# *Kaempferia parviflora* Extract Diminishes Hyperglycemia and Visceral Fat Accumulation in Mice Fed with High Fat and High Sucrose Diet

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## Abstract

High fat and high sucrose (HFHS) diet are a nutritional condition that accounts for the largest incidence of metabolic syndrome (MetS) which comprises important risk factors that increase the incidence of cardiovascular disease and type 2 diabetes mellitus. *Kaempferia parviflora* extract (KPE) has been widely used for health promotion as a traditional medicine for improvement of blood flow and as an anti-inflammatory agent. However, there is little work investigation the effect of KPE on MetS. Therefore, the aim of this study was to investigate the effects of KPE on the reduction of body weight, blood glucose concentration and visceral fat accumulation induced by high fat and high sucrose diet in mice. C57BL/6Mlac mice were divided into 6 groups: Control (C), C+KPE10, C+KPE100, HFHS-C, HFHS+KPE10 and HFHS+KPE100. The HFHS groups were given a high fat and high sucrose diet while the C groups received normal diet for 20 weeks, after which they were further treated with KPE 10 or 100 mg/kg BW or distilled water for 8 weeks. Food intake and body weight were measured by weighing, fasting blood glucose was determined by using the glucose oxidase method and blood pressure was measured by tail cuff method. After the animals were sacrificed visceral fat was removed for weighing. The results showed that KPE treatment decreased blood glucose in the HFHS+KPE10 and HFHS+KPE100 groups and reduced visceral fat in the HFHS+KPE10 group when compared with the HFHS-C group. KPE treatment did not alter body weight in all groups. Blood pressure in all groups was not significantly different. In conclusion, KPE treatment is able to reduce symptoms found in high fat and high sucrose diet-induced mice by suppressing visceral fat accumulation and decreasing hyperglycemia.

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***Keywords:*** *Kaempferia parviflora, visceral fat, blood glucose, blood pressure, high fat and high sucrose diet*

Dietary intake rich in carbohydrates such as sucrose and fructose as well as high fat[1] are important in determining who may develop obesity. Presently, consumption of a Western-style diet characterized by a higher intake of high-fat and sugar-containing food has been more popular. Increasing Western-style dietary are associated with an increased risk of obesity, cardiovascular disease[2] and type 2 diabetes.[3] High fat and high sucrose (HFHS) diet are the major cause of abdominal obesity, body weight gain,[4] increased plasma triglyceride, plasma glucose and free fatty acids concentration[5] as well as hyperinsulinemia and impaired glucose tolerance.[6]

Obesity is a metabolic disorder, resulting in increased adipose tissue mass, especially increased visceral fat accumulation,[7] and leading to the onset of metabolic syndrome.[8] Metabolic disorders increases the risk of mortality from cardiovascular disease[9] and type 2 diabetes mellitus.[10] The prevalence of metabolic syndrome continues to grow. Around 20-25% of the world's adults suffer from metabolic syndrome. It is noted that in Thai adults the prevalence of metabolic syndrome was 15.2% in 2007[11] and increased to 32.6% in 2011.[12] Therefore, if one can prevent the progression of metabolic syndrome from the early stages of onset, the mortality rate would reduce.

*Kaempferia parviflora* (*K. parviflora*) Wall. ex Baker or kra-chai-dam, is a black rhizome plant that belongs to the family of Zingiberaceae. It has been reported that *K. parviflora* comprised of several methoxyflavone such as 5-hydroxy-3,7-dimethoxy-flavone, 3,5,7,4′-tetramethoxyflavone, 3,5,7-trimethoxyflavone, 5,7-dimethoxyflavone, 5-hydroxy-7-methoxyflavone, 5-hydroxy-3,7,4′-trimethoxyflavone, 5-hydroxy-3,7,3′,4′-tetramethoxyflavone, 5-hydroxy-7,4′-dimethoxyflavone, 5,7,4′-trimethoxyflavone, 5-hydroxy-7,4′-dimethoxyflavone and 3,5,7,3′,4′-pentamethoxyflavone.[13] In Thailand the black rhizome of *K. parviflora* has been widely used for health promotion and as a traditional medicine to improve blood flow and impotence. In addition extract from *K. parviflora* has various effects

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including treatment of gastric ulcer,[14] inhibition of nitric oxide synthase and prostaglandin $E_2$,[15] as well as anti-inflammatory property.[16] Thus**,** *K. parviflora* has been increasingly used as an alternative medicine and more potential therapeutic functions.

Recently, it has been shown that *K. parviflora* had anti-obesity effect and improved insulin resistance, abnormal glucose and lipid metabolism in spontaneously obese type 2 diabetes, Tsumura, Suzuki, Obese Diabetes (TSOD) mice.[17] However, there are no scientific reports that explain the beneficial effects of *K. parviflora* extract (KPE) to reduce various symptoms caused by prolonged consumption of unhealthy diet, such as plenty fat and sugar containing food. There was evidences indicated that mice fed with high fat and high sucrose diet increased body weight and abdominal fat deposition and developed hyperinsulinemia, hyperglycemia, dyslipidemia and hyperleptinemia.[18] Therefore, this study aimed to investigate the effects of *K. parviflora* on various symptoms found in high fat and high sucrose diet-induced mice by focusing on body weight increase, hyperglycemia and abdominal fat deposition.

## Materials and Methods

**Plant material and preparation of crude extract**

*K. parviflora* was collected from cultivable sources in Phitsanulok, Thailand and kept at Faculty of Pharmaceutical Sciences, Naresuan University, Phitsanulok, Thailand. The rhizomes were cut into small pieces, shade dried by hot air oven at 45$^{o}$C for 24 h before being milled into rough powder. The dried rhizome powder was macerated at room temperature with hexane for 3 days. After that the residue was macerated with 95% ethanol for 3 days, and were filtered and extracted again. The residue solvent was removed by lyophilization to obtain a dry *K. parviflora* extract (KPE). The crude *K. parviflora* was kept at -20$^{o}$C until use. On the day of the experiment, *K. parviflora* was dissolved with Tween-80 before adding distilled water to doses of 10 and 100 mg/kg body weight (BW).

**Animals and treatments**

Three weeks old male C57BL/6Mlac mice (body weight 15-20 g) were used in the experiments. All animals were obtained from the National Laboratory Animal Center, Mahidol University, Salaya, Nakhon Pathom, Thailand. The mice were acclimatized for at least one week before starting the experiment. They were housed (5 animals per cage) at a constant temperature of 24 ± 2$^{o}$C, humidity 50 ± 10%, under a standard light/dark cycle (12:12 h) and allowed free access to standard pellet chow and water *ad libitum* prior to the study. The experimental protocols were approved by the Animal Ethics Committee of Naresuan University, Phitsanulok, Thailand.

Mice were divided into 2 groups: control (C; n=30) and high fat high sucrose diet (HFHS; n=30). The HFHS group was given high fat and high sucrose diet (58% of calories from fat and 26% from carbohydrate)[19] while the C group received standard pellet chow. Animals were allowed free access to diet and water *ad libitum* for 20 weeks in order to develop the abnormal signs of metabolic syndrome in the HFHS group.

After the inducing period, the animals were treated with vehicle (distilled water plus Tween-80) or KPE suspension, once daily at 9:00-10:00 am by oral gavage, for further 8 weeks. The C and HFHS groups were each randomly divided into 3 subgroups as follows: 1) C group as normal mice receiving vehicle (n=10); 2) C+KPE10 group as normal mice receiving KPE suspension 10 mg/kg BW (n=10); 3) C+KPE100 group as normal mice receiving KPE suspension 100 mg/kg BW (n=10); 4) HFHS-C group as high fat high sucrose-induced mice receiving vehicle (n=7); 5) HFHS+KPE10 group as high fat high sucrose-induced mice receiving KPE suspension 10 mg/kg BW (n=10); 6) HFHS+KPE100 group as high fat high sucrose-induced mice receiving KPE suspension 100 mg/kg BW (n=13).

**Body weight and food intake measurement**

The body weight of each animal was measured once a week (1 pm) from the beginning until the end of the experiment by weighing. Food intake was measured three times a week (3 pm) throughout the 28 weeks of the experiment by weighing. The average food intake was calculated as the mean value per each mouse by the difference in amount between total food and the remaining food divided by the number of mice in each cage.

**Fasting blood glucose measurement**

Fasting blood glucose concentration was assessed 3 times; at the beginning of the experiment as a baseline, at the end of inducing period with high fat high sucrose diet or before given KPE suspension, and at the end of the 8-week oral KPE administration. After 12 h of fasting, each mouse was restricted in a restrainer and blood sample was collected from the lateral tail vein (4-5 µl). The blood glucose concentration was determined by using an Accu-Chek glucometer (Accu-CHEX Performa, USA).

**Blood pressure measurement**

The blood pressure was measured before and after treatment with KPE suspension. Blood pressure of mice was determined with a non-invasive blood pressure meter under normal consciousness. Each mouse was restricted in a restrainer, the tail was inserted into the tail cuff and the finger pulse transducer was attached to the tail. The tail cuff and the finger pulse transducer were connected to the non-invasive blood pressure-pulse transducer system and the pressure output was connected to the PowerLab system, which recorded to the computer (Chart v5; AD Instruments, Bella Vista, NSW 2153,

Australia). After cardiovascular parameters were stabilized, systolic blood pressure was recorded immediately.

**Visceral fat collection**

Animals were euthanized with overdose of pentobarbital by intraperitoneal (i.p.) injection. All amount of visceral fat was immediately removed and then blotted dry for weighing. Visceral fat pads in this study referred to the fat pads covering the abdominal cavity which comprises of mesenteric fat, packed between the lesser curvature of the stomach and sigmoid colon, retroperitoneal fat and epididymal fat.

**Statistical analysis**

All data were expressed as the mean ± standard error of the mean (SEM). Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) software version 17.0. The data analysis was conducted by one-way analysis of variance (ANOVA), with post hoc multiple comparison or independent sample *t*-test. In all comparisons, *P* values less than 0.05 were considered as significant.

## Results

**Effect of high fat and high sucrose diet on food intake and the abnormal development**

Food intake throughout the 20 weeks of the inducing period was significantly lower in the HFHS group than the C group ($P < 0.001$; Figure 1)**.** The food intake expressed as mean values of each mouse in the C group were 3.05 ± 0.03 g (during week1-4), 3.20 ± 0.02 g (during week 5-8), 3.10 ± 0.03 g (during week 9-12), 3.17 ± 0.02 g (during week 13-16) and 3.18 ± 0.03 g (during week 17-20). For the HFHS group the food intake of each mouse were 2.23 ± 0.02 g (during week 1-4), 2.25 ± 0.03 g (during week 5-8), 2.24 ± 0.03 g (during week 9-12), 2.40 ± 0.04 g (during week 13-16) and 2.72 ± 0.02 g (during week 17-20). At the beginning, the body weight was not significantly different in all mice. During the period of induction with high fat high sucrose diet, the body weight was significantly lower in the HFHS group ($P < 0.05$) when compared with the C group at 4$^{th}$, 8$^{th}$ and 12$^{th}$ week. However, at the end of 20$^{th}$ week, the body weight was significantly higher in the HFHS group when compared with the C group (32.38 ± 0.38 *vs* 31.17 ± 0.36 g; $P < 0.05$; Figure 2).

Similarly to the body weight, at the beginning the fasting blood glucose concentration was not significantly different in all mice. After inducing with high fat high sucrose diet for 20 weeks, the blood glucose concentration was significantly increased in the HFHS group when compared to the C group (163.83 ± 6.09 *vs* 114.07 ± 3.54 mg/dl; $P < 0.001$; Figure 3).

Systolic blood pressure, however, could not be detected in three week-old mice at the beginning of the experiment. After 20 weeks of the inducing period, systolic blood pressure was measured again

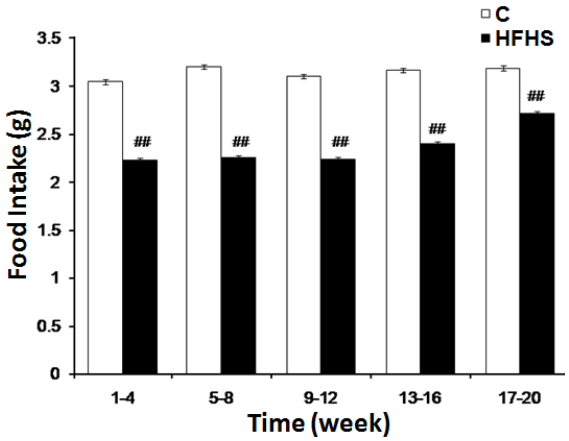



**Figure 1** Effect of high fat and high sucrose (HFHS) diet on food intake in mice during 4-week intervals throughout the 20 weeks of inducing period. Each bar represents mean ± SEM of food intake per mouse (n=30) of control (C) and high fat and high sucrose (HFHS) groups. $^{\#\#}P < 0.01$ *vs* C (unpaired Student's *t*-test).

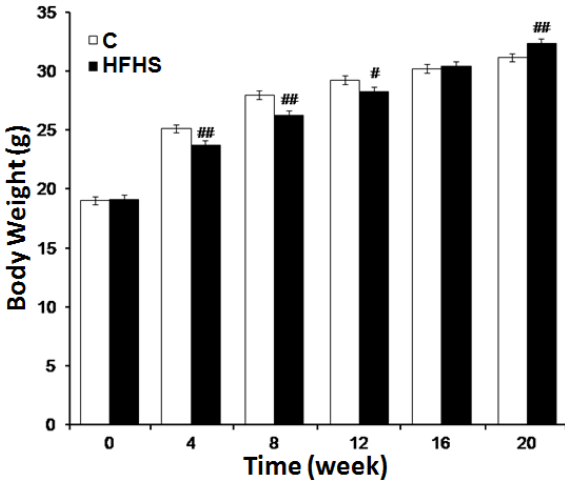



**Figure 2** Effect of high fat and high sucrose (HFHS) diet on body weight in mice during 20 weeks of the inducing period. Each bar represents mean ± SEM (n=30) of control (C) and high fat and high sucrose (HFHS) groups. $^{\#\#}P < 0.01$ or $^{\#}P < 0.05$ *vs* C (unpaired Student's *t*-test).

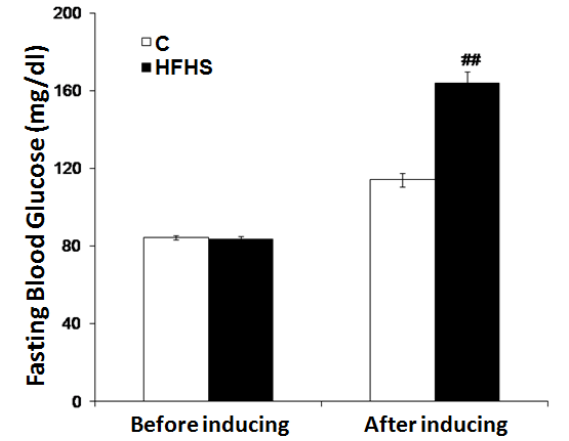



**Figure 3** Effect of high fat and high sucrose (HFHS) diet on fasting blood glucose concentration in mice before and after 20 weeks of inducing period. Each bar represents mean ± SEM (n=30) of control (C) and high fat and high sucrose (HFHS) groups. $^{\#\#}P < 0.01$ *vs* C (unpaired Student's *t*-test).

and showed that there was no significantly difference between the HFHS and C groups and the values were in normal range (111.24 ± 3.12 *vs* 113.00 ± 2.47 mmHg; Figure 4).

**Effect of *K. parviflora* extract on food intake**

During the 8-week treatment with *K. parviflora*, the pattern of food intake in all the HFHS and C groups was similar to that before treatment. In the first 4 weeks of *K. parviflora* extract treatment the amount of food intake (g) in all HFHS groups were less than all C groups: HFHS-C, 2.58 ± 0.04; HFHS+KPE10, 2.61 ± 0.05; HFHS+KPE100, 2.70 ± 0.06; C, 3.00 ± 0.05; C+KPE10, 3.02 ± 0.04; and C+KPE100, 3.07 ± 0.07. The same pattern was found in the later 4 weeks of *K. parviflora* extract treatment. Besides, the food intake of the *K. parviflora* extract-treated groups was not significantly different from their own control (Figure 5).

**Effect of *K. parviflora* extract on body weight**

After administration of *K. parviflora* extract for 8 weeks, the body weight in all the HFHS and C treated groups was not significantly different from their own control; the HFHS-C and C groups respectively (Figure 6). In other words, *K. parviflora* extract did not alter the body weight either in mice fed with HFHS diet or normal diet.

**Effect of *K. parviflora* extract on blood glucose**

Before treatment with *K. parviflora* extract, the fasting blood glucose concentration in HFHS-C group was significantly higher than the C group (168.43 ± 9.25 *vs* 117.20 ± 6.70 mg/dl*; P* < 0.001), while the blood glucose concentration was not significantly different between the HFHS-C (168.43 ± 9.25 mg/dl) and all HFHS treated groups (HFHS+KPE10, 158.60 ± 9.42 and HFHS+KPE100, 165.38 ± 12.39 mg/dl, respectively).

After administration of *K. parviflora* extract for 8 weeks, the blood glucose concentration in the HFHS+KPE10 and HFHS+KPE100 groups were significantly decreased when compared with the HFHS-C group (116.30 ± 7.66, 118.54 ± 2.74 and 172.43 ± 9.07 mg/dl, respectively; *P* < 0.001). In contrast, the blood glucose concentration in the C+KPE10 and C+KPE100 groups was not significantly different from that of the C group (Figure 7).

**Effect of *K. parviflora* extract on visceral fat accumulation**

At the end of the experiment, the visceral fat accumulation in the HFHS-C group was significantly higher than the C group (0.50 ± 0.01 *vs* 0.13 ± 0.01 g*; P* < 0.001). After 8 weeks of treatment with *K. parviflora* extract the accumulation of visceral fat was significantly lower in the HFHS+KPE10 group only when compared with the HFHS-C group (0.32 ± 0.01 *vs* 0.50 ± 0.01 g; *P* < 0.01). However, the fat deposition in the HFHS+KPE100 group was slightly

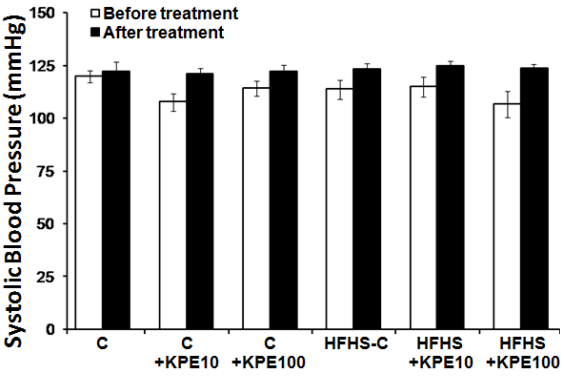



**Figure 4** Effect of *K. parviflora* extract (KPE) on systolic blood pressure before and after 8 weeks of administration. Each bar represents mean ± SEM of control group (C; n=10), C treated with KPE 10 mg/kg group (C+KPE10; n=10), C treated with KPE 100 mg/kg group (C+KPE100; n=10), high fat and high sucrose control group (HFHS-C; n=7), HFHS treated with KPE 10 mg/kg group (HFHS+KPE10; n=10), and HFHS treated with KPE 100 mg/kg group (HFHS+KPE100; n=13); one-way ANOVA followed by post hoc LSD test.

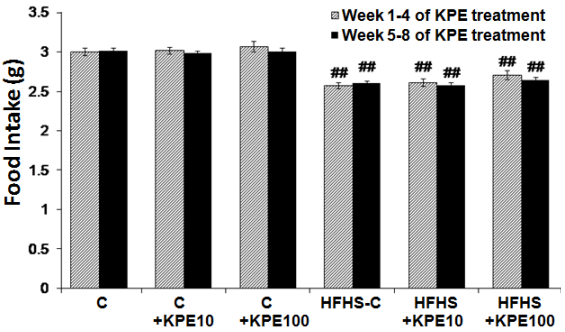



**Figure 5** Effect of *K. parviflora* extract (KPE) on food intake during 8 weeks of administration. Each bar represents mean ± SEM of control group (C; n=10), C treated with KPE 10 mg/kg group (C+KPE10; n=10), C treated with KPE 100 mg/kg group (C+KPE100; n=10), high fat and high sucrose control group (HFHS-C; n=7), HFHS treated with KPE 10 mg/kg group (HFHS+KPE10; n=10), and HFHS treated with KPE 100 mg/kg group (HFHS+KPE100; n=13). $^{\#\#}P < 0.01$ *vs* C; one-way ANOVA followed by post hoc LSD test.

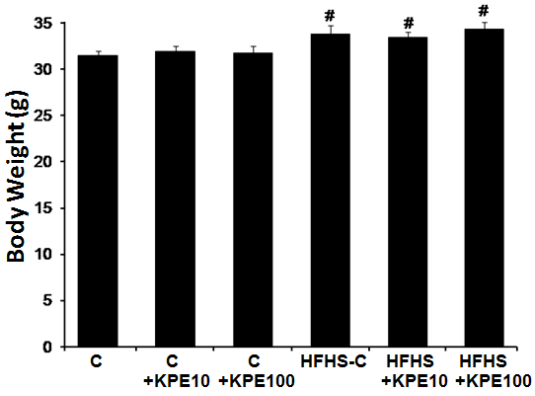



**Figure 6** Effect of *K. parviflora* extract (KPE) on body weight after 8 weeks of administration. Each bar represents mean ± SEM of control group (C; n=10), C treated with KPE 10 mg/kg group (C+KPE10; n=10), C treated with KPE 100 mg/kg group (C+KPE100; n=10), high fat and high sucrose control group (HFHS-C; n=7), HFHS treated with KPE 10 mg/kg group (HFHS+KPE10; n=10), and HFHS treated with KPE 100 mg/kg group (HFHS+KPE100; n=13). $^{\#}P < 0.05$ *vs* C; one-way ANOVA followed by post hoc LSD test.

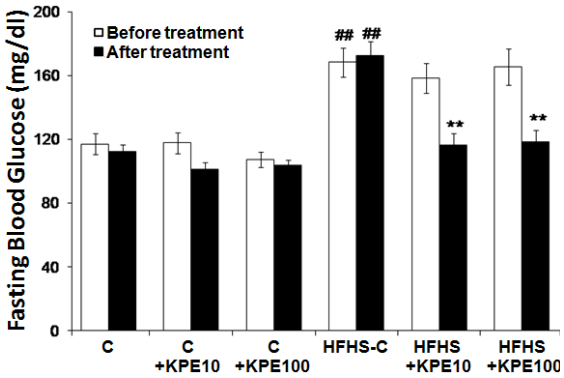



**Figure 7** Effect of *K. parviflora* extract (KPE) on blood glucose concentration before and after 8 weeks of administration. Each bar represents mean ± SEM of control group (C; n=10), C treated with KPE 10 mg/kg group (C+KPE10; n=10), C treated with KPE 100 mg/kg group (C+KPE100; n=10), high fat and high sucrose control group (HFHS-C; n=7), HFHS treated with KPE 10 mg/kg group (HFHS+ KPE10; n=10), and HFHS treated with KPE 100 mg/kg group (HFHS+KPE100; n=13).$^{\#\#}P < 0.01$ *vs* C, $^{**}P < 0.01$ *vs* HFHS-C; one-way ANOVA followed by post hoc LSD test.

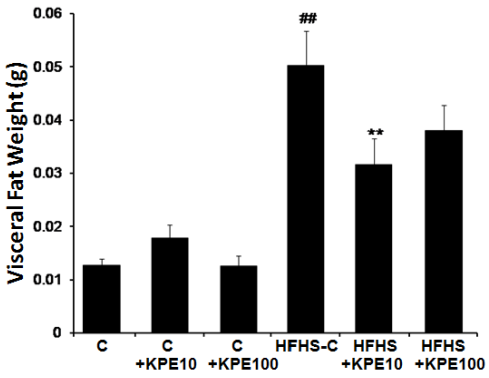



**Figure 8** Effect of *K. parviflora* extract (KPE) on visceral fat accumulation after 8 weeks of administration. Each bar represents mean ± SEM of control group (C; n=10), C treated with KPE 10 mg/kg group (C+KPE10; n=10), C treated with KPE 100 mg/kg group (C+KPE100; n=10), high fat and high sucrose control group (HFHS-C; n=7), HFHS treated with KPE 10 mg/kg group (HFHS+ KPE10; n=10), and HFHS treated with KPE 100 mg/kg group (HFHS+KPE100; n=13).$^{\#\#}P < 0.01$ *vs* C, $^{**}P < 0.01$ *vs* HFHS-C; one-way ANOVA followed by post hoc LSD test.

decreased but not significant when compared to the HFHS-C group (Figure 8).

## Discussion

This study demonstrated that C57BL/6Mlac mice fed with high fat (58%) and high sucrose (26%) diet for 20 weeks had increased body weight, blood glucose concentration and visceral fat accumulation, whereas systolic blood pressure was unchanged. Increasing consumption of high fat and high sucrose diet in this model is closely correlated with the occurrence of metabolic syndrome in human. In particular, an excess fat deposition in abdominal cavity also plays an important role in the onset of metabolic syndrome.[8] Our results were in agreement with several previous studies, for instance, Survit et al, found that the C57BL/6J mice fed on high fat combine with high sucrose diet for 16 weeks could develop obesity, hyperglycemia and hyperinsulinemia.[20] It has been reported that these mouse strain fed a high fat diet was able to increase body weight and blood glucose level which occur after 8 weeks diet consumption.[21] A recent study found that high fat diet induced obesity in C57BL/6J strain developed features of non-alcoholic fatty pancreatic disease (NAFPD), hepatic fat accumulation, insulin resistance and an increased abdominal fat deposition which were characterized mainly cause metabolic syndrome.[22]

Our results show that at the end of the inducing period with high fat and high sucrose diet the body weight of the HFHS group was not extremely greater, albeit significantly different, than the C group, even though the body weight of the HFHS group was less than the C group during the 1-12 weeks. These might be due to the less food consumption of the HFHS group throughout the period of induction with high fat and high sucrose diet (Figure 1). The control of food intake is responsible by the feeding and satiety centers in the hypothalamus. There were studies demonstrated that in low glycemic index diet, the slowly digested food, which produced a gradual rise in blood glucose, increased satiety[23] and thereby reduced food intake.[24] The given high amount of fat might reduce the appetite, leading to less food consumption found in the HFHS group, plausibly causing less body weight increase during the 1-12 weeks. However, even though the HFHS group consumed less amount of food, the calculated calories the HFHS group received were still huge, approximately five folds above the C group. In addition, excess amount of fat (58%) and sucrose (26%) consumed by the HFHS group gave rise to fat deposition and plausible increased in body weight that were seen at the end of the 20 weeks inducing period. The possible explanation for prolonged less food intake or less appetite found in the HFHS group may involve the higher secretion of leptin from the increased adipose tissues. Leptin acts on the hypothalamus to suppress appetite which then decreases food intake. Although mice in the HFHS group ate less amount of food throughout the experiment, their body weight was significantly higher than mice in the C group at the end of the inducing period. Moreover, mice in the HFHS group had more visceral fat accumulation than mice in the C group. Therefore it could be implied that mice fed on high fat and high sucrose diet in this study developed obesity.

The potential treatment effects of *K. parviflora* extract on various symptoms, focusing on body weight increase, high blood glucose concentration and visceral fat accumulation, which were found in mice fed with high fat and high sucrose diet, were determined in this study. After oral *K. parviflora* extract administration for 8 weeks, we found that the

treatment could decrease visceral fat accumulation in the HFHS+KPE10 group (Figure 8). Our result partly agrees with a previous study which demonstrated that both the Tsumura, Suzuki, Non-obesity (TSNO) and Obese Diabetes (TSOD) mice, the genetic disease animal model in metabolic syndrome, received *K. parviflora* powder mix with Powder Feed *ad libitum* for 8 weeks, had a reduction in visceral fat deposition, implying that *K. parviflora* had an anti-obesity effect.[17] On the contrary, we found that *K. parviflora* extract could not reduce visceral fat accumulation in all groups fed on normal diet. Therefore, it could be summarized that *K. parviflora* extract had capability to decrease visceral fat deposition in mice that consume high fat high sucrose diet, but no effect in those consuming normal diet.

Our results also showed that *K. parviflora* extract decreased fasting blood glucose concentration in the HFHS group but had no effect in the C group (Figure 7). This means that *K. parviflora* extract had an anti-hyperglycemic effect. Abdominal obesity people have an increased adipose tissue mass. The expansion of adipocytes leads to dysregulated adipocytokine secretion such as proinflammatory (tumor necrosis factor-α (TNF-α), interleukin 6 (IL-6) and inflammatory cytokines, resulting in insulin resistance.[25] Recently, it has been reported that a flavonoid from *Salvia plebeian* could improve insulin sensitivity by inhibiting TNF-α and IL-6 mRNA expression.[26] The main active compounds of *K. parviflora* extract are flavonoids attached with many methoxyl groups.[13] It could be postulated that the *K. parviflora* extract might decrease blood glucose concentration by suppressing the proinflammatory cytokines secretion, such as TNF-α and IL-6, from an adipose tissue, leading to increased insulin sensitivity, which finally resulting in decreased blood glucose levels. Further study about insulin sensitivity or levels of plasma insulin will help better clarify this anti-hyperglycemic effect of *K. parviflora* extract.

The body weight of the *K. parviflora*-treated HFHS and C groups was not different from their control groups (Figure 6) indicating that the administration of *K. parviflora* extract for 8 weeks could not alter the body weight of mice fed with either normal or unhealthy diet. The body weight remained high in the HFHS-treated group despite visceral fat decrease might be explained by considering that fat has very light weight (low in density) compared to the body weight. Therefore the decrease in visceral fat had less impact on body weight. Another possible explanation might concern the doses of *K. parviflora* extract which were smaller than those used in a previous study[17] which showed inhibitory effects on body weight in TSOD mice.

This study demonstrated that the C57BL/6Mlac mice fed high fat and high sucrose diet for 20 weeks did not develop hypertension (Figure 4). It has been reported that *K. parviflora* powder improves hypertension symptoms in TSOD mice that genetically develop hypertension.[17] We did not find any effect of *K. parviflora* extract on systolic blood pressure, since hypertension did not develop in these mice as we had expected.

## Conclusion

The administration of *K. parviflora* extract for eight weeks could reduce symptoms found in high fat and high sucrose diet-induced mice by suppressing visceral fat accumulation and decreasing hyperglycemia. These effects appeared only in the mice fed on diet containing plenty of fat and sugar that is frequently found in Western-style food. Thus *K. parviflora* extract is one medical plant that has the potential preventive effects, helping to interrupt the progression of metabolic syndrome from the early stages of onset.

Future studies are needed to evaluate the effect of *K. parviflora* extract on serum insulin concentration, insulin resistance and lipid profile in high fat and high sucrose diet-induced mice.

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### Conflict of interest

None to declare.

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