Research in Pharmaceutical Sciences, February 2026; 21(1): 47-63
Received: 13-07-2023
Peer Reviewed: 15-08-2023
Revised: 01-12-2025
Accepted: 24-12-2025
Published: 26-12-2025

School of Pharmacy & Pharmaceutical Sciences
Isfahan University of Medical Sciences

Original Article

Dietary fibers of *Canna edulis* and *Maranta arundinacea* rhizomes ameliorate metabolic diseases and gut dysbiosis in mice fed a high-fat diet

Putra Santoso* **and Rita Maliza**

Department of Biology, Faculty of Mathematics and Natural Sciences, Universitas Andalas, Padang, Indonesia.

Abstract

Background and purpose: *Canna edulis* (*C. edulis*) and *Maranta arundinacea* (*M. arundinacea*) are potential medicinal plants. This study investigated the preventive effect of dietary fibers from *C. edulis* and *M. arundinacea* rhizomes against metabolic diseases and gut dysbiosis promoted by a high-fat diet (HFD).
Experimental approach: Twenty-four male mice were divided into 4 groups and fed a low-fat diet, HFD, or HFD combined with 10% *C. edulis* fiber or *M. arundinacea* fiber for 12 weeks. Subsequently, the indicators of metabolic syndromes and gut microbiota composition were investigated.
Findings/Results: *C. edulis* fiber effectively prevented obesity and counteracted HFD-induced dyslipidemia. *C. edulis* and *M. arundinacea* fibers prevented type 2 diabetes, but *C. edulis* fiber was more effective in regulating glucose tolerance and insulin than *M. arundinacea*. *C. edulis* fiber also reduced steatosis and inflammation in the liver. 16S rRNA sequencing of fecal microbiota revealed that the fibers decreased the abundance of *Desulfobacterota*, but only *C. edulis* increased *Bacteroidota* while decreasing *Firmicutes*. *C. edulis* fiber elevated the abundance of beneficial microbiota, including *Lactobacilus reuteri*, *L. johnsonii*, and *Bacteroides fragilis*, while lowering the pathogenic species *Mucispirillum* sp. Otherwise, *M. arundinacea* fiber increased the beneficial species *L. murinus* and *Faecalibacterium prausnitzii*, and pathogenic species *Mucispirillum* sp.
Conclusion and implications: *C. edulis* and *M. arundinacea* fibers exerted ameliorative effects against metabolic diseases and gut dysbiosis caused by HFD. However, *C. edulis* fiber was more effective than *M. arundinacea*. Therefore, *C. edulis* fiber could be a candidate for supplements preventing metabolic diseases and gut dysbiosis.

Keywords: *Desulfobacterota*; Dyslipidemia; Hepatic steatosis; Hyperinsulinemia; Metabolic syndromes; *Mucispirillum*.

INTRODUCTION

Canna edulis (*C. edulis*, Cannaceae) and *Maranta arundinacea* (*M. arundinacea,* Marantaceae) rhizomes are among the potentially valuable yet relatively unexplored medicinal plants in tropical and subtropical regions. The starch is extracted from the rhizomes and consumed as a staple or complementary food, while the by-product, which is rich in dietary fiber, is usually considered waste. However, a previous report demonstrated that the by-product of *C. edulis* rhizomes was rich in dietary fiber fractions comprising hemicelluloses (including arabinoxylans, glucuronoxylans, and xyloglucans), cellulose, pectin, and lignin (1). In addition, the by-product contains highly concentrated phenolic compounds and exerts antioxidant activity (1). A study using *in vitro* and *in silico* approaches also revealed that extracted lignin from *C. edulis* rhizomes had a potent inhibitory effect against α-d-glucosidase. α-d-glucosidase is a digestive enzyme that plays a pivotal role in blood glucose increment (2).

*Corresponding author: P. Santoso
Tel: +62-81374151306, Fax: +62-73118
Email: putrasantoso@sci.unand.ac.id

Access this article online
Website: http://rps.mui.ac.ir
DOI: 10.4103/RPS.RPS_129_23

Likewise, another study found that extracted fiber from the rhizomes of *M. arundinacea,* in combination with the starch, could be developed as a high-quality biopolymer eliciting antimicrobial activity (3). Thus, the fibers from *C. edulis* and *M. arundinacea* rhizomes were suggested to have various potential health benefits. However, until recently, the effectiveness of *C. edulis* and *M. arundinacea* fibers in counteracting diseases, particularly metabolic diseases and gut dysbiosis, remained less investigated.

Several studies have demonstrated the close association between excessive high-fat diet intake (HFD) and metabolic dysregulation and dysbiosis (4-6). A study in mice indicated that chronic consumption of HFD could lead to excessive weight gain toward obesity, dyslipidemia, inflammation, insulin resistance, and hepatic steatosis (7). Another report also showed that HFD caused profound alteration in gut microbiota composition by increasing pathogenic microbiota and decreasing health-promoting microbiota (8). Conversely, the incorporation of extracted fiber from the jicama tuber (*Pachyrhizus erosus*) could substantially preclude the development of obesity, type 2 diabetes, systemic inflammation, as well as gut dysbiosis in mice fed with HFD (9). Moreover, it has been revealed that the dietary fiber of bamboo shoots (*Dendrocalamus brandisii*) potently exerts a hypoglycemic effect by inhibiting enzymes involved in the digestion of carbohydrates (10). Overall, the findings indicated that dietary fibers have high potential benefits in preventing and alleviating metabolic dysregulation and gut dysbiosis caused by HFD. Unfortunately, to date, the studies on the health benefits of fibers extracted from *C. edulis* and *M. arundinacea* rhizomes against the detrimental effects of HFD remain limited.

Various dietary fibers have been indicated to affect the absorption of nutrients in the digestive tract and regulate glucose and lipid metabolism (11,12). Soluble fiber, for instance, can bind to cholesterol and prevent its absorption, thereby precluding dyslipidemia (13,14). The fiber could also regulate blood glucose levels by slowing down glucose absorption into the bloodstream (15). In addition, some fiber types can simulate the growth of beneficial gut microbiota, which can reduce inflammation (16). Importantly, the gut microbiota can also ferment fibers to produce short-chain fatty acids (SCFAs), which have been shown to elicit various metabolic benefits, such as sustaining blood glucose and lipid homeostasis in the body (17). However, the effectiveness of dietary fiber in counteracting metabolic diseases and gut dysbiosis may differ, depending on their resources and constituents (18). Accordingly, it was hypothesized that the effectiveness of fiber extracted from *C. edulis* rhizomes might be unequal to that of *M. arundinacea* in precluding HFD-induced metabolic diseases and gut dysbiosis. Therefore, this present study was performed to investigate whether dietary fibers extracted from rhizomes of *C. edulis* and *M. arundinacea* could elicit anti-metabolic diseases and gut dysbiosis caused by HFD, and if so, whether they could exert different levels of effectiveness against HFD.

MATERIALS AND METHODS

Materials

The analytical-grade chemical reagents used in this study were purchased from Sigma Aldrich (St. Louis, Missouri, USA). The ELISA kits for insulin, fibroblast growth factor 21 (FGF21), and glucagon-like peptide 1 (GLP-1), as well as kits for plasma lipid measurement, were obtained from Bioassay Technology Laboratory (Shanghai, China). The kits for molecular analysis were purchased from Qiagen (Germany) and Bio-Rad (USA) Laboratories. A kit for measuring human insulin (Actrapid) was purchased from Novo Nordisk (Denmark). The standard chow diet (Ratbio) for mice and ketamine were obtained from Citra Inna Fedmill (Jakarta, Indonesia) and Pantex (Holland), respectively.

Fiber extraction from rhizomes

Fresh samples of *C. edulis* and *M. arundinacea* rhizomes were collected from Air Pikat (Rejang Lebong district, Bengkulu province, Indonesia). The species were identified and validated by a plant taxonomist in Herbarium Anda (Biology Department, Universitas Andalas; the voucher specimens

were deposited with ID numbers 021-Anda-113/UA for *C. edulis* and 022-Anda-113/UA for *M. arundinacea*). The fiber extraction was performed based on the protocol previously described (19). Briefly, the samples were washed with distilled water 4-5 times. After peeling and slicing, the samples were pulverized using an electric grater until the porridge texture was achieved. Next, samples were soaked in distilled water (1:4) and stored at 4 ℃ for 12 h in an isolated container. The fibers that floated as supernatant at the upper side of the container were collected and filtered. After steaming at 100 ℃ for 30 min, the samples were dried for 16 h at 67 ℃. Finally, the dried samples were subjected to grinding to produce fine fiber powder and stored until used in the experiment.

Animals

Adult male ddy mice (4 weeks old and 25-27 g of body weight) were provided by Balai Veteriner Baso (Bukittinggi, West Sumatra, Indonesia). Before the experiment, mice were acclimated for one week in the animal house of the Animal Physiology Laboratory of the Department of Biology, Universitas Andalas, with regulated temperature (25.0-26.1 ℃), humidity (68-70%), and light-dark cycle (12 h dark/12 h light). All animals were fed *ad libitum* with a standard chow diet and had free access to water bottles filled with distilled water. All experimental procedures for animals in this study were in accordance with the standard guidelines for animal use and handling and approved by the Committee of Research Ethics of Universitas Andalas (Ethical No. 528-UN.16.2-KEP-FK-21). The male mice were considered to be used as animal models in this study to minimize the variability of their responses to the treatments due to fluctuation in hormone levels that usually occur in female mice during the estrous cycle.

Experimental design

After being acclimatized, 24 animals were assigned randomly into 4 groups according to their respective diets, including (1) low-fat diet (LFD), (2) high-fat diet (HFD), (3) HFD combined with 10% (w/w) of *C. edulis* fiber extract (HFD + CE), and (4) HFD combined with 10% (w/w) *M. arundinacea* fiber extract (HFD + MA). LFD, composed of 10% kcal of energy from fat, was a commercial diet purchased from Citra Ina Feedmill (Jakarta, Indonesia). The HFD, consisting of 53% kcal from fat, was prepared based on the previous study (7). The groups received the respective diets *ad libitum* for 12 weeks. After that, the treatments were terminated, and the animals were sacrificed for further analysis.

The LFD-fed group was assigned as a control group in this study. The LFD was composed of 40 g fat/kg of diet, equal to 10% kcal of energy contribution to the total energy content (as described by the manufacturer). Based on a previous report (20), LFD exerted a similar effect as a normal diet (ND) on body weight, blood glucose, and metabolic hormones (leptin and adiponectin) in male mice after an 18-week diet treatment. Moreover, LFD elicited comparable effects as ND on high-density lipoprotein (HDL) and triglyceride (TG) levels and behavior of mice (20). Thus, the use of LFD for the control group in the present study was reasonable since it had similar outcomes as ND on several metabolic and behavioral aspects in male mice as experimental models.

A 10% single dose of fiber was decided to use in the experiment based on a previous study showing that 10% of extracted dietary fiber from jicama (*Pachyrhizus erosus*) was the best dose to counteract metabolic disorders and gut dysbiosis in HFD-fed mice (9). Although a higher dose of jicama fiber (20%) was effective, it was different in *C. edulis* and *M. arundinacea* fibers. Our preliminary observations found that the supplementation of *C. edulis* and *M. arundinacea* fibers at a dose of 20% substantially reduced food intake while increasing water intake in 2 weeks of diet treatment. Hence, a 10% single dose of *C. edulis* and *M. arundinacea* fibers was chosen for this study. The fibers of *C. edulis* and *M. arundinacea* were separately mixed with HFD (10% of fiber and 90% of HFD), and the diets were given to the animals for 12 consecutive weeks.

Assessments of blood glucose homeostasis

Random blood glucose measurements were conducted at the beginning and at the end of treatments, while other measurements were performed at the end of treatments only.

Random blood glucose levels of mice were determined using an automated glucometer (AGM-4000, All Medicus Co., South Korea) with blood samples drawn from the tail vein in the morning (09.00-10.00) under *ad libitum* conditions. On the latest day of the experiment, after 18 h of fasting, blood glucose levels were subsequently measured using a similar procedure to random blood glucose measurement. A glucose tolerance test (GTT) was conducted after the intraperitoneal injection of d-glucose (20 g/kg) under a 6-h fasting condition. Furthermore, the insulin tolerance test (ITT) was performed after an intraperitoneal insulin injection at 0.75 IU/kg under a 6-h fasting condition. The area under the curve (AUC) for GTT and ITT was eventually determined based on the blood glucose levels from the tests.

Biochemical, molecular, and histopathological measurements

The body weight of mice was measured using a sensitive balance (NVT.220-1, Ohaus, USA). On the latest day of treatment, mice were anesthetized with ketamine (lethal dose of 200 mg/kg, intraperitoneally). Then, blood samples were collected through cardiac puncture. The plasma samples were obtained by a 10-min centrifugation (3000 rpm at 4 °C) and kept at -80 °C to measure metabolic hormones and plasma lipids. Then, the epididymal white adipose tissue (eWAT) samples were removed and weighed to determine the mass. Eventually, tissue samples were collected for molecular and histopathological observations.

Histopathological observations of eWAT and liver

Samples of eWAT and liver tissues were freshly collected and immediately fixed in the 10% neutral-buffered formalin (Sigma-Aldrich, USA) for 12 h. Thereafter, the tissues were subjected to histological preparations and stained using hematoxylin and eosin (21). Five representative tissue slices were observed in each animal under a microscope (Olympus CX31, Tokyo). The quantitative measurements on the tissues were performed using Image J software for Windows (NIH, USA; available at: https://imagej.nih.gov/ij/download.html).

Measurements of metabolic hormones

Following the manufacturer's protocols, the plasma insulin, GLP-1, and FGF21 levels were measured using assay kits for mice. The microplate ELISA reader (x-Mark-1681150, Bio-Rad, USA) was used to measure the absorbance of samples.

Measurements of plasma lipids

Subsequently, plasma total cholesterol (TC), TG, low-density lipoprotein (LDL), and HDL were determined by the enzymatic colorimetric assays following the previously described procedures (22).

Gut microbiota composition analysis by 16S rRNA next-generation sequencing

The caeca fecal samples of mice were freshly collected (50 mg) using a sterilized spatula and gloves and quickly stored at -80 °C. The microbial genomic DNA from the samples was extracted as per the protocols described by the manufacturer (DNeasy PowerSoil Pro kit, Qiagen). Furthermore, the V3-V4 hypervariable regions of the bacterial 16S rRNA gene were amplified using PCR with specific primers as follows: 341F (5'-CCTAYGGGRBGCASCAG-3') and 806R (5'-GGACTACNNGGGTATCTAAT-3') (23). Subsequently, the purified samples of PCR product were sequenced using a next-generation sequencing (NGS) (Illumina Novaseq 6000 platform) in Novogene (Singapore). The NGS data were analyzed using 16S Metagenomics software (GAIA version 2.0, Sequentia Biotech).

Statistical analysis

Data were presented as mean ± SEM. The data were analyzed by one-way ANOVA followed by Bonferroni post-hoc test using IBM SPSS Statistics Base 22.0 for Windows. P-values < 0.05 were considered statistically significant.

RESULTS

Effect of C. edulis and M. arundinacea fibers on body weight and eWAT profile

The body weight measurements revealed weight gain in all groups after the 12-week diet treatments. However, the significant increments of body weight appeared only in the HFD and HFD + MA groups compared with their initial

body weight (Fig. 1A). Furthermore, the significant gain of body weight was 3 times and 2 times higher in HFD and HFD + MA groups than that in the LFD group, respectively (Fig. 1B). Although the increment of body weight was not significant between HFD + CE and LFD groups (Fig. 1B).

The gross morphology of WAT found that HFD increased the excessive accumulation of eWAT in all groups compared with the LFD group (Fig. 1C). Moreover, the measurements of eWAT mass and index also confirmed that HFD substantially elevated WAT weight and index in HFD and HFD + MA groups compared with LFD and HFD + CE groups (Fig. 1D and 1E). However, those fed with HFD + MA exhibited no significant changes in eWAT mass and index in comparison to the HFD group (Fig. 1D and 1E).

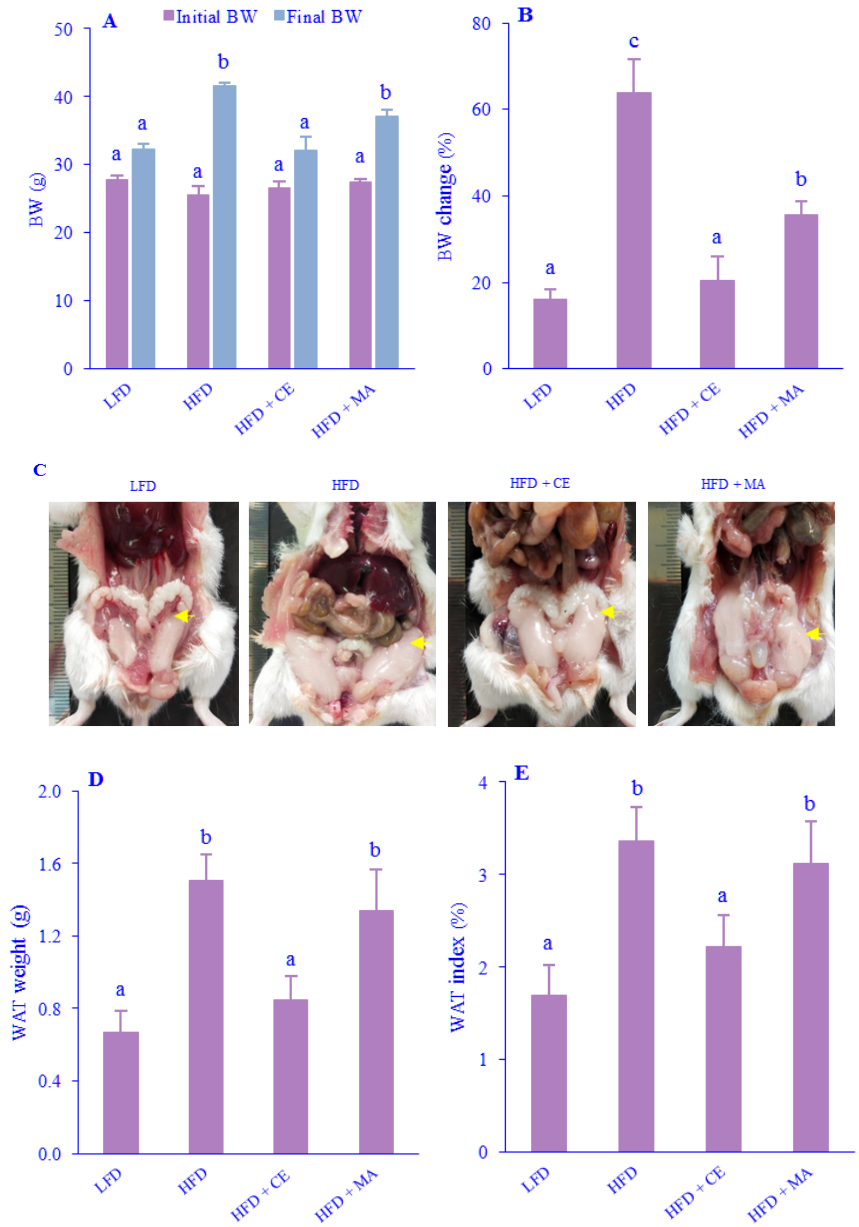

Fig. 1. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on BW and WAT of HFD-fed mice. (A) Initial and final body weight; (B) body weight gain; (C) gross morphology of eWAT; (D) eWAT mass; (E) eWAT index presented against body weight. Data were expressed as mean ± SEM. Different lowercase letters indicate significant differences. BW, Body weight; WAT, white adipose tissue; eWAT, epididymal white adipose tissue; LFD, low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea*.

The microscopic observations on eWAT found that HFD induced significant enlargement in adipocytes compared to the LFD group (Fig. 2A and 2B). Otherwise, mice fed HFD + *C. edulis* fiber had comparable adipocyte size to LFD. The adipocyte size and number were significantly smaller and more in the HFD + CE group, respectively, than in the HFD group (Fig. 2B and 2C). Moreover, the group treated with HFD + *M. arundinacea* exhibited larger and fewer adipocytes than the HFD + CE group (Fig. 2). However, there were no significant differences between the HFD and HFD + MA groups in the adipocyte size and number (Fig. 2).

Effect of C. edulis and M. arundinacea fibers on plasma lipid profile

Plasma lipid measurements at the end of treatment indicated that mice fed with HFD exhibited significant rises in TC, LDL, and TG levels compared with the LFD group (Fig. 3A-C). On the other hand, the HDL level decreased in the HFD group and HFD groups supplemented with *C. edulis* and *M. arundinacea* fibers compared with the LFD-fed group, which was significant in the HFD and HFD + CE groups (Fig. 3D). Also, the group treated with *C. edulis* fiber had lower TC, LDL, and TG levels than the HFD group, significantly. However, the mentioned parameters found no significant changes when compared with the LFD group (Fig. 3A-C). Moreover, group fed with *M. arundinacea* fiber showed higher TC and TG levels compared with LFD and HFD + CE groups. In contrast, the levels of TC and TG remained lower in the HFD + MA group than those in the HFD group, significantly (Fig. 3A and 3C).

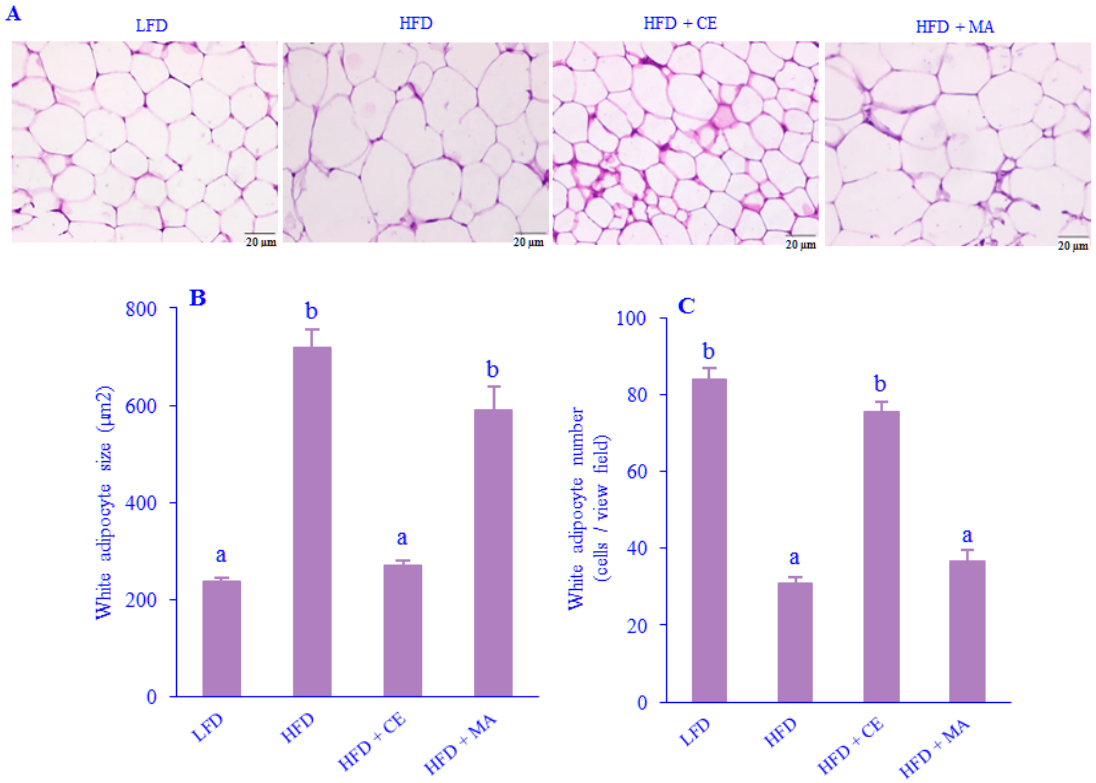

Fig. 2. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on the adipocyte number and size of eWAT in HFD-fed mice. (A) Representative photomicrograph of eWAT; (B) adipocyte size; (C) number of adipocytes/view field. Tissues were stained with hematoxylin and eosin (magnification 40×). Data were expressed as mean ± SEM. Different lowercase letters indicate significant differences. LFD, Low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea*.

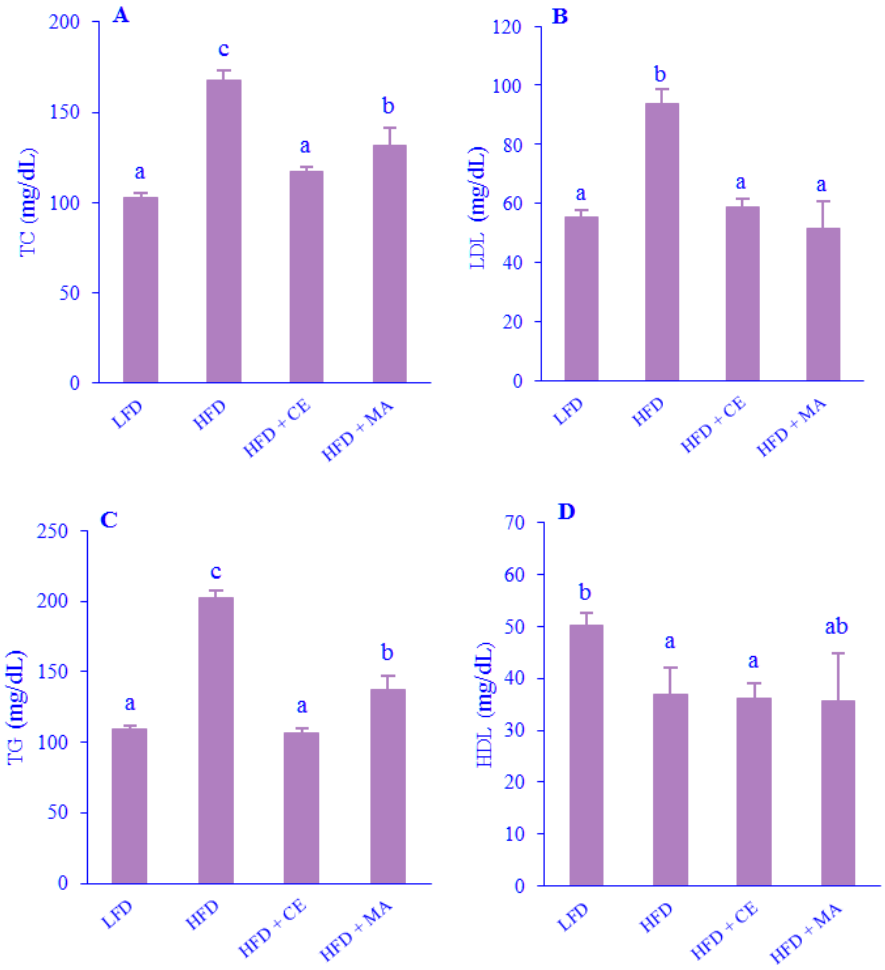

Fig. 3. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on plasma lipid profile in HFD-fed mice. (A) TC; (B) LDL; (C) TG; (D) HDL. Data were expressed as mean ± SEM. Different lowercase letters indicate significant differences. TC, Total cholesterol; LDL, low-density lipoprotein; TG, triglyceride; HDL, high-density lipoprotein; LFD, low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea*.

Effect of C. edulis and M. arundinacea fibers on blood glucose homeostasis and metabolic hormones

Monitoring of blood glucose levels found that HFD caused substantial elevation of random and fasting blood glucose at the end of treatment compared with the LFD group. On the other hand, mice fed with dietary fiber from *C. edulis* and *M. arundinacea* had substantially lower blood glucose levels than the HFD group; however, no significant difference with those fed LFD (Fig. 4A and 4B) was observed. The assessment of glucose tolerance revealed that HFD exhibited glucose intolerance as indicated by significantly higher blood glucose levels at the major time points of measurements after glucose injection (15, 30, 60, and 90 min) (Fig. 4C) and had higher AUC values compared with LFD group (Fig. 4D). Otherwise, mice fed with fibers from *C. edulis* and *M. arundinacea* had a sustained lower blood glucose at the major time points after glucose injection as well as lower AUC values (significantly in the only HFD + CE group) compared with HFD group (Fig. 4C and 4D). The assessment of insulin tolerance also revealed that HFD caused apparent intolerance to insulin as indicated by sustained higher blood glucose levels compared with other groups at 15-, 30-, 60-, and 90-min post insulin administration (Fig. 4E). Moreover, the AUC of ITT indicated that the HFD group had higher AUC values compared with other groups (Fig. 4F).

The measurement of plasma insulin levels at the end of treatment demonstrated that the HFD group had a significantly higher insulin level compared with the LFD and HFD + CE groups; however, it was not significant in comparison to the HFD + MA group (Fig. 4G). Furthermore, plasma GLP-1 levels were elevated in HFD and HFD + MA groups compared with the LFD group, while it was not statistically significant in HFD + CE group compared with other groups (Fig. 4H). Also, plasma FGF21 levels were substantially elevated in the HFD, HFD + CE, and HFD + MA groups compared with the LFD group. Moreover, the level of FGF21 in the HFD + MA group was significantly higher than in the HFD group (Fig. 4I).

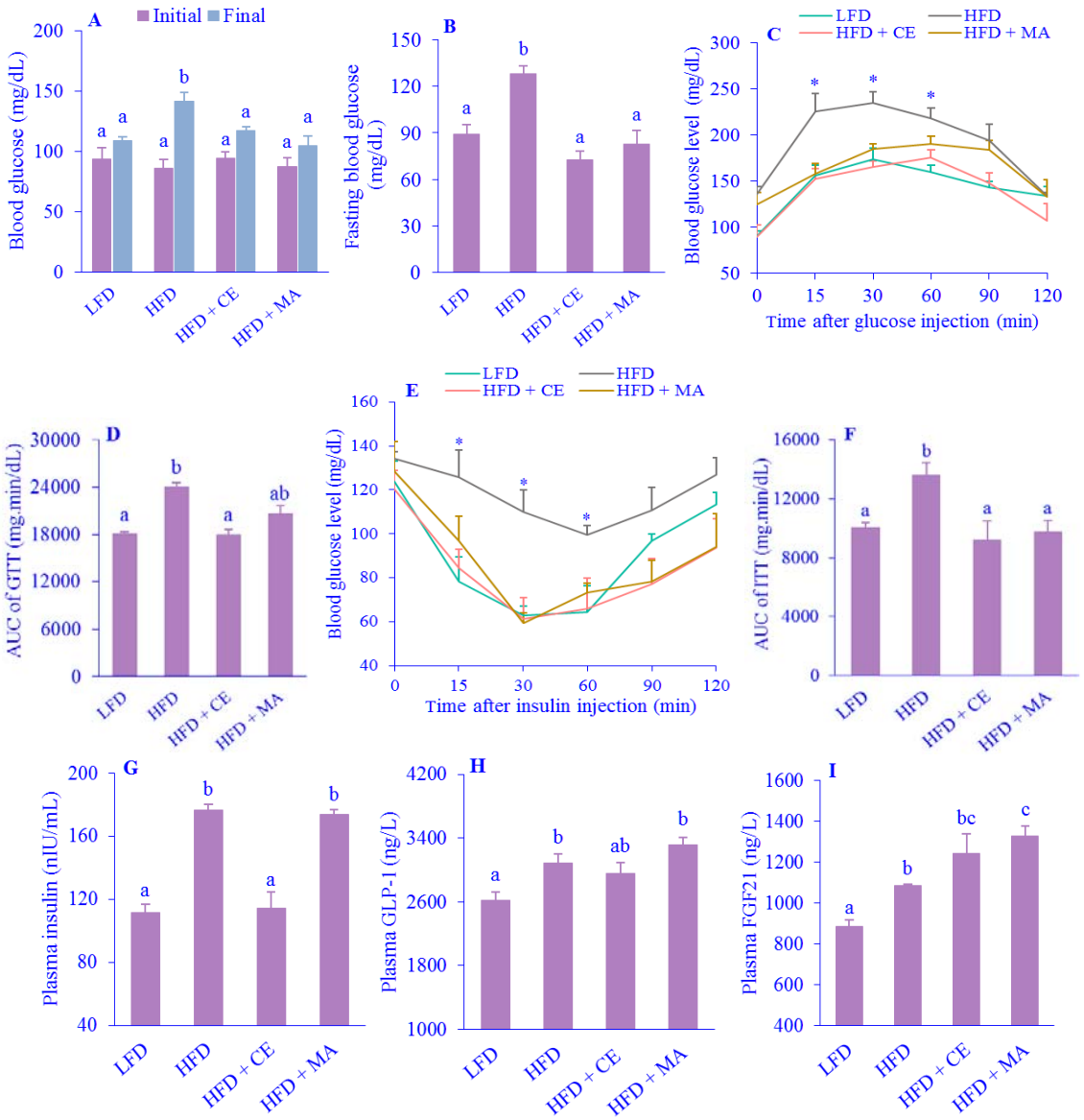

Fig. 4. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on blood glucose homeostasis and metabolic hormones in HFD-fed mice. (A) Random blood glucose measured at the beginning and the end of treatment; (B) fasting blood glucose measured at the end of treatment; (C) blood glucose levels during GTT; (D) AUC of GTT; (E) blood glucose levels during ITT; (F) AUC of ITT; (G) plasma levels of insulin; (H) GLP-1 plasma levels; (I) FGF21 plasma levels. Data were expressed as mean ± SEM. Different lowercase letters indicate significant differences. $^{*}P < 0.05$ indicates a significant difference compared with other groups. AUC, Area under curve; GTT, glucose tolerance test; ITT, insulin tolerance test; GLP-1, glucagon-like peptide 1; FGF21, fibroblast growth factor 21; LFD, low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea.*

Effect of C. edulis and M. arundinacea fibers on the liver

The histopathological examination of the liver exhibited high pathological alterations in the HFD group compared with the LFD group (Fig. 5A). The steatosis in the liver tissue was markedly higher in the HFD group than the LFD group (Fig. 5B), and the number of inflammatory cells also elevated significantly in the HFD group than the LFD group (Fig. 5C). Moreover, the percentage of degenerated hepatocytes was substantially increased in the HFD group than the LFD group (Fig. 5D). In addition, the percentage of abnormal sinusoids were also markedly increased in HFD group compared with LFD group (Fig. 5E), and the diameter of the central vein was significantly higher in HFD group than LFD group (Fig. 5F). Otherwise, mice fed with *C. edulis* fiber had markedly lower steatosis compared with HFD group, whereas, those fed with *M. arundinacea* fiber exhibited a slight decrease in steatosis. The number of inflammatory cells, degenerated hepatocytes, and sinusoid edema also depicted a similar tendency, showing that *C. edulis* fiber exerted a significantly higher effect than *M. arundinacea* fiber. However, the central vein diameter was statistically comparable in mice fed with *C. edulis* and *M. arundinacea* fibers to the LFD group (Fig. 5).

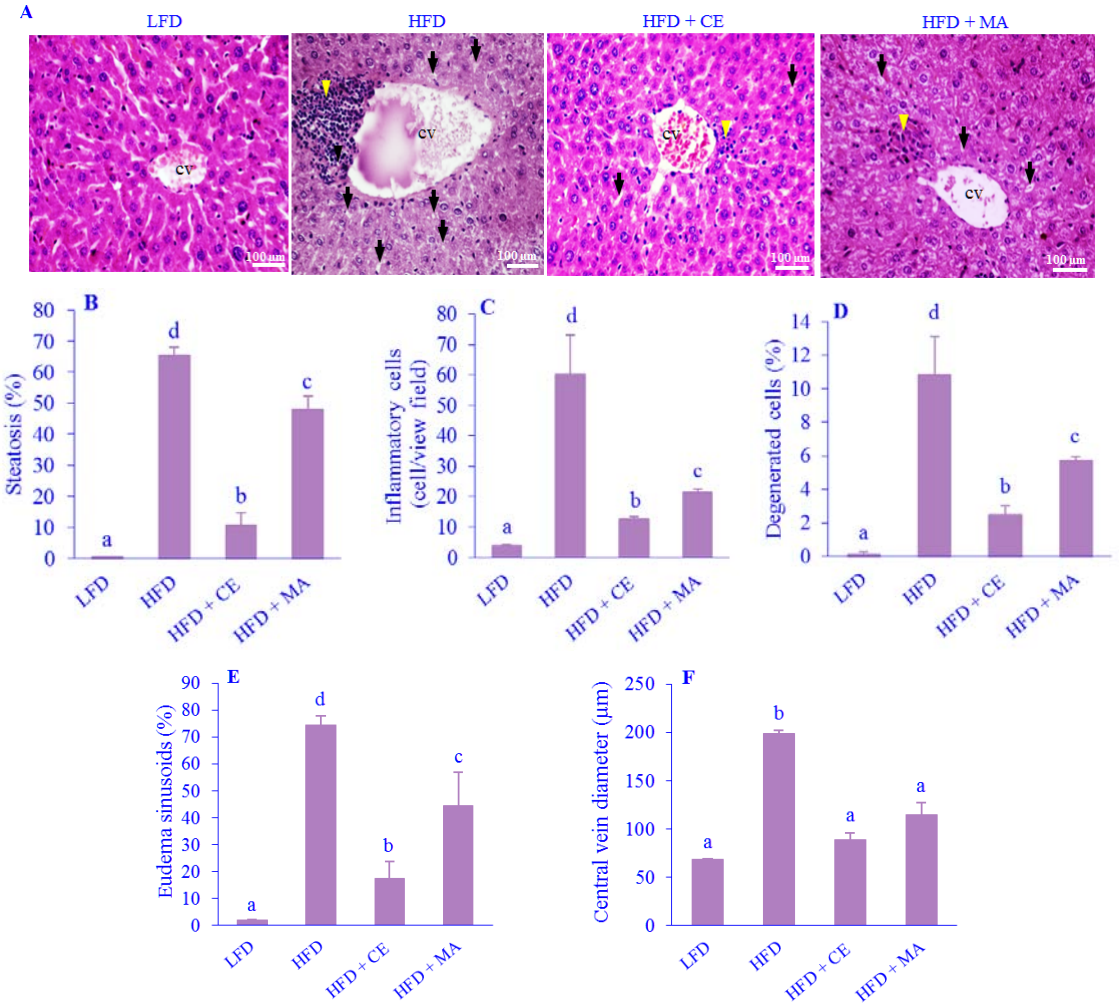

Fig. 5. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on liver in HFD-fed mice. (A) Representative photomicrographs of liver tissue (magnification 10×); (B) percentage of hepatic steatosis; (C) number of inflammatory cells; (D) number of degenerated hepatocytes; (E) incidence of edema in sinusoids; (F) diameter of central vein. Tissues were stained with hematoxylin and eosin. Data were expressed as mean ± SEM. Different lowercase letters indicate significant differences. CV, Central vein; yellow arrows, inflammatory cells; black arrows, steatosis; LFD, low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea*.

Effect of C. edulis and M. arundinacea fibers on gut microbiota composition

As shown in Fig. 6A, the sequencing depth deployed in the present study was reliable in elucidating gut microbial diversity. The observed operational taxonomy units (OTUs) were varied among treatment groups. The highest observed OTUs were in the HFD group (123412 observed OTUs), followed by the HFD + CE group (121410 OTUs) and HFD + MA group (115876 OTUs), while the LFD group was the lowest (88927 OTUs).

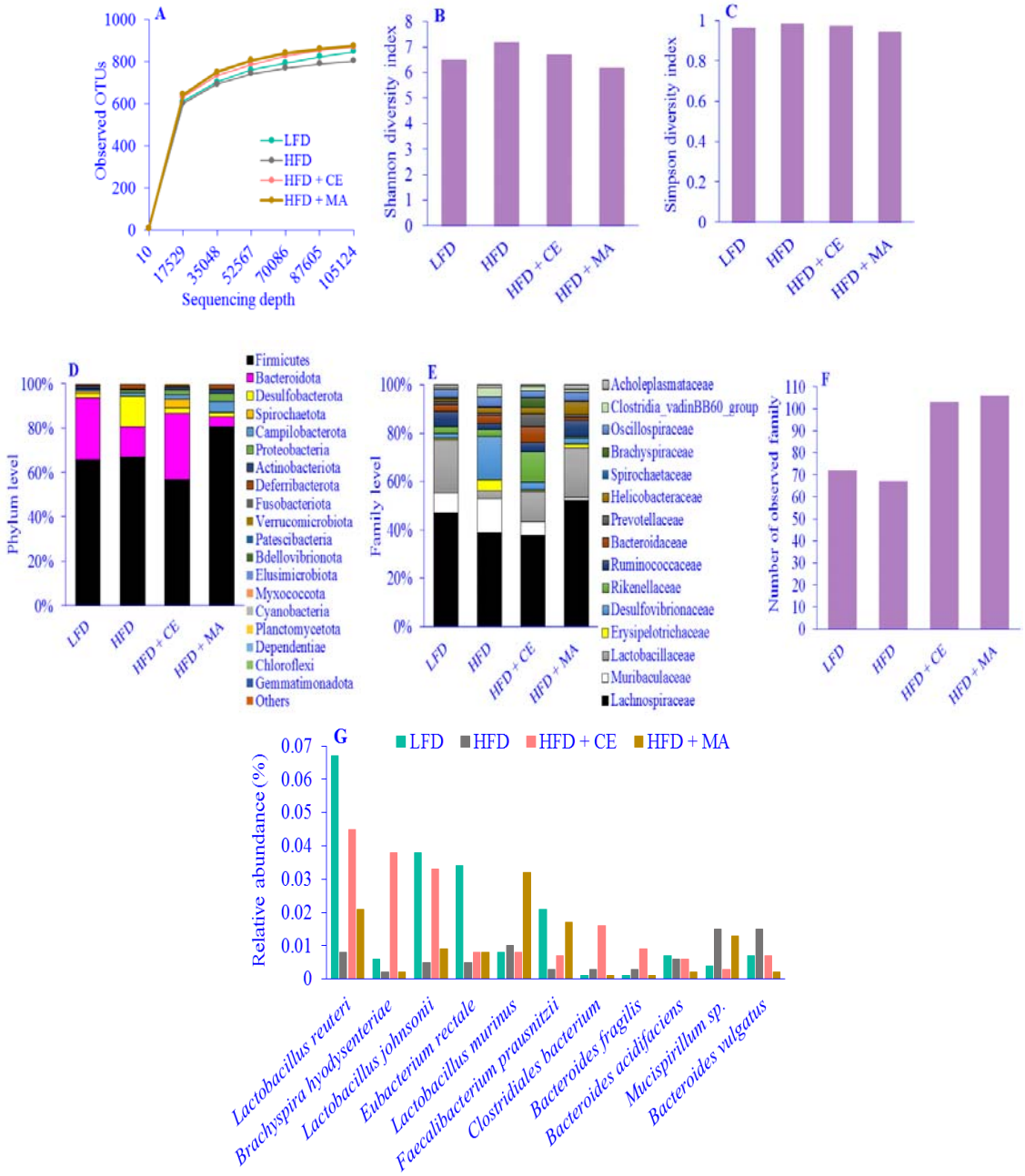

Fig. 6. Effect of dietary fiber of *C. edulis* and *M. arundinacea* rhizomes on gut microbiota composition in HFD-fed mice. (A) Observed OTUs against 16S rRNA sequencing depth on fecal sample; (B) Shannon diversity index; (C) Simpson diversity index; (D) predominant microbiota composition at phylum levels; (E) predominant microbiota composition at family levels; (F) number of families; (G) relative abundance of predominant species. Data were expressed as the mean. OTUs, Operational taxonomy units; LFD, low-fat diet; HFD, high-fat diet; CE, *C. edulis;* MA, *M. arundinacea*.

The Shannon and Simpson diversity indexes of gut microbiota revealed no substantial differences among all groups of treatment but showed highly diverse gut microbiota in all groups (Fig. 6B and 6C). Further analysis of composition at the phylum level revealed that the HFD group had a higher abundance of *Firmicutes* and lower *Bacteroidota* than the LFD group. Conversely, mice fed with *C. edulis* fiber had an increased abundance of *Bacteroidota* and a decreased abundance of *Firmicutes*. On the other hand, HFD + MA group exhibited a profound increase in *Firmicutes* and a decrease in *Bacteroidota* than other groups (Fig. 6D). In addition to *Firmicutes*, mice in HFD group had a high abundance of *Desulfobacterota*, while it was decreased in other groups including LFD, HFD + CE, and HFD + MA (Fig. 6D). Moreover, *Spirochaetota* was specifically predominant in HFD + CE group, while *Campilobacterota* was specifically predominant in HFD + MA group (Fig. 6D). Further analysis at the family level revealed that *Lachnospiraceae* was the most predominant family of gut microbiota in all groups, but its abundance was lower in HFD and HFD + CE groups compared with LFD and HFD + MA groups (Fig. 6E). The *Muribaculaceae* increased in HFD group, but decreased in HFD + CE and HFD + MA groups. Otherwise, *Lactobacillaceae* decreased in HFD, but increased in other groups (Fig. 6E). Furthermore, *Desulfovibrionaceae* increased in HFD, but decreased in other groups. The *Rikenellaceae* specifically increased in the HFD + CE group, whereas the *Erysipelotrichaceae* were specifically higher in the HFD group (Fig. 6E). The total number of observed families was lowest in the HFD group (67 families) and increased in mice treated with dietary fibers, either *C. edulis* (103 families) or *M. arundinacea* (106 families) (Fig. 6F). Figure 6G demonstrates the relative abundance of 11 predominant species in all groups. The HFD group had a significantly higher abundance of *Mucispirillum* sp. and *Bacteroides vulgatus* compared with the LFD group. However, mice fed with *C. edulis* fiber had a higher abundance of beneficial species, including *Lactobacilus reuteri, L. johnsonii,* and *Bacteroides fragilis.* Moreover, a pathogenic species such as *Brachyspira hyodysentriae* also increased in the HFD + CE group. Furthermore, mice fed with *M. arundinacea* fiber had a substantial elevation of *L. murinus, Faecalibacterium prausnitzii*, and *Mucispirillum* sp (Fig. 6G).

DISCUSSION

This present experimental study using adult male mice revealed the ameliorative effects of fibers extracted from *C. edulis* and *M. arundinacea* rhizomes against metabolic syndromes and gut dysbiosis promoted by HFD. It was found that the fibers could prevent obesity, dyslipidemia, type 2 diabetes, and hepatic steatosis. In addition, the fibers elicited a substantial effect on the microbiota community in the gut.

It was demonstrated that dietary fibers from the rhizomes of *C. edulis* and *M. arundinacea* have differential effects on body weight gain, adipose tissue mass, adipocyte size, and plasma lipid profile in mice fed with HFD. The HFD-fed mice exhibited significant weight gain, excessive accumulation of eWAT, adipocyte hypertrophy, and dyslipidemia, which were significantly attenuated by the addition of *C. edulis* dietary fiber. Conversely, *M. arundinacea* dietary fiber was found to have a lesser or no significant effect on body weight gain or eWAT mass, but resulted in larger adipocyte size and higher plasma TC and TG levels than *C. edulis* fiber. The findings suggested that *C. edulis* fiber elicited a protective role against obesity and dyslipidemia by reducing adipose tissue mass and adipocyte hypertrophy and improving lipid metabolism. These findings were consistent with a previous study, showing that dietary fiber intake from bamboo shoots could reduce body weight gain and adiposity, and improve lipid profiles in HFD-fed mice (23). Likewise, another report also found that type 3 resistance starch from *C. edulis* effectively improved plasma lipid profiles, while preventing obesity in mice (24). The differential effects of *C. edulis* and *M. arundinacea* fibers on body weight gain, adiposity, and lipid metabolism may be due to differences in their physicochemical properties

and/or composition. For example, *C. edulis* fiber has been reported to be high in soluble fiber and resistant starch (25), which may be effective in modulating lipid absorption rate and its metabolism (13). In contrast, *M. arundinacea* fiber is high in insoluble fiber (26), thus blunting its effectiveness in counteracting the development of obesity and dyslipidemia promoted by HFD. This reason may also explain why adipocyte hypertrophy was still observed in animals treated with *M. arundinacea* fiber. In addition, it should be noted that while *C. edulis* fiber effectively improved several plasma lipid components (including TC, TG, and LDL), but it did not effectively increase HDL levels. Given that the metabolism of HDL involves multiple pathways and mechanisms, including biosynthesis, maturation, and clearance (27), *C. edulis* fiber might only play a limited role in one aspect of HDL metabolism. This could prevent its optimal effect on raising HDL levels under an HFD challenge. Collectively, the findings suggested that dietary fiber from different sources may have distinct effects on body weight and lipid metabolism and highlighted the significance of considering dietary fiber diversity in the development of dietary interventions for obesity and related metabolic disorders. However, further studies are needed to elucidate the mechanisms underlying the effects of dietary fiber on adipose tissue biology and lipid metabolism and to evaluate the long-term effects of dietary fiber intake on health outcomes.

The present study also indicated that dietary fibers extracted from *C. edulis* and *M. arundinacea* had a noticeable effect in sustaining blood glucose homeostasis against HFD-induced type 2 diabetes development. Interestingly, *C. edulis* fiber was more effective in preventing glucose intolerance and hyperinsulinemia than *M. arundinacea* fiber, though both fibers exerted comparable effects on blood glucose levels and insulin tolerance. In the previous study, it was also found that HFD caused the hypertrophy of the islet of Langerhans along with the type 2 diabetes development, whereas the supplementation of extracted fiber from the tuber of *P. erosus* was able to diminish it (28). A clinical investigation found that snacks made from the whole powder of *M. arundinacea* rhizomes, in the form of chips, effectively managed blood glucose levels in patients with type 2 diabetes (29). Additionally, the resistant starch from *C. edulis* has been demonstrated to improve blood glucose homeostasis in rats suffering from type 2 diabetes (30). In line with the reports, our present study also indicated the effectiveness of the dietary fiber of *C. edulis* rhizomes in preventing type 2 diabetes development. This could be attributed to the ability of dietary fiber from the *C. edulis* rhizomes to anticipate insulin resistance caused by HFD. Otherwise, the glucose intolerance and hyperinsulinemia observed under *M. arundinacea* fiber supplementation could be a subsequent impact of a persistent tissue microhypoxic state and sustained inflammatory response. Taken together, our present study provided evidence that dietary fiber from *C. edulis* rhizomes has potential therapeutic benefits for managing type 2 diabetes development by improving blood glucose homeostasis and insulin sensitivity. Further investigations are required to define its underlying mechanisms.

Overconsumption of HFD has been shown to dysregulate metabolic hormones, including GLP-1 and FGF21 (31,32). Similarly, the findings of the present study found that HFD noticeably elevated plasma GLP-1 and FGF21 levels, which may indicate hormonal resistance. The supplementation of *C. edulis* and *M. arundinacea* fibers in HFD also increased GLP-1 and FGF21 with a higher magnitude. A study performed on HFD-fed mice demonstrated that HFD supplementation substantially increased the secretion of GLP-1 plausibly through the increment of SCFAs as a product of fiber fermentation by gut microbiota (33). Another study also indicated that fiber supplementation elicited a modulatory effect on FGF21 secretion by involving the alterations in gut microbiota composition (34). Hence, it was speculated that *C. edulis* and *M. arundinacea* fibers per se may also elicit a stimulatory effect on GLP-1 and FGF21 secretion. Future investigation is required to elucidate it.

The findings of this study also indicated that HFD caused profound pathological alterations in the liver, particularly hepatic steatosis and

other histopathological outcomes, including an increase in macrophage infiltration in the liver tissue. It has been demonstrated that HFD promotes liver damage and triggers inflammation, leading to non-alcoholic fatty liver disease (7). Interestingly, the supplementation of dietary fiber from rhizomes of *C. edulis* and *M. arundinacea* in the diet could substantially lower the level of liver pathology. Likewise, an experiment in HFD-fed mice also revealed that type 3 resistant starch of *C. edulis* could alleviate hepatic steatosis (24). However, in our study, the magnitude of reduction in liver pathology was higher in mice treated with *C. edulis* fiber compared with *M. arundinacea* fiber, suggesting the predominant effect of *C. edulis* fiber over *M. arundinacea* in precluding the development of HFD-induced liver diseases. It was speculated that the composition and phytochemical constituents of the fibers may be attributed to this difference. Further comparative study emphasizing the composition and constituents of *C. edulis* and *M. arundinacea* is needed to confirm it.

The microbiota composition in the gut is closely related to the metabolic homeostasis of the body (35), while some dietary fibers have been suggested to exert an apparent impact on the gut microbiota composition (12,16). In line with the previous reports (6,7), the present study showed that HFD caused profound alterations in the gut microbiota composition at the phylum level by markedly promoting *Firmicutes* over *Bacteroidota* and a substantial increment of *Desulfobacterota*. Otherwise, the supplementation of *C. edulis* fiber, but not *M. arundinacea* fiber, in HFD reduced the *Firmicutes* abundance, while noticeably increasing the abundance of *Bacteroidota*. In addition, both *C. edulis* and *M. arundinacea* fibers reduced the abundance of *Desulfobacterota*. It has been indicated that an overgrowth of *Firmicutes* is associated with obesity development and widespread inflammatory responses in the body, leading to type 2 diabetes (35). Moreover, *Desulfobacterota* overgrowth is closely related to type 2 diabetes and obesity (9,35). Conversely, the increment of *Bacteroidota* has been linked to beneficial outcomes on metabolic regulation (36). Taken together, the previous reports and the present results suggested that a modulatory effect elicited particularly by *C. edulis* fiber on the composition of *Firmicutes*, *Bacteroidota*, and *Desulfobacterota* may underlie its higher effectiveness in counteracting HFD-induced metabolic diseases compared with *M. arundinacea* fiber.

According to analysis at the family level, both *C. edulis* and *M. arundinacea* fibers caused a reduction in the abundance of *Desulfovibronaceae*, while increasing the abundance of *Lactobacillaceae*. Some studies have suggested that *Desulfovibronaceae* is predominantly composed of pathogenic species and is closely associated with obesity and fatty liver disease in both rodents and humans (37,38). On the other hand, *Lactobacillaceae* has been shown to counteract *Desulfovibrionaceae* in aggravating hepatic steatosis in mice fed HFD (36). In addition, *Lactobacillaceae* is implicated in managing dyslipidemia (39). Further investigation at the species level found that *C. edulis* fiber specifically promoted beneficial species, including *L. reuteri* and *L. johnsonii*. These species have been demonstrated to be implicated in the enhancement of gut barrier function (40), thereby preventing inflammatory response and subsequent impacts on metabolic homeostasis. Moreover, *M. arundinacea* fiber limitedly promoted beneficial species of *L. murinus* and *F. prausnitzii* that have also been reported to contribute to gastrointestinal health (41,42). However, *C. edulis* fiber noticeably elevated the abundance of pathogenic species *B. hyodysentriae,* but suppressed another pathogenic species, namely *Mucispirillum* sp. In contrast, *M. arundinacea* fiber supported the overgrowth of *Mucispirillum* sp. to be comparable to that of HFD alone. Although the rise of *B. hyodysentriae* is reported to commonly link to gastrointestinal problems (43), the present results indicated that it may not link to dysregulated metabolic homeostasis and inflammation in mice fed *C. edulis* fiber. This could be due to the inhibitory effect elicited by health-promoting species that were predominant under *C. edulis* fiber treatment against *B. hyodysentriae*. Meanwhile, the overgrowth of *Mucispirillum* sp. has been

strongly associated with obesity and inflammation (44,45). Hence, the increment of its abundance under *M. arundinacea* fiber treatment might underlie the lesser effectiveness of the fiber compared to *C. edulis* fiber in precluding HFD effects.

Previous reports have highlighted the benefits of dietary fiber from *C. edulis*, particularly in the form of type 3 resistant starch, and the powder of *M. arundinacea* rhizomes, on gut microbiota composition. A study on type 2 diabetic mice showed that the resistant starch from *C. edulis* increased gut microbiota diversity and specifically boosted the abundance of SCFA-producing species (30). Additionally, the resistant starch from *C. edulis* promoted the growth of *Clostridium* in mice (46). In addition, another experiment in rats revealed that supplementation of *M. arundinacea* powder in the diet enhanced the growth of Lactobacilli bacteria, but failed to suppress the overgrowth of pathogenic *Escherichia coli* and *Clostridium perfringens* (26). These findings suggested that *C. edulis* is more effective in promoting health-associated gut microbiota compared to *M. arundinacea*. Similarly, our current study aligns with these findings, indicating that the extracted dietary fiber from *C. edulis* rhizomes has a more positive impact on gut microbiota composition than the fiber from *M. arundinacea*. Specifically, *C. edulis* fiber proved more effective than *M arundinacea* fiber in promoting beneficial species and suppressing pathogenic microbiota. This difference might arise from the distinct physicochemical properties of the fibers, which in turn influence their effects on microbiota composition. A previous report indicated that *C. edulis* fiber is rich in phenolic compounds (1) that have been suggested to enhance beneficial gut bacteria, while inhibiting pathogenic species (47). A phytochemical analysis comparing the phenolic content between *C. edulis* and *M. arundinacea* fibers is necessary to validate this hypothesis.

Although the current study provided some interesting findings, it had several limitations that should be considered. Firstly, the study only investigated the effect of dietary fiber from two sources (*C. edulis* and *M. arundinacea* rhizomes) with a single dose of 10%, thus it is unclear whether the other sources of dietary fiber and dosage variations may exert different effects against HFD. Secondly, the study did not investigate the composition and chemical constituents of the fibers that may be attributed to their variable effectiveness in exerting beneficial health effects. Thirdly, this study used LFD as a control diet, which may not perfectly represent the normal physiological responses of the animal models fed with ND. In addition, this study did not determine the level of SCFAs in the gut and blood plasma. As a result, it is still unclear whether the SCFAs are implicated in the counteractive effects of *C. edulis* and *M. arundinacea* fibers against HFD. Moreover, the other metabolic hormones (such as leptin, ghrelin, and glucagon) were not measured. Hence, it remains unknown whether the *C. edulis* and *M. arundinacea* fibers could also modulate metabolic hormone levels. Next, this study only deployed the 16S rRNA sequencing technique to elucidate the gut microbiota composition. A further exploration using whole-genome sequencing is highly suggested to comprehensively reveal the dynamics of the microbiota community under dietary fiber treatments. In addition, future investigations deploying molecular approaches are required to better understand the mechanisms of these dietary fibers in preventing the development of metabolic diseases and gut dysbiosis. Furthermore, incorporating female animal models into the research is crucial for understanding the sex-specific physiological responses to dietary fiber treatments.

CONCLUSION

This present study revealed the ameliorative effect of dietary fibers extracted from rhizomes of *C. edulis* and *M. arundinacea* against metabolic diseases caused by HFD (obesity, dyslipidemia, type 2 diabetes, and liver steatosis) and gut dysbiosis. In regard to their effectiveness in preventing metabolic diseases, *C. edulis* dietary fiber was more effective than *M. arundinacea*. Therefore, the dietary fiber from the *C. edulis* rhizomes could be formulated as a potent candidate for the supplement against metabolic diseases and gut dysbiosis.

Acknowledgments

This study was supported by a World Class Research Grant (WCR) from the Directorate of Research, Technology and Society Service of the Ministry of Education, Culture, Research and Technology of Indonesia (DRTPM KEMENDIKBUDRISTEK RI, No. 012/E5/PG.02.00.PL/2023).

Conflict of interest statement

The authors declared no conflict of interest in this study.

Authors' contributions

P. Santoso conceptualized and designed the research, conducted the animal studies, assessed metabolic parameters, and analyzed the results; R. Maliza executed the molecular experiments and undertook histopathological evaluations; P. Santoso and R. Maliza collaborated in drafting and preparing the manuscript. All authors have read and approved the finalized article. Each author has fulfilled the authorship criteria and affirmed that this article represents honest and original work.

AI declaration

The authors did not use any AI-assisted technologies in the preparation of this manuscript.

REFERENCES

1. Zhang J, Wang ZW. Soluble dietary fiber from *Canna edulis* Ker by-product and its physicochemical properties. Carbohydr Polym. 2013; 92(1):289-296. DOI: 10.1016/j.carbpol.2012.09.067.
2. Xie F, Gong S, Zhang W, Wu J, Wang Z. Potential of lignin from *Canna edulis* ker residue in the inhibition of α-d-glucosidase: kinetics and interaction mechanism merging with docking simulation. Int J Biol Macromol. 2017;95:592-602. DOI: 10.1016/j.ijbiomac.2016.11.100.
3. Tarique J, Sapuan SM, Khalina A, Ilyas RA, Zainudin ES. Thermal, flammability, and antimicrobial properties of arrowroot (*Maranta arundinacea*) fiber reinforced arrowroot starch biopolymer composites for food packaging applications. Int J Biol Macromol. 2022;213:1-10. DOI: 10.1016/j.ijbiomac.2022.05.104.
4. Malesza IJ, Malesza M, Walkowiak J, Mussin N, Walkowiak D, Aringazina R, *et al*. High-fat, western-style diet, systemic inflammation, and gut microbiota: a narrative review. Cells. 2021;10(11):3164,1-31. DOI: 10.3390/cells10113164.
5. Thomas MS, Blesso CN, Calle MC, Chun OK, Puglisi M, Fernandez ML. Dietary influences on gut microbiota with a focus on metabolic syndrome. Metab Syndr Relat Disord. 2022;20(8):429-439. DOI: 10.1089/met.2021.0131.
6. Yang J, Wei H, Zhou Y, Szeto CH, Li C, Lin Y, *et al*. High-fat diet promotes colorectal tumorigenesis through modulating gut microbiota and metabolites. Gastroenterology. 2021;162(1):135-149. DOI: 10.1053/j.gastro.2021.08.041.
7. Li J, Wu H, Liu Y, Yang L. High-fat diet-induced obesity model using four strains of mice: kunming, C57BL/6, BALB/c and ICR. Exp Anim. 2020;69(3):326-335. DOI: 10.1538/expanim.19-0148.
8. Tanes C, Bittinger K, Gao Y, Friedman ES, Nessel L, Paladhi UR, *et al*. Role of dietary fiber in the recovery of the human gut microbiome and its metabolome. Cell Host Microbe. 2021;29(3):394-407. DOI: 10.1016/j.chom.2020.12.012.
9. Santoso P, Maliza R, Rahayu R, Astrina Y, Syukri F, Maharani S. Extracted yam bean (*Pachyrhizus erosus (L.)* Urb.) fiber counteracts adiposity, insulin resistance, and inflammation while modulating gut microbiota composition in mice fed with a high-fat diet. Res Pharm Sci. 2022;17(5):558-571. DOI: 10.4103/1735-5362.355213.
10. Dong Y, Li Q, Guo Y, Zhao Y, Cao J. Comparison of physicochemical and in vitro hypoglycemic activity of bamboo shoot dietary fibers from different regions of Yunnan. Front Nutr. 2023;9:1102671,1-11. DOI: 10.3389/fnut.2022.1102671.
11. Rebello CJ, O'Neil CE, Greenway FL. Dietary fiber and satiety: the effects of oats on satiety. Nutr Rev. 2016;74(2):131-147. DOI: 10.1093/nutrit/nuv063.
12. Hijová E, Bertková I, Štofilová J. Dietary fibre as prebiotics in nutrition. Cent Eur J Public Health. 2019;27(3):251-255. DOI: 10.21101/cejph.a5313.
13. Ho HVT, Sievenpiper JL, Zurbau A, Blanco MS, Jovanovski E, Au-Yeung F, *et al*. The effect of oat β-glucan on LDL-cholesterol, non-HDL-cholesterol and apoB for CVD risk reduction: a systematic review and meta-analysis of randomised-controlled trials. Br J Nutr. 2016;116(8):1369-1382. DOI: 10.1017/S000711451600341X.
14. Surampudi P, Enkhmaa B, Anuurad E, Berglund L. Lipid lowering with soluble dietary fiber. Curr Atheroscler Rep. 2016;18(12):75,1-13. DOI: 10.1007/s11883-016-0624-z.
15. Powthong P, Jantrapanukorn B, Suntornthiticharoen P, Luprasong C. An *in vitro* study on the effects of selected natural dietary fiber from salad vegetables for lowering intestinal glucose and lipid absorption. Recent Pat Food Nutr Agric. 2021;12(2):123-133. DOI: 10.2174/2212798412666210311163258.
16. Cronin P, Joyce SA, O'Toole PW, O'Connor EM. Dietary fibre modulates the gut microbiota. Nutrients. 2021;13(5):1655,1-22. DOI: 10.3390/nu13051655.

17. Xu C, Cheng C, Zhang X, Peng J. Inclusion of soluble fiber in the gestation diet changes the gut microbiota, affects plasma propionate and odd-chain fatty acids levels, and improves insulin sensitivity in sows. Int J Mol Sci. 2020;21(2):635,1-14. DOI: 10.3390/ijms21020635.
18. Lattimer JM, Haub MD. Effects of dietary fiber and its components on metabolic health. Nutrients. 2010; 2(12):1266-1289. DOI: 10.3390/nu2121266.
19. Kumalasari ID, Nishi K, Harmayani E, Raharjo S, Sugahara T. Immunomodulatory activity of bengkoang (*Pachyrhizus erosus*) fiber extract *in vitro* and *in vivo*. Cytotechnology. 2014;66(1):75-85. DOI: 10.1007/s10616-013-9539-5.
20. Almeida-Suhett CP, Scott JM, Graham A, Chen Y, Deuster PA. Control diet in a high-fat diet study in mice: regular chow and purified low-fat diet have similar effects on phenotypic, metabolic, and behavioral outcomes. Nutr Neurosci. 2019;22(1):19-28. DOI: 10.1080/1028415X.2017.1349359.
21. Yao L, Wei J, Shi S, Guo K, Wang X, Wang Q, *et al*. Modified lingguizhugan decoction incorporated with dietary restriction and exercise ameliorates hyperglycemia, hyperlipidemia and hypertension in a rat model of the metabolic syndrome. BMC Complement Altern Med. 2017;17(1):132,1-12. DOI: 10.1186/s12906-017-1557-y.
22. Ding S, Jiang J, Yu P, Zhang G, Zhang G, Liu X. Green tea polyphenol treatment attenuates atherosclerosis in high-fat diet-fed apolipoprotein E-knockout mice *via* alleviating dyslipidemia and up-regulating autophagy. PLoS One. 2017;12(8):e0181666,1-18. DOI: 10.1371/journal.pone.0181666.
23. Li X, Guo J, Ji K, Zhang P. Bamboo shoot fiber prevents obesity in mice by modulating the gut microbiota. Sci Rep. 2016;6(1):32953,1-11. DOI: 10.1038/srep32953.
24. Qiu M, Jin XY, Zhang CJ, Zhang C, Wang M, Lu YB, *et al*. Effect of *Canna edulis* type 3 resistant starch in reducing body weight, serum lipid and acute toxicity. Chinese J Exp Trad Med Form. 2019;24:149-156. DOI: 10.13422/j.cnki.syfjx.20190130.
25. Praseptiangga D, Wandasari, Widyaastuti D. Chemical and physical properties of canna (*Canna edulis*) and jack bean (*Canavalia ensiformis*)-based composite flours. Food Res. 2022;6(2):354-367. DOI: 10.26656/fr.2017.6(2).292.
26. Harmayani E, Kumalasari ID, Marsono Y. Effect of arrowroot (*Maranta arundinacea L.*) diet on the selected bacterial population and chemical properties of caecal digesta of Sprague Dawley rats. Int Res J Microbiol. 2011;2:278-284.
27. Zhang Q, Ke Y, Hong H. HDL and lipid metabolism. Adv Exp Med Biol. 2022;1377:49-61. DOI: 10.1007/978-981-19-1592-5_4.
28. Santoso P, Maliza R, Insani SJ, Fadhilah Q, Rahayu R. Preventive effect of jicama (*Pachyrhizus erosus*) fiber against diabetes development in mice fed with high-fat diet. J Appl Pharm Sci. 2021;11(1):137-143. DOI: 10.7324/JAPS.2021.110116.
29. Novitasari D, Sunarti S, Fatmawati A. Garut chips (*Maranta arundinacea Linn*) as snacks and blood glucose levels, plasma angiotensin II and blood pressure in patients with type 2 diabetes mellitus (DMT2). Med Medika Indonesiana. 2011;45(1):53-58.
30. Zhang C, Ma S, Wu J, Luo L, Qiao S, Li R, *et al*. A specific gut microbiota and metabolomic profiles shifts related to antidiabetic action: the similar and complementary antidiabetic properties of type 3 resistant starch from *Canna edulis* and metformin. Pharmacol Res. 2020;159:104985,1-41. DOI: 10.1016/j.phrs.2020.104985.
31. Richards P, Pais R, Habib AM, Brighton CA, Yeo GS, Reimann F, *et al*. Highfat diet impairs the function of glucagon-like peptide-1 producing L-cells. Peptides. 2016;277:21-27. DOI: 10.1016/j.peptides.2015.06.006.
32. Nygaard EB, Møller CL, Kievit P, Grove KL, Andersen B. Increased fibroblast growth factor 21 expression in high-fat diet-sensitive non-human primates (*Macaca mulatta*). Int J Obes (Lond). 2014;38(2):183-191. DOI: 10.1038/ijo.2013.79.
33. Suzuki S, Aoe S. High β-Glucan barley supplementation improves glucose tolerance by increasing GLP-1 secretion in diet-induced obesity mice. Nutrients. 2021;13(2):527,1-11. DOI: 10.3390/nu13020527.
34. Martin A, Ecklu-Mensah G, Ha CWY, Hendrick G, Layman DK, Gilbert J, *et al*. Gut microbiota mediate the FGF21 adaptive stress response to chronic dietary protein-restriction in mice. Nat Commun. 2021;12(1):3838,1-11. DOI: 10.1038/s41467-021-24074-z.
35. Qin J, Li Y, Cai Z, Li S, Zhu J, Zhang F, *et al*. A metagenome-wide association study of gut microbiota in type 2 diabetes. Nature .2012;490(7418):55-60. DOI: 10.1038/nature11450.
36. Zhai X, Lin D, Zhao Y, Li W, Yang X. Effects of dietary fiber supplementation on fatty acid metabolism and intestinal microbiota diversity in C57BL/6J mice fed with a high-fat diet. J Agric Food Chem. 2018;66(48):12706-12718. DOI: 10.1021/acs.jafc.8b05036.
37. Lin YC, Lin HF, Wu CC, Chen CL, Ni YH. Pathogenic effects of *Desulfovibrio* in the gut on fatty liver in diet-induced obese mice and children with obesity. J Gastroenterol. 2018;57(11):913-925. DOI: 10.1007/s00535-022-01909-0.
38. Shahi SK, Ghimire S, Lehman P, Mangalam AK. Obesity induced gut dysbiosis contributes to disease severity in an animal model of multiple sclerosis. Front Immunol. 2022;3:966417,1-13. DOI: 10.3389/fimmu.2022.966417.
39. Frappier M, Auclair J, Bouasker S, Gunaratnam S, Diarra C, Millette M. Screening and characterization of some *lactobacillaceae* for detection of cholesterol-lowering activities. Probiotics Antimicrob Proteins. 2022;14(5):873-883. DOI: 10.1007/s12602-022-09959-9.

40. Liu HY, Roos S, Jonsson H, Ahl D, Dicksved J, Lindberg JE, *et al*. Effects of *Lactobacillus johnsonii* and *Lactobacillus reuteri* on gut barrier function and heat shock proteins in intestinal porcine epithelial cells. Physiol Rep. 2015;3(4):e12355,1-13. DOI: 10.14814/phy2.12355.
41. Hu J, Deng F, Zhao B, Lin Z, Sun Q, Yang X, *et al*. *Lactobacillus murinus* alleviate intestinal ischemia/reperfusion injury through promoting the release of interleukin-10 from M2 macrophages *via* Toll-like receptor 2 signaling. Microbiome. 2022;10(1):38,1-21. DOI: 10.1186/s40168-022-01227-w.
42. Xu J, Liang R, Zhang W, Tian K, Li J, Chen X, *et al*. *Faecalibacterium prausnitzii*-derived microbial anti-inflammatory molecule regulates intestinal integrity in diabetes mellitus mice *via* modulating tight junction protein expression. J Diabetes. 2020;12(3):224-236. DOI: 10.1111/1753-0407.12986.
43. Lin SJ, Helm ET, Gabler NK, Burrough ER. Acute infection with *Brachyspira hyodysenteriae* affects mucin expression, glycosylation, and fecal MUC5AC. Front Cell Infect Microbiol. 2023;12:1042815,1-18. DOI: 10.3389/fcimb.2022.1042815.
44. Herp S, Durai Raj AC, Salvado SM, Woelfel S, Stecher B. The human symbiont *Mucispirillum schaedleri*: causality in health and disease. Med Microbiol Immunol. 2021;210(4):173-179. DOI: 10.1007/s00430-021-00702-9.
45. Ussar S, Griffin NW, Bezy O, Fujisaka S, Vienberg S, Softic S, *et al*. Interactions between gut microbiota, host genetics and diet modulate the predisposition to obesity and metabolic syndrome. Cell Metab. 2015;22(3): 516-530. DOI: 10.1016/j.cmet.2015.07.007.
46. Tanaka M, Koida A, Miyazaki A, Tabata K, Takei Y, Tanimoto Y, *et al*. Canna starch improves immune functions and the intestinal environment in mice. Biosci Microbiota Food Health. 2023;42(2):131-137. DOI: 10.12938/bmfh.2022-068.
47. Rodríguez-Daza MC, Pulido-Mateos EC, Lupien-Meilleur J, Guyonnet D, Desjardins Y, Roy D. Polyphenol-mediated gut microbiota modulation: toward prebiotics and further. Front Nutr. 2021;8:689456,1-24. DOI: 10.3389/fnut.2021.689456.