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Peaches (*Prunus persica* L.) pulp mitigate type 2 diabetic mice by modulating of glucose metabolism and gut microbiota

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ABSTRACT

Peach are a fruit with high nutritional and economic value, but their safety and suitability for diabetic patients have been questioned. This study investigated the effects and potential mechanisms of peach pulp (PP) on type 2 diabetes mellitus (T2DM) in mice induced by a high-fat diet (HFD) combined with streptozotocin (STZ). The results showed that PP alleviated hyperglycemia, hyperlipidemia, hyperuricemia, and tissue dysfunction in T2DM mice through the synergistic effect of nutrients and non-nutrient compounds. Analysis of mRNA expression levels revealed that PP improved glucose metabolism in T2DM mice by promoting glycogen synthesis and inhibiting gluconeogenesis. Furthermore, elevated levels of PP resulted in an increase in acetic acid content following a 4 weeks intervention period. Additionally, it led to the restoration of gut microbiota balance by decreasing the Firmicutes/Bacteroidota (F/B) ratio and enhancing the presence of *Romboutsia*, *Allobaculum*, *Alloprevotella*, and *Bacteroides* after an 8 weeks intervention. Ultimately, our results suggest that PP may offer advantages for individuals with diabetes.

1. Introduction

Type 2 diabetes mellitus (T2DM) is a non-insulin-dependent form of diabetes characterized by hyperglycemia resulting from insulin resistance and relative insulin deficiency[1]. According to the International Diabetes Federation, approximately 537 million adults aged 20 to 79 years worldwide were affected by diabetes in 2021. This number is projected to increase to 643 million by 2030 and 783 million by 2045, with T2DM accounting for more than 90% of all diabetes cases[2]. Complications arising from T2DM, such as cardiovascular disease, end-stage renal disease, and vitreoretinopathy, pose significant threats to both patient health and public health services. Given the high costs and side effects associated with traditional antidiabetic medications, dietary interventions involving whole foods are increasingly recognized as primary prevention methods for diabetes[3].

Studies have revealed that dietary education intervention could not only significantly reduce the body mass index, glycosylated hemoglobin, and fasting blood glucose (FBG) levels, but also decrease the risk of diabetic complications[4]. The current popular low-carb and ketogenic diets, vegan, and Mediterranean diets have been shown to improve glycemic control[5]. It is worth noting that eating the right amount of fruit was a common feature of vegan and Mediterranean diets. However, the traditional concept was that fruits have a high sugar content, and eating fruits will lead to the deterioration of glycemic management, resulting in diabetic patients only eating a limited variety of low-sugar fruits, such as cucumbers[6].

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Conversely, studies have shown that consumption of dragon fruit has no significant effect on FBG and 2-h postprandial blood glucose in T2DM patients compared with the non-intervention group[7]. In fact, fresh fruits are rich in polysaccharides, organic acids, and antioxidants such as vitamins and polyphenols, which help to regulate the composition and metabolic activity of gut microbiota and reduce the deterioration of diabetes and its complications[6]. In 3 prospective cohort studies, the consumption of fruits such as apples, bananas, and grapefruit were significantly negatively correlated with the risk of T2DM[8]. In addition, there was a significant negative correlation between fruit intake and insulin sensitivity markers, which means that the consumption of fruits helps to improve hyperinsulinemia in T2DM patients[9].

Peach (*Prunus persica* L.) is an important kind of Rosaceae family fruit and has always been a part of the human diet worldwide. Research has reported that this fruit is rich in polysaccharides, polyphenols, vitamins, organic acids, and other phytochemicals, and has an excellent substance basis for hypoglycemic activity[10]. *In vitro* and *in vivo* studies have revealed that polyphenols extracted from peaches have excellent antioxidant, hypoglycemic, and hypolipidemic properties and improve obesity in high-fat diet (HFD) feeding mice by affecting the structure and composition of gut microbiota, thereby preventing pre-diabetes symptoms[11-12]. Research has studied the antioxidant, anti-glycation and anti-inflammatory activities of three different peach-based suspensions in fresh peach pulp, peel, and preserved peach pulp. Results showed that fresh peach pulp and peel had good antioxidant and anti-inflammatory effects in rat liver, kidney and cerebral cortex[13]. In addition, in a previous study, we found that polysaccharides extracted from peaches played an important anti-diabetic role in the construction of diabetic mice induced by HFD/streptozotocin (STZ)[14].

Despite the beneficial effects of bioactive compounds and various peach extracts on diabetes-related metabolism, there is no direct evidence certifying whether whole peaches exhibit positive effects on hypoglycemic properties. Furthermore, whole peaches and peach pulp (PP) represent the main forms of peach consumption, with PP offering greater convenience in food applications, thus attracting more attention. Therefore, this study aimed to evaluate the comprehensive regulatory effects of PP on T2DM by assessing variations in physiological, histological, and biochemical parameters in diabetic mice induced HFD/STZ. Additionally, we analyzed the effect of PP on the structure and composition of gut microbiota to explore potential underlying mechanisms. The novelty of this study lies in the discovery of the beneficial effects of PP on T2DM, which were achieved through improvements in glucose metabolism and modulation of gut microbiota. These findings confirm the positive impact of PP on diabetes, enhance its safety and acceptability for diabetic patients, and offer guidance for the development of PP products.

2. Materials and methods

2.1 Plant materials and chemicals

Peaches (*Prunus persica* L. cv. Hujingmilu), used in the present study, were harvested at commercial maturity in Fenghua, Ningbo, Zhejiang Province, China. Normal diets (D12450J, 10 kcal% fat, 3.85 kcal/g) and high-fat diet (D12492, 60 kcal% fat, 5.24 kcal/g) were purchased from Research Diets, Inc. (New Brunswick, NJ, America). STZ (CAS: 18883-66-4), acetic acid (CAS: 64-19-7), propionic acid (CAS: 79-09-4), *n*-butyric acid (CAS: 107-92-6), *i*-butyric acid (CAS: 79-31-2), *n*-valeric acid (CAS: 109-52-4), *i*-valeric acid (CAS: 503-74-2) and eosin (CAS: 15086-94-9) were obtained from Solarbio Biochemical Technology Co., Ltd. (Beijing, China). Hematoxylin (CAS: 517-28-2) was supplied by Sigma-Aldrich (St. Luis, MO, USA). All other reagents were of analytical grade and purchased from Shanghai Chemicals and Reagents Co., Ltd. (Shanghai, China).

2.2 Preparation and composition analysis of PP

The fruit firmness, total soluble solids (TSS), and titratable acidity (TA) of whole peaches were determined using previously described methods after harvesting[15]. Additionally, the whole peaches were decorticated, pitted, frozen with liquid nitrogen, and crushed into powder form. This powder was then freeze-dried in a vacuum freeze dryer to obtain PP. The PP powder was stored in brown drying dishes at room temperature for subsequent analysis and usage. The moisture, ash, protein, polysaccharide, flavonoid, and total phenol contents of PP were measured according to a previous report[16].

The equivalents of total phenols and flavonoids were calculated using gallic acid and rutin, respectively. The content of soluble sugars was analyzed using a modified version of a previously reported method[17]. Briefly, a 0.1 g/mL PP homogenate prepared with deionized water was stirred at 80 ℃ for 30 min and then centrifuged at 12 000 r/min for 10 min to obtain the supernatant. The supernatant passed through a 0.22 μm aqueous membrane for high-performance liquid chromatography (HPLC) analysis. The HPLC system (Model 2695, Waters, USA) utilized a Sugarpak1 column (6.5 mm × 300 mm) equipped with a differential refractive index detector. A 10 μL sample was injected into the system, with water serving as the mobile phase at a total flow rate of 0.4 mL/min, and the column temperature maintained at 85 ℃.

2.3 Antioxidant capacity of PP

According to the reported method, the antioxidant capacity of PP was quantified using 2,2-Diphenyl-1-picrylhydrazyl (DPPH) and 2,2′-azino-bis (3-ethylbenzothiazoline-6-sulfonic acid) (ABTS) free radical scavenging assays. Ascorbic acid served as the positive control, and the IC_{50} value was calculated using the non-linear regression method after transforming concentrations to logarithms[18].

2.4 Animals and experimental design

A totaly of 32 5-week-old male C57BL/6J mice ((19 ± 1) g) were obtained from Vital River Laboratory Animal Technology Co., Ltd. (Zhejiang, China). All mice used in the experiment were treated in strict accordance with China Legislation and approved by the Welfare and Ethics Committee at the Laboratory Animal Center of Ningbo University (Approval No. NBU20220093). They were housed under conditions of relative humidity of (50 ± 10)% , a temperature of (23 ± 1) ℃ , a 12-h light-dark cycle, and provided with ad libitum access to food and water. The establishment of the diabetic mouse model was carried out according to a previously published method, as illustrated in Fig. 2A[19]. Briefly, after 1 week of acclimation, the mice

were randomly divided into 2 groups for 5 weeks of dietary manipulation: a normal diet group (n = 8) and a high-fat diet (HFD) group (n = 24). Mice in the HFD group were intraperitoneally injected with a 1% cold STZ solution (dissolved in 0.1 mol/L freshly prepared citrate buffer, pH = 4.5) at a dose of 50 mg/kg BW for 5 days, while mice in the normal group received an equal volume of cold citrate buffer intraperitoneally. Three days after STZ injection, FBG levels were measured using an ACCU-CHEK glucometer (Roche Diagnostics, Mannheim, Germany) *via* tail-cleavage. Mice with FBG levels ≥ 11.1 mmol/L, accompanied by symptoms of polydipsia, polyuria, and weight loss, were considered successful T2DM mouse models (Fig. S1).

Subsequently, the T2DM mice were randomly divided into 3 groups (n = 8 per group) to receive different interventions by gastric gavage for 8 weeks: a diabetes model group (DM, administered with an equal volume of 0.9% sterile saline), a low-dose PP group (LP, administered with 1.0 g/kg BW PP), and a high-dose PP group (HP, administered with 2.0 g/kg BW PP). The normal control (NC) group of mice received an equal volume of 0.9% sterile saline throughout the experimental intervention process. Throughout the experiment, food intake and water consumption were determined daily, and body weight (BW) and FBG levels were recorded weekly. At the end of the experiment, all mice fasted for 12 h and were euthanized by cervical dislocation after anesthesia with isoflurane. Biological samples such as liver, kidney, and pancreas were collected for histological and biochemical analysis. The liver index was expressed by calculating the ratio of liver weight to body weight.

2.5 Oral glucose tolerance test (OGTT)

In the 8th week, OGTT was performed after an overnight fast. FBG was measured using the tail-cleavage method and served as the baseline blood glucose at 0 min. Subsequently, each mouse received a 20% glucose solution (2.0 g/kg BW) by gastric gavage, and blood glucose concentrations were monitored at 30, 60, 90, and 120 min. The area under the curve (AUC) of glucose was calculated using GraphPad Prism 9.0 (GraphPad Software, La Jolla, USA) according to a previously reported method[20].

2.6 Serum biochemical parameter and hepatic glycogen analysis

Serum was collected from each mouse using a previously reported method[21]. Serum insulin levels were determined using a commercial insulin ELISA kit (Qiaodu, Shanghai, China) according to the manufacturer's instructions. Homeostasis model assessment of insulin resistance (HOMA-IR) and homeostasis model assessment of β-cell function (HOMA-β) were calculated using FBG and insulin levels as previously reported[22]. An automatic biochemical analyzer (Beckman Coulter AU5800, California, USA) was used to detect serum levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein (TP), albumin (ALB), uric acid (UA), and urea. Additionally, the level of globulin (GLO) was calculated by the difference between TP and ALB, and the albumin-to-globulin ratio (A/G) was calculated as the ratio of ALB to GLO.

Moreover, hepatic glycogen was determined using a previously reported method with slight modifications[23]. Briefly, a 0.15 g liver sample was added to 1.5 mL of 30% KOH solution and extracted at 100 ℃ for 30 min. The mixture was vortexed once every 10 min and then 0.5 mL of 0.1 mol/L Na_2SO_4 and 1.5 mL of anhydrous ethanol were added. The mixture was centrifuged at 12 000 r/min for 5 min, and the precipitated glycogen was washed with 1.5 mL of anhydrous ethanol. The precipitate was dissolved in 1.5 mL of deionized water, and the glycogen concentration was determined at 620 nm by anthrone colorimetry and normalized to the liver weight.

2.7 Histological examination

Histopathological analysis of the liver, kidney, and pancreas was conducted using hematoxylin-eosin (H&E) staining, and periodic acid-Schiff (PAS) staining was employed for hepatic glycogen analysis[1]. Tissues were fixed in 4% paraformaldehyde solution (Beyotime, Shanghai, China) overnight at 4 ℃, dehydrated in a series of ascending ethanol concentrations, transparentized in xylene, embedded in paraffin, and sectioned into 4 μm slices. Sections were then deparaffinized in xylene and rehydrated in descending ethanol concentrations. Subsequently, they were stained with H&E and PAS staining solution, sealed with neutral resin, and observed under a microscope (Olympus CX-21, Tokyo, Japan).

2.8 Quantitative real-time PCR

Total RNA was extracted from the liver to evaluate the relative mRNA expression levels of *PEPCK*, *G6Pase*, *GSK-3β*, and *FoxO1* using quantitative real-time PCR, as previously reported[14]. *GAPDH* served as the reference gene, and the relative levels of these genes were calculated using the $2^{-\Delta\Delta CT}$ method. Primer sequences were listed in Table S1 and sequenced by Youkang Biotechnology Co., Ltd. (Hangzhou, China).

2.9 pH and short-chain fatty acids (SCFAs) of feces

The pH and SCFAs of mouse feces in different periods were measured as previous described with slight modifications[24]. Fecal samples were diluted with deionized water at a ratio of 1:9 (m/V) and mixed intermittently for 5 min, followed by sonication at 20 ℃ for 5 min. The samples were then placed in ice water for 20 min and centrifuged at 4 ℃, 8 000 × g, for 20 min. The supernatant was transferred for pH measurement (FE28, METTLER TOLEDO, China). For SCFA analysis, fecal samples were similarly diluted at a ratio of 1:5 (m/V) with deionized water, followed by the same procedures. The supernatant was analyzed by gas chromatography (GC) after passing through a 0.22 μm aqueous membrane. The analytical conditions of GC are shown in Table S2.

2.10 Gut microbiota analysis by 16S sequencing

Genomic DNA of gut microbiota from fecal samples was extracted and sequenced by LC-Bio Technology Co., Ltd. (Hangzhou, China). The cetyltrimethylammonium bromide (CTAB) method was used for DNA extraction, and the V3 − V4 hypervariable regions of 16S rDNA were amplified with 341F (5′-CCTACGGGNGGCWGCAG-

3′) and 805R (5′-GACTACHVGGGTATCTAATCC-3′) primers according to the manufacturer's instructions. After quantification by Qubit (Invitrogen, Waltham, USA), paired-end sequencing (2 × 250 bp) was performed on NovaSeq PE250 platforms (Illumina, San Diego, USA) to obtain amplicon sequence variants (ASVs). Data analysis was performed based on a previous method[25]. Additionally, Spearman's clustering correlation heatmap and network map were used to evaluate the correlation between T2DM-related indicators and gut microbiota at the genus level using OmicStudio tools (https://www.omicstudio.cn).

2.11 Statistical analysis

All results were expressed as means ± standard error (mean ± SE). Statistical differences were analyzed using two-tailed Student's *t*-test and one-way ANOVA followed by Scheffe multiple comparisons tests conducted by SPSS software (Version 26.0, Chicago, USA). A *P*-value of < 0.05 was considered statistically significant. The results were plotted using GraphPad Prism 9.0 (GraphPad Software, La Jolla, USA), and different letters indicated significant differences. All experiments were performed in triplicate at least.

3. Results

3.1 Characterization of peach fruits and pulp

The levels of fruit firmness, total soluble solids (TSS), and titratable acidity (TA) were measured at 50.44 N, 15.05° Bx, and 0.28%, respectively. The PP powder contained 6.92% moisture, 15.02% polysaccharides, 0.67% polyphenols, 0.22% protein, and 3.55% ash. Soluble sugars in PP varied, with sucrose being the most dominant type at 45.94%, followed by glucose (10.82%), fructose (9.06%), and sorbitol (4.48%) (Table 1). These results from the characterization of fresh peach fruits and dried pulp contribute to further research on the physiological and metabolic effects of PP on health.

Table 1
Quality of peach fruit and chemical composition of PP.

Items	Levels
Quality of fresh peach fruit	
Moisture (%)	85.84 ± 0.58
Firmness (N)	50.44 ± 1.00
TSS (°Bx)	15.05 ± 0.37
TA (%)	0.28 ± 0.02
Composition of PP (dry basis, *m*/*m*, %)	
Moisture	6.92 ± 0.05
Polysaccharide	15.02 ± 0.27
Total phenols (gallic acid)	0.67 ± 0.01
Total flavonoids (rutin)	1.66 ± 0.37
Protein	0.22 ± 0.00
Ash	3.55 ± 0.03
Sucrose	45.94 ± 0.18
Glucose	10.82 ± 0.08
Fructose	9.06 ± 0.03
Sorbitol	4.48 ± 0.00

3.2 Antioxidant capacity of PP

As depicted in Fig. 1, PP demonstrated effective capacity in scavenging DPPH and ABTS free radicals, with antioxidant capacity positively correlated with the tested concentration. At experimental concentrations, the scavenging rate of PP on DPPH and ABTS free radicals was significantly lower than that of ascorbic acid ($P < 0.01$), but both showed similar scavenging abilities at the maximum test concentration, exceeding 90%. At a concentration of 5.0 mg/mL, the DPPH free radical scavenging rates of PP and ascorbic acid were 94.00% and 96.36%, respectively (Fig. 1A). At a concentration of 20.0 mg/mL, the ABTS free radical scavenging rates were 92.30% and 93.39%, respectively (Fig. 1B). Meanwhile, the IC_{50} values of PP for DPPH and ABTS free radicals were 0.52 and 5.02 mg/mL, respectively. These results demonstrate the effective antioxidant capacity of PP.

3.3 Effect of PP on nutrient consumption, body weight, liver weight

In Table 2, it is observed that the DM group showed no symptoms of polyphagia compared to the NC group. Furthermore, PP intervention did not significantly affect the food intake of diabetic mice at any experimental doses ($P > 0.05$). The daily water consumption of the DM group was significantly higher than that of the NC group ($P < 0.01$). However, after PP intervention, no significant effects were observed in water consumption compared to the DM group ($P > 0.05$). The BW of the DM group continued to decline compared to the NC group with the progression of diabetes ($P < 0.01$). Notably, PP intervention ameliorated the trend of emaciation in the early stage (0–3 weeks). Furthermore, PP intervention reversed the trend of emaciation, with the change in BW mirroring that of the NC group in the later stage (4–8 weeks). At the end of the intervention, a significant difference in BW was observed between the LP and HP groups compared to the DM group ($P < 0.01$). These results indicate that PP did not exacerbate symptoms of polyphagia and polydipsia in T2DM mice, and the positive effect of PP on BW was not associated with nutrient consumption but may be related to the potential functional substances contained in PP.

Liver weight was measured after dissection to evaluate the effect of PP on abnormal hepatomegaly in T2DM mice (Fig. 2C). The liver weight in the DM group was notably higher, indicating hepatomegaly compared to the NC group ($P < 0.01$). PP intervention decreased the liver weight of diabetic mice, particularly in the HP group, followed by the LP group ($P < 0.01$). The trend of the liver index was consistent with the results of liver weight (Fig. 2D). These results suggest that PP has a beneficial effect on abnormal hepatomegaly in a dose-dependent manner.

3.4 Effects of PP on FBG and glucose homeostasis

After fasting overnight, FBG was measured weekly *via* tail cleavage to evaluate the effect of PP on blood glucose levels. As depicted in Fig. 2B, over the 8-week monitoring period, FBG levels in diabetic mice were significantly higher than those in normal mice ($P < 0.01$). Initially, all diabetic mice exhibited a fluctuating upward

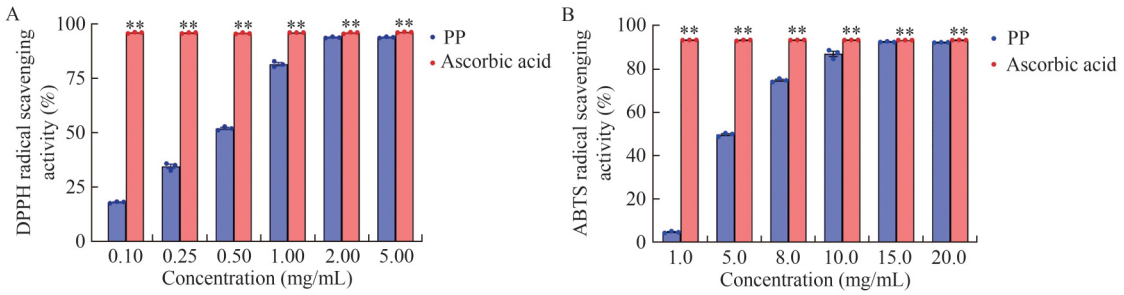

Fig. 1 Antioxidant capacity of PP. (A) DPPH radical scavenging activity, (B) ABTS radical scavenging activity. Data were presented as the means ± SE. Significant differences were indicated as $^{**}P < 0.01$ comparison with the PP group.

trend in FBG levels. Following 4 weeks of PP administration, FBG levels in the PP intervention groups began to decrease compared to the DM group ($P < 0.05$). By the end of the experiment, FBG levels in the LP and HP groups were lower than those in the DM group, with a significant difference observed between the HP group and the DM group ($P < 0.01$).

Table 2
Effect of PP on nutrient consumption.

Time (week)	NC	DM	LP	HP
	Food intake (g/day)			
1	2.62 ± 0.12^{a}	2.23 ± 0.07^{b}	2.28 ± 0.10^{b}	2.22 ± 0.09^{b}
2	2.69 ± 0.15^{a}	2.76 ± 0.04^{a}	2.52 ± 0.07^{ab}	2.41 ± 0.04^{b}
3	2.75 ± 0.19^{a}	2.54 ± 0.09^{a}	2.69 ± 0.10^{a}	2.61 ± 0.09^{a}
4	2.73 ± 0.16^{a}	2.38 ± 0.12^{a}	2.51 ± 0.10^{a}	2.43 ± 0.09^{a}
5	2.73 ± 0.16^{a}	2.38 ± 0.12^{a}	2.51 ± 0.10^{a}	2.43 ± 0.09^{a}
6	2.77 ± 0.13^{a}	2.35 ± 0.10^{a}	2.56 ± 0.11^{a}	2.45 ± 0.05^{a}
7	2.81 ± 0.09^{a}	2.38 ± 0.07^{a}	2.53 ± 0.13^{a}	2.40 ± 0.12^{a}
8	2.77 ± 0.14^{a}	2.40 ± 0.09^{a}	2.50 ± 0.05^{a}	2.49 ± 0.10^{a}
	Water consumption (mL/day)			
1	3.60 ± 0.10^{b}	8.90 ± 0.22^{a}	8.97 ± 0.27^{a}	8.09 ± 0.23^{a}
2	4.22 ± 0.37^{c}	10.72 ± 0.63^{a}	9.77 ± 0.41^{ab}	8.52 ± 0.35^{b}
3	3.81 ± 0.19^{c}	9.80 ± 0.33^{b}	10.99 ± 0.52^{a}	9.60 ± 0.32^{b}
4	5.69 ± 0.36^{b}	9.64 ± 0.73^{a}	10.39 ± 0.57^{a}	9.52 ± 0.98^{a}
5	4.03 ± 0.18^{c}	9.58 ± 0.22^{a}	8.95 ± 0.26^{ab}	8.56 0.38^{b}
6	4.23 ± 0.22^{b}	8.98 ± 0.43^{a}	8.61 ± 0.55^{a}	8.52 ± 0.28^{a}
7	4.31 ± 0.07^{b}	9.05 ± 0.40^{a}	9.65 ± 0.33^{a}	8.97 ± 0.35^{a}
8	4.90 ± 0.16^{c}	9.37 ± 0.40^{ab}	10.03 ± 0.34^{a}	9.02 ± 0.25^{b}
	Body weight (g)			
1	24.90 ± 0.39^{a}	23.75 ± 0.67^{a}	24.04 ± 0.55^{a}	24.06 ± 0.27^{a}
2	25.40 ± 0.42^{a}	23.46 ± 0.52^{b}	23.94 ± 0.51^{b}	24.00 ± 0.26^{b}
3	25.51 ± 0.40^{a}	23.18 ± 0.45^{b}	23.89 ± 0.43^{b}	23.93 ± 0.18^{b}
4	26.04 ± 0.43^{a}	23.08 ± 0.40^{c}	24.94 ± 0.46a^{b}	24.76 ± 0.25^{b}
5	26.30 ± 0.50^{a}	23.08 ± 0.38^{b}	24.86 ± 0.54^{b}	24.38 ± 0.23^{c}
6	26.29 ± 0.45^{a}	22.80 ± 0.28^{c}	25.00 ± 0.52^{b}	24.85 ± 0.20^{b}
7	26.65 ± 0.46^{a}	22.60 ± 0.35^{c}	24.60 ± 0.46^{b}	24.54 ± 0.23^{b}
8	27.10 ± 0.42^{a}	22.78 ± 0.23^{c}	25.45 ± 0.42^{b}	24.93 ± 0.27^{b}

Note: Different letters indicated statistically significant differences ($P < 0.05$).

OGTT was utilized to evaluate glucose tolerance in diabetic mice after 8 weeks of PP administration (Fig. 2E). The glucose tolerance curve initially showed an increase, peaking at 30 min, and then decreased. After 120 min of oral glucose administration, blood glucose levels in the DM group were significantly higher than those in the NC group ($P < 0.01$), indicating impaired glucose tolerance. High-dose PP intervention significantly reduced blood glucose levels in diabetic mice ($P < 0.01$), whereas low-dose PP did not exhibit significant improvement. The OGTT curve was quantified by the AUC. As illustrated in Fig. 2F, compared to the NC group, the AUC value in the DM group was significantly increased, while high-dose PP intervention significantly reduced the AUC value in diabetic mice ($P < 0.01$). Additionally, there was no statistical significance in the AUC between the LP group and the DM group. These results suggest that the glucose tolerance of T2DM mice induced by HFD/STZ deteriorates significantly. Low-dose PP did not exacerbate impaired glucose tolerance, while high-dose PP improved glucose tolerance to a certain extent.

3.5 Effects of PP on serum insulin and lipid profiles

To elucidate the effects of PP on hyperinsulinemia, serum insulin levels were determined (Fig. 3A). Compared with the NC group, serum insulin levels were significantly increased in diabetic mice ($P < 0.01$). After 8 weeks of PP administration, serum insulin levels in the PP intervention groups decreased, although there was no statistical difference ($P > 0.05$). Furthermore, the homeostasis model assessment of insulin resistance (HOMA-IR) index was calculated to quantify the degree of insulin resistance (Fig. 3B). The HOMA-IR value in the DM group was significantly increased compared to the NC group ($P < 0.01$). High-dose PP intervention significantly decreased the HOMA-IR value compared with the DM group ($P < 0.01$).

T2DM is often accompanied by hyperlipidemia. Therefore, serum lipid profiles were measured to evaluate the effect of PP on hyperlipidemia (Figs. 3C-F). Compared to the NC group, levels of TC, TG, LDL-C, and HDL-C were significantly elevated in the DM group ($P < 0.01$), indicating severe hyperlipidemia in T2DM mice. After 8 weeks of high-dose PP intervention, hyperlipidemia was notably ameliorated, with reductions observed in TG, LDL-C, and HDL-C levels ($P < 0.01$). However, no significant differences were noted in TC, TG, LDL-C, and HDL-C levels between the DM group and the LP group ($P > 0.05$). Compared with the LP group, the HP group was more effective in ameliorating hyperlipidemia in diabetic mice. These results indicate that PP plays a crucial role in reducing hyperlipidemia and exhibits a dose-dependent effect in alleviating insulin resistance.

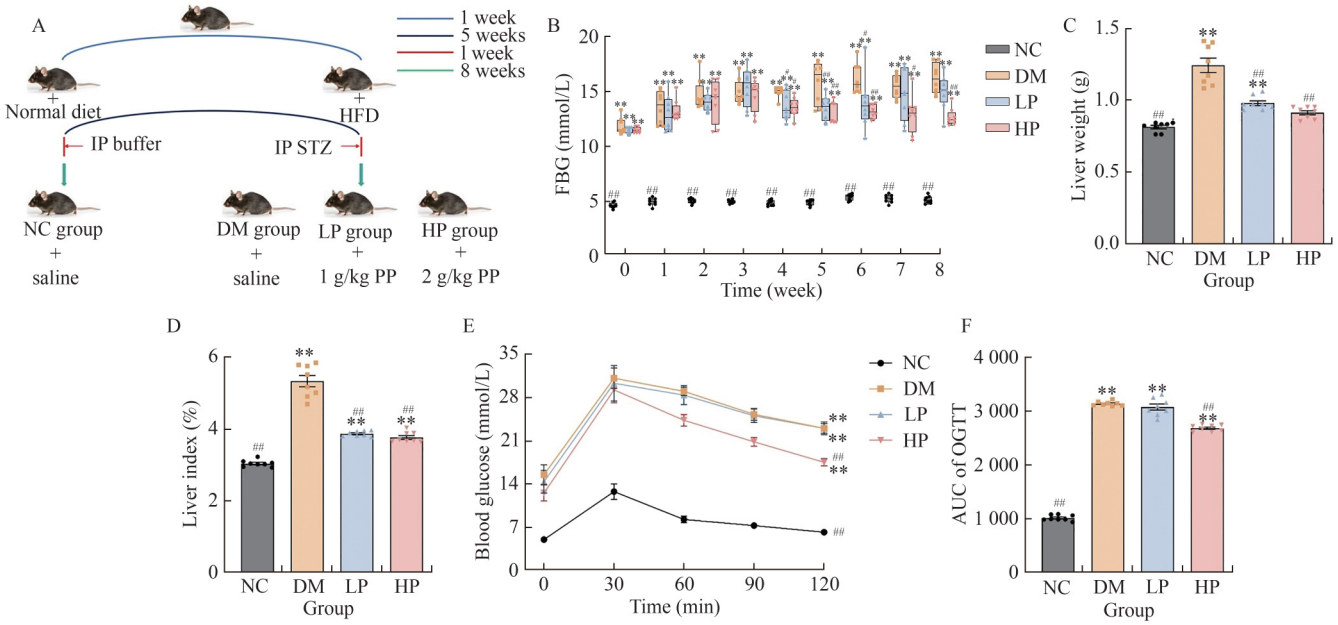

Fig. 2 Effects of PP on some symptoms of T2DM mice. (A) Experimental protocol, (B) FBG levels, (C) Liver weight, (D) Liver index, (E) OGTT, (F) AUC of OGTT. Data were presented as the means ± SE. $^{**}P < 0.01$ comparison with NC group; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ for comparison with the DM group.

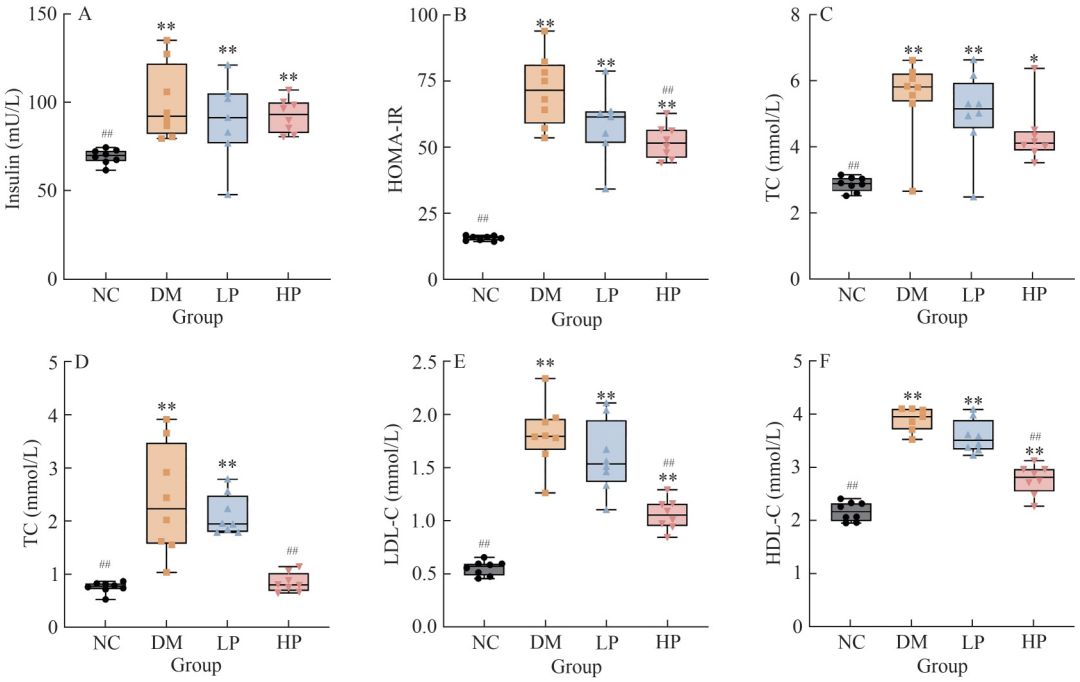

Fig. 3 Effects of PP on insulin resistance and dyslipidemia. (A) Serum insulin, (B) HOMA-IR, (C) Serum TC, (D) Serum TG, (E) Serum LDL-C, (F) Serum HDL-C. $^{*}P < 0.05$, $^{**}P < 0.01$ comparison with NC group; $^{\#\#}P < 0.01$ for comparison with the DM group.

3.6 Effects of PP on liver histopathology and function

Hepatic glycogen content was evaluated through periodic acid-Schiff (PAS) staining (Fig. 4A). In the DM group, hepatic glycogen content was notably lower compared to the NC group. High-dose PP intervention amplified the purple-red staining intensity in liver sections, whereas the LP group exhibited staining akin to that of the DM group. Corresponding with PAS staining outcomes, high-dose PP intervention significantly augmented hepatic glycogen content in the livers of diabetic mice ($P < 0.01$, Fig. 4C).

Furthermore, H&E staining was utilized to assess liver histopathology (Fig. 4B). Compared with the NC group, the DM group exhibited obvious dilation of hepatic sinusoids, disordered hepatocyte arrangement, severe vacuolar degeneration, hepatocyte necrosis, inflammatory factor infiltration, and numerous white fat vacuoles of varying sizes. After 8 weeks of PP intervention, the liver condition in each dose group improved to varying degrees. While a large number of fat vacuoles and inflammatory factors were still present in the LP group, hepatic sinusoid dilatation was reduced, hepatocyte arrangement tended to be regular, and occasional

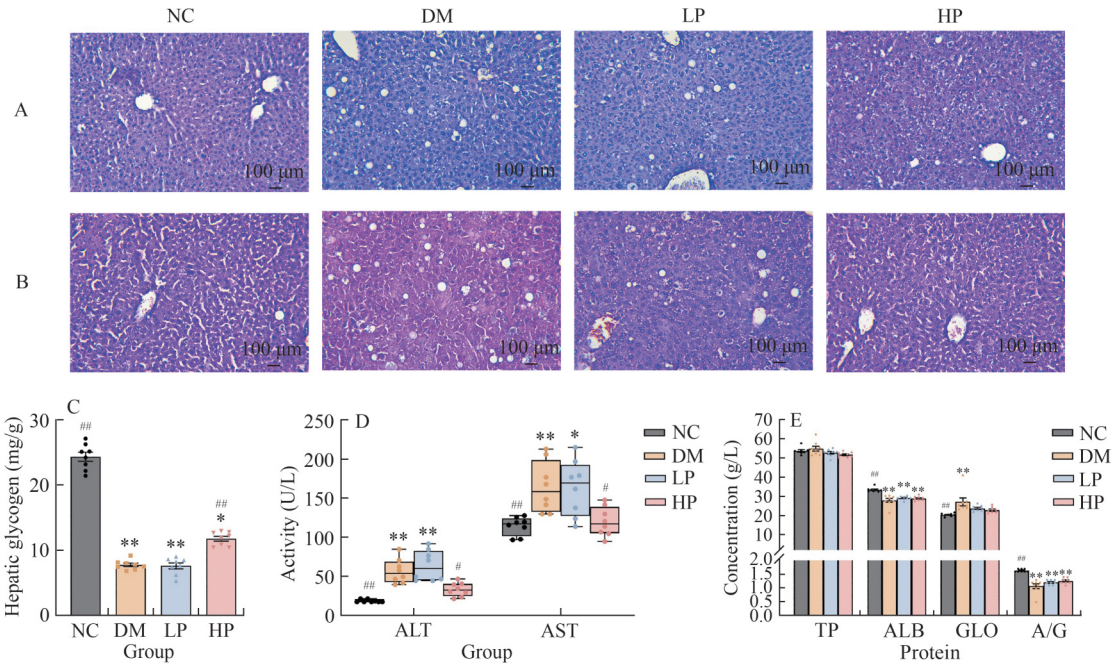

Fig. 4 Effects of PP on liver functions. (A) PAS staining, (B) HE staining, (C) The levels of hepatic glycogen, (D) The activities of ALT and AST, (E) Serum protein. $^{*}P < 0.05$, $^{**}P < 0.01$ comparison with NC group; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ for comparison with the DM group.

ballooning degeneration and necrosis were observed. In the HP group, hepatocytes exhibited a distinct and organized arrangement, with a notable reduction in balloon degeneration and fat vacuoles. However, inflammatory infiltration was still observed.

Furthermore, serum ALT and AST levels were utilized for the assessment of liver function (Fig. 4D). The DM group exhibited significantly elevated ALT and AST activities compared to the NC group, indicating the presence of severe liver dysfunction ($P < 0.01$). High-dose PP demonstrated hepatoprotective properties by decreasing AST and ALT activities ($P < 0.05$), whereas low-dose PP did not yield any significant effects ($P > 0.05$). Analysis of serum protein indexes revealed that PP intervention did not have a notable impact on TP and ALB levels, but did lead to a reduction in GLO levels (Fig. 4E). These findings suggest that liver injury in T2DM mice induced by HFD/STZ is severe, characterized by diminished hepatic glycogen content and elevated ALT and AST activities. Although high-dose PP intervention can enhance liver tissue morphology and structure to some extent, and decrease ALT and AST activities, it is unable to mitigate inflammatory factor infiltration.

3.7 Effects of PP on pancreas and kidney histopathology

The H&E staining of the pancreas and kidney from mice in various groups is depicted in Fig. 5. The islet morphology of mice in the NC group appeared complete and roughly oval, with a distinct and tidy boundary between the islet and the surrounding acinar tissue. In contrast, the islet shape of mice in the DM group was irregular, with a significantly reduced islet area and some inflammatory cell infiltration at the periphery of the islet cell mass. Following PP intervention, the islet morphology in all dose groups remained irregular, but there was an improvement in islet atrophy compared to the DM group. Both the LP and HP groups helped alleviate the accumulation of islet nuclei and inflammatory infiltration (Fig. 5A).

The H&E staining results of the kidney are presented in Fig. 5B. The NC group exhibited distinguishable glomeruli and tubules. Conversely, the DM group showed an enlarged glomerulus, thickened basement membrane, widened glomerular saccular space, and a significant amount of inflammatory cell infiltration. PP intervention was able to alleviate the kidney's pathological condition, characterized by a gradual reduction in glomerular volume and basement membrane thickening, although it had no obvious effect on glomerular cysts and inflammatory cell infiltration. Furthermore, PP intervention led to a significant reduction in serum UA levels in a dose-dependent manner compared to the DM group ($P < 0.05$) but did not have a significant impact on UREA levels (Figs. 5C and D). These findings indicate that PP supplementation may slightly alleviate the symptoms of pancreas and kidney in T2DM mice, but does not have a significant effect on the infiltration of inflammatory factors.

3.8 Effect of PP on key genes of glucose metabolism in liver

The relative mRNA expressions of glucose metabolic key genes were measured by real-time quantitative PCR and diagramed in Fig. 6. In the DM group, the relative mRNA expression of *GSK3β*, *FoxO-1*, *G6Pase* and *PEPCK* were increased significantly compared with NC group ($P < 0.05$). As shown in Fig. 6A, PP intervention significantly decreased the expression of *GSK3β* in T2DM mice ($P < 0.01$), which means consumption of PP may have benefit effects on hepatic glycogen synthesis in liver. Figs. 6B-D showed all doses of PP reversed the increased expression of *FoxO-1* and *G6Pase* and *PEPCK* in diabetic mice ($P < 0.05$). These results indicated that PP could mediate blood glucose homeostasis by regulating the key genes about glucose metabolism in T2DM mice, which was manifested by inhibiting gluconeogenesis and increasing glycogen synthesis in liver.

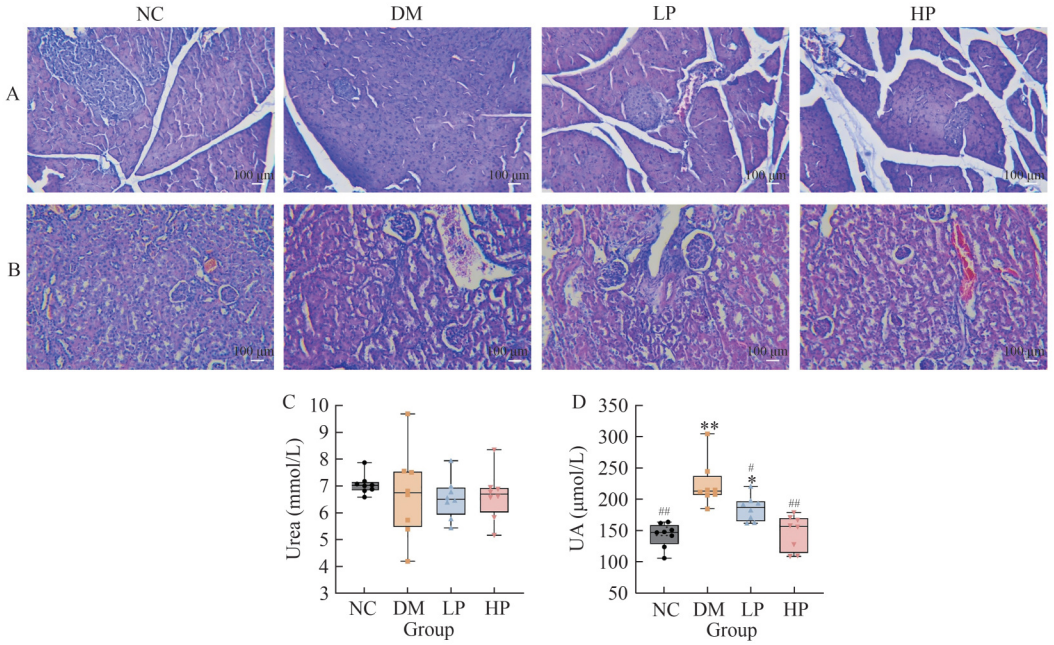

Fig. 5 Effects of PP on kidney and pancreas functions. (A) HE staining of the pancreas tissue, (B) HE staining of the kidney tissue, (C) Serum urea, (D) Serum UA. $^{*}P < 0.05$, $^{**}P < 0.01$ comparison with NC group; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ for comparison with the DM group.

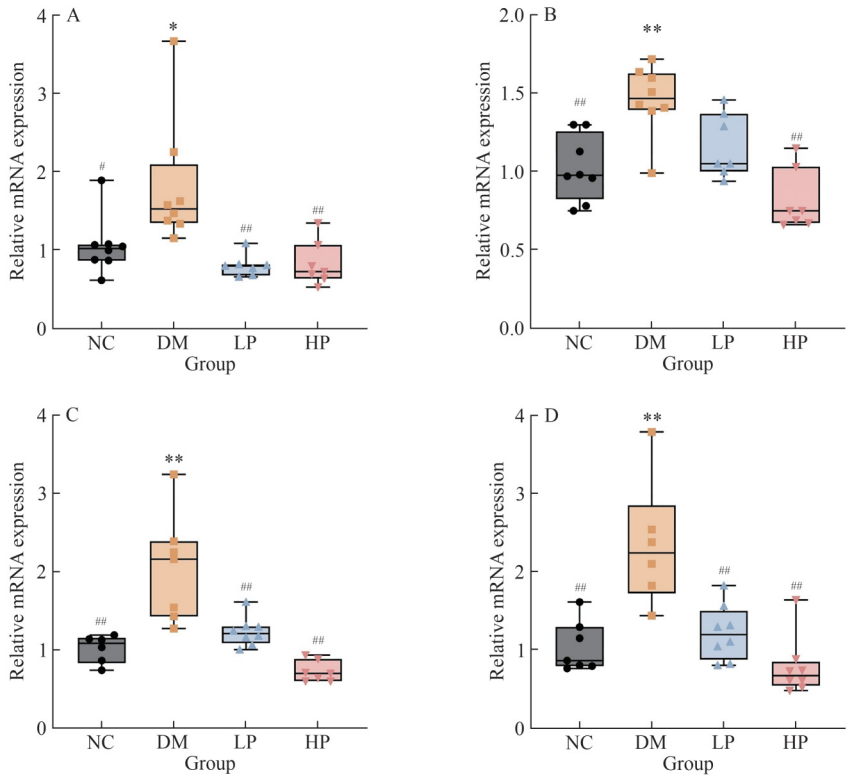

Fig. 6 Effects of PP on the relative mRNA expression of (A) *GSK3β*, (B) *FoxO-1*, (C) *G6Pase*, (D) *PEPCK*. $^{*}P < 0.05$, $^{**}P < 0.01$ comparison with NC group; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ for comparison with the DM group.

3.9 Effects of PP on SCFAs concentration and pH value of feces

SCFAs are secondary metabolites known for their beneficial effects on intestinal health and metabolic diseases. In this study, pH and SCFAs in the feces of all groups were examined at different time points (Fig. 7). In the first week, the total SCFA content in feces from the NC group was significantly lower than that in the DM group, primarily due to lower levels of acetic acid and propionic acid, while the *n*-butyric acid content in the NC group was significantly higher than that in the DM group ($P < 0.01$). PP intervention led to levels of acetic acid and propionic acid that were similar to those in the NC group (Fig. 7A). By the fourth week, except for propionic acid, the

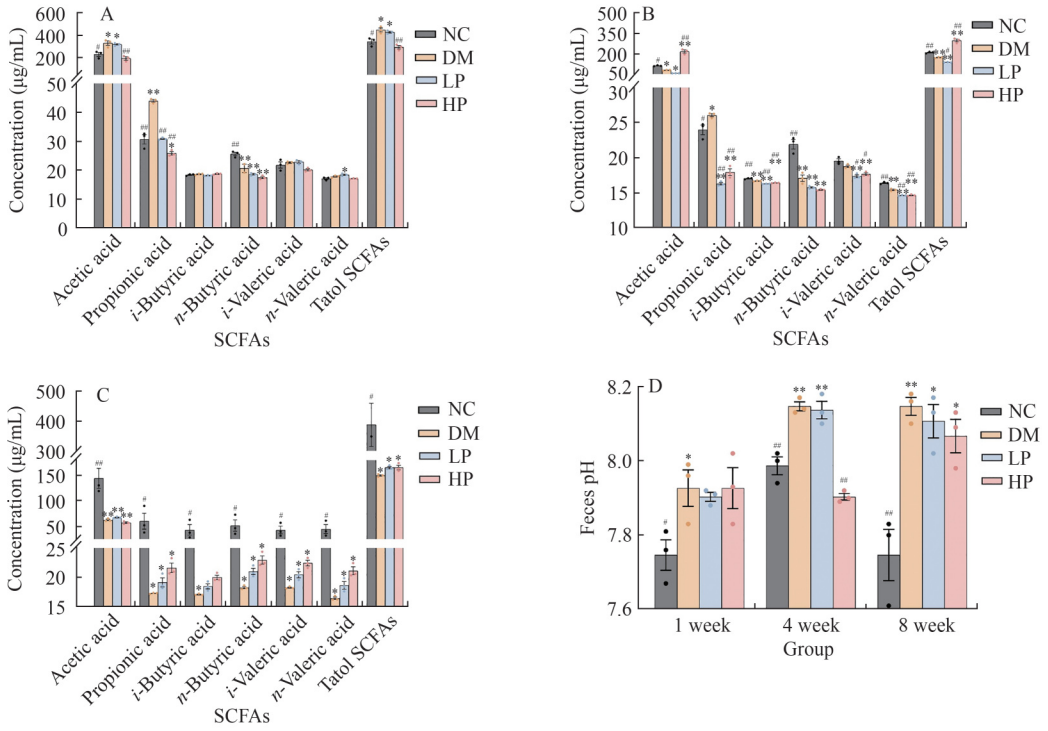

Fig. 7 Effects of PP on feces SCFAs concentration in different periods. (A) 1 week, (B) 4 weeks, (C) 8 weeks; (D) Effects of PP on pH value in different periods. Different letters indicated statistically significant differences ($P < 0.05$).

SCFA level in the NC group was significantly higher than that in the DM group ($P < 0.05$). High-dose PP intervention notably increased the content of acetic acid in the feces of diabetic mice, surpassing that in the NC group (Fig. 7B). In the final week of the experiment, compared with the DM group, the contents of various SCFAs (except acetic acid) in PP intervention groups showed an upward trend, although without statistical significance (Fig. 7C, $P > 0.05$). Additionally, the pH value of feces in different periods was inversely proportional to the total SCFA content (Fig. 7D). These results suggest that PP intervention might enhance the production of SCFAs in the feces of T2DM mice induced by HFD/STZ.

3.10 Effects of PP on gut microbiota

3.10.1 Effect of PP on diversity and structure of gut microbiota

Considering the superior effects of high-dose PP on T2DM mice compared to low-dose PP, high-throughput sequencing of the V3-V4 region of the 16S rRNA gene of the gut microbiota of mice in the NC, DM, and HP groups was conducted. Analyses of the Chao 1 index curve and rank abundance curve indicated that the sequencing depth covered most of the diversity, making the data suitable for further analysis (Fig. S2). Simpson and Pielou_e indexes analyses revealed that gut microbiota diversity and evenness were significantly increased in the DM group compared with the NC group ($P < 0.05$). However, PP intervention significantly decreased the Chao 1 index compared with the DM group ($P < 0.05$), bringing it close to the NC group level. Furthermore, there was no significant difference in Shannon, Simpson, and Pielou_e indexes between the HP group and the DM group (Table 3).

Table 3
Effects of PP on gut microbiota diversity.

Index	NC	DM	HP
OTUs	509.75 ±18.68^{b}	820.25 ± 51.85^{a}	547.25 ± 70.03^{b}
Chao 1	513.11 ± 19.48^{b}	824.44 ± 52.33^{a}	550.89 ± 74.21^{b}
Shannon	4.77 ± 0.69^{b}	6.89 ± 0.26^{a}	6.48 ± 0.20^{a}
Simpson	0.82 ± 0.08^{a}	0.98 ± 0.01^{a}	0.97 ± 0.01^{a}
Pielou_e	0.53 ± 0.08^{b}	0.72 ± 0.02^{a}	0.72 ± 0.02^{a}
Goods coverage	1.00 ± 0.00^{a}	1.00 ± 0.00^{a}	1.00 ± 0.00^{a}

Note: Different letters indicated statistically significant differences ($P < 0.05$).

Consistently, a Venn diagram showed that T2DM increased the number of specific amplicon sequence variants (ASVs), while PP intervention reversed this trend, with the number of specific ASVs approaching that of the NC group (Fig. 8A). These results indicated that high-dose PP may reduce the abundance of certain harmful bacteria. Additionally, hierarchical clustering (based on Jaccard distance) and principal coordinates analyses (PCAs) exhibited a clear separation between the DM and HP group, suggesting that high-dose PP exerted a significant impact on the gut microbiota of T2DM mice (Figs. 8B and C).

3.10.2 Effect of PP on the composition of gut microbiota

Taxonomic analysis revealed that *Firmicutes*, *Bacteroidota*, *Actinobacteriota*, *Campylobacterota*, and *Desulfobacterota* were the dominant phyla, and their relative abundances varied among different groups (Fig. 9A). Specifically, the DM group exhibited a higher relative abundance of Firmicutes and a lower relative abundance of

Bacteroidota and *Campylobacterota* compared with the NC group. After administration of high-dose PP, a lower relative abundance of Firmicutes and a higher relative abundance of *Bacteroidota* were observed in the HP group. Additionally, the F/B ratio in the DM group was significantly higher than in the NC group ($P < 0.05$). PP intervention for 8 weeks resulted in a decrease in the F/B ratio in T2DM mice, although without statistical significance (Fig. S3). At the family level, the DM group presented a higher abundance of

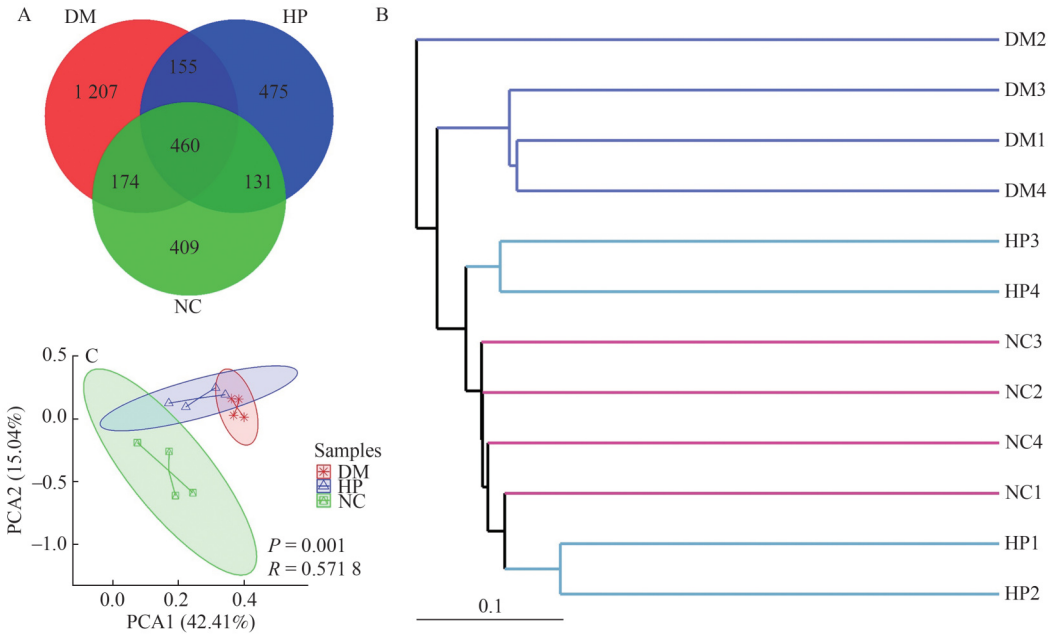

Fig. 8 Effects of PP on the diversity and structure of gut microbiota. (A) Venn diagram, (B) Hierarchical clustering, (C) PCA plots.

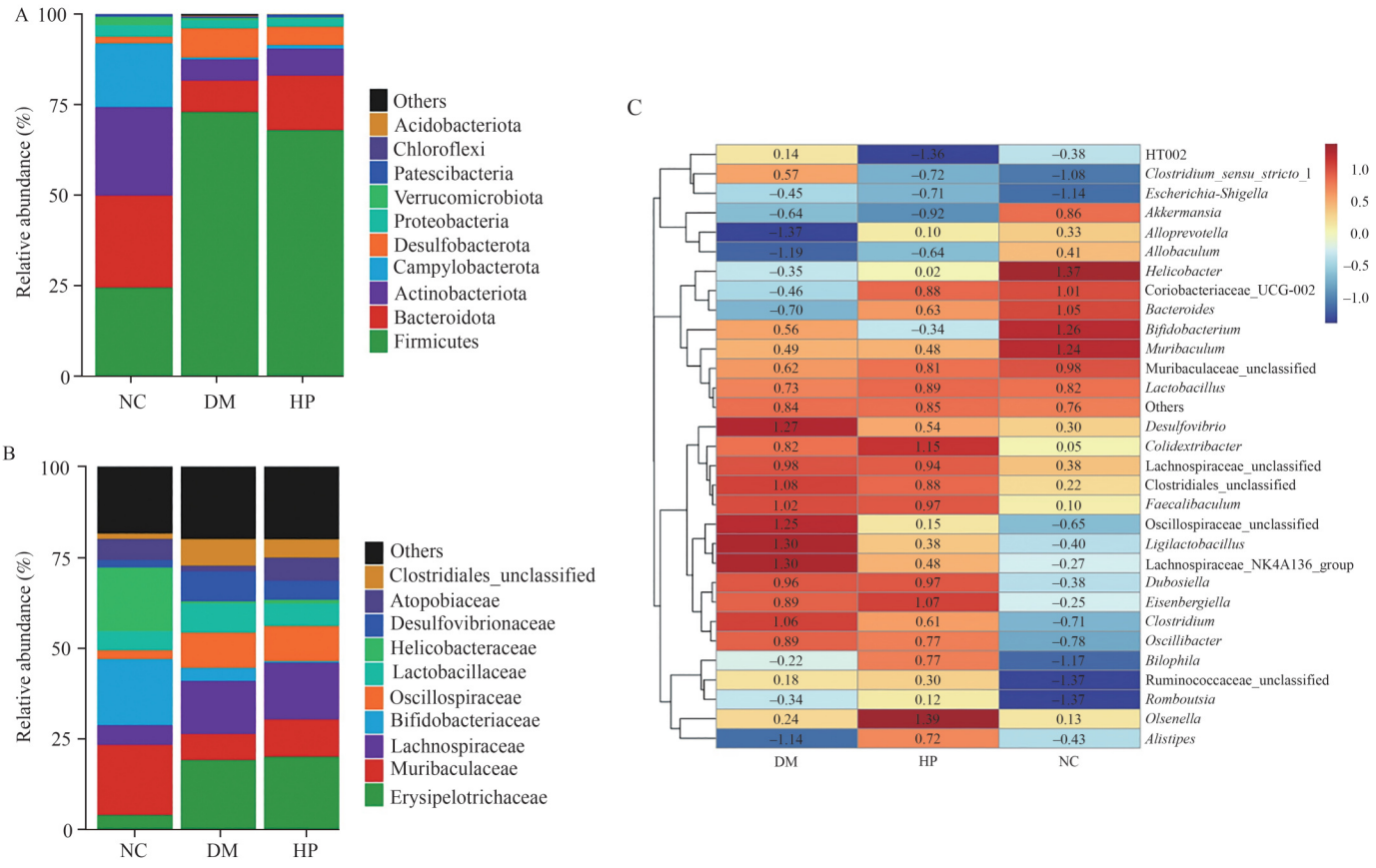

Fig. 9 Effects of PP on composition and LEfSe analysis of gut microbiota. (A) Phylum level, (B) Family level, (C) Clustering heat map at the genus level, (D) LEfSe analysis of LDA value histogram. The LDA score threshold ≥ 3 and adjusted $P < 0.05$, (E) Taxonomic cladogram.

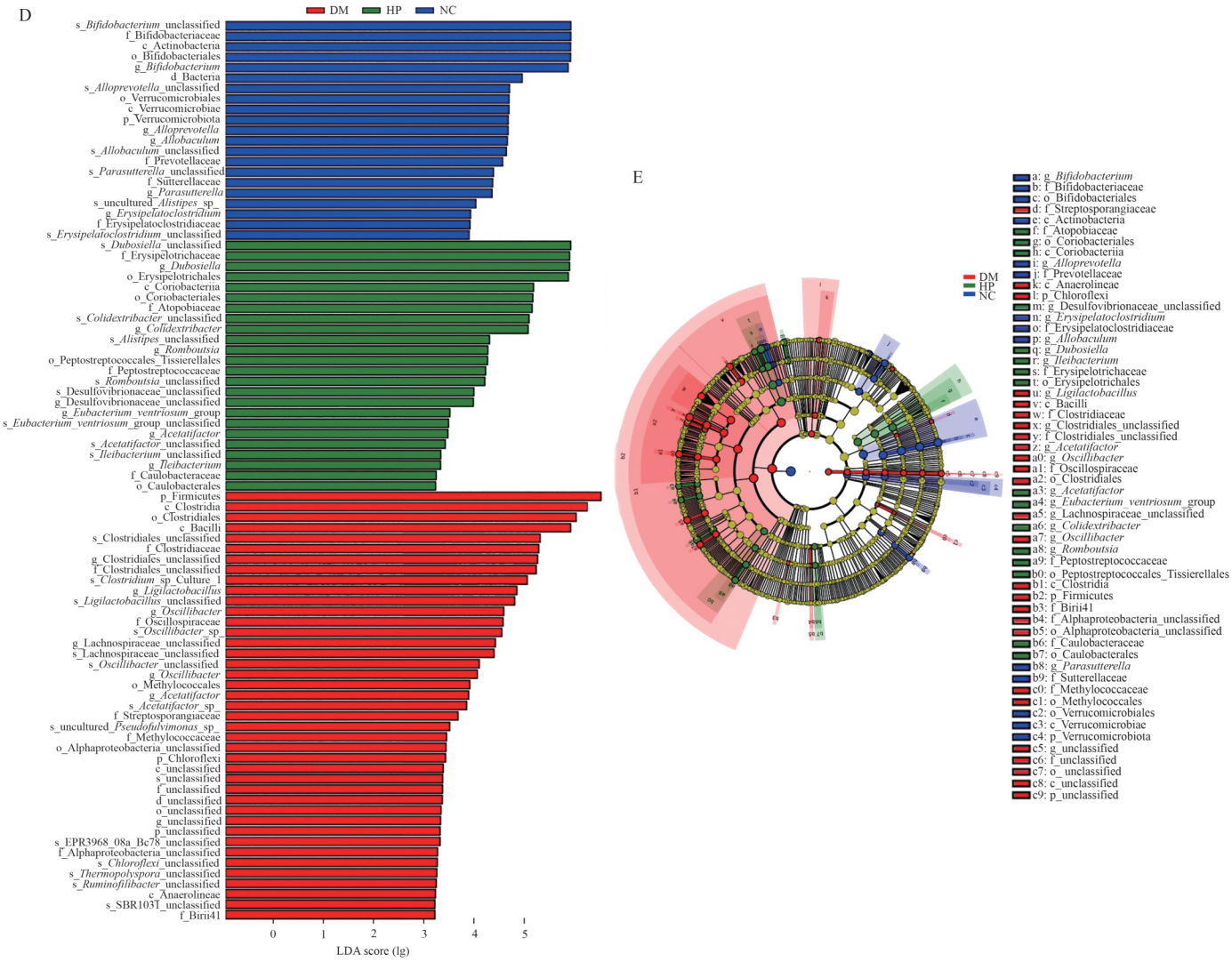

Fig. 9 (*Continued*)

Erysipelotrichaceae, Lachnospiraceae, Oscillospiraceae, Desulfovibrionaceae, and Clostridiales_unclassified, and a lower abundance of Muribaculaceae and Atopobiaceae compared with the NC group ($P < 0.05$). HP group resulted in an increasing abundance of Atopobiaceae, while Bifidobacteriaceae was decreased after administration with high-dose PP ($P < 0.05$, Fig. 9B). Furthermore, a clustering heatmap was used to compare the relative abundance of gut microbiota at the genus level (Fig. 9C). Compared with the DM group, the relative abundance of *Romboutsia*, *Allobaculum*, *Alloprevotella*, and *Bacteroides* in the HP group was increased, while the relative abundances of *Clostridium_sensu_stricto*_1, Oscillospiraceae_unclassified, HT002, and *Ligilactobacillus* were significantly decreased ($P < 0.05$). These bacterial profiles of the HP group at the genus level tended to resemble those of the NC group.

To further explore the effect of PP on the gut microbiota of T2DM mice, LEfSe analysis was used to find biomarkers with different relative abundances at different taxonomic levels among groups, and the threshold of liner discriminant analysis (LDA) for discriminative features was beyond 3.0. The results were shown in Fig. 9D, high-dose PP intervention enriched 24 bacterial taxa in HP group. At the class level, PP administration enrich the bacterial of *Coriobacteriia*. Furthermore, the abundance of Erysipelotrichaceae, Atopobiaceae, Peptostreptococcaceae and Caulobacteraceae were enrich in family level, and *Dubosiella*, *Colidextribacter*, *Romboutsia*, Desulfovibrionaceae_unclassified, *Acetatifactor*, *Ileibacterium* were unique genus bacteria in HP group (Fig. 9F). These results revealed that PP significantly altered the structure and composition of gut microbiota in T2DM mice, and the effect was directional.

3.10.3 Correlation of gut microbiota and T2DM-related indicators

In the Spearman correlation analysis (Fig. 10A), several key associations between specific genera of gut microbiota and T2DM-related biochemical indicators were observed. *Ligilactobacillus*, *Dubosiella*, and *Clostridium* were found to be positively associated with ALT, GLO, HDL-C, insulin, LDL-C, TC, and TG, while they showed negative correlations with body weight (BW) and hepatic glycogen levels ($P < 0.05$). Conversely, *Allobaculum* and *Alloprevotella* displayed negative associations with diabetic phenotypes and were positively correlated with BW and hepatic glycogen levels ($P < 0.05$). Furthermore, the correlation network map (Fig. 10B) highlighted

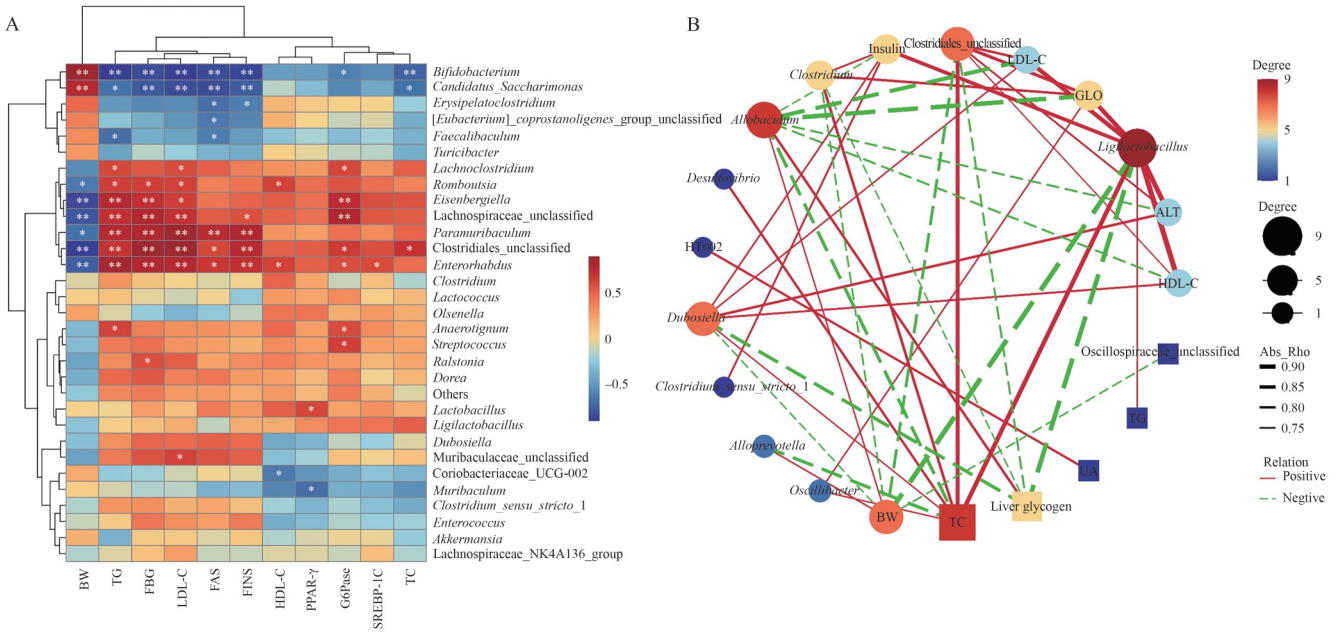

Fig. 10 Spearman correlation analysis between T2DM-related indicators and gut microbiota at the genus level. (A) Heatmap, (B) Network map. Significant correlations were indicated as $*P < 0.05$, $**P < 0.01$ and $***P < 0.001$.

specific relationships between certain genera and biochemical indicators. Notably, *Ligilactobacillus* showed a unique positive correlation with TG (Rho = 0.74, $P < 0.01$), indicating a potential role of this genus in lipid metabolism. Additionally, HT002 exhibited a significant positive correlation with UA, suggesting its potential as a biomarker for hyperuricemia (Rho = 0.78, $P < 0.01$). Combining these correlation results with the changes observed in gut microbiota composition following PP intervention (Fig. 9C), it appears that the genera enriched by PP were negatively correlated with harmful diabetes-related indicators, while the genera inhibited by PP were positively correlated with these indicators. However, further research is needed to elucidate the underlying mechanisms driving these associations between gut microbiota characteristics and T2DM pathology.

4. Discussion

T2DM is a heterogeneous disease characterized by hyperglycemia and insulin resistance, resulting in imbalance of glycolipids metabolism[26]. As one of the most common metabolic diseases worldwide, it can be normalized through appropriate dietary intervention[27]. Peach, with its low glycemic index and glucose load, is rich in polyphenols, flavonoids, polysaccharides, and other functional substances beneficial to T2DM[8,10]. *In vitro* and *in vivo* studies have reported that individual components and extracts of peach fruit exhibit hypoglycemic and hypolipidemic activities[11-12,14]. However, the traditional view that the sucrose-based soluble sugars contained in peach may exacerbate the imbalance of glycolipid metabolism in T2DM patients has been challenged. Accumulated research reports suggest possible synergistic effects of nutrients and non-nutrient compounds, demonstrating health benefits through reasonable mechanisms of action[28]. Therefore, this study evaluates the comprehensive effects of consuming PP on T2DM mice and provides a preliminary exploration of the underlying mechanisms. Results show that PP can alleviate hyperglycemia, hyperlipidemia, and hyperuricemia, with these effects mediated by gluconeogenesis and glycogen synthesis, as well as modulation of the gut microbiota and SCFAs.

Excessive production of reactive oxygen species in the body leads to oxidative stress, further damaging pancreatic β-cells, and promoting the production of inflammatory factors, thereby exacerbating the deterioration of T2DM[29]. PP alleviate the accumulation of islet nuclei and inflammatory infiltration in the pancreas, attributed to their effective antioxidant capacity. Furthermore, PP intervention reversed the weight loss observed in T2DM mice, consistent with previous findings where the administration of peach polysaccharides reversed weight loss trends in similar studies[14]. Notably, polysaccharides are considered a major functional component contributing to the health benefits of whole fruit, and PP's beneficial effects on body weight may be attributed to the anti-diabetic effects of peach polysaccharides[30]. A previous prospective study has shown that consuming whole fruits can be a preventive measure against diabetes[8]. Chlorogenic acid, the main phenolic compound in peach, has been shown to mediate the beneficial effects of coffee consumption on diabetes risk[11,31]. Studies indicate that chlorogenic acid increases muscle uptake of glucose in diabetic mice by enhancing glucose transport and inhibiting the activity of α-glucosidase, thereby reducing FBG levels[32-33]. Vegetarian diets emphasize fruit intake, and daily PP supplementation enriches the diversity of vegetarian diets. Previous studies have demonstrated the benefits of vegetarian diets in reducing TC, TG, and LDL-C, thus regulating dyslipidemia[34]. Similar effects have been observed in a study where the addition of 5% and 10% sweet cherry (*Prunus avium* L.) dry fruits to a high-fat cholesterol diet improved serum lipid profiles and reduced liver lipid accumulation in Wistar rats[35].

Hepatic glycogen plays a critical role in glucose homeostasis[36]. T2DM disrupts endogenous metabolic processes, hindering the conversion of glucose to hepatic glycogen and resulting in decreased

hepatic glycogen content. Previous studies have reported that decreased expression of *GSK3β* increases the dephosphorylation and activity of hepatic glycogen synthase, thereby enhancing hepatic glycogen synthesis[1]. *FoxO1*, a transcription factor encoding gluconeogenesis genes, inhibition of *FoxO1* phosphorylation induces glucokinase gene expression and increases glycogen biosynthesis[37]. Additionally, overexpression of *G6Pase* and *PEPCK* promotes glucose production. Downregulating the expression of *G6Pase* and *PEPCK*, the 2 rate-limiting enzymes in hepatic gluconeogenesis, ultimately helps improve hyperglycemia symptoms in T2DM mice[38].

T2DM decreases the levels of gut SCFAs, exacerbating gut microecology imbalance and T2DM progression[39]. High-dose PP intervention significantly elevated acetic acid levels midway through the experiment. Research indicates that acetic acid alters liver glucose metabolism by reducing xylose-5-phosphate accumulation in the liver, thereby mitigating glucose metabolic disorders by affecting glycolysis and gluconeogenesis cycles in T2DM individuals[39]. Additionally, various SCFA levels in the PP intervention groups showed an upward trend in the last week of the experiment. Previous studies have shown that propionic acid can mediate intestinal hormone release to alleviate insulin resistance[40]. Interestingly, propionic acid is primarily produced by fermenting glucose, xylose, and arabinose, while peaches' polysaccharides are primarily composed of galactose, arabinose, and xylose[14,39]. Butyric acid can improve glucose uptake by activating intestinal gluconeogenesis through the gut-brain axis, with gut butyric acid production stemming from xylose fermentation[39,41].

Furthermore, T2DM etiology or progression is related to dysregulation of the gut microbiota. In this study, oral administration of high-dose PP reduced the F/B ratio compared to the DM group, consistent with previous studies showing an inverse relationship between the F/B ratio and blood glucose levels in diabetes[42]. At the genus level, PP administration increased the abundance of *Romboutsia* and *Bacteroides* while decreasing *Clostridium_sensu_stricto*_1, HT002, Oscillospiraceae_unclassified, and *Ligilactobacillus*. *Romboutsia* and *Bacteroides* are known to inhibit diabetes and other metabolic diseases. Liu et al.[43], reported that liraglutide as an analogue of glucagon-like peptide-1 could enrich the abundances of *Romboutsia* in the gut microbiota composition of *db/db* mice. Probiotics such as *Roseburia*, *Bacteroides*, and *Akkermansia* have been shown to improve glucose metabolism disorders and insulin sensitivity[44]. Additionally, polysaccharides from *Lycium barbarum* have been reported to reduce the relative abundance of *Clostridium_sensu_stricto*_1, alleviating T2DM, and *Clostridium_sensu_stricto*_1 was positively correlated with HDL-C, HOMA-IR, ALT, AST and TC[45]. PP also suppressed the growth of Oscillospiraceae_unclassified, which belong to the Oscillospiraceae family, a potentially pathogenic bacterium associated with gut inflammation[46]. *Cordyceps militaris* polysaccharides decreased *Ligilactobacillus* abundance to improve metabolic disorders, positively affecting lipid metabolism and inflammation-related indicators[47]. Furthermore, PP administration increased the relative abundance of *Allobaculum* and *Alloprevotella*, negatively correlated with diabetic phenotypes. Ethanol extraction of Simiao Wan increased the abundance of *Akkermansia* and *Allobaculum* in diabetic mice, negatively correlated with plasma TG, TC, LDL-C, glucose levels, and HOMA-IR values[48]. Similarly, *Alloprevolella* abundance was negatively associated with HOMA-IR, TG, and epididymal fat in rats fed a high-sugar diet after the theabrownin administration[49]. Huang et al.[50] also reported increased *Alloprevotella* and *Romboutsia* abundance after administering a *Sanghuangporus vaninii* mixture in T2DM mice, negatively correlated with TG, TC, HOMA-IR, FBG, LDL-C, tumor necrosis factor-α, and free fatty acids. However, while this study focused on glucose metabolism and intestinal flora to elucidate PP's positive effects on T2DM, further in-depth research is needed to explore potential mechanisms and whether PP regulates signaling pathways in other organs.

5. Conclusion

In conclusion, PP are rich in functional substances and demonstrate effective antioxidant capacity *in vitro*. Animal experiments have shown that supplementing with PP as a beneficial fruit in HFD/STZ induced T2DM mice, results in mitigated hyperglycemia, hyperlipidemia, and improved tissue functions. The synergistic effects of nutrients (such as polysaccharides, polyphenols, and organic acids) and non-nutrient compounds in PP may ultimately lead to the alleviation of T2DM. The positive impacts of PP may be linked to enhancing glycogen synthesis, suppressing gluconeogenesis, and regulating gut microenvironment balance. These findings have broadened our understanding of the nutritional benefits of PP, enhancing its safety and appeal for individuals with diabetes. Additionally, our research reinforces the existing guidelines advocating for higher PP intake as a component of a balanced diet to reduce the risk of developing T2DM.

Conflict of interest

There is no conflict of interest regarding the publication of this article.

Acknowledgements

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Appendix A. Supplementary information

Supplementary data associated with this article can be found, in the online version, at http://doi.org/10.26599/FSHW.2024.9250246.

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