-fat chow for 6 weeks. Values indicate means±S.E.M. of 7 or 8 rats. a) p<0.05 vs chow-fed diet; significantly different from the chow-fed control group (N); b) p<0.05; c) p<0.005; d) p<0.001 vs high-fat diet group.

n=8, see Table 3). However, PM-F2-OB reduced the observed gains in kidney fat weight induced by the high-fat diet by approximately 28% (high-fat diet-fed rats (C), 2.599±0.278 g, n=8; PM-F2-OB treatment rats (PM-F2-OB), 1.875±0.170 g, n=8, see Table 3).

No Effect of PM-F2-OM on Body Weight Gain in Normal-Chow Diet It is widely known that a common toxicity associated many drugs is the inhibition of normal body weight gain, resulting in significant body weight loss. It was thus investigated whether PM-F2-OM had any effects on body weight gain in normal diet group to evaluate the toxicity of PM-F2-OB. A significant change in body weights from 1 week was only detected between the normal diet and PM-F2-OB treatment groups (n=7 or 8, p<0.05, see Table 4). Such a change might be due to adaptation by the rats to PM-F2-OB in their body. However, no significant changes in body weight in 2 weeks to 6 weeks were detected between the normal diet and PM-F2-OB treatment groups (see Table 4). These results together suggest that PM-F2-OB treatment did not significantly reduce the increment of body weight gain induced by chow-fed diet, a clear sign there was no toxicity.

Inhibitory Effect of PM-F2-OM on the Increase of Serum Lipids induced by High-Fat Diet Significant increases of serum lipids, such as total cholesterol, LDL-cholesterol, free-fatty acid, phospholipids, triglycerides and total lipids are typically observed in obese animals and people. In addition, a decrease in the HDL/LDL ratio is detected in obese human and animal subjects. Thus, alteration of these lipid profiles can be used as an index of obesity.

Based on these facts, the concentrations of serum lipids, such as total cholesterol, LDL-cholesterol, HDL-cholesterol, free-fatty acid, phospholipids, triglycerides and total lipids were measured in normal, control and PM-F2-OB treatment groups in this study. In terms of cholesterol profile, the high-fat diets substantially increased the concentrations of total cholesterol, LDL-cholesterol, and HDL-cholesterol compared with the normal group: cholesterol, N 75.375±1.972 mg/dl vs. C 95.750±3.283 mg/dl, p<0.001; LDL-cholesterol, N 14.5±0.982 mg/dl vs. C 19.5±1.488 mg/dl, p<0.05; HDL-cholesterol, N 29.375±0.730 mg/dl vs. C 33.75±1.031 mg/dl, p<0.05 (see Fig. 1). In contrast, PM-F2-
OB treatment significantly inhibited increases in the concentrations of total cholesterol and LDL-cholesterol, which were induced by high-fat diets: cholesterol, C 95.750 ± 3.283 mg/dl vs. PM-F2-OB 83 ± 3.262 mg/dl, p < 0.01; LDL-cholesterol, C 19.5 ± 1.488 mg/dl vs. PM-F2-OB 15.125 ± 1.302 mg/dl, p < 0.05 (see Fig. 1).

Similarly, in the lipid profiles, high-fat feeding increased the concentrations of triglycerides, free fatty acids, phospholipids and total lipids compared with the normal group: triglycerides, N 135.625 ± 11.108 mg/dl vs. C 167 ± 19.369 mg/dl, 23% increase, p > 0.05; free fatty acid, N 642 ± 70.436 mg/dl vs. C 680 ± 52.516 mg/dl, 6% increase, p > 0.05; phospholipids, N 139.750 ± 4.938 mg/dl vs. C 174.125 ± 7.691 mg/dl, 25% increase, p < 0.001; total lipids, N 375.5 ± 11.326 mg/dl vs. C 445.750 ± 22.215 mg/dl, 19% increase, p < 0.01 (see Fig. 2). Once again, PM-F2-OB treatment inhibited increases in the concentrations of triglycerides, free fatty acids, phospholipids and total lipids induced by the high-fat diet: triglycerides, C 167 ± 19.369 mg/dl vs. PM-F2-OB 136.625 ± 6.383 mg/dl, 22% decrease, p > 0.05; free fatty acid, C 680 ± 52.516 mg/dl vs. PM-F2-OB 537.250 ± 30.074 mg/dl, 27% decrease, p > 0.05; phospholipids, C 174.125 ± 7.691 mg/dl vs. PM-F2-OB 158 ± 2.752 mg/dl, 10% decrease, p = 0.051; total lipids, C 445.750 ± 22.215 mg/dl vs. PM-F2-OB 376.750 ± 8.998 mg/dl, 18% decrease, p < 0.005 (see Fig. 2). This result suggests that PM-F2-OB treatment reduced the increase in serum lipids induced by the high-fat diet, thus causing an anti-obesity effect.

**Effect of PM-F2-OM on Glucose Level** A significant increase in glucose concentration in obesity is known to be an indication of obesity-induced diabetes, the severity of obesity. To investigate the severity of obesity, the changes in glucose concentrations in the normal, control and PM-F2-OB treatment groups were measured. There were no significant changes in the glucose concentrations in the normal, control and PM-F2-OB treatment groups: N 167.250 ± 8.497 vs. C 155.750 ± 6.514 mg/dl vs. PM-F2-OB 135.750 ± 11.412 mg/dl, p > 0.05. This result indicates that obesity induced by the high-fat diet in rats did not cause diabetes.

**Effect of PM-F2-OM on Liver and Kidney Functions** Significant increases in L-alanine aminotransferase (ALT or SGPT) and L-aspartate aminotransferase (AST or SGOT) activities are typically observed in the blood of obese animals and people. Such increases in enzymes are attributed to damage of fat. Thus, alteration of these enzymes can be used as an index of obesity, and inhibition of this change by these treatments is considered to be an indication of an anti-obesity effect.

Therefore, this study also focused on the activities of SGPT and SGOT measured in the normal, control and PM-F2-OB treatment groups. There were no significant changes in SGPT in the normal, control and PM-F2-OB treatment groups: N 50.250 ± 1.709 U/l vs. C 55.375 ± 3.386 U/l vs. PM-F2-OB 55.375 ± 3.386 mg/dl, p > 0.05. PM-F2-OB treatment significantly decreased the activity of SGOT induced by the high-fat diet: C 184.375 ± 19.288 U/l vs. PM-F2-OB 138.125 ± 18.328 U/l, 33% decrease, p < 0.05 (see Fig. 3). However, the value of SGOT in the high-fat diet group was almost the same as the normal diet group. Thus, together they suggest that liver function was not significantly changed in the high-fat diet. In addition, significant increases in creatinine and BUN are typically observed in the blood of obese animals and people. These increases are attributed to damaged kidneys induced by a high-fat diet and an excess accumulation of fat. Thus, alteration of these factors can be...
used as an index of obesity and inhibition of changes in these factors can be used as an index of an anti-obesity effect. Based on this, the concentrations of creatinine and BUN were measured in the normal, control and PM-F2-OB treatment groups. There were no significant changes in the creatinine concentrations in the normal, control and PM-F2-OB treatment groups: N 0.637 ± 0.0263 mg/dl vs. C 0.612 ± 0.0125 mg/dl vs. PM-F2-OB 0.587 ± 0.0125 mg/dl, p > 0.05 (see Fig. 4). There were significant changes in the BUN concentrations in the normal, control and PM-F2-OB treatment groups: N 24.138 ± 0.929 mg/dl vs. C 19.8 ± 0.876 mg/dl vs. PM-F2-OB 19.0 ± 0.794 mg/dl, p < 0.005 (see Fig. 4). These results suggest that kidney function was not significantly changed or damaged even though the high-fat diet induced obesity.

**DISCUSSION**

Obesity, which affects up to 30% of the adult population in developed countries, is associated with serious mortalities including a high incidence of type 2 diabetes, hyperlipidemia, hypercholesterolemia, cardiovascular disease, osteoarthritis, as well as an increased risk of many forms of cancer. When body weight increases to 20% above the average, the likelihood of mortality rises by 20% for men and 10% for women. When greater quantities of energy (in the form of food) than can be expended enter the body, body weight increases. Therefore, obesity is caused by excess energy input over energy output. Because mortality risk through development of various deadly diseases is dramatically increased in obese patients, a quick and effective treatment is required.

Various animal models of obesity have been used to emulate an obesity-like condition in humans in order to develop effective anti-obesity treatments. Among the animal models of obesity, rats that are fed a high-fat diet are considered useful; a high percentage of fat in their diet is considered to be an important factor in the development of obesity, leading to accumulation of body fat even in the absence of an increase in caloric intake.

Based on this model, the anti-obesity effect of PM-F2-OB in rats fed a high-fat diet was investigated by analyzing the changes in body weight, kidney fat weight and blood biochemicals. In this study, we have shown that PM-F2-OB in a high-fat diet tends to reduce body weight, blood lipids, and kidney fat weight, which is an index of excess accumulation of body fat. This result suggests that PM-F2-OB has a potential role in therapy for obesity-related disorders.

The anti-obesity effect of PM-F2-OB on a high-fat diet was thoroughly studied. In the blood lipid profile, PM-F2-OB significantly reduced the total cholesterol, LDL-cholesterol and total lipids. Also, PM-F2-OB in the high-fat diet showed a reduction of free fatty acids and phospholipids in serum. A substantial reduction of total cholesterol in serum by PM-F2-OB could be attributed to a reduction in the activities of the liver enzyme 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, which is a rate-limiting enzyme in cholesterol biosynthesis. Also, a substantial reduction in

---

**Fig. 3. Protective Effect of PM-F2-OM on Liver**

Changes in activities of GOT (A) and GPT (B) were measured in the normal, control and PM-F2-OB treatment group rats using multifunctional biochemistry analyzers to detect a recovery of liver damage induced by a high-fat-diet with PM-F2-OB. Each bar presents the mean ± S.E.M. from eight rats per group and three groups according to treatment: the normal group fed a standard rat diet (N), the control group fed a high-fat diet (C) and the PM-F2-OB treatment group fed a high-fat diet (PM-F2-OB). * Significantly different from control group (p < 0.05) based on ANOVA followed by a post-hoc test, the S-N-K test.

**Fig. 4. Protective Effect of PM-F2-OM on Kidney**

The changes in concentrations of Creatinine (A) and BUN (B) were measured in the normal, control and PM-F2-OB treatment groups using multifunctional biochemistry analyzers to detect a recovery in kidney damage induced by a high-fat-diet with PM-F2-OB. Each bar presents the mean ± S.E.M. from eight rats per group and three groups according to treatment: the normal group fed a standard rat diet (N), the control group fed a high-fat diet (C) and the PM-F2-OB treatment group fed a high-fat diet (PM-F2-OB). **Significantly different from control group (p < 0.005) based on ANOVA followed by a post-hoc test, the S-N-K test.
LDL-cholesterol and total cholesterol level in serum could be achieved by decreased production of total cholesterol by liver tissue and/or efficient removal of the LDL-cholesterol by various tissues without subsequent renewal. Reductions in free fatty acids, triglycerides, phospholipids and total lipids in serum could be attributed to the inhibition of lipid absorption in the gastrointestinal tract, such as gastrointestinal lipase inhibition. Generally, all lipids are absorbed into the liver to be converted into free fatty acids, triglycerides, phospholipids and total lipids. Reductions in LDL-cholesterol and total cholesterol level in serum could be achieved further before a firm conclusion is drawn.

Acknowledgments We thank Ms. Mi-Jung Kim for her excellent technical assistance in this study.

REFERENCES