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Biochemistry

Effects of overfeeding on the fatty acid profile and stearoyl-CoA desaturase-1 indices in the liver and subcutaneous adipose tissue in cats

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ABSTRACT. To evaluate the effect of overfeeding on fatty acid distribution and metabolism, especially stearoyl-CoA desaturase-1 (SCD-1) indices, 8 cats in the experimental and control groups (4 per group) were evaluated in this study. The experiments involved feeding the experimental group cats twice their daily energy requirement with a commercial diet for 4 weeks. The control group was fed the estimated daily energy requirement with the same diet. Body weight, feline body mass index, body condition score, several zoometry measurements, and plasma metabolites/hepatic injury markers were measured in all the cats before and after the experiment. In addition, the fatty acid profiles in the liver and subcutaneous adipose tissue were measured after the experiment. After 4 weeks of overfeeding, the experimental group demonstrated significant increases in hepatic C18:1, plasma triglyceride, and nonesterified fatty acid (NEFA) concentrations and in alanine aminotransferase activity. Furthermore, hepatic SCD-1 indices were positively correlated with body weight, feline body mass index, body condition score, and plasma NEFA concentration, although subcutaneous adipose tissue did not demonstrate any increase in SCD-1 indices in this study. The increase in hepatic SCD-1 indices might be enhanced by the inflow of plasma NEFA into the liver, and NEFA toxicity might stimulate C18:1 synthesis by SCD-1.

KEY WORDS: fatty acid profile, feline, overfeeding, stearoyl CoA desaturase-1 index

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In Japan, the prevalence of felines that are overweight and obese is approximately 56% [22], suggesting that the risk of fat accumulation in the liver and adipose tissue is increasing. Increases in nonesterified fatty acid (NEFA) in the blood are derived from ectopic fat accumulation and prevent glucose (Glu) and lipid metabolism [26]. These deleterious effects have been termed lipotoxicity [28].

When lipotoxicity is considered, the histionic fatty acid profile is important. Saturated fatty acids (SFAs), particularly C16:0 and C18:0, induce strong lipotoxicity, leading to inflammation, insulin resistance, and subsequent hepatocyte apoptosis [17, 31].

Unsaturated fatty acids (UFAs), particularly C18:1 and omega-3 fatty acids, exert preventive effects against metabolic disorders [19, 27]. However, little information is available regarding the relationship between overfeeding and accumulated fatty acid composition in cats.

Stearoyl-CoA desaturase-1 (SCD-1), known as Δ-9 desaturase, synthesizes monounsaturated fatty acids (MUFAs: C16:1 and C18:1) from SFAs (C16:0 and C18:0) [1, 24]. SFAs, such as C16:0, stimulate SCD-1 mRNA expression *in vitro* [26]. Enzyme activity or the level of RNA expression should be directly measured to detect the potential effect of SCD-1 on health. However, the levels of SCD-1 indices C16:1/C16:0 and C18:1/C18:0 are more often used for practical or ethical reasons [1], although their effects on health are controversial.

Some researchers have reported protective effects of SCD-1 [12, 17, 26]. Additionally, a recent report stated that SCD-1 inhibition promotes SFA-induced atherosclerosis in mice [5]. This previous study demonstrated that SCD-1 antisense oligonucleotide treatment in mice with an SFA diet produced a 2.7-fold increase in the total aortic lesion area compared to that from control SCD-1 antisense oligonucleotide treatment. The study hypothesized that hepatic SCD-1 inhibition stimulates

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inflammation by macrophages via the secretion of SFA-rich very-low-density lipoproteins, followed by the secretion of SFA-rich low-density lipoproteins. Furthermore, Li *et al*. [17] reported that SCD-1 inhibition in hepatocytes increases cell sensitivity to SFA-induced apoptosis. These data suggest that hepatic desaturation by SCD-1 may be a protective mechanism against SFA-mediated lipotoxicity.

Conversely, other researchers have reported the negative effects of SCD-1 activation, such as a deterioration of fat accumulation [25], metabolic syndrome [1], insulin resistance [26], and hepatic steatosis [17]. The inhibition of SCD-1 has been targeted for the treatment of obesity, metabolic syndrome, and type 2 diabetes [1, 8].

Overfeeding contributes to metabolic deterioration during the early phases of obesity [21]. To the best of the authors' knowledge, no studies on SCD-1 have been reported in cats. The aim of this study was to evaluate the effect of overfeeding on fatty acid distribution in the liver and subcutaneous adipose tissue as well as to assess fatty acid metabolism, especially SCD-1 indices, in cats.

MATERIALS AND METHODS

Animals

Eight intact cross-bred male cats (1–2 years old, nonlittermates) were utilized. Four cats [body weight (BW) 3.9 ± 0.2 kg and body condition score (BCS) 3.3 ± 0.3] were defined as the experimental group and fed a commercial diet [Royal Canin Sensible 33, Royal Canin Japon, Inc., Minato, Japan; moisture, 7.0%; crude protein, 33.0%; ether extract, 22.0%; crude fiber, 2.8%; crude ash, 6.8%; metabolizable energy (ME)/100 g, 443.6 kcal] consisting of 2/3 of their daily energy requirement (DER; $1.4 \times 70 \times BW^{0.75}$ kcal) 3 times daily, at 9:00 am, 1:00 pm, and 5:00 pm (the total amount was designated as 2-fold their DER), for 4 weeks.

Four cats [BW 3.7 ± 0.2 kg, BCS 2.8 ± 0.3] were defined as the control group and fed the same diet given to the experimental group consisting of half their DER 2 times daily at 9:00 am and 5:00 pm (the total amount was designated as their DER) for 4 weeks.

The amount of food consumption and leftovers was recorded daily. The cats were housed in individual cages at a temperature of 24 ± 2°C and a relative humidity of 55.0 ± 10.0% with a 12:12 hr light:dark cycle (lights on: 8:00 am to 8:00 pm) at the AQS Co., Ltd. (Narita, Japan).

The BW, BCS, head and body length (HBL), length from the top of patella to the end of calcaneus (PCL), neck girth (NG), chest girth (CG), abdominal girth (AG), and hip girth (HG) were measured before and after the experiment. The BCS was determined using the following five-point scale: 1, thin; 2, lean; 3, ideal; 4, overweight; and 5, obese. HBL was determined by the length from the top of the nose to the joint between the sacrum and the coccyx. The PCL was determined by the length from the top of patella to the end of calcaneus. NG, CG, AG, and HG were determined by the circumference of the atlas, 6–7th rib, 13th rib, and pelvis, respectively. These zoometry measurements were measured using a commercial tape measure. The feline body mass index (fBMI) was calculated with the following formula: BW (kg)/PCL (m) [13].

All procedures and protocols were performed in accordance with the Ethical Codes for Laboratory Animal established by the Nippon Veterinary and Life Science University.

Blood and tissue sample collection

Preprandial blood was drawn from the jugular vein into heparinized plastic tubes concurrently with BW measurements. The blood samples were immediately centrifuged at 3,000 rpm for 5 min in a refrigerated centrifuge (4°C) to obtain plasma. The samples were stored at −25°C for further use.

At the end of each experimental period, the cats were premedicated with 0.1 mg/kg BW of Medetomidine (Domitor, Meiji Co., Ltd., Tokyo, Japan) after overnight fasting and then anesthetized with isoflurane. Then, laparotomy was performed, and 5 g of liver and abdominal subcutaneous adipose tissue samples were immediately obtained from all the cats under minimal stress conditions to the animal. The tissue samples were immediately stored at −80°C for further use.

Plasma biomarker analysis

Plasma Glu, triglyceride (TG), total cholesterol (T-Cho), and total protein (TP) concentrations as well as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) activities were determined using an autoanalyzer (AU2700, Olympus Corp., Tokyo, Japan) with the manufacturer's reagents. Plasma NEFA and insulin concentrations were determined using a commercial kit (NEFA-C test WAKO, Wako Pure Chemical Industries, Ltd., Osaka, Japan, and Cat Insulin ELISA kit, SHIBAYAGI Co., Ltd., Shibukawa, Japan, respectively).

Fatty acid sample preparation and profiling analysis

Fat that accumulated in the liver and subcutaneous adipose tissues was extracted with chloroform/methanol [(2/1; v/v)] using a modification of the method of Folch *et al.* [11]. Fatty acid profiles were measured as methyl esters using a gas chromatograph (GC-2000 Shimadzu Gas Chromatograph, Shimadzu Corp., Kyoto, Japan) with a 0.2 μm (film thickness) × 0.25 mm (internal diameter) × 100 m capillary column (RT-2560, Shimadzu GLC Ltd., Tokyo, Japan) and a flame ionization detector.

Statistical analysis

The values are expressed as the means ± standard error. Statistically significant differences between the experimental and control groups were assessed using the Mann-Whitney *U*-test. To identify the statistical power between the experimental group and control

group for SCD-1 indices, a *post hoc* power analysis was performed using software (G* Power 3.1, Kiel University, Germany) [9].

To identify the relationships of SCD-1 indices with zoometry and plasma metabolites, Pearson's regression analysis was performed, and a regression coefficient was obtained. BW, fBMI, BCS, Glu, TG, NEFA, T-Cho, Insulin, TP, ALT, AST, and LDH were each set as dependent values. Each SCD-1 index (C16:1/C16:0 and C18:1/C18:0) was set as an independent value. Statistical significance was set at $P<0.05$.

RESULTS

Zoometry measurements and plasma biomarker analysis

After 4 weeks of overfeeding, the BW, fBMI, BCS, NG, AG, HG, plasma TG, NEFA, TP concentration, and plasma ALT activity in the experimental group were significantly higher than those in the control group ($P<0.05$) (Table 1). No significant differences were found between the experimental and control groups before initiating this experiment.

Fatty acid profiling

After overfeeding, the C18:1 level in the livers of the experimental group were significantly increased compared to those in the livers of the control group ($P<0.05$) (Table 2). The 3 most abundant fatty acids (in descending order) in the livers from the experimental group were C18:2, C18:0, and C18:1, and the most abundant fatty acids from the control group were C18:0, C18:2, and C16:0. These 3 fatty acids in the livers from the experimental and control groups comprised 62.33 and 57.49%, respectively, of the total fatty acids. The statistical power of each SCD-1 index in the liver between the experimental and control groups was 0.36 and 0.35, respectively.

The C10:0, C14:0, C20:4, C20:5 and C22:6 levels were significantly increased, and C20:0 was significantly decreased in the subcutaneous adipose tissue of the experimental group compared to that of the control group (Mann-Whitney *U*-test, $P<0.05$) (Table 2).

The 3 most abundant fatty acids in the subcutaneous adipose tissue from both the experimental and control groups (in descending order) were C18:1, C16:0, and C18:2, and they comprised 73.13 and 77.90%, respectively, of the total fatty acids. The statistical power of each SCD-1 index in subcutaneous adipose tissue between the experimental and control groups was 0.05 and 0.25, respectively.

The hepatic SCD-1 indices C16:1/C16:0 and C18:1/C18:0 demonstrated significant positive correlations with BW, fBMI, BCS, and NEFA concentration after overfeeding ($P<0.05$) (Tables 3 and 4). The SCD-1 indices in subcutaneous adipose tissue did not show any significant correlations with zoometry measurements or plasma metabolites.

Table 1. Comparison of zoometry measurements and plasma biomarkers before and after 4 weeks of overfeeding in the experimental and control group cats

	0 week			4 weeks		
	Experiment (4)	Control (4)	*P* value	Experiment (4)	Control (4)	*P* value
Zoometry measurements						
BW (kg)	3.9 ± 0.2	3.7 ± 0.2	0.38	4.7 ± 0.3[a)]	3.7 ± 0.2	0.03
fBMI (kg/m)	26.4 ± 0.8	24.6 ± 1.4	0.25	33.1 ± 1.8[a)]	25.1 ± 1.3	0.02
BCS (/5)	3.3 ± 0.3	2.8 ± 0.3	0.19	4.3 ± 0.3[a)]	2.8 ± 0.3	0.02
HBL (cm)	56.5 ± 0.7	57.6 ± 1.5	0.56	56.4 ± 1.6	56.3 ± 1.4	1.00
PCL (cm)	14.6 ± 0.5	14.9 ± 0.5	0.56	14.3 ± 0.5	14.6 ± 0.4	0.77
NG (cm)	19.9 ± 0.5	19.9 ± 0.3	1.00	21.0 ± 0.4[a)]	18.8 ± 0.5	0.03
CG (cm)	30.3 ± 1.0	30.8 ± 1.0	0.77	32.8 ± 0.9	30.3 ± 0.7	0.06
AG (cm)	35.0 ± 1.1	35.3 ± 1.2	0.88	39.1 ± 1.8[a)]	34.9 ± 0.8	0.02
HG (cm)	30.9 ± 1.3	31.0 ± 1.2	0.88	34.8 ± 1.9[a)]	30.0 ± 0.8	0.04
Plasma metabolites						
Glu (mg/d*l*)	117.8 ± 18.9	162.3 ± 18.1	0.15	148.0 ± 24.2	141.3 ± 19.8	0.88
TG (mg/d*l*)	28.8 ± 5.5	23.8 ± 2.3	0.77	48.3 ± 3.8[a)]	21.8 ± 1.4	0.02
NEFA (mEq/*l*)	0.29 ± 0.06	0.21 ± 0.01	0.25	0.38 ± 0.02[a)]	0.27 ± 0.01	0.02
T-cho (mg/d*l*)	122.8 ± 3.4	133.5 ± 13.7	0.88	191.8 ± 9.4	200.8 ± 16.1	0.38
Insulin (*n*g/m*l*)	1.8 ± 0.1	2.5 ± 0.4	0.25	1.6 ± 0.1	1.7 ± 0.1	0.08
TP (mg/d*l*)	6.4 ± 0.1	6.2 ± 0.2	0.66	6.8 ± 0.1[a)]	6.1 ± 0.1	0.02
ALT (IU/*l*)	69.3 ± 7.2	64.5 ± 2.1	1.00	131.3 ± 14.2[a)]	68.0 ± 4.1	0.02
AST (IU/*l*)	18.3 ± 0.9	20.0 ± 2.7	0.77	34.3 ± 6.6	35.3 ± 6.4	1.00
LDH (IU/*l*)	103.5 ± 14.0	71.8 ± 8.1	0.15	132.8 ± 36.5	105.0 ± 23.9	0.66

Values are presented as means ± standard error. The numbers in parenthesis indicate the number of animals examined. a) Significantly different from the control group (Mann-Whitney *U*-test, $P<0.05$). BW: Body weight, fBMI: feline body mass index, BCS: Body condition score, HBL: Head and body length, PCL: length from the top of patella to the end of calcaneus, NG: Neck girth, CG: Chest girth, AG: Abdominal girth, HG: Hip girth, Glu: Glucose, TG: Triglyceride, NEFA: Non-esterified fatty acid, T-Cho: Total cholesterol, TP: Total protein, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LDH: Lactate dehydrogenase.

Table 2. Comparison of the fatty acid profiles in the liver and subcutaneous adipose tissue after 4 weeks of overfeeding in the experimental and control group cats

(%)	Liver			Subcutaneous adipose tissue		
	Experiment (4)	Control (4)	*P* value	Experiment (4)	Control (4)	*P* value
C8:0	1.00 ± 0.45	6.26 ± 3.16	0.39	0.09 ± 0.01	0.07 ± 0.01	0.25
C10:0	0.05 ± 0.05	3.05 ± 1.77	0.32	0.44 ± 0.09[a)]	0.18 ± 0.04	0.02
C12:0	1.26 ± 0.66	3.01 ± 0.99	0.25	5.75 ± 0.62	3.40 ± 1.05	0.08
C14:0	1.93 ± 0.14	1.04 ± 0.41	0.25	5.94 ± 0.39[a)]	4.12 ± 0.33	0.02
C14:1	1.33 ± 0.45	0.62 ± 0.16	0.15	0.49 ± 0.01	0.46 ± 0.03	0.56
C16:0	16.97 ± 0.45	14.70 ± 2.50	0.39	22.78 ± 0.55	22.62 ± 0.39	1.00
C16:1	3.15 ± 0.76	1.62 ± 0.38	0.08	4.03 ± 0.07	3.51 ± 0.54	0.25
C18:0	21.61 ± 2.55	22.44 ± 1.90	0.77	7.17 ± 0.40	7.96 ± 0.48	0.25
C18:1	17.90 ± 2.46[a)]	12.16 ± 1.56	0.04	30.26 ± 1.22	34.65 ± 1.73	0.08
C18:2	22.82 ± 0.68	20.35 ± 1.91	0.56	20.10 ± 0.63	20.62 ± 0.49	0.39
C18:3	0.60 ± 0.35	1.27 ± 0.87	0.77	1.36 ± 0.11	0.96 ± 0.10	0.08
C20:0	0.12 ± 0.07	0.14 ± 0.10	1.00	0.17 ± 0.02[a)]	0.24 ± 0.00	0.02
C20:1	0.08 ± 0.05	0.12 ± 0.08	0.76	0.40 ± 0.05	0.56 ± 0.06	0.08
C20:4	9.51 ± 0.94	8.79 ± 0.52	0.77	0.40 ± 0.01[a)]	0.29 ± 0.02	0.02
C20:5	0.80 ± 0.47	2.49 ± 1.02	0.24	0.36 ± 0.03[a)]	0.15 ± 0.01	0.02
C22:6	0.91 ± 0.55	1.94 ± 1.23	0.55	0.26 ± 0.01[a)]	0.19 ± 0.03	0.04
SFAs	42.93 ± 3.18	50.64 ± 1.97	0.08	42.34 ± 1.11	38.60 ± 1.70	0.15
UFAs	57.08 ± 3.17	49.36 ± 1.97	0.08	57.66 ± 1.11	61.40 ± 1.70	0.15
C16:1/C16:0	0.19 ± 0.05	0.11 ± 0.01	0.25	0.18 ± 0.01	0.16 ± 0.03	0.25
C18:1/C18:0	0.89 ± 0.21	0.54 ± 0.05	0.15	4.26 ± 0.26	4.41 ± 0.40	0.77

Values are presented as means ± standard error. The numbers in parentheses indicate the number of animals examined. a) Significantly different from the control group (Mann-Whitney *U*-test, $P<0.05$). SFAs: Saturated fatty acids, UFAs: Unsaturated fatty acids.

Table 3. Correlations between stearoyl CoA desaturase-1 (SCD-1) indices in liver or subcutaneous adipose tissue and zoometry measurements after 4 weeks of overfeeding in cats

	BW	fBMI	BCS
SCD-1 indices in liver			
C16:1/C16:0	0.80[a)]	0.82[a)]	0.73[a)]
C18:1/C18:0	0.79[a)]	0.83[a)]	0.76[a)]
SCD-1 indices in subcutaneous adipose tissue			
C16:1/C16:0	0.08	0.16	0.40
C18:1/C18:0	0.19	0.35	0.62

Values are presented as regression coefficients. a) Significant correlation by Pearson's regression analysis ($P<0.05$). BW: Body weight, fBMI: feline body mass index, BCS: Body condition score.

Table 4. Correlations between stearoyl CoA desaturase-1 (SCD-1) indices in liver or subcutaneous adipose tissue and plasma metabolites after 4 weeks of overfeeding in cats

	Glu	TG	NEFA	T-cho	Insulin	TP	ALT	AST	LDH
SCD-1 indices in liver									
C16:1/C16:0	−0.42	0.47	0.75[a)]	−0.36	−0.08	0.53	0.56	−0.58	−0.14
C18:1/C18:0	−0.36	0.54	0.74[a)]	−0.22	−0.04	0.42	0.62	−0.48	−0.03
SCD-1 indices in subcutaneous adipose tissue									
C16:1/C16:0	0.37	0.23	0.09	0.25	−0.07	0.28	0.24	0.05	0.36
C18:1/C18:0	0.38	0.57	0.30	−0.16	−0.05	0.43	0.49	0.58	0.39

Values are presented as regression coefficients. a) Significant correlation by Pearson's regression analysis ($P<0.05$). Glu: Glucose, TG: Triglyceride, NEFA: Non-esterified fatty acid, T-cho: Total cholesterol, TP: Total protein, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LDH: Lactate dehydrogenase.

DISCUSSION

The determinants of the fatty acid profiles in tissue were species specificity, diet, and metabolism (elongation, desaturation, and saturation). The fatty acid profile in feline adipose tissue was similar to that in humans, and the 3 most abundant fatty acids in the

tissues from humans (in descending order) were C18:1, C16:0, C18:2 [15], which agreed with our results.

Subcutaneous adipose tissue primarily operates as a storage organ for excessive dietary fat during overfeeding. Field *et al*. [10] showed that the ratio of dietary polyunsaturated fatty acids/SFAs was significantly related to the saturated and polyunsaturated content of subcutaneous adipose tissue. van Niel and Beynen also reported a strong correlation in C18:2 and C18:3 levels between diet and subcutaneous adipose tissue in cats [30], as in other species [16, 18, 20, 29]. In our previous trial [14], the fatty acid profile in subcutaneous adipose tissue in experimental and control cats also reflected the dietary fatty acid profile. However, the dietary effects were not controlled in the previous trial because diets containing different percentages of fat were given in each group; the experimental cats were fed a high-fat diet, and the control cats were fed a moderate-fat diet. To prevent dietary effects, an identical diet was used in the current study.

The main source of fat is poultries fat in the used diet. According to the USDA Food Composition Databases (https://ndb.nal.usda.gov/ndb/), the chicken oil, rather than the oil from turkey poultry [6], is the more similar to the fatty acid profile in subcutaneous adipose tissue in the current study. Thus, fatty acid profile in the subcutaneous adipose tissue mainly reflected that in chicken oil.

C16:0 is an end product of fatty acid biosynthesis [3]. The significant differences in C14:0 in subcutaneous adipose tissue may suggest minor interference with elongation of C14:0 to C16:0 during overfeeding. However, the impact on the fatty acid profile is relatively small. In addition, the significant differences in the fatty acid profile between the experimental and control groups in subcutaneous adipose tissue were within 2.00%. Thus, subcutaneous adipose tissue showed a similar fatty acid profile pattern in both the experimental and control groups (post hoc power analysis). Similarly, the nonsignificant differences in the SCD-1 indices from subcutaneous adipose tissue between the experimental and control groups suggest that SCD-1 in subcutaneous adipose tissue was not activated by overfeeding.

Conversely, both hepatic SCD-1 indices showed significant correlations with plasma NEFA concentration. These differences between subcutaneous adipose tissue and liver suggest a specific fatty acid metabolism in the liver during overfeeding. Increased hepatic SCD-1 indices after overfeeding might be enhanced by an increased inflow of plasma NEFA into the liver in cats. An excess of chylomicrons induced by overfeeding is catabolized by lipoprotein lipase found in several tissues, such as adipose tissue and muscle, and synthesizes NEFAs [4]. Although most NEFAs were absorbed into the adipose tissue, the remainder, which exceeds the absorbency ability, is maintained in the blood and contributes to increasing its concentration in blood. An increase in the plasma NEFA concentration has been reported to accelerate β-oxidation and the subsequent synthesis of reactive oxygen species in the liver [23]. The toxicity of NEFA might stimulate the synthesis of C18:1 by SCD-1. Finally, the increase in MUFAs, especially C18:1, contributed to the reduced occupancy of SFA in the liver. These sequential hepatic metabolism steps might increase the antioxidant capacity by MUFAs [2] and might reduce the harmful effect of SFA in the liver [17, 31].

According to the mild increase in fat accumulation and hepatic injury markers after overfeeding, the experimental group was considered to have early stage hepatic steatosis before hepatic lipidosis. In humans, the development of nonalcoholic steatohepatitis (NASH) is explained as “two-hit-theory” [7]. In the first step, hepatic fat accumulation and steatosis is developed without severe inflammation and symptoms by hypernutrition and lack of exercise. Oxidative stress and lipid peroxidation, the second step, contribute to mitochondrial injury, the release of inflammatory cytokines, insulin resistance, and further hepatic inflammation. The experimental group in our study countervailed the first step of NASH in humans [17]. The progression of the pathophysiology should be interrupted at this first stage.

To treat or prevent hepatic lipidosis, dietary C18:1 may compensate for the synthesis of C18:1 by hepatic SCD-1. However, as shown by the elevated ALT activity in the experimental group after overfeeding, the protective effect of MUFAs appeared to be insufficient for the liver. To avoid damage to the liver, additional treatments, such as supplementation with several antioxidants, may be needed.

Unfortunately, this study did not show any significant differences in SCD-1 indices between the experimental and control groups due to the low statistical power, which was related to animal ethics standards. Additionally, our research did not include any oxidative stress markers. Further research is needed to show the mechanism of SCD-1 and MUFAs in protecting against oxidative stress.

In conclusion, this study demonstrated differences in fatty acid profiles and metabolism in the liver and subcutaneous adipose tissues. After overfeeding, the experimental and control groups had similar fatty acid profiles in the subcutaneous adipose tissue. However, an increase in hepatic C18:1 levels in the experimental group and relationships among BW, fBMI, BCS, plasma NEFA concentration and hepatic SCD-1 indices were demonstrated. The increase in hepatic SCD-1 indices after overfeeding might be enhanced by the increased inflow of plasma NEFA into the liver in cats.

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