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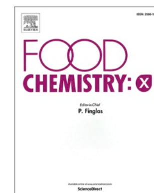

Soluble dietary fiber from *Dendrocalamus brandisii* (Munro) Kurz shoot improves liver injury by regulating gut microbial disorder in mice

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ABSTRACT

Bamboo shoot has long been regarded as a nutritious and healthy food. It is low in calorie and rich in high-quality dietary fiber (DF), making them a potential DF resource. However, the protective mechanism of soluble dietary fibers from *Dendrocalamus brandisii* (Munro) Kurz shoot (DS-SDF) on methionine and choline deficient (MCD) diet-induced non-alcoholic fatty liver disease (NAFLD) is still unclear. This study was aimed to investigate the regulation of DS-SDF on gut microbiota in MCD diet-induced mice and its potential protective effect on liver injury. The NAFLD model was induced by the MCD diet for 8 weeks. Through observation of changes in liver function and gut microorganisms, it was found that DS-SDF supplementation could inhibit liver inflammation, improve liver injury, regulate the diversity of gut microorganisms, increase the abundance of beneficial bacteria and short-chain fatty acid-producing bacteria, and reverse the gut disorders induced by the MCD diet in mice. This study showed that DS-SDF supplementation could treat NAFLD by regulating gut microbiota composition, improving liver function, and inhibiting the inflammatory response. It might broaden the idea of high-value utilization of DS-SDF.

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) refers to liver lesions characterized by excessive deposition of intrahepatic fat caused by the exclusion of alcohol and other clear causes. Hepatocyte degeneration, necrosis, and fibrosis often occur, which may evolve into cirrhosis in severe cases (Ebrahimzadeh Leylabadlo et al., 2020). Currently, NAFLD has become a major threat to global public health, with most countries experiencing an increase of >25% between 2009 and 2019 (Golabi et al., 2021).

Gut microbiota has been widely studied as an influencing factor closely related to NAFLD. The microbiota that coexists peacefully with the host is called gut microbiota (Neish, 2009).Gut microbiota accounts for >70% of human microorganisms and plays an important role in protecting the host from pathogen infection, changing the digestion and absorption of nutrients, and regulating the host immune system (Sekirov, Russell, Antunes, & Finlay, 2010; Wu et al., 2019). Gut microbiota mainly affects the development of NAFLD by changing the composition of gut microecology, inhibiting the expression of inflammatory factors, and regulating metabolites such as short-chain fatty acids (SCFAs), bile acids (BAs), and endogenous ethanol (Weixuan, Lu, Suzhen, Yi, & Hongliang, 2022).

Dietary fiber (DF) helps to promote and maintain gut health, and selectively promotes the proliferation and metabolic activity of beneficial bacteria during fermentation, thus attracting increasing attention (Mackie, Bajka, & Rigby, 2016). DFs may produce probiotic fermentation metabolites in the colon, such as SCFAs, which can promote gut health (Moro Cantu-Jungles, & do Nascimento, G. E., Zhang, X., Iacomini, M., Cordeiro, L. M. C., & Hamaker, B. R., 2019). For people with long-term DF intake, the number of bacteria (such as *Bifidobacteria*) capable of degrading DF in feces increased significantly, and the content of SCFAs increased significantly, which was conducive to regulating gut microbiota and protecting (Goff, Cowland, Hooper, & Frost, 2013).

Soluble dietary fibers (SDF) can be highly fermented by gut microbiota. Once in the intestine, they can be rapidly utilized by gut microbiota to produce probiotics (such as SCFAs), thereby promoting gut health (Comino, Williams, & Gidley, 2018). Therefore, foods with high SDF content can effectively improve gut health. Compared with insoluble dietary fibers (IDF), SDF has stronger antioxidant capacity. DF is the "seventh largest nutrient" for the human body and has many positive effects on health. Studies have shown that different types of DF have different functional characteristics. For example, SDF extracted from

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food by-products such as citrus peels (Du et al., 2021) show different functional characteristics, thus providing new opportunities for food development.

Bamboo is an evergreen perennial herb, widely planted all over the world, especially in Asia (He, Wang, Qin, Shui, Zhu, Wu, et al., 2014). Bamboo shoot is an immature young bamboo on the rhizome node of bamboo. It has low calories and is rich in DF (Chongtham, Bisht, & Haorongbam, 2011). It has long been regarded as nutritious and healthy food. Edible bamboo shoots have been shown to have beneficial effects on lipid and gut function (Park & Jhon, 2009). However, the mechanism by which bamboo shoot SDFs regulate gut microbiota to improve NAFLD is unclear. This study aims to investigate the effect of SDFs from *Dendrocalamus brandisii* (Munro) Kurz shoots (DS-SDF) on diet-induced NAFLD in mice by regulating gut microbiota. The effects of DS-SDF on the species diversity and composition of gut microbiota were determined, shedding light on the potential mechanism of DS-SDF in preventing NAFLD and contributing to the development and utilization of DS-SDF as a functional food.

2. Materials and methods

2.1. Materials and reagents

Dendrocalamus brandisii (Munro) Kurz shoots were harvested from Jinghong Dai Autonomous Prefecture of Xishuangbanna (Yunnan, China). High-temperature resistant α-amylase (4×10^4 U/g) was obtained from Yuan-chun Biotechnology Co., Ltd. (Shanghai, China). Neutral protease (1×10^5 U/g) and glucosidase (1×10^5 U/g) were acquired from Yingxin Laboratory Equipment Co., Ltd. (Shanghai, China). Methionine and choline deficient (MCD) (MD12052) was obtained from Jiangsu Medicience Biomedicine Co., Ltd. (Yangzhou, China). Alanine aminotransferase (ALT, 70111) and aspartate aminotransferase (AST, 70110) were purchased from Shandong Biobase Biodusty Co., Ltd. (Jinan, China). Enzyme-linked immunosorbent assay kits for tumor necrosis factor (TNF)-α (*E*-EL-M3063), interleukin (IL)-1β (E-EL-M0037c), and IL-6 (E-EL-M0044c) were purchased from Wuhan Elabscience Biotechnology Co., Ltd. (Wuhan, China). Fecal genomic DNA extraction kit and universal DNA purification kit were purchased from Tiangen Biotech Co., Ltd. (Beijing, China). The NEB Next® Ultra TM II FS DNA polymerase chain reaction (PCR)-free Library Prep Kit was obtained from New England Biolabs, Inc. (Massachusetts, USA). All other reagents are of pure analytical grade and were manufactured in China.

2.2. Preparation of DS-SDF

Following our previous method (Dong, Li, Zhao, & Cao, 2023), *Dendrocalamus brandisii* (Munro) Kurz shoots collected from Jinghong Dai Autonomous Prefecture of Xishuangbanna (Yunnan, China) were rinsed with distilled water and dried at 60 °C until a constant weight was achieved. The dried bamboo shoots were pulverized with a high-speed grinder, sifted through a 40-mesh sieve, and then stored at 4 °C for subsequent research. High-temperature resistant α-amylase (5 mL/100 g) was added to water at 95 °C for 60 min, and the pH was adjusted to approximately 6. Neutral protease (10 mL/100 g) was introduced at 60 °C for 60 min, followed by the supplement of glucosidase (10 mL/100 g) at 60 °C for 60 min. The mixture was then heated in a boiling water bath for 10 min to halt enzymatic hydrolysis. The supernatant was gathered, combined with 95% ethanol (1:4 *v*/v), left to stand for 2 h, centrifuged at 6500 ×*g* for 10 min, and the resulting precipitate was DS-SDF.

2.3. Animal experiments

Forty male Kunming mice (6 weeks old, weighing 20 ± 2 g) were procured from SPF (Beijing) Biotechnology Co., Ltd., bearing license number SCXK (Jing) 2019–0010. The mice were housed in a temperature range of 20–26 °C and a humidity of 40–70%, and were fed a diet for growth and reproduction. After one week of adaptive feeding, the mice were randomly divided into 5 groups, each comprising 8 mice. These groups were fed normal diet (NFD), MCD diet (MD12052), MCD diet + low dose DS-SDF intervention (200 mg/kg/day, MCD + D-L), MCD diet + medium dose DS-SDF intervention (500 mg/kg/day, MCD + D-M), and MCD diet + high dose DS-SDF intervention (1000 mg/kg/day, MCD + D-H). DS-SDF was administered via gavage at doses of 200, 500, and 1000 mg/kg daily, respectively, while the control group received only drinking water. All experimental procedures adhered to the welfare standard for experimental animals, under experimental animal use permit SYXK (Gan) 2020–0001.

2.4. Sample collection and detection

At the 8th week of the experiment, the mice were fasted overnight and euthanized using anesthesia (Fig. 1). Fresh feces were collected from each mouse and placed in a 2 mL sterile centrifuge tube. The feces were frozen in liquid nitrogen and stored at −80 °C. Blood samples were collected in 1.5 mL centrifuge tubes and centrifuged at 4000 ×*g* for 15 min to obtain serum, which was then stored at −80 °C. Pancreatic tissue and liver were weighed, dissected, quickly frozen in liquid nitrogen for 10 min, and finally stored at −80 °C for further analysis.

2.5. Histopathological evaluation of liver and pancreas tissues

The liver and pancreatic tissues of mice were roasted, dewaxed, hydrated, stained with hematoxylin for 3–5 min, washed with running water, differentiated with 1% hydrochloric acid alcohol, counterstained with eosin for 3–5 min, and observed under a microscope (CX43, Olympus, Tokyo, Japan).

2.6. Detection of liver function and inflammatory factor release

Biochemical indexes were determined using a microplate reader (WD-2012B, Liuyi biotechnology, Beijing, China). The serum of mice was utilized, and the optical density (OD) value of each well at 510 nm wavelength was measured using commercial kits (Shandong Biobase Biodusty, China) to detect the activity of ALT and AST.

The expression of TNF-α, IL-1β, and IL-6 in liver tissue was assessed using enzyme-linked immunosorbent assay kits. Tissue homogenate was prepared from animal tissue, centrifuged at 5000 ×*g* for 10 min to obtain tissue supernatant, and then tested according to the kit instructions.

2.7. Detection of fecal SCFAs

Mouse fecal samples were mixed with 75% methanol, vortexed, and centrifuged at 17000 ×*g* for 15 min. The supernatant was filtered using a 0.22 μm organic membrane filter. Liquid chromatography-mass spectrometry (AB 6500, USA) was used to collect primary and secondary mass spectrometry data based on multiple-reaction monitoring (MRM) mode. Negative mode acquisition, electrospray ionization (ESI) ion source parameter settings were as follows: ion source dry gas temperature (Gas Temp): 400 °C, nitrogen flow (Gas Flow): 50 L/min, sheath gas flow rate (Sheath Gas Flow): 15 L/min, sheath gas temperature (Sheath Gas Temp): 350 °C, capillary voltage (VCap): 2500 V. The sample concentration was calculated using the internal standard method and MultiQuant 3.0.3 analysis software.

2.8. Fecal genomic DNA extraction and PCR amplification

Fecal samples were processed using a fecal genomic DNA extraction kit (TianGen, Beijing, China). Subsequently, 1% agarose gel electrophoresis was used to determine the purity and concentration of DNA. An appropriate amount of sample DNA was taken in a centrifuge tube and

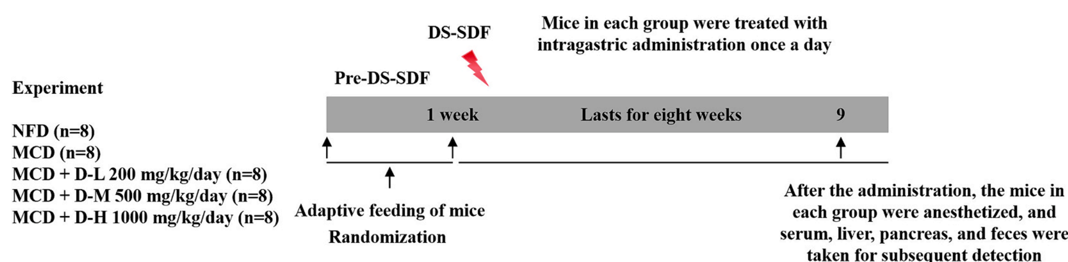

Fig. 1. Experimental timeline.

diluted to 1 ng/μL with sterile water. Genomic DNA was extracted and polymerase chain reaction (PCR) amplification was carried out using 16S rRNA V3-V4 region primers 341F (5′-CCTAYGGGRBGCASCAG-3′) and 806R (5′-GGACTACNNGGGTATCTAAT-3′). The PCR products, purified by magnetic beads, were combined in equal amounts based on concentration. Following mixing, the PCR products were analyzed via 2% agarose gel electrophoresis, and the target bands were recovered using a universal DNA purification kit (TianGen, Beijing, China).

2.9. High-throughput sequencing

The NEB Next® Ultra TM II FS DNA PCR-free Library Prep Kit (New England Biolabs, Massachusetts, USA) was used to prepare the library. Quantification of the constructed library was performed using Qubit and quantitative PCR (Q-PCR). After qualification, PE 250 sequencing was carried out using NovaSeq 6000 from Illumina (California, USA). The sequenced data underwent splicing and quality control, followed by filtering. Subsequently, the sequences were clustered into operational taxonomic units (OTUs) with 97% consistency and annotated using the Illumina database. Based on the species annotation, further analysis was conducted on alpha (α)-diversity and beta (β)-diversity. Principal coordinates analysis (PCoA) sorted a series of eigenvalues and eigenvectors to extract essential elements and structures from multidimensional data. Principal component analysis (PCA) reduced dimensionality based on the relative abundance distribution of amplicon sequence variant (ASV) to extract crucial elements and structures from the data. Non-metric multi-dimensional scaling (NMDS) was performed to reflect species information in a two-dimensional plane in the form of points, aiming to overcome the limitations of linear models and better reveal differences between community structures of two groups.

2.10. Statistical analysis

All experiments were repeated three times, and results were expressed as mean ± standard deviation. One-way analysis of variance was performed using Graphpad Prism 9.5.0 software and Origin 2023 software. Other data were statistically analyzed and plotted. A significance level of $P < 0.05$ indicated a statistically significant difference.

3. Results

3.1. Effects of DS-SDF on histopathology of liver and pancreas tissues

The results of pathological analysis of liver tissue in mice were depicted in Fig. 2. In the NFD group, hepatic lobule structure was clear, hepatocytes appeared normal, with no cytoplasmic fat vacuoles, steatosis, or necrosis, and the pancreatic tissue exhibited clear boundaries and orderly arrangement. Conversely, the MCD group displayed steatosis, vacuolar steatosis, inflammatory infiltration, blurred pancreatic cell mass edges, and inflammatory infiltration. However, in a dose-dependent manner, the MCD + D-L, MCD + D-M, and MCD + D-H groups showed significant improvement. These findings indicated that DS-SDF intervention significantly alleviated liver steatosis.

3.2. Effects of DS-SDF on liver function and release of inflammatory factors

3.2.1. Biochemical index

As shown in Fig. 3A-B, after 8 weeks of intervention, AST and ALT levels in the MCD group were significantly increased compared to the NFD group. However, in the three dose groups, levels of AST and ALT were decreased. Notably, the MCD + D-L group showed a significant decrease in AST and ALT levels. These results indicated that DS-SDF reduced AST and ALT values in liver-injured mice, suggesting a protective effect on liver function damage in mice.

3.2.2. Inflammatory factor

Gut inflammation is characterized by increased production of pro-inflammatory cytokines and inflammatory cell infiltration in the colon. Serum levels of pro-inflammatory factors TNF-α, IL-1β, and IL-6 were significantly elevated in the MCD group compared to the NFD group. Following DS-SDF intervention, serum levels of TNF-α, IL-1β, and IL-6 in the MCD + D-L, MCD + D-M, and MCD + D-H groups were

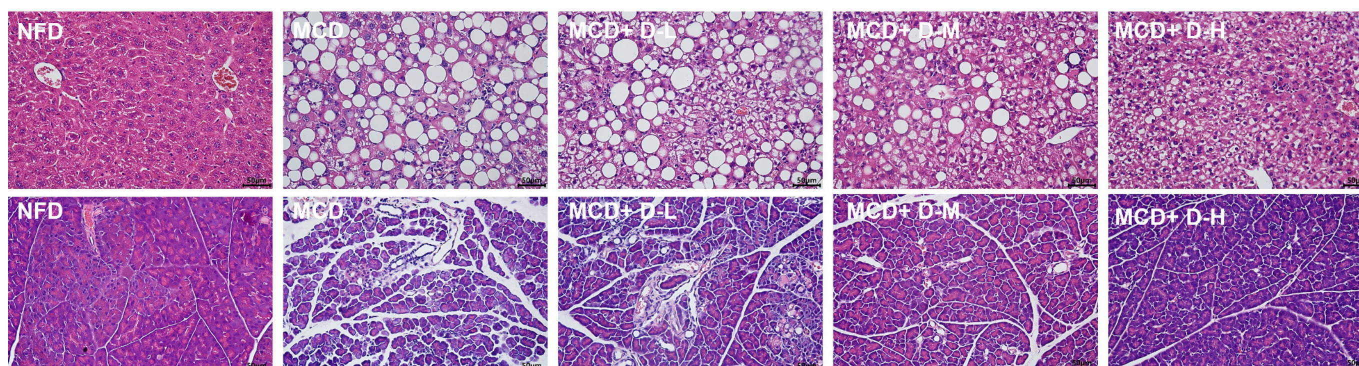

Fig. 2. H&E staining of liver and pancreas tissues in different groups of mice.

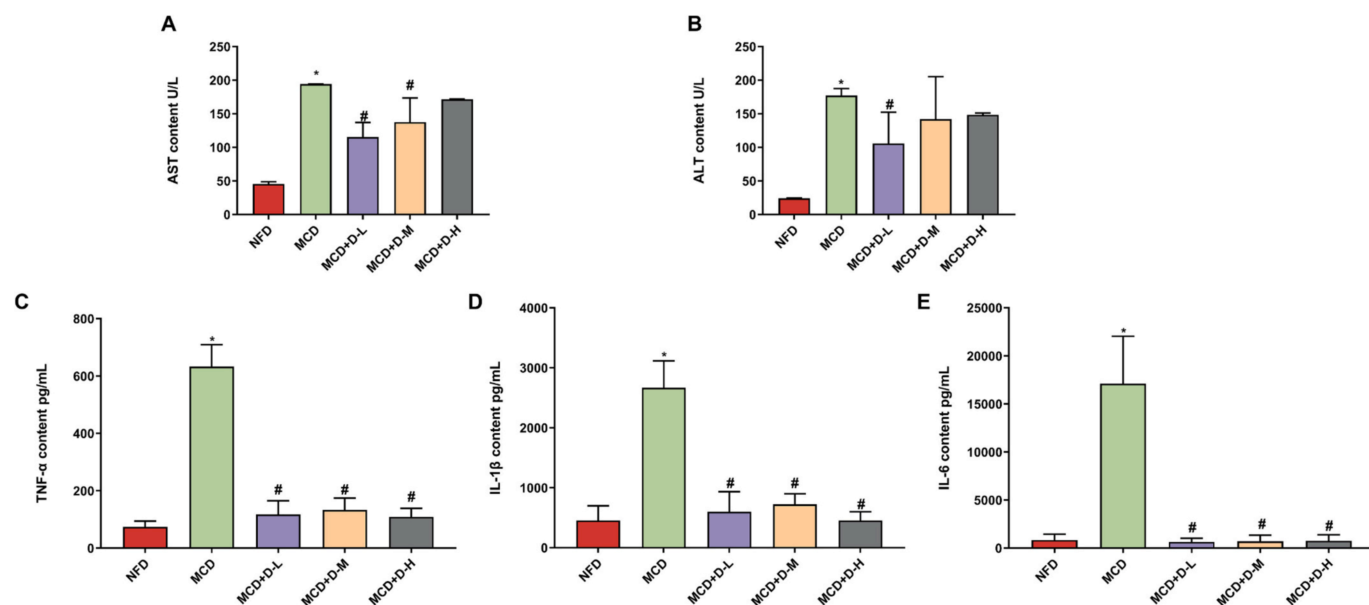

Fig. 3. Effects of DS-SDF on liver biochemical indexes and inflammation. (A) Liver AST level, (B) Liver ALT activity, (C) Liver TNF-α level, (D) Liver IL-1β level, (E) Liver IL-6 levels. $^{*}P < 0.05$ vs NFD, $^{\#}P < 0.05$ vs MCD.

significantly decreased (Fig. 3C-E).

3.3. Effects of DS-SDF on fecal SCFAs

Following 8 weeks of MCD diet feeding and DS-SDF gavage intervention, the content of SCFAs in the feces of mice changed as shown in Fig. 4A-E. Compared to the NFD group, the MCD group showed significantly reduced contents of acetic acid, propionic acid, *n*-butyric acid, *i*-butyric acid, and total SCFA. However, low, medium, and high doses of DS-SDF increased SCFA levels in both serum and fecal samples, demonstrating a dose-dependent improvement in SCFA levels in feces of MCD-fed mice.

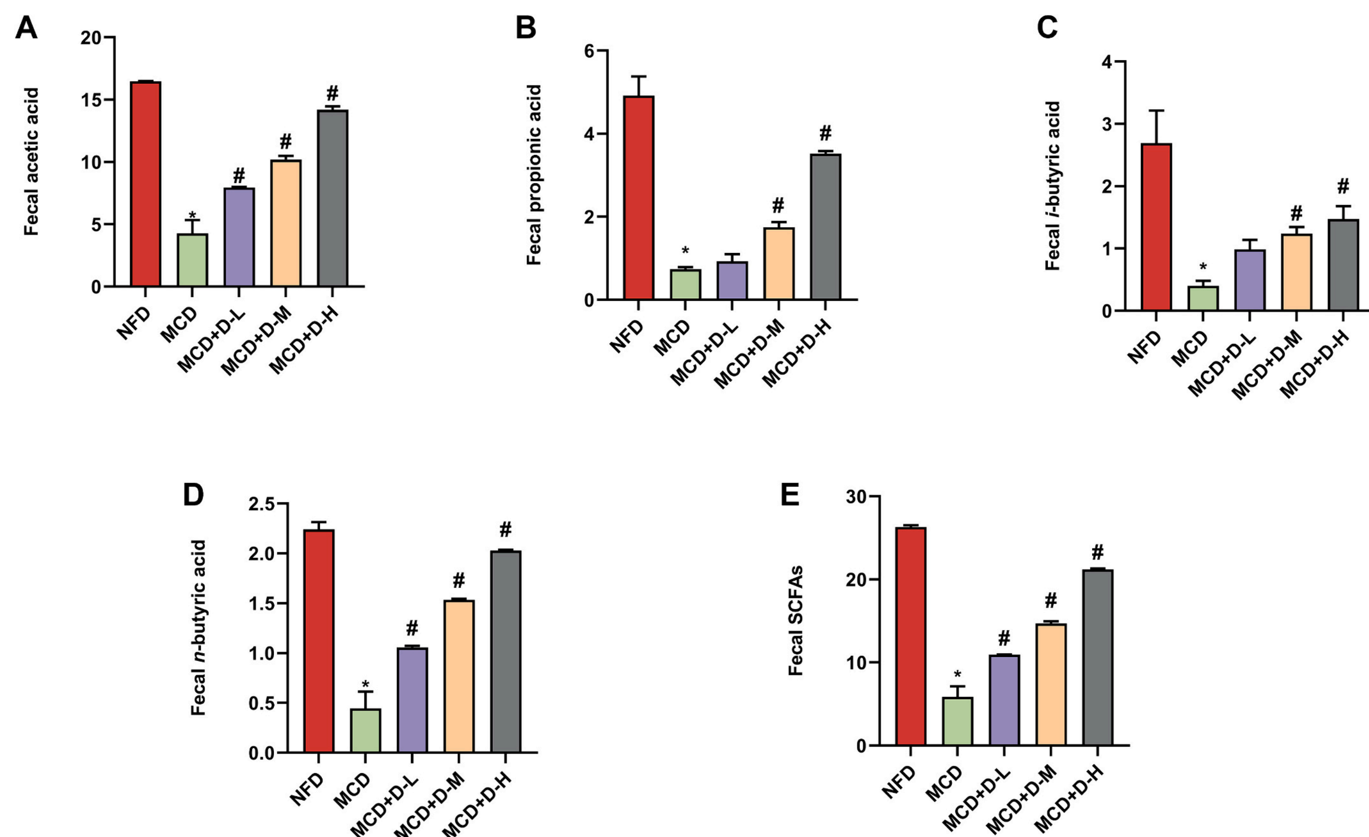

Fig. 4. Changes of SCFAs content in different groups of mice. (A) Acetic acid concentration, (B) Propionic acid concentration, (C) *I*-butyric acid Concentration, (D) *N*-butyric acid concentration, (E) Total SCFAs concentration. $^{*}P < 0.05$ vs NFD, $^{\#}P < 0.05$ vs MCD.

3.4. Effects of DS-SDF on microbial diversity in mouse feces

High-throughput 16S rRNA sequencing was used to analyze the microbial composition of fecal samples. After filtering, a total of 1,115,480 valid sequences were obtained. Each group of samples contained 128 OTUs, with 226 unique OTUs in the NFD group (Fig. 5A). The α-diversity analysis using Chao1, Simpson, and Shannon indices revealed that compared to the NFD group, the MCD group showed significantly reduced values. However, the Chao1 index of the MCD + D-M and MCD + D-H groups increased significantly, while the Simpson index decreased compared to the MCD group. These results showed that DS-SDF promoted gut microbial richness and diversity. The β-diversity analysis, including PCoA, PCA, and NMDS, further illustrated the impact of adding DS-SDF on overall structural changes of gut microbiota (Fig. 6A-C). The results indicated that the MCD group and the NFD group were significantly separated, and the MCD + D-M and MCD + D-H groups were closer to the NFD group, highlighting the regulation of gut microbial structure by DS-SDF supplementation in a dose-dependent manner.

3.5. Effects of DS-SDF on microbial composition in fecal of mice

After conducting high-throughput sequencing of gut microbiota, we obtained the composition and abundance of gut microbiota in mice by comparing it with the database. To observe the impact of DS-SDF on the diversity and species composition of gut microbiota in MCD diet mice, the differences in gut microbiota at phylum and genus levels were compared and analyzed.

As shown in Fig. 7A, at the phylum level, Firmicutes and Bacteroidota were the dominant phyla in the intestine, constituting over 85%. In comparison with the NFD group, the relative abundance of Firmicutes and Bacteroidota in the MCD group's intestines significantly decreased. However, following DS-SDF intervention, the relative abundance of Firmicutes increased significantly while the presence of *Proteobacteria* and *Verrucomicrobiota* increased, and *Deferribacterota* decreased after MCD feeding. Moreover, there was a varying increase in *Deferribacterota* abundance within each dose group compared to the MCD group.

After 8 weeks of DS-SDF intervention, at the genus level (Fig. 7B), compared with the NFD group, the abundance of *Lactobacillus*, *Muribaculaceae*, *Helicobacter*, and *Clostridia_UCG-014* in the MCD group decreased, while *Akkermansia* and *Alloprevotella* increased. Interestingly, DS-SDF partially reversed the MCD diet-induced changes in bacterial abundance. The relative abundance of *Bifidobacterium*, *Lachnospiraceae*, *Ileibacterium*, *Lactobacillus,* and *Faecalibaculum* increased in the DS-SDF group. These results indicate that DS-SDF improves MCD diet-induced intestinal flora imbalance.

3.6. Correlation between gut microbiota and liver inflammation

Through Spearman correlation analysis, key microbial groups and their association with liver biochemical and inflammatory indicators were shown by heat maps (Fig. 8). *Lactobacillus* in the MCD + D-L, MCD

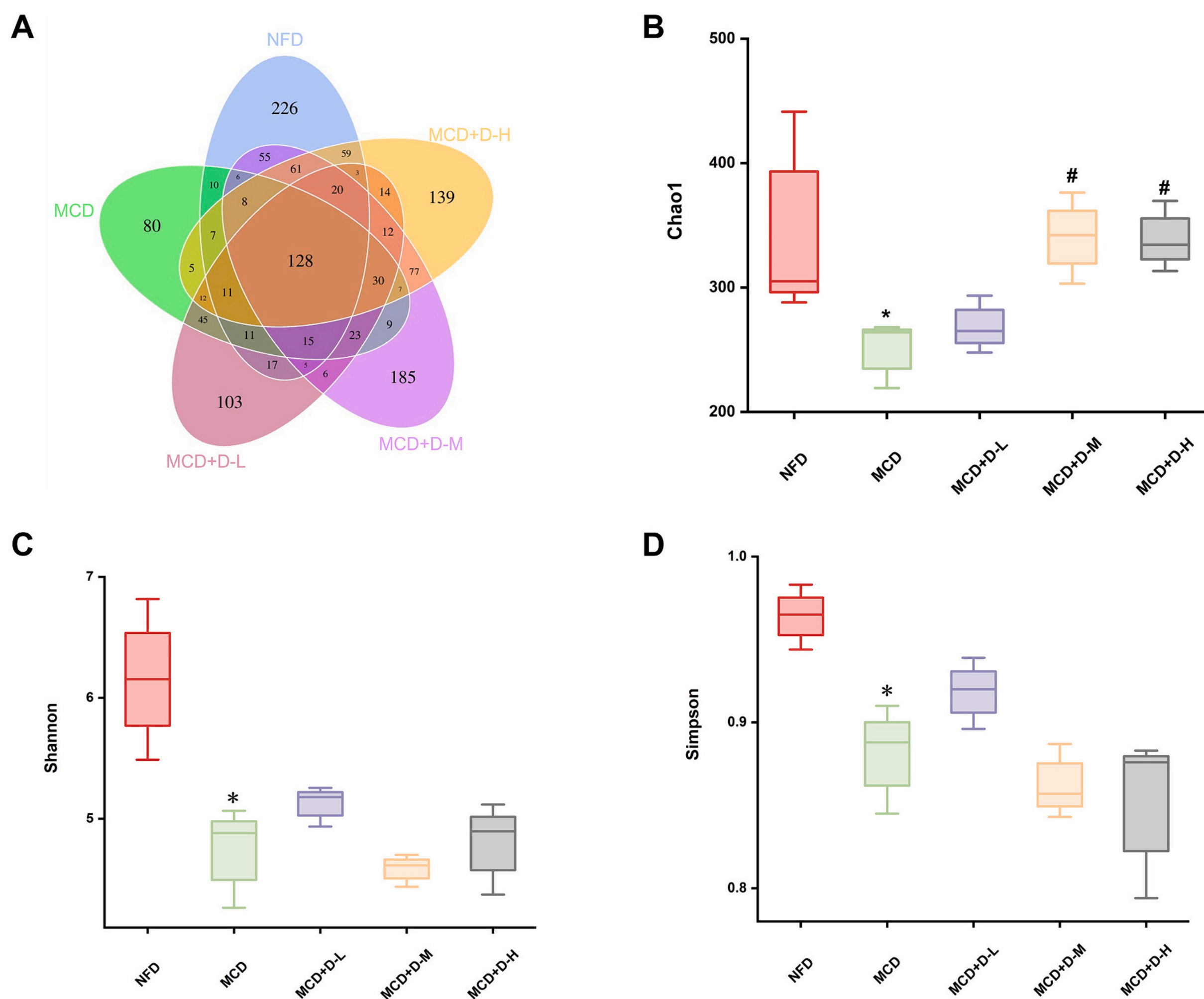

Fig. 5. OTU and alpha diversity of different groups of mice. (A) Venn graph, (B) Chao1 index, (C) Shannon index, (D) Simpson index. *P $<$ 0.05 vs NFD, $^{\#}$P $<$ 0.05 vs MCD.

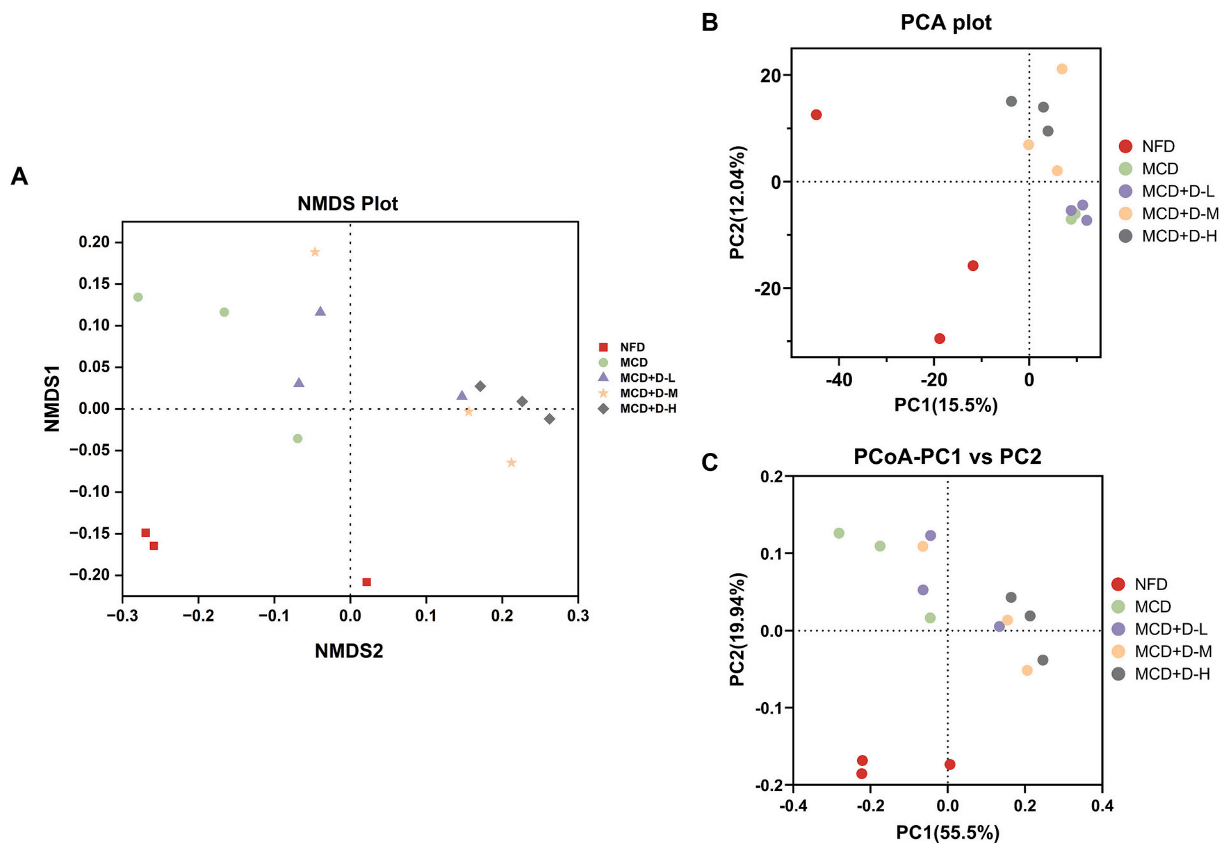

Fig. 6. Beta diversity of different groups of mice. (A) NMDS analysis of gut microbiota, (B) PCA analysis of gut microbiota, (C) PCoA analysis of gut microbiota.

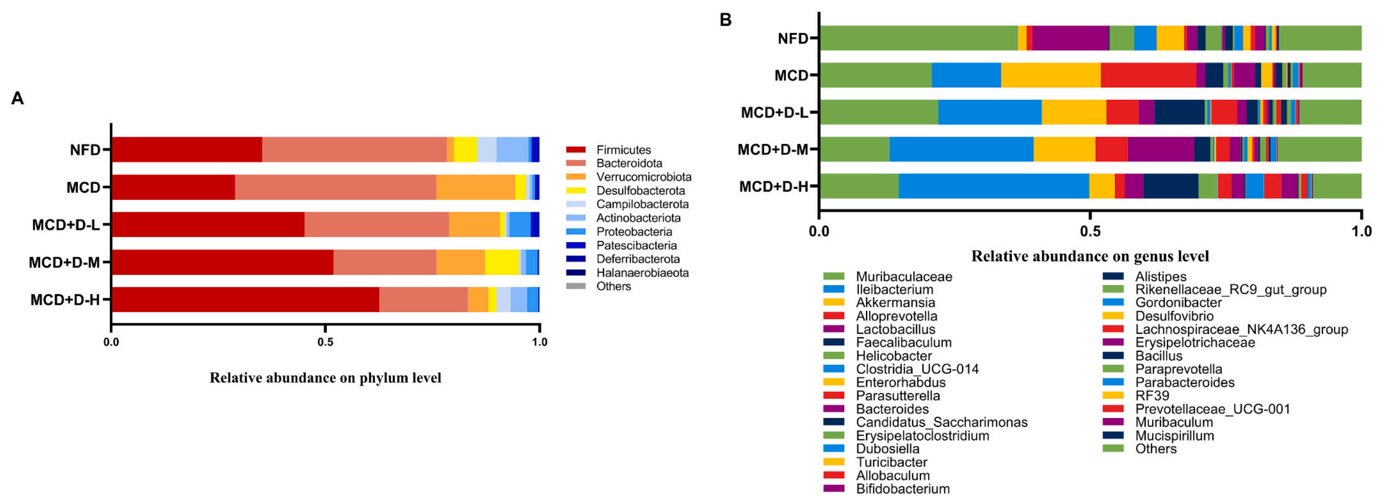

Fig. 7. Analysis of dominant species composition of gut microbiota in different groups of mice. (A) Relative abundance on phylum level, (B)Relative abundance on genus level.

+ D-M, MCD + D-H groups increased to varying degrees and showed a significant negative correlation with inflammation-related parameters. Notably, *Akkermansia* was significantly enriched in MCD-induced NAFLD mice and positively correlated with liver biochemical and inflammatory parameters.

4. Discussion

Studies have highlighted the potential role of gut microbiota changes in the treatment and improvement of NAFLD (Aron-Wisnewsky, Warmbrunn, Nieuwdorp, & Clément, 2020; Iwaki et al., 2021). Currently, dietary intervention, drug treatment, and increased exercise have been recognized to impact human gut microbiota, of which dietary intervention is an important approach to treatment (Cronin, Joyce, O'Toole, & O'Connor, 2021). Consuming a diet rich in DFs is beneficial for human health and plays a crucial role in modifying human gut microbiota and addressing various inflammatory diseases (W. Ma, Nguyen, Song, Wang, Franzosa, Cao, et al., 2021; Mocanu et al., 2021). Compared with drug therapy, improving dietary structure and increasing intake of DFs are healthier and safer. Specifically, SDFs can be fermented by gut microbiota, promoting the growth of beneficial microorganisms, producing beneficial metabolites such as SCFAs,

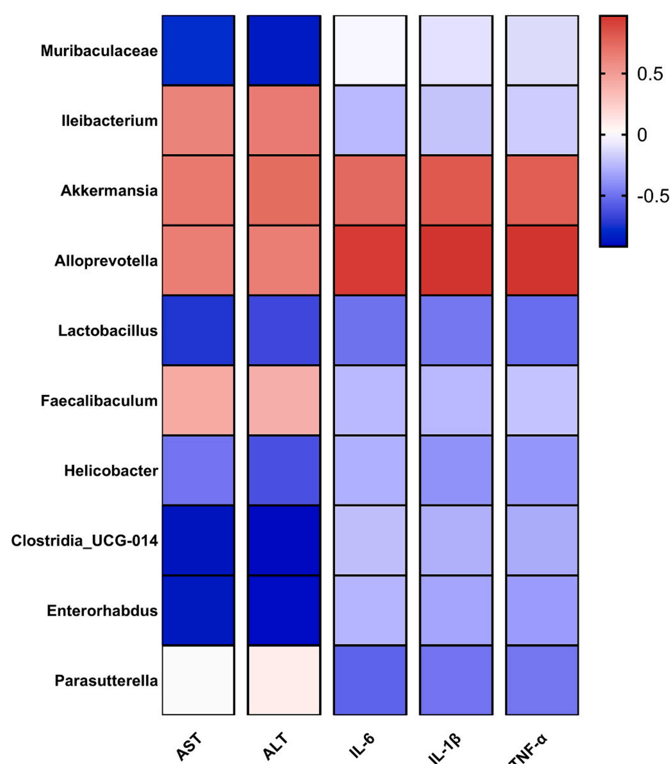

Fig. 8. Correlation between gut microbiota and liver metabolic indexes.

regulating gut microbiota composition (Zhu et al., 2021), and consequently treating NAFLD.

In this study, histopathological observation revealed that 8 weeks of MCD diet feeding successfully induced NAFLD in Kunming mice, resulting in steatosis. Adding DS-SDF to the diet significantly improved hepatic steatosis. AST and ALT activity serve as decisive biochemical parameters for evaluating the degree of liver injury (Chen et al., 2020). DS-SDF inhibited AST and ALT levels in the liver and enhanced liver function. Furthermore, the levels of TNF-α, IL-1β, and IL-6 in the liver of the MCD group substantially increased, and DS-SDF intervention reversed this change. Based on these results, DS-SDF supplementation effectively reduced lipid accumulation and inflammatory response, thereby preventing NAFLD.

Simultaneously, the gut microbiota has been considered a potential therapeutic target for NAFLD (Burz et al., 2021; Suk & Kim, 2019). Gut microbiota imbalance can disrupt liver metabolism and affect the balance of the liver's inflammatory response, leading to the onset of NAFLD (Aron-Wisnewsky et al., 2020; Ni, Ni, Zhuge, & Fu, 2020). This study found that DS-SDF effectively improved the structure of gut microflora in MCD diet-induced mice and increased the relative abundance of some key bacterial genera. The Chao1 index showed a decrease in the MCD group. Following DS-SDF intervention, the Chao1 index of the MCD + D-M and MCD + D-H groups significantly increased, while the Simpson index decreased. It indicated that after 8 weeks of DS-SDF intervention, the gut microbiota diversity of MCD diet mice was improved to varying degrees. In addition, β-diversity analysis demonstrated different levels of separation between the five groups. DS-SDF intervention positively influenced the migration of the microbial community composition along PC1, indicating that DS-SDF restored the gut microflora disorder of MCD diet mice to a certain extent.

It is worth noting that at the gate level, DS-SDF significantly increased the reduced *Firmicutes/Bacteroidota* ratio in mice on an MCD diet. The relative abundance of *Firmicutes* and *Bacteroidota* affects the metabolism of the body and controls the rate of body mass growth (Ma et al., 2019). When the relative abundance of *Firmicutes* is higher than that of *Bacteroidota*, the body canmore effectively absorb nutrients. Compared to the NFD group, the gut microbiota of mice on an MCD diet was disordered at the genus level, with reduced relative abundance of genera such as *Bifidobacterium*, *Lachnospiraceae*, and *Ileibacterium*. Fig. 7B shows that the MCD diet induced a decrease in the relative abundance of some SCFA-producing bacteria, such as *Lactobacillus*, in the intestines of mice. After the DS-SDF diet intervention, compared to the mice on the MCD diet, there was an overall increase in the abundance of beneficial bacteria and SCFAs-producing bacteria, reaching or even surpassing the levels found in normal mice. Among them, *Lactobacillus* was particularly enriched in the MCD + D-M and MCD + D-H groups. *Lactobacillus*, a rod-shaped member of the lactic acid bacteria, has a strong ability to decompose carbohydrates. With eight weeks of DS-SDF intervention, DS-SDF can be fermented and utilized by the beneficial and SCFAs-producing bacteria in the intestine, adjusting the gut pH value (T, P. N., & C, M. E., 2017), and thus inhibiting pathogenic bacteria. SCFAs help maintain gut morphology and function and have a regulatory effect, which can be used to evaluate the species and activity of bacteria in the intestine (Q. Ma et al., 2019). Therefore, DS-SDF can regulate the gut microbiota environment in mice, promote gut microbiota health, and subsequently treat NAFLD. *Lactobacillus* has shown a strong positive correlation with liver biochemistry and inflammation, consistent with many previous studies. In these studies, *Lactobacillus* was significantly positively correlated with some liver biochemical indicators and inflammation-related markers (Kim et al., 2016; Ou, Lin, Tsai, & Lin, 2011). It has also been reported that *Akkermansia* is involved in host-microbe interactions that affect immune function and gut health. While MCD feeding significantly increased *Akkermansia* levels, high-dose DS-SDF intervention significantly reduced them. However, contrary to our results, most previous studies have shown that *Akkermansia* is a genus of gut microbes beneficial to liver metabolism (Wang et al., 2022). In conclusion, DS-SDF intervention significantly changed the structure of the gut microbiota and offered a new avenue for the treatment of NAFLD.

5. Conclusions

In summary, this study discovered that DS-SDF supplementation could effectively reduce liver inflammation in mice. It also regulated the abundance and diversity of gut microflora in vivo, reversed the imbalance of gut microbiota caused by an MCD diet, and improved the intestinal environment in mice. Further evidence demonstrated that DS-SDF could efficiently manage gut disorders and alleviate liver injury in mice with NAFLD induced by an MCD diet. The findings indicated that DS-SDF might have the potential to treat NAFLD by regulating the gut microbiota, providing a novel approach to NAFLD treatment and a theoretical foundation for the use of DS-SDF as a functional food.

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CRediT authorship contribution statement

Yufan Dong: Writing – original draft, Visualization, Methodology, Investigation, Conceptualization. **Yuhong Guo:** Validation, Resources, Data curation. **Qin Li:** Validation, Software, Data curation. **Yihe Zhao:** Writing – review & editing, Resources, Project administration, Funding acquisition. **Jianxin Cao:** Writing – review & editing, Supervision, Project administration, Methodology.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could affect the work reported in this paper.

Data availability

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

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