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ORIGINAL ARTICLE

Effects of WiKim0124 starter-fermented kimchi on hepatic lipid metabolism, colonic inflammation, and barrier-related gene expression in mice with high-fat diet-induced obesity

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Popular scientific summary

- WiKim0124 starter-fermented kimchi (SFK) reduced fat accumulation in the liver by suppressing lipid synthesis and promoting fatty acid oxidation.
- SFK alleviated obesity-related inflammation in the liver and intestine by regulating inflammatory gene expression.
- SFK partially restored the colonic mRNA expression of tight junction-related genes, including *Zo1* and *Occludin*.

Abstract

Background: The anti-obesity effects of lactic acid bacteria (LAB) isolated from kimchi have been widely investigated; however, few studies have examined kimchi fermented with LAB starter cultures. Our previous cellular, animal, and clinical studies showed anti-obesity effects of specific starter-fermented kimchi (SFK), warranting evaluation of kimchi produced with newly developed starters.

Objective: We evaluated the effects of *Lactococcus lactis* WiKim0124 SFK on body composition, serum biochemical parameters, hepatic lipid metabolism, inflammation, and colonic barrier-related gene expression in high-fat diet (HFD)-induced obese mice.

Design: Mice were assigned to normal-diet, HFD, and SFK groups. Body weight, food efficiency ratio (FER), tissue weights, serum liver enzyme activities, adipokines, lipid profiles, hepatic lipid accumulation, and hepatic and colonic gene expression related to lipid metabolism, inflammation, and barrier integrity were assessed.

Results: SFK significantly attenuated HFD-induced increases in body weight, fat mass, and liver weight without reducing food intake. SFK showed significantly lower FER than HFD group ($P < 0.0001$). SFK normalized serum aspartate aminotransferase and alanine aminotransferase activities, decreased leptin, increased adiponectin, improved serum lipid profiles, and reduced hepatic triglyceride and total cholesterol levels. SFK also reduced hepatic lipid droplet accumulation, downregulated lipogenic genes (*Srebp1c, Lxrα, Fas, and C/ebpα*), and upregulated fatty acid oxidation genes (*Pparα, Cpt1α, and Acox1*). Additionally, SFK decreased hepatic and colonic *Tnfα* and *Il6* mRNA expression, increased hepatic *Il10* mRNA expression, and partially restored colonic mRNA expression of *Zo1* and *Occludin*.

Discussion: The anti-obesity effects of SFK appear to involve the coordinated regulation of hepatic lipid metabolism, inflammatory responses, and colonic barrier-related gene expression.

Conclusions: SFK attenuated HFD-induced obesity in association with suppressed hepatic lipogenesis, enhanced fatty acid oxidation, reduced hepatic and colonic inflammation, and partial restoration of colonic mRNA expression of tight junction-related genes, indicating its potential as a functional food for obesity management.

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Keywords: *fermented foods*; *lipogenesis*; *lipid metabolism*; *inflammation*; *intestinal barrier function*; *obesity*

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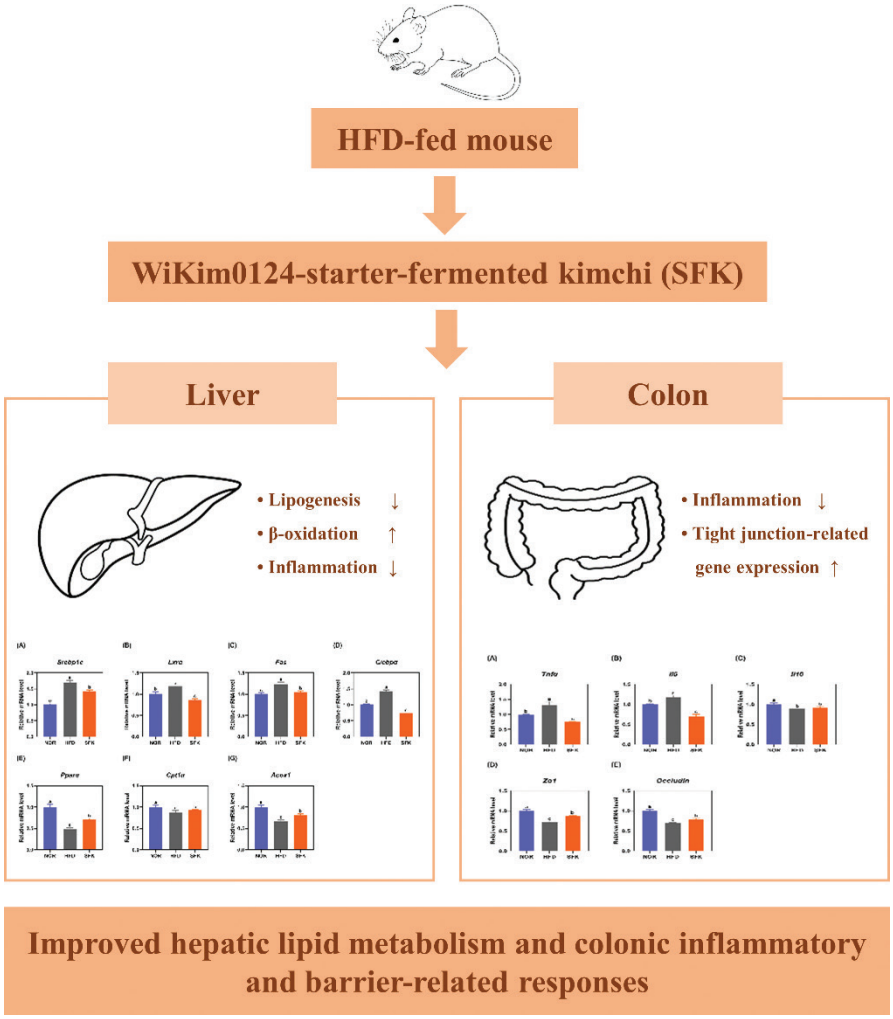
Graphic Abstract



Obesity is a chronic metabolic disorder characterized by excessive fat accumulation and is a major risk factor for various metabolic diseases (1, 2). The global prevalence of obesity has increased rapidly over the past few decades, and it is now one of the most important public health challenges worldwide (3). Previous studies have shown that high-fat diet (HFD)-induced obesity causes metabolic dysfunction through hepatic steatosis, adipose tissue expansion, chronic inflammation, and impaired lipid metabolism (4). In particular, excessive hepatic lipid accumulation results from increased de novo lipogenesis and reduced fatty acid β-oxidation, which are regulated by key transcription factors and metabolic enzymes (5). Accordingly, several studies have investigated strategies to suppress obesity by regulating lipogenesis and fatty acid β-oxidation.

The gut microbiota and intestinal barrier integrity also play pivotal roles in obesity-related metabolic disorders (6). An HFD disrupts intestinal tight junctions, increases intestinal permeability, and facilitates the translocation of bacterial endotoxins into the systemic circulation, thereby promoting chronic inflammation and metabolic endotoxemia (6, 7). Tight junction proteins such as zonula occludens-1 (ZO-1) and occludin, are critical for

maintaining epithelial barrier function; their disruption is associated with obesity-related intestinal dysfunction (8). Furthermore, obesity increases the production of pro-inflammatory cytokines and often suppresses anti-inflammatory cytokines, resulting in metabolic inflammation in hepatic and intestinal tissues (4, 9). These observations support investigating interventions that target hepatic lipid metabolism and inflammatory responses, as well as colonic barrier-related pathways.

Kimchi, a traditional Korean fermented food, has attracted considerable attention worldwide because of its various health-promoting effects (10). These beneficial effects have been attributed to bioactive components, metabolites, and lactic acid bacteria (LAB) produced during kimchi fermentation (11). Numerous studies have demonstrated that kimchi-derived LAB exert anti-obesity effects by regulating adipogenesis, lipid metabolism, inflammation, and gut microbial composition *in vitro* and *in vivo* (12–14). However, few studies have investigated the anti-obesity effects of kimchi fermented using functional starter cultures.

Recently, starter cultures have been investigated as a means of controlling kimchi fermentation and modulating microbial community composition and metabolite profiles to obtain desired product characteristics (15). In previous studies, kimchi fermented using selected starter cultures exhibited anti-obesity effects *in vitro*, *in vivo* and in a clinical trial (16–18). Both WiKim0124 and kimchi fermented with WiKim0124 as a starter culture exhibited anti-obesity activity in a cell-based screening assay (16). The functional properties of WiKim0124 have also been reported (19). Furthermore, the anti-obesity effects of WiKim0124 itself have recently been investigated in diet-induced obesity animal models (20). However, whether kimchi fermented with WiKim0124 as a starter culture exhibits similar anti-obesity effects in an animal model remains unclear. Therefore, this study evaluated the effects of WiKim0124 starter-fermented kimchi (SFK) on HFD-induced obese mice.

Here, the effects of oral administration of SFK, which exhibited strong antiobesity activity in screening, on hepatic lipid metabolism, hepatic and colonic inflammation, and colonic barrier-related gene expression were investigated in HFD-induced obese mice.

Materials and methods

Bacterial strain and kimchi preparation

The starter strain *Lactococcus lactis* WiKim0124, originally isolated from kimchi and deposited in the Korean Culture Center of Microorganisms (KCCM) under accession number KFCC11883P, was identified using 16S rRNA gene sequencing (1,418 bp; GenBank accession number MZ424472.1). It showed 100% sequence identity to *L. lactis* subsp. *lactis* DSM 20481. The strain was cultured in de Man, Rogosa, and Sharpe (MRS) broth and collected by centrifugation at 6,000 × *g* for 10 min at 4°C. SFK was prepared by mixing salted kimchi cabbage with kimchi seasoning at a ratio of 3:1 (w/w) and adding the starter culture at a final concentration of 10^7 CFU/mL, as previously described (16). WiKim0124 was used as a single starter strain, and non-sterilized salted kimchi cabbage and seasoning ingredients were used; therefore, indigenous microorganisms may have coexisted during fermentation. The kimchi was fermented at 6°C for 4 weeks until the pH reached 4.2 and was then freeze-dried for use in the experiments.

Reagents

TRIzol reagent was purchased from Invitrogen (Carlsbad, CA, USA), and the cDNA synthesis kit and SYBR Green premix were purchased from Enzynomics (Daejeon, Korea). Serum biochemical analysis kits were obtained from Asan Pharmaceutical (AM 102-K, 103-K, 157S-K, 201-K, and 202-K; Seoul, Korea). Adipokine assay kits were obtained from Abcam (ab199082 and ab226900; Cambridge, MA, USA).

Animals and diets

Six-week-old male C57BL/6N mice were purchased from Orient Bio Inc. (Seongnam, Korea) and housed at 21–23°C and 40–60% relative humidity under a 12-h light/dark cycle. The mice were housed at a density of 3–4 mice per cage. Feed was provided in predetermined amounts, and water was available *ad libitum*. The normal diet (D12450B; 10% kcal from fat) and HFD (D12492; 60% kcal from fat) were purchased from Research Diets (New Brunswick, NJ, USA). A mouse smart home (Datesand) was provided as environmental enrichment material. After 1 week of adaptation on a normal diet, the mice were allocated to experimental groups by body-weight-stratified randomization so that mean initial weights were comparable among groups: the normal-diet group (NOR; 10% kcal from fat; $n = 7$), HFD group (60% kcal from fat; $n = 7$), and SFK group (60% kcal from fat and 70 mg/kg body weight; $n = 7$). The sample size ($n = 7$) was based on our previous experience with animal experiments and published studies using similar HFD-induced obesity models; no a priori sample size calculation was performed. No prespecified exclusion criteria were applied, and no animals were excluded from the study. All 21 animals were included in the final analysis. The researcher conducting the experiments was aware of group allocation, and outcome assessment was not blinded. SFK was dissolved in phosphate-buffered saline (PBS) and administered orally to the SFK group three times per week for 8 weeks. The NOR and HFD groups received an equivalent volume of PBS on the same schedule.

The animal protocol was reviewed and approved by the Institutional Animal Care and Use Committee of the World Institute of Kimchi (approval no. WIKIM IACUC 202234), and all procedures were conducted in accordance with established ethical and scientific animal care procedures and the ARRIVE guidelines. All efforts were made to minimize animal suffering. The NOR and HFD groups used in this study were from the same animal experiment as that reported previously for naturally fermented kimchi (NK) and specific SFK (17). Allocation was performed simultaneously for all groups of the original experiment, including the NK and SK groups reported previously (17). The SFK group was part of the same experiment and was allocated, housed, treated, and sampled concurrently with these groups; its results have not been reported previously.

Food intake was monitored at regular intervals by measuring the amount of food provided and the remaining food in each cage. Food consumption was calculated as the difference between the amount of food provided and the remaining amount. The food efficiency ratio (FER) was calculated as body weight gain divided by total food intake (FER = body weight gain (g) / total food intake (g)).

At week 8, mice were fasted overnight, and fat mass and lean mass were measured using EchoMRI-500 (Echo Medical Systems, Houston, TX, USA) before dissection. Mice were anesthetized by CO_2 inhalation, and blood was collected from the abdominal aorta, which resulted in euthanasia by exsanguination. Cessation of respiration was confirmed before epididymal and abdominal adipose tissue, liver, and colon were collected. Adipose tissue and liver were weighed. Blood was allowed to clot at room temperature (22–25°C) for 30 min, and serum was obtained by centrifugation at 800 × *g* for 5 min. Liver and colon tissues were stored at –80°C until analysis.

Serum biochemical parameters

To evaluate the effects of SFK administration on hepatic injury, the activities of serum aspartate aminotransferase (AST; GOT) and alanine aminotransferase (ALT; GPT) were measured. Serum leptin and adiponectin concentrations were also measured to assess adipokine status.

Serum and hepatic lipid analyses

Triglyceride (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C) levels were measured in serum and liver samples. Serum low-density lipoprotein cholesterol (LDL-C) levels were estimated using the Friedewald formula (TC – [TG/5 + HDL-C]). Liver tissue (100 mg) was homogenized, and lipid extracts were isolated by centrifugation at 800 × *g* for 10 min. After overnight drying, the extracted lipids were dissolved in ethanol (100 μL), and lipid levels were measured using commercial assay kits and expressed as mg/dL of the lipid extract obtained from 100 mg of liver tissue.

Histological analysis

Liver tissues were embedded in an optimal cutting temperature (OCT) compound, frozen at –80°C, and sectioned at a thickness of 5 μm using a cryostat. Sections were stained with Oil Red O (ORO) and images were acquired using a microscope at 20× magnification (Ts2; Olympus, Tokyo, Japan). ORO-positive area was quantified using ImageJ software (version 1.53; NIH, USA).

Quantitative polymerase chain reaction

Total RNA was extracted with TRIzol and reverse-transcribed into cDNA using the TOPscript cDNA Synthesis Kit. Quantitative polymerase chain reaction was performed with SYBR Green premix and the gene-specific primers listed in Table 1. Relative mRNA expression was calculated using the $2^{\wedge(-\Delta\Delta Ct)}$ method and normalized to *Gapdh*.

Statistical analysis

Results are presented as mean ± standard deviation. Statistical analyses were performed using GraphPad Prism 9 (GraphPad Software Inc., San Diego, CA, USA). Significant differences were determined using one-way ANOVA followed by Tukey's multiple-comparison test, and adjusted $P < 0.05$ was considered significant. Mean differences (MD) were calculated as HFD minus the comparison group (NOR or SFK) and reported with 95% confidence intervals (CIs).

Results

Effects of SFK on body weight, food efficiency ratio, body composition, and organ weights

To evaluate the anti-obesity effects of SFK, body weight, food intake, FER, body composition, and tissue weights were measured (Table 2). Initial body weight did not differ significantly among the groups, confirming comparable baseline body weights before the dietary intervention. The HFD group had significantly higher final body weight than the NOR group. SFK administration reduced final body weight compared with HFD group (MD 8.87; 95% CI 5.42 to 12.32; $P < 0.0001$).

Food intake did not differ significantly between the SFK and HFD groups, suggesting that the observed effect was not due to decreased food intake. However, the FER in the SFK group was significantly lower than that in the HFD group (MD 0.030; 95% CI, 0.022 to 0.038; $P < 0.0001$), indicating that SFK reduced body weight relative to food intake by lowering the efficiency with which food was converted into body mass.

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Table 1. Quantitative polymerase chain reaction primer sequences

Primer	Forward	Reverse
Gapdh	GTATGACTCCACTCACGGCAAA	GGTGTGGCTCCTGGAAGATG
Lipogenesis		
Srebp1c	AGAGGGTGAGCCTGACAA	CCTCTGCAATTTCCAGAT
Lxrα	CTCAATGCCTGATGTTTCTCCT	TCCAACCCTATCCCTAAAGCAA
Fas	TCTGAGCAGGTGCAGGAGGA	GTTGTTCCTCCAGTTCCGATTTGTA
C/ebpα	AGGTGCTGGAGTTGACCAGT	CAGCCTAGAGATCCAGCGAC
Fatty acid β-oxidation		
Pparα	ATCTTAACTGCCGGATCCAC	TGGTGATTTGTCCGTTGTCT
Cpt1α	CTCCGCCTGAGCCATGAAG	CACCAGTGATGATGCCATTCT
Acox1	ACCTTCACTTGGGCATGTTC	TTCCAAGCCTCGAAGATGAG
Inflammation		
Tnfα	ACGGCATGGATCTCAAAGAC	AGATAGCAAATCGGCTGACG
Il6	CCTTCCTACCCCAATTTCCAA	AGATGAATTGGATGGTCTTGGTC
Il10	ACTGCACCCACTTCCCAGT	TGTCCAGCTGGTCCTTTGTT
Colonic barrier		
Zo1	CCAGCAACTTTCAGACCACC	AGGTGGGAGAATGCTATTGTCT
Occludin	TGTTCAAGAATTCTATGACCCCT	AGAAGGTGTTGGCTTGGGAT

Table 2. Body weight, food efficiency ratio, and tissue weights

Parameter	NOR	HFD	SFK
Initial weight (g)	22.04 ± 0.96[a]	22.56 ± 1.74[a]	21.70 ± 1.74[a]
Final weight (g)	30.74 ± 0.89[b]	42.31 ± 2.78[a]	33.44 ± 3.26[b]
Food intake (g/day/mouse)	3.87 ± 0.24[a]	2.98 ± 0.13[b]	3.03 ± 0.03[b]
Food efficiency ratio	0.03 ± 0.00[c]	0.08 ± 0.01[a]	0.05 ± 0.00[b]
Fat mass (g)	3.70 ± 1.85[c]	15.96 ± 2.87[a]	9.10 ± 3.63[b]
Lean mass (g)	24.84 ± 1.88[a]	24.26 ± 1.54[a]	24.44 ± 1.83[a]
Liver weight (g)	1.03 ± 0.03[b]	1.35 ± 0.13[a]	1.05 ± 0.07[b]
eWAT weight (g)	0.18 ± 0.10[c]	1.13 ± 0.14[a]	0.67 ± 0.34[b]
AF weight (g)	0.46 ± 0.26[c]	2.19 ± 0.19[a]	1.42 ± 0.63[b]

Results are presented as mean ± standard deviation (n = 7 per group).

Different superscript letters within the same row indicate significant differences among groups ($P < 0.05$; Tukey's multiple-comparison test following one-way ANOVA among the NOR, HFD, and SFK groups).

The NOR and HFD control groups were derived from the same animal experiment as Ref. (17) and are shared between the two studies.

Fat mass in the SFK group was significantly lower than that in the HFD group ($P = 0.0008$), whereas lean mass did not differ significantly among the groups. These findings indicate that the reduction in body weight was attributable mainly to reduced fat accumulation without a loss of lean mass. Consistent with the changes in body weight and fat mass, liver weight in the SFK group was significantly lower than that in the HFD group (MD 0.30; 95% CI, 0.18 to 0.42; $P < 0.0001$), suggesting attenuation of HFD-induced hepatomegaly. The weights of abdominal fat (AF) and epididymal white adipose tissue (eWAT) were also significantly lower in the SFK group than those in the HFD group ($P < 0.01$), although both remained higher than those in the NOR group. These findings indicate a significant but partial reduction in regional adiposity.

Effects of SFK on serum biochemical parameters

Serum hepatic enzyme activities and adipokine concentrations were measured to assess hepatic function and adipose tissue endocrine status (Table 3). Serum AST and ALT activities were markedly higher in the HFD group than those in the NOR group. SFK administration significantly reduced both enzyme activities to levels comparable with those in the NOR group (AST: MD 0.99; 95% CI, 0.60 to 1.38; ALT: MD 1.98; 95% CI, 1.16 to 2.80; both $P < 0.0001$), suggesting attenuation of HFD-induced hepatic injury.

Serum leptin concentrations in the HFD group were significantly higher than those in the NOR group and were significantly reduced by SFK administration ($P < 0.01$), consistent with the lower adiposity. Conversely, serum adiponectin concentration in the HFD group was significantly lower than that in the NOR group (MD −10.32; 95% CI, −12.55 to −8.09; $P < 0.0001$) and was significantly increased by SFK administration (MD −13.00; 95% CI, −15.23 to −10.77; $P < 0.0001$). Adiponectin concentrations in the SFK group exceeded those in the NOR group. This favorable shift in the leptin-to-adiponectin balance further supports the potential metabolic benefits of SFK.

Effects of SFK on lipid profiles

Serum and hepatic lipid profiles were analyzed to determine the effects of SFK on systemic and hepatic lipid accumulation (Table 4). Serum TG, TC, and LDL-C levels were markedly higher in the HFD group than in the NOR group. SFK administration significantly reduced serum TG, TC, and LDL-C levels (all $P < 0.0001$ vs. HFD). Serum TG, TC and LDL-C levels in the SFK group did not differ significantly from those in the NOR group, although serum TG was numerically higher. Serum HDL-C levels were slightly lower in the SFK group than in the HFD group (MD 6.41; 95% CI, 0.11 to 12.71; $P = 0.0457$) and did not differ significantly from those in the NOR group.

A similar pattern was observed for hepatic lipid levels. HFD group significantly increased hepatic TG and TC levels relative to the NOR group (both $P < 0.0001$), whereas hepatic HDL-C levels were unaffected. SFK administration significantly decreased hepatic TG and TC levels. Hepatic TG levels were restored to levels comparable with those in the NOR group. Hepatic TC level was markedly reduced but remained significantly higher than that in the NOR group, indicating substantial but incomplete normalization of hepatic lipid accumulation.

Inhibitory effects of SFK on hepatic lipid accumulation

To assess hepatic lipid accumulation visually and quantitatively, liver sections were stained with ORO to detect neutral lipid droplets (Fig. 1). Histological examination revealed minimal ORO-positive staining in the NOR group, consistent with low hepatic lipid accumulation. In contrast, the HFD group showed extensive ORO-positive staining, with numerous large lipid droplets throughout the hepatic parenchyma. SFK administration visibly reduced the number and size of ORO-positive lipid droplets relative to the HFD group, producing a staining pattern more similar to that of the NOR group.

Quantitative analysis supported these histological observations. The ORO-positive area was significantly greater in the HFD group than that in the NOR group and was significantly reduced by SFK administration. These findings indicate that SFK partially attenuated

Table 3. Serum biochemical parameters

Parameter	NOR	HFD	SFK
AST (IU/L)	3.65 ± 0.11^{b}	4.65 ± 0.47^{a}	3.66 ± 0.09^{b}
ALT (IU/L)	2.34 ± 0.36^{b}	4.60 ± 0.94^{a}	2.62 ± 0.25^{b}
Leptin (ng/mL)	1.10 ± 0.06^{c}	1.85 ± 0.25^{a}	1.49 ± 0.11^{b}
Adiponectin (ng/mL)	131.68 ± 1.23^{b}	121.36 ± 2.23^{c}	134.36 ± 1.20^{a}

Results are presented as mean ± standard deviation ($n = 7$ per group).

Different superscript letters within the same row indicate significant differences among groups ($P < 0.05$; Tukey's multiple-comparison test following one-way ANOVA among the NOR, HFD, and SFK groups).

The NOR and HFD control groups were derived from the same animal experiment as Ref. (17) and are shared between the two studies.

Table 4. Serum and hepatic lipid profiles

Parameter	NOR	HFD	SFK
Serum TG (mg/dL)	190.05 ± 2.68^{b}	254.93 ± 18.03^{a}	198.28 ± 11.22^{b}
Serum TC (mg/dL)	152.86 ± 6.78^{b}	196.23 ± 14.07^{a}	147.20 ± 3.41^{b}
Serum HDL-C (mg/dL)	77.29 ± 7.25^{ab}	78.40 ± 3.14^{a}	71.99 ± 1.24^{b}
Serum LDL-C (mg/dL)	37.56 ± 1.01^{b}	66.75 ± 7.31^{a}	35.55 ± 1.38^{b}
Hepatic TG (mg/dL)	211.41 ± 20.10^{b}	343.25 ± 5.57^{a}	221.08 ± 6.31^{b}
Hepatic TC (mg/dL)	113.45 ± 16.01^{c}	212.91 ± 10.25^{a}	159.25 ± 8.01^{b}
Hepatic HDL-C (mg/dL)	70.22 ± 5.15^{a}	72.32 ± 8.45^{a}	72.69 ± 2.21^{a}

Results are presented as mean ± standard deviation ($n = 7$ per group).

Different superscript letters within the same row indicate significant differences among groups ($P < 0.05$; Tukey's multiple-comparison test following one-way ANOVA among the NOR, HFD, and SFK groups).

Serum levels of low-density lipoprotein cholesterol (LDL-C) were estimated using the Friedewald formula (TC – [TG/5 + HDL-C]).

Hepatic lipid concentrations are expressed as mg/dL of the lipid extract obtained from 100 mg of liver tissue and dissolved in 100 µL of ethanol.

The NOR and HFD control groups were derived from the same animal experiment as Ref. (17) and are shared between the two studies.

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HFD-induced hepatic lipid droplet accumulation, consistent with the reductions in hepatic TG and TC levels shown in Table 4.

Effects of SFK on the hepatic expression of lipid metabolism-related genes

To evaluate the effects of SFK on HFD-induced dysregulation of hepatic lipid metabolism, the mRNA expression of key transcription factors and enzymes involved in hepatic lipogenesis and fatty acid β-oxidation was measured (Fig. 2). HFD markedly increased the expression of the lipogenic genes *Srebp1c*, *Lxrα*, *Fas*, and *Clebpα* relative to the NOR group. SFK administration significantly attenuated this HFD-induced upregulation of all four genes (all $P < 0.0001$). Expression level of *Clebpα* in the SFK group was lower than that in the NOR group, indicating a pronounced reduction in *Clebpα* expression.

Conversely, HFD significantly reduced expression of the fatty acid oxidation-related genes *Pparα*, *Cpt1α*, and *Acox1* relative to the NOR group (all $P < 0.0001$). SFK administration significantly restored *Pparα* and *Acox1* expression toward the levels observed with NOR group (both $P < 0.0001$). *Cpt1α* expression also increased significantly, although to a lesser extent (MD −0.060; 95% CI, −0.116 to −0.004; $P = 0.0355$). Collectively, these results indicate that SFK counteracted HFD-induced dysregulation of hepatic lipid metabolism by downregulating

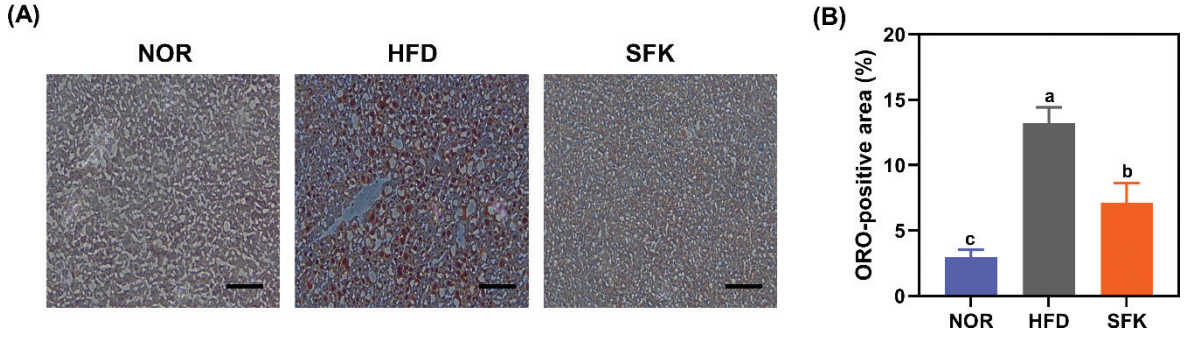

Fig. 1. Effects of WiKim0124 starter-fermented kimchi (SFK) on hepatic lipid accumulation in mice fed a high-fat diet (HFD). (A) Representative Oil Red O (ORO) staining images of liver sections from the NOR, HFD, and SFK groups (20 × magnification, scale bar = 100 μm) and (B) quantification of ORO-positive area (%). The results are presented as the mean ± standard deviation ($n = 7$ per group). Different letters above the bars indicate significant differences among groups (one-way ANOVA followed by Tukey's multiple comparisons test, $P < 0.05$).

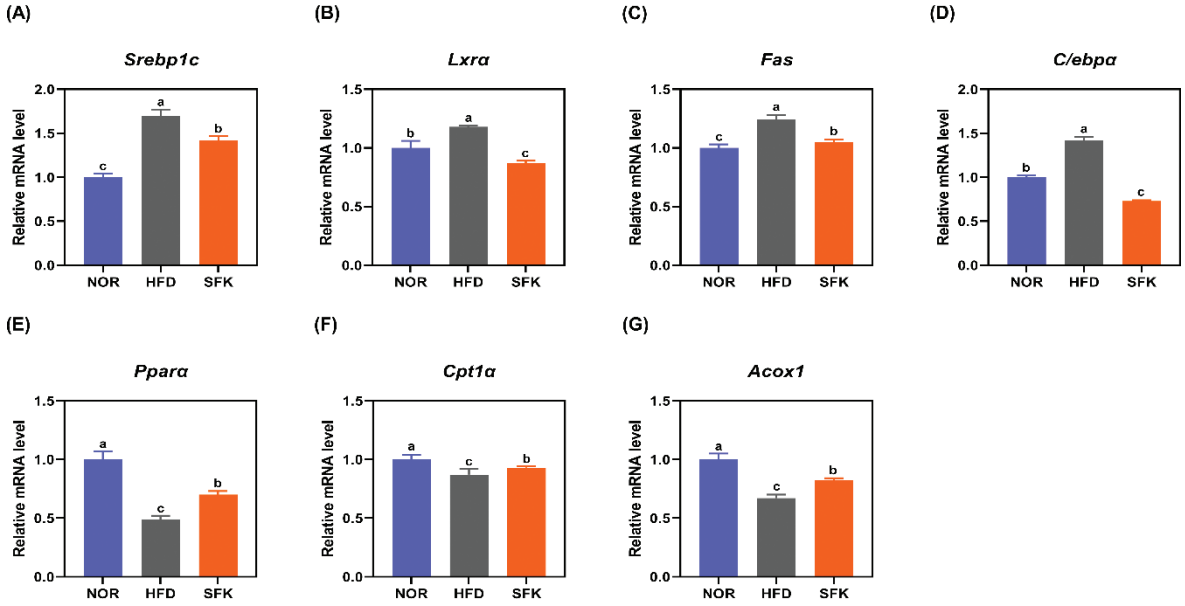

Fig. 2. Effects of WiKim0124 starter-fermented kimchi (SFK) on the hepatic mRNA expression of genes involved in lipogenesis and fatty acid β-oxidation in mice fed a high-fat diet (HFD). (A) *Srebp1c*, (B) *Lxrα*, (C) *Fas*, (D) *Clebpα*, (E) *Pparα*, (F) *Cpt1α*, and (G) *Acox1*. The results are presented as the mean ± standard deviation ($n = 7$ per group). Different letters above the bars indicate significant differences among groups (one-way ANOVA followed by Tukey's multiple comparisons test, $P < 0.05$).

lipogenic gene expression and upregulating fatty acid oxidation-related gene expression.

Effects of SFK on the hepatic expression of inflammation-related genes

The hepatic mRNA expression of pro-inflammatory and anti-inflammatory cytokines was assessed to determine whether SFK modulated HFD-induced hepatic inflammation (Fig. 3). HFD markedly increased hepatic *Tnfα* and *Il6* expression relative to the NOR group (both $P < 0.0001$). SFK administration significantly reduced the expression of both genes (both $P < 0.001$ vs. HFD), with *Tnfα* expression approaching the level observed in the NOR group.

Hepatic *Il10* expression in the HFD group was significantly reduced relative to the NOR group (MD −0.25; 95% CI, −0.31 to −0.19; $P < 0.0001$). SFK administration markedly increased *Il10* expression relative to the HFD group (MD −1.38; 95% CI, −1.44 to −1.32; $P < 0.0001$), resulting in levels higher than those in the NOR group. These findings suggest that SFK reduces pro-inflammatory gene expression and increases anti-inflammatory gene expression in the liver.

Effects of SFK on the colonic expression of inflammation- and barrier-related genes

To determine whether the hepatic effects of SFK were accompanied by changes in colonic inflammation and barrier-related gene expression, the colonic mRNA expression of inflammatory cytokines and tight junction-related genes was measured (Fig. 4). Colonic *Tnfα* and *Il6* expression levels in the HFD group were significantly higher than that in the NOR group (both $P < 0.0001$). SFK administration significantly reduced the expression of both genes relative to the HFD group (*Tnfα*: MD 0.57; 95% CI, 0.48 to 0.66; *Il6*: MD 0.48; 95% CI, 0.42 to 0.54; both $P < 0.0001$), resulting in levels lower than those in the NOR group.

Colonic *Il10* expression was modestly reduced in the HFD group but did not differ significantly between the HFD and SFK groups. This finding suggests that the colonic anti-inflammatory effects of SFK were associated mainly with suppression of pro-inflammatory cytokine expression rather than induction of *Il10*, in contrast to the liver.

HFD significantly reduced colonic expression of *Zo1* and *Occludin*, compared with the NOR group (both $P < 0.0001$). SFK administration significantly increased the expression of both genes relative to the HFD group (*Zo1*: MD −0.16; 95% CI, −0.19 to −0.13; *Occludin*: MD −0.09; 95% CI, −0.13 to −0.05; both $P < 0.0001$). These results are consistent with partial restoration of colonic tight junction-related gene expression. Additional statistical comparisons between the HFD and SFK groups, including mean differences and corresponding 95% CIs, are provided in Supplementary Table S1.

Discussion

In previous studies, the anti-obesity effects of NK and SK were demonstrated *in vitro, in vivo,* and in a clinical trial (16–18). Furthermore, the functional properties of WiKim0124 itself have been reported (19, 20). This study showed that oral administration of SFK prepared using WiKim0124 attenuated HFD-induced hepatic lipid dysregulation, hepatic and colonic inflammation, and changes in the expression of colonic barrier-related genes in obese mice. These findings suggest that the metabolic benefits of SFK may be associated with changes in hepatic metabolism and in colonic inflammation and barrier-related gene expression.

The phenotypic and biochemical findings presented in Tables 2–4 provide physiological support for the molecular

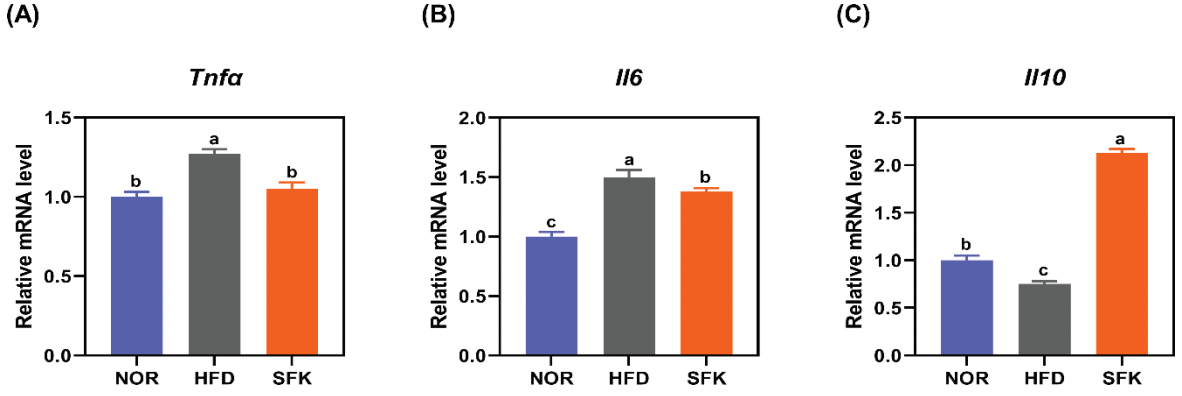

Fig. 3. Effects of WiKim0124 starter-fermented kimchi (SFK) on the hepatic mRNA expression of inflammation-related genes in mice fed a high-fat diet (HFD). (A) *Tnfα*, (B) *Il6*, and (C) *Il10*. The results are presented as the mean ± standard deviation ($n = 7$ per group). Different letters above the bars indicate significant differences among groups (one-way ANOVA followed by Tukey's multiple comparisons test, $P < 0.05$).

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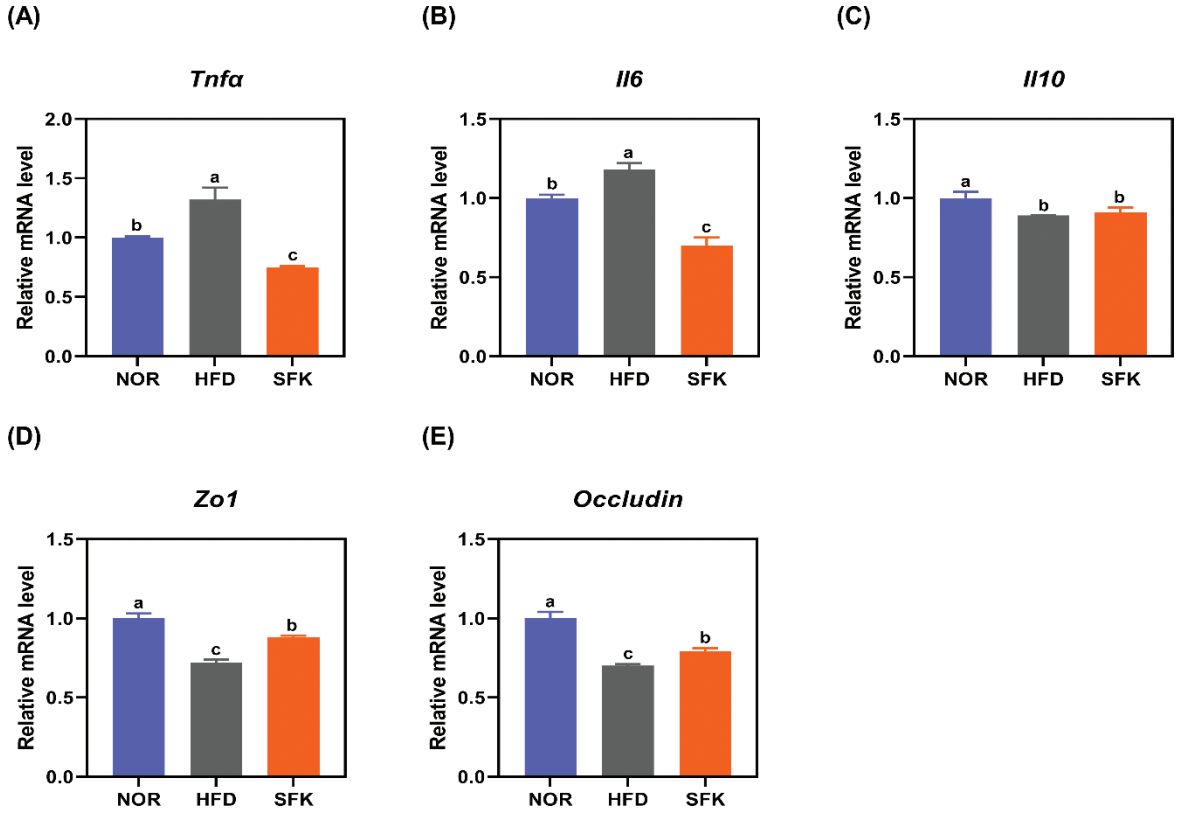

Fig. 4. Effects of WiKim0124 starter-fermented kimchi (SFK) on the colonic mRNA expression of genes associated with inflammation and colonic barrier-related gene expression in mice fed a high-fat diet (HFD). (A) *Tnfα*, (B) *Il6*, (C) *Il10*, (D) *Zo1*, and (E) *Occludin*. The results are presented as the mean ± standard deviation (n = 7 per group). Different letters above the bars indicate significant differences among groups (one-way ANOVA followed by Tukey's multiple comparisons test, $P < 0.05$).

changes shown in Figs. 2–4. SFK significantly attenuated HFD-increased body weight and fat accumulation without altering food intake, suggesting that these effects were not due to reduced food consumption. The lower FER indicates that SFK reduced body weight relative to food consumption. These results are consistent with previous studies showing that kimchi-derived LAB strains and LAB-fermented kimchi reduce body weight and adiposity in HFD-fed mice without reducing food intake (17, 21, 22). The preservation of lean mass, together with the selective reduction in fat mass, further supports an effect primarily involving adiposity. These phenotypic changes were consistent with the suppression of lipogenic genes and increased expression of fatty acid oxidation-related genes.

Previous clinical evidence also provides context for interpreting the potential relevance of kimchi consumption to body-weight management in humans. In our previous randomized, double-blind, placebo-controlled clinical trial (18), overweight participants consumed freeze-dried kimchi preparations corresponding to approximately 60 g of kimchi per day for 12 weeks. These preparations included kimchi fermented with *Leuconostoc mesenteroides* KCKM0828. Both the spontaneously fermented kimchi and SFK groups showed reductions in body fat mass and body fat percentage compared with the placebo group. In addition, the SFK group showed favorable changes in body weight and selected metabolic parameters. These findings suggest that consumption of kimchi preparations may have potential benefits for body-fat management in humans. However, the kimchi preparations and intervention conditions used in that clinical trial differed from those of this study, and the clinical findings cannot be directly attributed to WiKim0124 SFK. Accordingly, the present animal findings should not be extrapolated to commercially available or homemade kimchi, or to people who are already overweight or obese.

For reference, the mouse dose of 70 mg/kg BW used in this study corresponds to an estimated human-equivalent dose of approximately 5.7 mg/kg BW, based on body-surface-area scaling (mouse Km = 3 and human Km = 37) (23). This corresponds to approximately 0.34 g of freeze-dried SFK powder per administration for a 60-kg adult. Assuming an approximate freeze-drying yield of 10%, this amount corresponds to approximately 3.4 g of fresh kimchi per administration. However, SFK was administered three times per week in this study, so this estimate should not be interpreted as a daily intake recommendation.

Furthermore, this calculation provides only an approximate dose comparison and does not establish an effective or recommended intake level for humans.

The normalization of serum AST and ALT activities by SFK provides biochemical evidence of reduced hepatic injury. This finding is supported by the reductions in hepatic TG and TC levels shown in Table 4 and the suppression of hepatic lipogenesis-related gene expression shown in Fig. 2. Elevated serum transaminase activities in HFD-fed mice generally reflect hepatocellular injury associated with lipid accumulation and inflammation. Therefore, the concurrent reductions in hepatic *Tnfα* and *Il6* expression and in serum transaminase activities suggest that SFK may have alleviated HFD-associated hepatic injury, in association with its lipid-regulatory and anti-inflammatory effects.

The decrease in leptin and increase in adiponectin concentrations following SFK administration are notable because this adipokine profile is consistent with the favorable changes in adiposity observed in the SFK group. Leptin resistance and hypoadiponectinemia are characteristic features of diet-induced obesity and are mechanistically associated with adipose tissue inflammation and ectopic lipid deposition (24, 25). The adiponectin concentration in the SFK group was higher than that in the NOR group, resembling the pattern observed for hepatic *Il10* expression. These findings raise the possibility that bioactive components or metabolites derived from SFK may contribute to metabolic and inflammatory regulation beyond the prevention of HFD-induced deterioration.

The histological reduction in hepatic lipid droplet accumulation shown in Fig. 1 supports the improvement in hepatic lipid profiles shown in Table 4. This improvement may be explained, at least in part, by the concurrent suppression of lipogenic genes and increased expression of fatty acid oxidation-related genes shown in Fig. 2. The histological, biochemical, and transcriptional findings are consistent with SFK attenuating HFD-induced hepatic steatosis through coordinated regulation of hepatic lipid metabolism. Residual ORO-positive staining remained evident in the SFK group relative to the NOR group, consistent with incomplete normalization of hepatic TC levels. Thus, although SFK substantially alleviated HFD-induced hepatic lipid accumulation, some hepatic lipid parameters were not completely normalized. Further studies are needed to determine the effects of different SFK doses and treatment duration on hepatic lipid accumulation.

SFK suppressed the expression of lipogenic genes *Srebp1c*, *Lxrα*, and *Clebpα*, as well as *Fas*, which encodes a key enzyme in fatty acid synthesis. Simultaneously, SFK increased the expression of *Pparα* and its target genes *Cpt1α* and *Acox1*, which are involved in mitochondrial and peroxisomal fatty acid *β*-oxidation. This dual effect, involving suppression of lipid synthesis and promotion of fatty acid catabolism, is consistent with previous studies of kimchi-derived LAB and LAB-fermented kimchi. These interventions have been reported to downregulate hepatic lipogenesis-related genes and improve serum and hepatic lipid profiles in HFD-fed mice (17, 26, 27). Previous studies of kimchi fermented using selected LAB starter cultures also demonstrated similar suppression of adipogenic and lipogenic gene expression in 3T3-L1 adipocytes and in HFD-induced obese mice (16, 17), suggesting that similar changes in lipid metabolism-related gene expression may occur across different kimchi formulations.

The reduced hepatic expression of *Tnfα* and *Il6*, together with the marked upregulation of *Il10*, suggests that SFK attenuates HFD-induced hepatic inflammation while promoting an anti-inflammatory hepatic environment. The higher hepatic *Il10* expression in the SFK group than that in the NOR group may be associated with bioactive metabolites produced during LAB-mediated kimchi fermentation. This possibility is supported by previous studies demonstrating that hepatic *Il10* expression is modulated by fatty acid exposure and can be enhanced by probiotic interventions in HFD-fed mice (28, 29). Potential mediators may include short-chain fatty acids, exopolysaccharides, and bacteriocin-like compounds, although these compounds were not measured in this study.

At the intestinal level, SFK partially restored *Zo1* and *Occludin* mRNA expression, consistent with previous findings that probiotics and LAB can mitigate HFD-induced reduction in tight junction protein expression levels and help preserve intestinal barrier function (30–32). Disruption of the intestinal barrier by HFD may facilitate the translocation of bacterial lipopolysaccharide into the portal circulation, thereby contributing to metabolic endotoxemia and hepatic inflammation. Therefore, the concurrent suppression of colonic pro-inflammatory cytokine expression and restoration of tight junction-related gene expression observed in this study may be associated with the attenuation of hepatic inflammation. These parallel changes in colonic barrier-related genes and hepatic inflammatory markers are consistent with possible contribution of the gut–liver axis to the observed effects of SFK. Nevertheless, a direct causal relationship between intestinal barrier improvement and reduced hepatic inflammation was not established.

Unlike the marked induction of hepatic *Il10* expression, colonic *Il10* expression was not significantly altered by SFK, indicating a tissue-specific response to the intervention. This differential response may reflect distinct immunomodulatory mechanisms in the liver and colon, potentially arising from differences in local immune cell populations, including hepatic Kupffer cells and macrophages in the colonic lamina propria, or in the

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tissue-specific responses to bioactive components generated during SFK fermentation.

Collectively, these findings highlight the potential of SFK as a functional food that may simultaneously improve hepatic lipid metabolism, attenuate hepatic and colonic inflammation, and partially restore colonic barrier-related gene expression in diet-induced obesity. Furthermore, these findings provide a rationale for further investigating the roles of kimchi-derived LAB and their metabolites in modulating metabolic disturbances associated with obesity.

Limitations

This study has several limitations that should be considered when interpreting the findings. Firstly, gene expression changes were evaluated at the mRNA level, and validation at the protein-level is needed. Secondly, intestinal permeability, circulating endotoxin-related markers (e.g. serum LPS or LBP), glucose homeostasis (e.g. fasting glucose and insulin, or glucose and insulin tolerance tests), gut microbial composition, fermentation-associated metabolites, and renal and cardiovascular-related parameters were not evaluated, limiting the mechanistic and broader systemic interpretation of the findings. Thirdly, only male mice were used, and SFK was administered at a single dose concurrently with HFD for 8 weeks; therefore, the findings may not fully represent responses in females, longer-term treatment, or the therapeutic effects of SFK in established obesity. Further studies are needed to evaluate the effects of different SFK doses and treatment durations. In addition, the relative abundance of WiKim0124 and its viable counts after fermentation and freeze-drying were not determined. Thus, its dominance and the viable cell dose could not be confirmed, and the individual contributions of the starter strain, kimchi matrix, and fermentation-derived components could not be distinguished, and the strain's safety characteristics (e.g. antibiotic resistance, virulence factors, and biogenic amines) were not assessed. Finally, the NOR and HFD groups were part of the same animal experiment as the NK and SK groups reported previously (17), whereas the SFK group was an additional treatment group whose results had not been previously reported. This shared-control design was adopted to reduce unnecessary animal use in accordance with the 3Rs principles. Accordingly, this study should not be considered an independent replication of the previous experiment.

Conclusions

Oral administration of SFK prepared using WiKim0124 significantly attenuated HFD-increased body weight and fat accumulation without affecting food intake. These effects were accompanied by the downregulation of hepatic lipogenesis-related genes, regulation of fatty acid oxidation-related genes, reduced hepatic lipid droplet accumulation, and improved serum and hepatic lipid profiles. In addition, SFK attenuated hepatic and colonic inflammation and partially restored colonic expression of tight junction-related genes. Collectively, these findings suggest that SFK may exert anti-obesity effects in association with improvements in hepatic lipid metabolism, inflammatory responses, and colonic barrier-related gene expression, supporting its potential as a functional food for obesity management.

Acknowledgments

Not applicable.

Conflict of interests and funding

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Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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