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RESEARCH ARTICLE

Beneficial influences of *Amorphophallus konjac* k. koch in counteracting weight gain resulting from a high-fat diet and the use of antipsychotic drugs

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Abstract

Obesity and overweight represent a significant challenge to global health, necessitating immediate action to create safe and effective treatment options. This research aimed to evaluate the effectiveness of *Amorphophallus konjac* K. Koch (konjac) as a natural anti-obesity agent in mice that developed obesity due to a high-fat diet and olanzapine treatment. After administering *A. konjac* in the form of konjac flour orally to mice over eight weeks, we measured body weight gain, total body fat and visceral fat mass, along with the serum lipid profile. The study also focused on evaluating hepatic steatosis and liver injury. Konjac flour was shown to inhibit weight gain and reduce BMI in obese mice. Notably, the groups treated with konjac powder exhibited significant reductions in visceral fat mass compared to the untreated obese group. Moreover, the oral administration of konjac powder led to marked improvements in serum lipid levels and mitigated liver damage by decreasing fat accumulation in the liver and lowering aspartate aminotransferase activity. Research involving *Amorphophallus konjac* K. Koch (konjac) revealed its significant weight loss effects in obese mice fed a high-fat diet combined with olanzapine. These outcomes suggest that this herbal formulation could be a valuable addition to anti-obesity therapies, helping to prevent the accumulation of body fat and influence lipid metabolism.

Keywords: *Amorphophallus konjac*; high-fat diet; obesity; olanzapine; mice

Introduction

Overweight and obesity are defined as abnormal or excessive fat accumulation that can impair health. Body mass index (BMI) is a simple index of weight for height often used to classify overweight and obesity in adults. In adults, overweight or pre-obese is defined as a BMI of 25-29.9 kg/m^2, while a BMI ≥ 30 kg/m^2 is defined as obesity (1). In 2022, there were 2.5 billion adults aged 18 and older classified as overweight, with 890 million of them experiencing obesity. The global rate of obesity has more than doubled from 1990 to 2022 (1). High incidence rates of obesity were found in children, with an estimated 37 million children under the age of 5 years being overweight or obese in 2022 (1). A high-calorie diet and a sedentary lifestyle are factors that increase the obesity rate. Being overweight and obese increases the risk of many health problems, including dyslipidemia, cardiovascular disease (including hypertension, stroke and myocardial infarction), insulin resistance, impaired glucose metabolism, osteoarthritis and some cancers (2). Current treatments for obesity include dietary changes and increased physical activity or exercise, psychotherapy for people with eating disorders and pharmacotherapy. Present anti-obesity drugs act through several potential mechanisms, including increasing energy expenditure, suppressing appetite, inhibiting digestive enzymes, or interfering with the absorption of fat or sugar from food at the intestinal level. These drugs can cause many side effects such as depression, anxiety, headache, dizziness, nausea and fatigue, high blood pressure, irregular heartbeat, constipation, fatty stools and oil-soluble vitamin and essential fatty acid deficiencies (3). Therefore, there is a need to further develop alternative therapies with little or no side effects in the management of obesity and related complications. Drugs derived from medicinal herbs are an approach that is receiving great attention in the development of weight control and obesity treatment preparations (4) and konjac flour derived from the tubers of the konjac plant is one of the current potential directions (5).

Amorphophallus konjac K. Koch (konjac) is a perennial plant belonging to the family Araceae. It is grown as an edible plant in Asia countries. In China and Japan, konjac has been used as a food and food additive for over 1000 years (5, 6). Over the past few decades, purified konjac powder, commonly known as

konjac glucomannan (KGM), a water-soluble polysaccharide and considered dietary fiber, has been used as a food additive as well as a dietary supplement in the diet of many Asia and European countries for weight loss (6 - 8). Despite its widespread use, the safety and effectiveness of glucomannan have not been fully elucidated. We intend to present more substantial evidence highlighting the anti-obesity properties of konjac in our research.

The objective of this study was to assess the efficacy of *Amorphophallus konjac* K. Koch (konjac) in the form of tuber konjac flour in mice that experienced obesity because of a high-fat diet and olanzapine administration.

Materials and methods

Konjac flour preparation

Konjac product is supplied by Ngoc Thien Pharmaceutical and Trading Joint Stock Company, reaching the manufacturer's standard. Each sachet contains 5 g of tuber konjac (*Amorphophallus konjac* K. Koch) flour. The expected dose in humans is two sachets/day (equivalent to 10 g of tuber konjac flour daily).

The preparation of konjac flour involves several steps. Initially, the tubers are harvested, followed by cleaning and peeling. The tubers are then finely ground and the starch is extracted through a series of processes: dissolving in water, allowing it to settle and decanting through cotton to obtain the starch. This filtration process is conducted three times. Once the starch is extracted, it is dried to achieve a moisture content of less than 14 %. The final dried flour is then packaged in sealed bags, each containing 5 g. Konjac flour (KF) was suspended in pure water before daily intragastric administration.

Animals

Male and female adult *Swiss* mice were purchased from the National Institute of Hygiene and Epidemiology (Hanoi, Vietnam). The animal experiment was performed according to the guidelines for the care and use of laboratory animals (9). Animals were housed in ten per metal cage (H: 13 cm, W: 35 cm, D: 27.5 cm) in a temperature-controlled room (22 ± 1 °C) with a 12 hrs light-dark cycle (lights on 8 am). Mice were freely provided either a 45 % high-fat diet (HFD) or a normal diet, as well as water and libitum.

Experimental design

Olanzapine ODT 10 mg (OLZ) was purchased from An Thien Pharmaceutical Company Limited and dissolved in distilled water. OLZ was administered at the dose of 3 mg/kg, PO (10).

Mice were randomized into five groups of fifteen animals each and treated as follows:

Group 1: Normal control

Group 2: HFD plus olanzapine (HFD-OLZ) (Negative control)

Group 3: HFD-OLZ and Metformin (200 mg/kg/day) (HO-Met)

Group 4: HFD-OLZ and KF high dose (7.2 g/kg/day) (HO-KFH)

Group 5: HFD-OLZ and KF low dose (2.4 g/kg/day) (HO-KFL)

From the beginning of the experiment, mice in groups 2-5 received oral olanzapine administration (3 mg/kg, once daily, at 8:00 am). Mice in group 1 (normal control) were maintained on the normal diet and given drug vehicle alone. From the first day of OLZ (or vehicle) administration, the animals in the HO-Met group received metformin (as reference drug) at the dose of 200 mg/kg/day PO, while mice in the HO-KFL and HO-KFH groups were treated orally with 2.4 and 7.2 g/kg/day konjac flour, respectively. The above treatment lasted for 8 weeks, during which weight gain was measured every week.

Body weight analysis

The body weight (g) and body length (nose-to-anus length) (cm) of mice were measured weekly using a weighing scale and tape rule, respectively. The body weight and body length were used to estimate the following anthropometric parameter:

$$\text{Body mass index (BMI)} = \frac{\text{Body weight (g)}}{\text{Body length}^2\ (\text{cm}^2)}$$

Serum biochemical analysis

The arterial blood sample was obtained from the overnight (16 hrs) fasted mice after 24 hrs of the last dose. The blood was collected in Eppendorf tubes and then centrifuged at 3000 rpm (40 °C for 10 min). Serum was separated for the estimation of total cholesterol (TC), HDL-C (high-density lipoprotein cholesterol), TG (triglycerides), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) using commercially available kits (DIALAB GmbH, Austria) according to the manufacturer's protocol.

The following formulae were applied to calculate serum LDL-C (low-density lipoprotein cholesterol) value and atherogenic index of plasma (AIP):

LDL-C = (TC – HDL-C) – TG/2,2 (mmol/L) (11)

AIP = log 10 (Triglyceride/HDL-C) (12).

Identification of adipose depots

Abdominal fat is primarily categorized into two types: subcutaneous adipose tissue (SAT), located between the skin and the abdominal wall and visceral adipose tissue (VAT), which encases the abdominal organs. A midline laparotomy was conducted along the sagittal plane, leading to the resection of the intestines and exposure of the retroperitoneal space. Samples of various fat types, including subcutaneous, gonadal, mesenteric, retroperitoneal and perirenal fat (collectively referred to as visceral fat), were obtained and weighed from five distinct groups.

The adiposity index (AI) is used as a marker of obesity because the degree of fat tends to increase with obesity, allowing an accurate assessment of body fat percentage. The AI was calculated using the following formula (13):

$$\text{Adiposity index} = \frac{[\text{TBF (SAT + VAT) (g)} \times 100}{\text{Final body weight (g)}}$$

Where, TBF = Total body fat, SAT = Subcutaneous adipose tissue and VAT = Visceral adipose tissue.

Histopathological analysis

Part of the liver samples from all groups were preserved in 10 % neutral formalin for histopathological analysis. Paraffin tissue sections of 5-6 µm thickness were cut and stained with hematoxylin –eosin (H & E) and examined under a light microscope (14).

Statistical analysis

Microsoft Excel 2010 and IBM SPSS Statistics 22.0 software were used to process and analyze the data. The values are shown as Mean ± Standard Deviation (SD) and presented in tables and figures. One way ANOVA and Bonferroni post hoc test were used to analyze the difference between the means of groups. A statistically significant difference was identified at $p < 0.05$.

Results

Effects on body weight and BMI

Table 1 illustrates that the mean weight changes in the HFD-OLZ group (group 2) were significantly higher than the normal control group from the first week until the last week of the study (P <0.0 01). Co-administration of metformin at the dose of 200 mg/kg with olanzapine significantly decreased HFD plus olanzapine-induced body weight gain from the first week to the 8th week. KF was able to significantly decrease the weight gain induced by HFD plus olanzapine in all two doses of 2.4 and 7.2 mg/kg from the 3rd week. The mean weight in the negative group increased by 119.41 % at the end of the 8-week administration compared to the normal control group. This weight gain in the groups receiving 2.4 and 7.2 g/kg of KF was 79.30 % and 93.89 %, respectively, compared to the HFD-OLZ group (Table 1).

The BMI of obese mice treated with KF is shown in Table 2. A significant ($p < 0.001$) increase in BMI was observed in obese-untreated mice when compared with the normal control from the 2nd week onward. Animals treated with metformin and KF, both low dose (2.4 g/kg) and high dose (7.2 g/kg), showed significantly reduced BMI compared to the obese control group from week 3 onward.

Effects on adipose depots

At the end of the 8 week study period, the body fat mass of mice was assessed (Fig. 1). In the HFD-OLZ group, the total body fat and AI significantly increased ($p < 0.001$) compared with the normal control. The KF groups showed a decrease in VAT ($p < 0.05$). No distinction on SAT and AI was observed between the KF and HFD-OLZ groups.

Effects on blood lipid profile

The serum lipid profiles of obese mice receiving KF treatment are illustrated in Fig. 2. On the final day of the study, the serum concentrations of total cholesterol (TC), triglycerides (TG) and low density lipoprotein cholesterol (LDL-C) were significantly elevated in the HFD-OLZ group compared to the normal control group. In contrast, animals treated with metformin (200 mg/kg) exhibited a notable reduction in serum TC and LDL-C levels, along with a significant increase in serum high-density lipoprotein cholesterol (HDL-C) levels compared to untreated obese mice. The only notable difference in TG levels was found between KF-treated obese mice and the HFD-OLZ group. At doses of 2.4 and 7.2 g/kg/day, KF did not affect TC, HDL-C, or LDL-C levels.

Atherogenic indices of obese mice treated with KF are presented in Fig. 3. A significant ($p < 0.05$) increase in all the atherogenic indices was observed in obese-untreated animals compared to the normal control, animals treated with metformin and all the investigated doses of KF.

Effects on structure and function of the liver

Changes in serum liver function parameters, including AST and ALT levels, are indicated in Table 3. The levels of the liver function parameters were significantly increased in the HFD-OLZ group when compared with those in the normal control group. Oral administration of metformin at the dose of 200 mg/kg led to a decrease in the level of AST and ALT, with a significant difference

Table 1. Alterations in the average body weight gain (%) of mice within the experimental groups during the study

Groups	Average body weight gain (%)							
	1st week	2nd week	3rd week	4th week	5th week	6th week	7th week	8th week
Normal control	13.25 ±3.84***	24.48 ± 6.87***	37.07 ± 11.25***	50.88 ± 16.10***	59.78 ± 17.45***	66.07 ± 20.23***	69.63 ± 21.33***	62.64 ± 8.34***
HFD-OLZ	31.13 ± 7.28	61.64 ± 10.85	91.95 ± 10.23	103.40 ± 14.77	117.27 ± 13.23	115.43 ± 10.99	116.10 ± 14.47	119.41 ± 15.76
HO-Met	21.88 ± 6.98**	50.88 ±12.87*	72.74 ± 17.83**	78.41 ± 21.02**	90.40 ± 25.06**	91.36 ± 24.32**	89.61 ± 24.38**	94.51 ± 23.78**
HO-KFH	27.86 ± 8.09	60.91 ± 14.43	72.36 ± 20.76**	87.68 ± 21.75*	98.29 ± 20.22**	97.73 ± 20.07*	94.94 ± 13.47***	93.89 ± 19.11***
HO-KFL	26.85 ± 4.79	55.49 ± 11.04	67.11 ± 15.08***	78.92 ± 17.42**	87.76 ± 18.43***	83.35 ± 19.88***	79.03 ± 18.19***	79.30 ± 19.78***

*p < 0.05; **p < 0.01; ***p < 0.001 as compared with HFD-OLZ

Table 2. BMI of obese mice treated with KF for 8 weeks

BMI (g/cm²)	Normal control	HFD-OLZ	HO-Met	HO-KFH	HO-KFL
Baseline	0.28 ± 0.04	0.26 ± 0.02	0.28 ± 0.02	0.27 ± 0.02	0.27 ± 0.02
1st week	0.30 ± 0.03	0.31 ± 0.02	0.32 ± 0.03	0.32 ± 0.03	0.33 ± 0.03
2nd week	0.31 ± 0.03	0.37 ± 0.03***	0.36 ± 0.03	0.38 ± 0.03	0.38 ± 0.01
3rd week	0.33 ± 0.03	0.41 ± 0.03***	0.38 ± 0.03+	0.37 ± 0.05+	0.37 ± 0.02++
4th week	0.34 ± 0.04	0.42 ± 0.05***	0.38 ± 0.03+	0.38 ± 0.04+	0.38 ± 0.03+
5th week	0.34 ± 0.04	0.44 ± 0.04***	0.40 ± 0.03+	0.40 ± 0.04+	0.38 ± 0.03++
6th week	0.35 ± 0.04	0.42 ± 0.04***	0.39 ± 0.03+	0.39 ± 0.03+	0.37 ± 0.03++
7th week	0.33 ± 0.04	0.41 ± 0.04***	0.38 ± 0.03+	0.38 ± 0.02+	0.36 ± 0.02++
8th week	0.32 ± 0.03	0.40 ± 0.04***	0.37 ± 0.03+	0.37 ± 0.04+	0.35 ± 0.03++

++p < 0.01; +p < 0.05 compared to HFD-OLZ, ***p < 0.001 compared to normal control

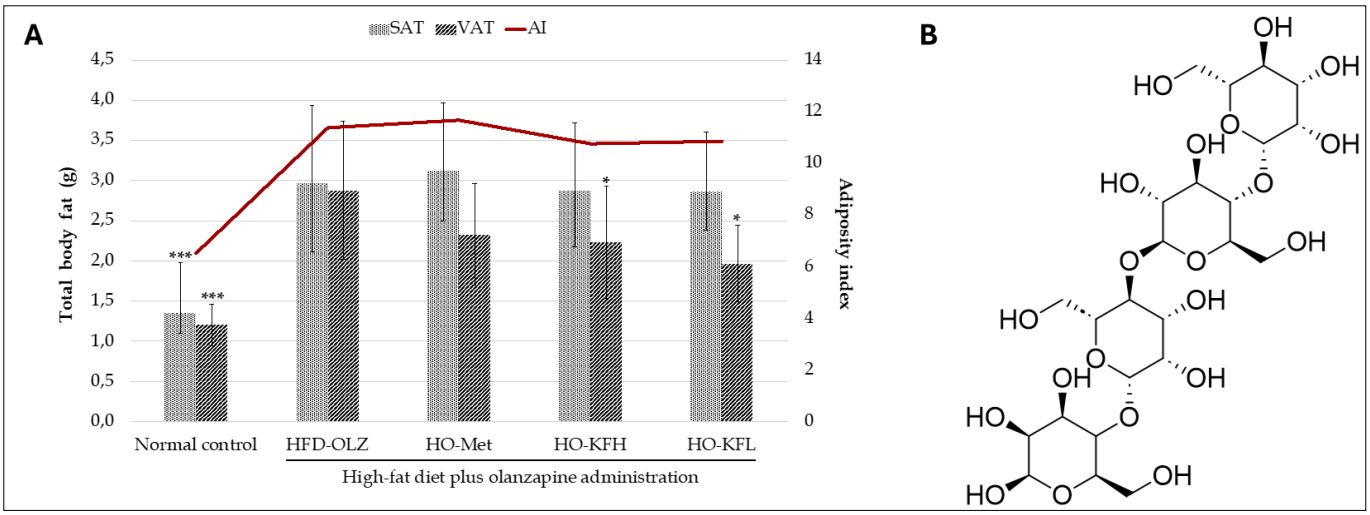

Fig. 1. (A) Effects of KF on Total Body Fat and AI; **(B)** Main component of Konjac glucomannan. AI: adiposity index; **SAT**: subcutaneous adipose tissue; **VAT**: visceral adipose tissue. ***p < 0.001; *p < 0.05 compared to HFD-OLZ.

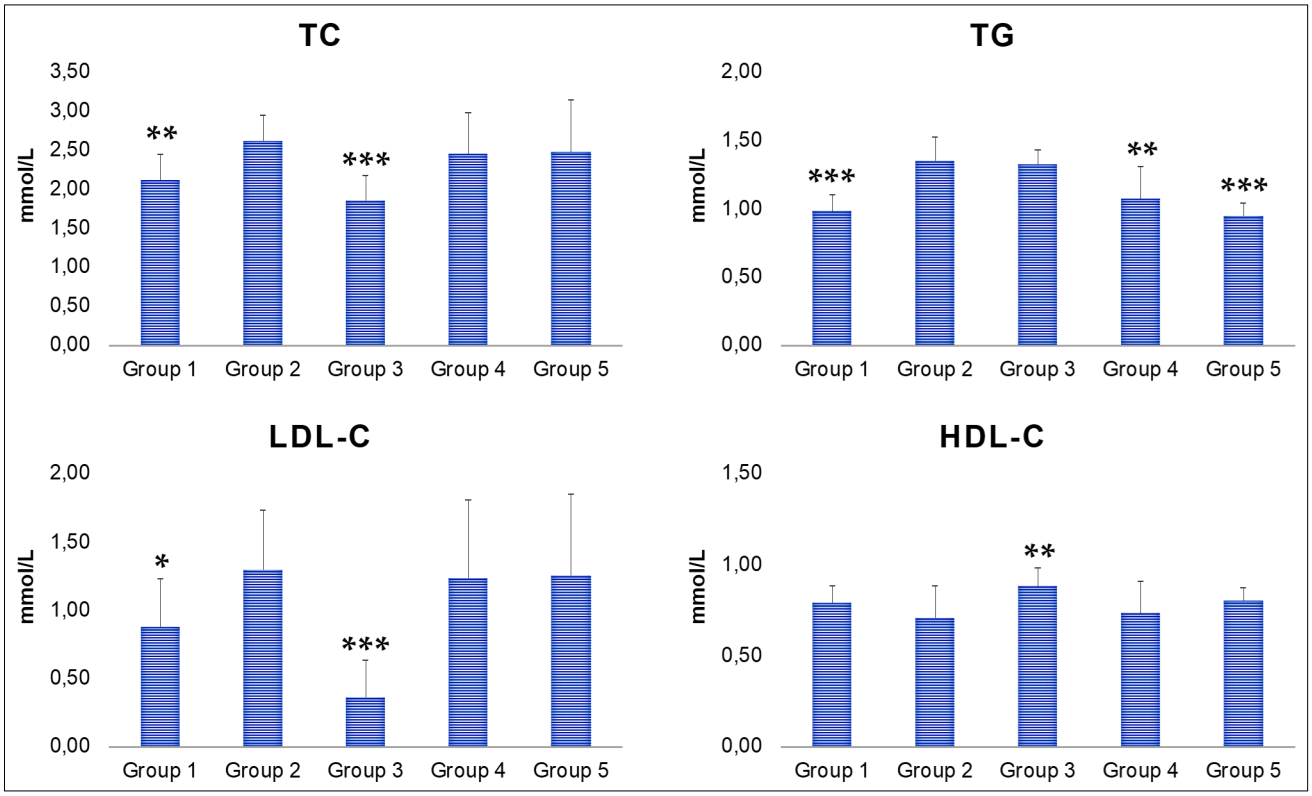

Fig. 2. Effects of KF on lipid profile. HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TC: total cholesterol; TG: triglycerides. ***p < 0.001; **p < 0.01; *p < 0.05 compared to HFD-OLZ (Group 2).

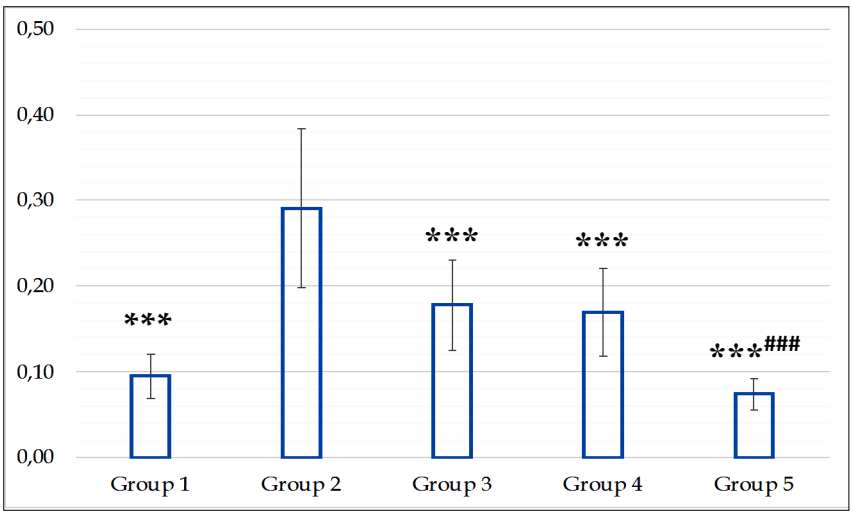

Fig. 3. Effects of KF on the atherogenic index of plasma. ***p < 0.001 compared to HFD-OLZ (Group 2); ≠≠≠p < 0,001 compared to HO-KFH (Group 4).

obtained only with ALT ($p<0.001$). KF administration at both doses significantly decreased the AST levels in HFD-OLZ mice. There was no significant difference in the ALT levels between the HFD-OLZ group and both KF-treated groups.

As presented in Fig. 4, the fat accumulation was increased significantly in the HFD-OLZ group in comparison with that observed in the normal control group. KF at both doses and metformin administration tended to decrease the fat accumulation in the mice with HFD plus OLZ-induced obesity.

Discussions

Obesity represents a significant global health concern, primarily resulting from an imbalance between energy consumption and expenditure. Additionally, the use of antipsychotic medications is another contributing factor to the development of obesity (15).

Table 3. Effect of KF on serum transaminase levels

Groups	AST (UI/L)	ALT (UI/L)
Normal control	170.70 ± 31.59*	58.40 ± 5.46**
HFD-OLZ	225.38 ± 60.39	71.23 ± 12.90
HO-Met	215.36 ± 54.97	53.64 ± 10.86***
HO-KFH	165.77 ± 35.94**	77.85 ± 23.79
HO-KFL	165.88 ± 23.01*	64.63 ± 16.47

*p < 0.05, **p < 0.01,***p < 0.001 compared to HFD-OLZ

Olanzapine is classified as an atypical (second-generation) antipsychotic and is commonly used to treat conditions such as schizophrenia, bipolar disorder and depression. While it is effective in managing these disorders, olanzapine is associated with a significant risk of weight gain and obesity, along with potential metabolic issues, including dyslipidemia and elevated blood glucose levels (16).

In this research, administering a high-fat diet (comprising 45 % of calories from fat) to mice resulted in an increase in both body weight and fat accumulation, specifically in subcutaneous and visceral abdominal areas. The impact was further intensified when olanzapine was present. As a result of the weight gain, the BMI in the HFD-OLZ (negative control) group was notably elevated compared to the normal control group. Metformin was chosen as a positive control to evaluate the anti-obesity effectiveness of KF. Metformin serves as the primary medication for managing type 2 diabetes by suppressing gluconeogenesis in the liver (17). Recent clinical trials have demonstrated the efficacy of metformin in countering weight gain associated with olanzapine; however, the underlying mechanism of action remains unclear (18-20) Metformin administration at doses ranging from 100 mg/kg to 300 mg/kg effectively inhibited weight gain induced by olanzapine in rodent models (21-23). In this investigation, metformin was given at a dosage of 200 mg/kg. The findings of this study indicated that metformin has a beneficial impact on alleviating metabolic and nutritional issues associated with a high-fat diet combined with olanzapine. Specifically, there was a reduction in body weight and a decrease in BMI. Additionally, dyslipidemia improved, evidenced by lower levels of

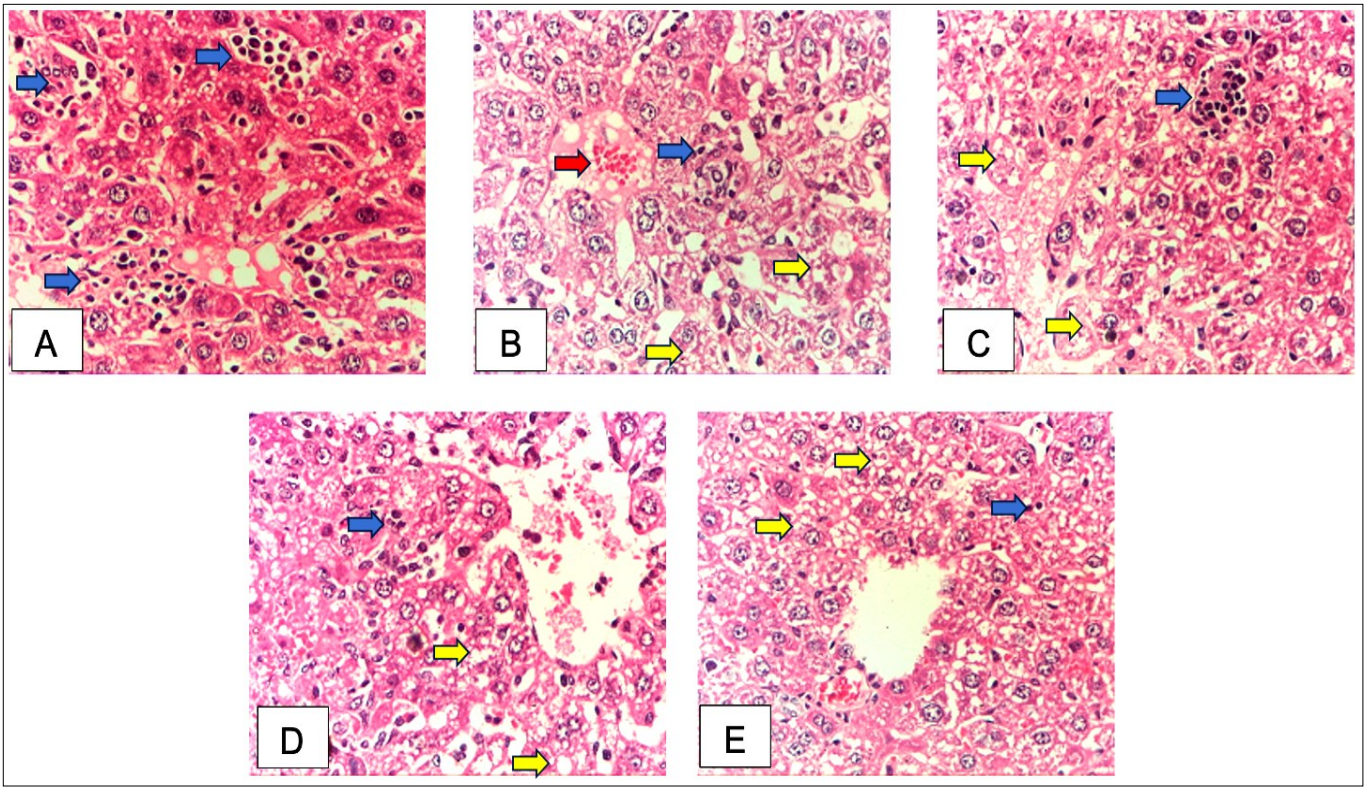

Fig. 4. Effect of KF on liver histology by hematoxylin and eosin staining. (A) Normal control: Mild hepatocyte degeneration, with inflammatory infiltrates (blue arrow); (B) HFD-OLZ: Severe vacuolar and fatty degeneration of hepatocytes (yellow arrow) with inflammatory infiltrates (blue arrow). Blood vessels are engorged with red blood cells and fat droplets (red arrow); (C) HO-Met: Hepatocytes with severe vacuolar degeneration (yellow arrow), with limited areas of fatty degeneration and many inflammatory cells infiltrated forming large foci (blue arrow); (D) HO-KFH: Hepatocytes with severe vacuolar degeneration, moderate fatty degeneration (yellow arrow), accompanied by inflammatory infiltrates (blue arrow); (E) HO-KFL: Hepatocytes with moderate vacuolar degeneration, few areas of fatty degeneration (yellow arrow), with inflammatory infiltrates (blue arrow).

total cholesterol and non-HDL-C, alongside an increase in HDL-C, which was accompanied by a reduction in the atherogenic index. Furthermore, metformin helped to limit fat accumulation in the liver and decreased transaminase activity, particularly ALT.

Konjac (*Amorphophallus konjac* K. Koch) is a perennial herb belonging to one of the largest genera in the Araceae Family, originating from China and the subtropical regions of Southeast Asia (5). Initially, it was valued for its medicinal properties (such as lowering lipid and sugar levels, combating obesity, promoting wound healing and providing anti-inflammatory benefits). Konjac tubers gradually evolved into food ingredients (5-8). It is noteworthy that konjac is the only plant identified globally with a high glucomannan content, which can account for nearly 50 % of the dry weight of its bulb (5). For purposes of weight reduction and the management of type 2 diabetes, insulin-resistance syndrome and dyslipidemia, a daily intake of glucomannan ranging from 3 to 13 grams is advised (24). The recommended dosage for a typical 50 kg individual of the konjac flour product utilized in this research was 10 g daily. Utilizing a human-to-mouse conversion factor of 12 (25), the dosages administered to assess the weight loss effects of konjac flour in mice were 2.4 and 7.2 g/kg/day, which align with the intended dosage and a threefold increased dosage respectively.

Weight reduction is a primary objective in the management of obesity, as studies indicate that losing weight in overweight or obese individuals correlates with a decreased risk of mortality (26). Monitoring the weekly weight change, we observed that the body weight of mice treated with metformin and KF groups increased more slowly than in the HFD-OLZ group from the first study week and a significant decrease was observed after three weeks of administration (Table 1). Corresponding to the reduction in the percentage of weight gain, the BMI in the KF groups was also significantly lower than that of the HFD-OLZ group after three study weeks (Table 2). The accumulation of body fat in mice treated with KF was also less than that of the HFD-OLZ mice, especially visceral abdominal fat, which was significantly reduced in the obese mice treated with KF compared with the untreated obese mice (Fig. 1). Several studies have also provided evidence of the safety and effectiveness of konjac flour in weight loss in experimental animals and humans (27, 28). KGM is recognized as one of the most viscous fibers, possessing an average molecular weight of approximately one million daltons. It has the capacity to absorb water up to 50 times its own weight, leading to expansion within the digestive system. This bulking characteristic contributes to a sensation of satiety, which can decrease appetite and slow the absorption process in the intestines (28).

Obesity is more than just weight gain; it is a risk factor for many other diseases, including dyslipidemia. Appropriate treatment for obesity, in addition to weight control, should include the management of this complication (29). In our study, the combined effects of a high-fat diet and olanzapine increased serum lipid parameters such as TG, total cholesterol and non-HDL. This dyslipidemia increases the risk of developing cardiovascular diseases, especially atherosclerotic cardiovascular diseases, with a high atherogenic index in the negative control group. The data in Fig. 2 showed the tendency of KF to correct dyslipidemia in obese mice, in which the test product showed the highest effect on the serum triglyceride levels. KF's effect of reducing the levels of lipid parameters that increase the risk of atherosclerosis may help reduce the risk of cardiovascular disease with a significantly reduced atherogenic index of plasma compared to the HFD-OLZ group ($p < 0.001$) (Fig. 3). Research involving human subjects has documented the impact of konjac flour on dyslipidemia regulation. Findings consistently indicate its efficacy in lowering blood lipid levels associated with an increased risk of atherosclerosis, including total cholesterol, LDL- C and triglycerides (TG) (30, 31).

Obesity leads to the accumulation of excess fat in the liver, resulting in fatty liver disease. If not addressed, this condition can progress to hepatitis, cirrhosis, disorders of fat metabolism and a heightened risk of developing gallstones (32). The concentration of biochemical markers indicating the degree of liver damage (AST, ALT) rose in the HFD-OLZ group and lipid accumulation in liver tissues was higher than in the normal control group. KF at both administered doses demonstrated a significant reduction in serum AST levels and a decrease in fatty degeneration of liver tissue. These results indicate that KF may effectively diminish fat accumulation in the liver and help avert liver damage associated with steatohepatitis.

Conclusion

Refined konjac flour has shown weight loss effects in obese mice induced by a high-fat diet plus olanzapine. KF at doses of 2.4 g/kg and 7.2 g/kg reduced weight gain and BMI, lowered body fat accumulation, decreased blood triglyceride (TG) levels, improved fatty liver degeneration and prevented liver damage related to steatohepatitis. KF may be developed into a highly effective natural product, thereby providing benefits to a greater number of patients experiencing metabolic changes induced by antipsychotic medications.

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Authors' contributions

All authors collaborated on the study's conception and design. VAPT, THDT and PTM prepared the materials, collected data and analyzed it. THDT and PTM authored the first draft of the manuscript. VAPT, XPP, TTT, TDD, HAD, TCN, TTP, VHV and PTM provided feedback on earlier versions. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: There are no conflicts of interest that the authors need to report.

Ethical issues: The scientific committee of Hanoi Medical University, Hanoi, Vietnam, approved all experimental procedures (Decision No. 775/QĐ-ĐHYHN, issued on April 8, 2022).

References

1. World Health Organization. Obesity and overweight fact sheet. 2024. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

2. Ansari S, Haboubi H, Haboubi N. Adult obesity complications: challenges and clinical impact. Ther Adv Endocrinol Metab. 2020;11:2042018820934955. https://doi.org/10.1177/2042018820934955
3. Ruban A, Stoenchev K, Ashrafian H, Teare J. Current treatments for obesity. Clin Med (Lond). 2019;19:205-12. https://doi.org/10.7861/clinmedicine.19-3-205
4. Aziz MA, Millat MS, Akter T, Hossain MS, Islam MM, Mohsin S, et al. A comprehensive review on clinically proven medicinal plants in the treatment of overweight and obesity, with mechanistic insights. Heliyon. 2023;9(2):e13493. https://doi.org/10.1016/j.heliyon.2023.e13493
5. Jain A, Sarsaiya S, Gong Q, Wu Q, Shi J. *Amorphophallus konjac*: traditional uses, bioactive potential and emerging health applications. Front Plant Sci. 2025;16:1530814. https://doi.org/10.3389/fpls.2025.1530814
6. Behera S, Ray R. Nutritional and potential health benefits of konjac glucomannan, a promising polysaccharide of elephant foot yam, *Amorphophallus konjac* K. Koch: review. Food Rev Int. 2016;33(1):22-43. https://doi.org/10.1080/87559129.2015.1137310
7. Devaraj RD, Reddy CK, Xu B. Health-promoting effects of konjac glucomannan and its practical applications: a critical review. Int J Biol Macromol. 2019;126:273-81. https://doi.org/10.1016/j.ijbiomac.2018.12.203
8. Mah E, Chen O, Liska DJ, Blumberg JB. Dietary supplements for weight management: a narrative review of safety and metabolic health benefits. Nutrients. 2022;14(9):1787. https://doi.org/10.3390/nu14091787
9. National Research Council (US) Committee for the Update of the Guide for the Care and Use of Laboratory Animals. Guide for the care and use of laboratory animals. 8th ed. Washington (DC): National Academies Press (US); 2011. https://doi.org/10.17226/12910
10. Shertzer HG, Kendig EL, Nasrallah HA, Johansson E, Genter MB. Protection from olanzapine-induced metabolic toxicity in mice by acetaminophen and tetrahydroindenoindole. Int J Obes (Lond). 2010;34(6):970-9. https://doi.org/10.1038/ijo.2009.291
11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-502.
12. Karimpour Reyhan S, Yadegar A, Samimi S, Nakhaei P, Esteghamati A, Nakhjavani M, et al. Atherogenic index of plasma (AIP): the most accurate indicator of overweight and obesity among lipid indices in type 2 diabetes-findings from a cross-sectional study. Endocrinol Diabetes Metab. 2024;7(6):e70007. https://doi.org/10.1002/edm2.70007
13. Martins T, Castro-Ribeiro C, Lemos S, Ferreira T, Nascimento-Gonçalves E, Rosa E, et al. Murine models of obesity. Obesities. 2022;2(2):127-47. https://doi.org/10.3390/obesities2020012
14. Slaoui M, Bauchet AL, Fiette L. Tissue sampling and processing for histopathology evaluation. Methods Mol Biol. 2017;1641:101-14. https://doi.org/10.1007/978-1-4939-7172-5_4
15. Nasrallah H. A review of the effect of atypical antipsychotics on weight. Psychoneuroendocrinology. 2003;28 Suppl 1:83-96. https://doi.org/10.1016/S0306-4530(02)00114-2
16. Bhana N, Foster RH, Olney R, Plosker GL. Olanzapine: an updated review of its use in the management of schizophrenia. Drugs. 2001;61:111-61. https://doi.org/10.2165/00003495-200161010-00011
17. Viollet B, Guigas B, Sanz Garcia N, Leclerc J, Foretz M, Andreelli F. Cellular and molecular mechanisms of metformin: an overview. Clin Sci (Lond). 2012;122:253-70. https://doi.org/10.1042/CS20110386
18. Maayan L, Vakhrusheva J, Correll CU. Effectiveness of medications used to attenuate antipsychotic-related weight gain and metabolic abnormalities: a systematic review and meta-analysis. Neuropsychopharmacol. 2010;35:1520-30. https://doi.org/10.1038/npp.2010.21
19. Kumar GC, Sarala N, Reddy MM, Manjunath H. Effect of metformin on olanzapine-induced weight gain: a 12-week randomised open-label study. J Clin Sci. 2025;22(1):11-7. https://doi.org/10.4103/jcls.jcls_62_24
20. Hiluy JC, Nazar BP, Gonçalves WS, et al. Effectiveness of pharmacologic interventions in the management of weight gain in patients with severe mental illness: a systematic review and meta-analysis. Prim Care Companion CNS Disord. 2019;21(6):19r02483. https://doi.org/10.4088/PCC.19r02483
21. Singh R, Bansal Y, Sodhi RK, Singh DP, Bishnoi M, Kondepudi KK, et al. Berberine attenuated olanzapine-induced metabolic alterations in mice: targeting transient receptor potential vanilloid type 1 and 3 channels. Life Sci. 2020;247:117442. https://doi.org/10.1016/j.lfs.2020.117442
22. Woods SE, Genter MB, Shertzer HG. A comparison of the effectiveness of metformin and acetaminophen in preventing olanzapine toxicity in mice. Pharmacologia. 2012;3(7):233-8. https://doi.org/10.5567/pharmacologia.2012.233.238
23. Kim J, Lee N, Suh SB, Jang S, Kim S, Kim DG, et al. Metformin ameliorates olanzapine-induced disturbances in POMC neuron number, axonal projection and hypothalamic leptin resistance. BMB Rep. 2022;55(6):293-8. https://doi.org/10.5483/BMBRep.2022.55.6.026
24. Keithley J, Swanson B. Glucomannan and obesity: a critical review. Altern Ther Health Med. 2005;11(6):30-4.
25. Nair AB, Jacob S. A simple practice guide for dose conversion between animals and human. J Basic Clin Pharm. 2016;7(2):27-31. https://doi.org/10.4103/0976-0105.177703
26. Poobalan AS, Aucott LS, Smith WCS, Avenell A, Jung R, Broom J. Long-term weight loss effects on all cause mortality in overweight/obese populations. Obes Rev. 2007;8:503-13. https://doi.org/10.1111/j.1467-789X.2007.00393.x
27. Aoe S, Kudo H, Sakurai S. Effects of liquid konjac on parameters related to obesity in diet-induced obese mice. Biosci Biotechnol Biochem. 2015;79:1141-6. https://doi.org/10.1080/09168451.2015.1018119
28. Keithley JK, Swanson B, Mikolaitis SL, DeMeo M, Zeller JM, Fogg L, et al. Safety and efficacy of glucomannan for weight loss in overweight and moderately obese adults. J Obes. 2013;2013:610908. https://doi.org/10.1155/2013/610908
29. Ministry of Health, Vietnam. Guidelines for diagnostic and treatment of obesity. Issued with Decision No. 2892/QD-BYT. 2022.
30. Zhang MY, Huang CY, Wang X, Hong JR, Peng SS. The effect of foods containing refined konjac meal on human lipid metabolism. Biomed Environ Sci. 1990;3:99-105.
31. Arvill A, Bodin L. Effect of short-term ingestion of konjac glucomannan on serum cholesterol in healthy men. Am J Clin Nutr. 1995;61:585-9. https://doi.org/10.1093/ajcn/61.3.585
32. Fabbrini E, Sullivan S, Klein S. Obesity and nonalcoholic fatty liver disease: biochemical, metabolic and clinical implications. Hepatology. 2010;51(2):679-89.

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