GGH(1) improves visceral fat mass in high fat diet induced obese mice

Heeyoung Lee*, Hyerim Lee**, Michung Yoon***, Seungbae Choi† and Soonshik Shin‡

Abstract — This study was undertaken to verify the effects of GGH(1) on obesity using high fat diet induced male mice. Eight-week old C57BL/6N mice were used for all experiments. Standard chow diet fed mice were used as lean control and high fat diet induced obese mice were randomly divided into 4 groups: obese control, GGH(1)-125 mg/kg, GGH(1)-250 mg/kg, and GGH(1)-500 mg/kg. After mice were treated with oral administration for 8 weeks, body weight, feeding efficiency ratio, plasma triglyceride level and visceral adipose tissue weights were measured. Compared with obese controls, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg treated mice had significantly lower body weight gain and feeding efficiency ratio. Consistent with the effects on body weight gain, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg significantly decreased the weights of visceral adipose tissues. GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg significantly decreased plasma levels of triglyceride. Consistent with the effects on feeding efficiency ratio, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg decreased plasma leptin concentrations. Plasma AST and ALT were within the physiological range and organs were not different following GGH(1) treatment compared with obese controls, indicating that GGH(1) does not show any toxic effects on liver. These results suggest that GGH(1) reduces obesity by regulating appetite and visceral lipid metabolism in C57BL/6N mice. Of the 3 GGH(1) concentrations, GGH(1)-500mg/kg seems to be most effective in improving obesity and visceral lipid disorders.

Keyword: MGGH(1); triglyceride; visceral lipid metabolism.

1 Introduction

Gambigyeongsinhwang(1)(GGH(1)) is extracted from 95% ethanol as prescription composed by three kinds such as turmeric, kelp and Amorphophalms konjac. In this animal experiment, we examine whether there are obese effect of GGH(1) using high fat diet-fed male C57BL/6N obese mice. An extract give by concentration of 125, 250, 500mg/kg/day. And in this study, we analyzed by blood biochemically, morphology and organically weight change, feeding efficiency ratio, body weight. A aim of this study is to compare with obese controls, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg treated mice had significantly lower body weight gain and feeding efficiency ratio. In Section 2, we introduce experiment design for our study and present its results in Section 3. Concluding remarks is given in Section 4.

2 Experiment design

2.1 Experiment conditions

1) Experiment animal

As a public announcement analysis, we used 35 eight-week old C57BL/6N mice be supplied from

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KOATECH (Gyeonggi province, South Korea). 7 mice per each group are used in our experiment after we randomly divided into groups in order not to receive influence of weight. We maintained a situation of SPF (specific pathogen free) during experiment duration with breeding environment as follows; temperature (21 ± 2°C), humidity (55 ± 5%), the number of ventilation (15-17 times/hour), intensity of illumination (150-300 lux) and illumination (control 12 hours by light and darkness; lighting (06:00), lights-out (18:00)). We supplied in freedom with Solidity feed (Harlan, USA) and water.

(2) Experiment materials
Experiment materials used the 95% an ethanol extract of the Gambigyeongsinhwan(1). The manufacturing as follows;

Step 1: A composition medicine of Gambigyeongsinhwan(1) bought from the Hwarim pharmaceutical factory and pulverized and selected by department of formula science, college of oriental medicine, dongeui university.

Step 2: The pulverized Gambigyeongsinhwan(1) is concentrated filtrating by a filter after extract during 180 minutes in a temperature 70°C by 95% ethanol.

Step 3: We obtained an 32.88% extract rate from concentrated result.

(3) Experimental groups and medication method
We arranged 7 male mice per group and obese control was gave medication water and GGH(1) was gave medication during 8 weeks by dosage of 125, 250 and 500mg/kg (Table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Number</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Low fat</td>
<td>7</td>
<td>male</td>
</tr>
<tr>
<td></td>
<td>High fat + GGH(1) (mg/kg BW)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>water</td>
<td>7</td>
<td>male</td>
</tr>
<tr>
<td>GGH(1)-1</td>
<td>125</td>
<td>7</td>
<td>male</td>
</tr>
<tr>
<td>GGH(1)-2</td>
<td>250</td>
<td>7</td>
<td>male</td>
</tr>
<tr>
<td>GGH(1)-3</td>
<td>500</td>
<td>7</td>
<td>male</td>
</tr>
</tbody>
</table>

2.2 Experience method

(1) Measure of the weight gain rate
In order to investigate how weight gain rate changing according to GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg, we calculated a change in body weight gain based on measured value the weight 2 times every week during eight weeks.

(2) Measure of the feeding efficiency ratio
To examine GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg, are have a close relation to an appetite, weight is measured 2 times every week and feed intake is measured one times every week during eight weeks. We calculated the feeding efficiency ratio based on above those. The formula for feeding efficiency ratio is given in equation (2.1).

feeding efficiency ratio (FER%) = \(\frac{\text{change in body weight gain (g)} \times \text{feed intake (g)}}{100}\)  

As the formula of the feeding efficiency ratio means, it can think there are the effect of obese control

Table 1: Experimental groups
which change in body weight gain is low although feed intake is high. Therefore, the feeding efficiency ratio can consider as a measurement representing obese and which the value of it is low means there are the effect of obese control.

(3) Statistical analysis
All statistics of this study present mean ± standard deviation and we used OriginLab Version 7.5 (OriginLab Corporation, MA, USA) which is a statistical package to work a statistical analysis.

3 Experiment result

3.1 Body weight gain

The analysis results for changes in body weight gain considering time in Figure 1. We used one-way ANOVA as analysis method. Data are expressed as the mean ± SD. * P<0.05, ** P<0.01, *** P<0.001 significantly different from control. GGH(1) = Gambigyeongsinhwan(1). It appeared that there are the effect of weight reduction 14.90% of the GGH(1)-125mg/kg, 13.48% of the GGH(1)-250mg/kg group and 17.16% in the GGH(1)-500mg/kg group than control group. And among three concentration groups, we can see that GGH(1)-500mg/kg group is high in the effect of weight reduction from Figure 1.

![Body weight gain graph](image)

Figure: 1. Changes in body weight gain of high fat diet-fed obese mice. GGH(1)-1, GGH(1)-125mg/kg; GGH(1)-2, GGH(1)-250mg/kg; GGH(1)-3, GGH(1)-500mg/kg

3.2 feeding efficiency ratio

It appeared that the feeding efficiency ratio is low 13.74% in the GGH(1)-125mg/kg, 13.06% in the GGH(1)-250mg/kg group and 17.56% in the GGH(1)-500mg/kg than control group in Figure 2. Contrast It came out that all three groups are statistically significant in contrast to control group and the GGH(1)-500mg/kg group among three groups is the highest in the extent of significance.
This study was undertaken to verify the effects of GGH(1) on body weight gain, feeding efficiency ratio, and obesity-related factors in plasma as well as histology of liver and adipose tissues using high fat diet-fed male C57BL/6N obese mice. Compared with obese controls, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg treated mice had significantly lower body weight gain and feeding efficiency ratio. Consistent with the effects on body weight gain, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg decreased the weights of visceral adipose tissues. GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg significantly decreased plasma levels of triglyceride. Consistent with the effects on feeding efficiency ratio, GGH(1)-125mg/kg, GGH(1)-250mg/kg and GGH(1)-500mg/kg decreased plasma leptin concentrations. Plasma AST and ALT were in the physiological range and organs were not different following GGH(1) treatment compared with obese controls, indicating that GGH(1) does not show any toxic effects on liver. In conclusion, these results suggest that GGH(1) reduces obesity by regulating appetite and visceral lipid metabolism in C57BL/6N mice. Of the 3 GGH(1) concentrations, GGH(1)-500mg/kg seems to be most effective in improving obesity and visceral lipid disorders.

References