

Effect of dietary protein quality and essential fatty acids on fatty acid composition in the liver and adipose tissue after rapid weight loss in overweight cats

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Objective—To examine effects of dietary protein quality (casein [CA] vs corn gluten [CG]) and dietary lipids (corn oil [CO] vs oil blend [OB] rich in long-chain polyunsaturated fatty acids [LCPUFAs]) on fatty acid composition in liver and adipose tissue after weight loss in overweight cats.

Animals—24 ovariohysterectomized adult cats.

Procedure—Cats were allowed ad libitum access to a high-quality diet until they weighed 30% more than their ideal body weight. Cats were then randomly assigned to 1 of 4 weight-reduction diets (6 cats/diet) and were fed 25% of maintenance energy requirements per day. Diets consisted of CG-CO, CA-CO, CG-OB, and CA-OB, respectively, and were fed until cats lost weight and returned to their original lean body mass. Liver biopsy specimens and samples of perirenal, subcutaneous, and abdominal fat were obtained and analyzed for fatty acid content.

Results—Following weight loss, fatty acid composition of the liver and adipose tissue was primarily affected by protein quality in that cats fed CA had significantly higher percentages of 20:4(n-6) and 22:6(n-3) fatty acids than those fed CG. Cats fed the CG-CO diet had the lowest concentrations of LCPUFAs, suggesting that dietary lipids and protein quality each influence fatty acid composition in tissues.

Conclusions and Clinical Relevance—These data provide direct evidence that dietary protein quality alters fatty acid composition of tissues during weight loss in cats. The fatty acid patterns observed suggest that protein quality may alter fatty acid composition through modulation of desaturase activity. (*Am J Vet Res* 2003;64:310–315)

A large number of nutritional idiosyncrasies in cats can be regarded as evolutionary adaptations to a carnivorous diet and may, in particular, influence fatty acid metabolism in the liver and the requirements for essential fatty acids (EFAs) in cats. Cats fed purified

diets containing vegetable oils that provided EFAs only as linoleate or as a mixture of linoleate (18:2[n-6]) and α -linolenate (18:3[n-3]) developed clinical signs compatible with EFA deficiency.¹ Analysis of plasma phospholipid concentrations in those cats revealed that they all had extremely low amounts of long-chain polyunsaturated fatty acids (LCPUFAs). It was concluded in that study and elsewhere² that cats fed 18:2(n-6) or 18:3(n-3) become deficient in EFAs, because they lack the ability to convert dietary EFAs into LCPUFAs. It has been postulated that this was attributable to a lack of Δ 6- and Δ 8-desaturases. It also has been suggested that cats lack the Δ 5-desaturase enzyme.³⁻⁵ Therefore, it has been implied that cats require preformed LCPUFAs, such as arachidonic, eicosapentaenoic and docosahexaenoic fatty acids, in their diet. However, the investigators have not successfully duplicated these earlier experiments. Cats maintained up to 8 years on diets that contained safflower seed oil (linoleate) as the sole source of dietary lipid appeared healthy but had problems with reproduction (≤ 2 viable litters) and had hepatic lipidosis. Differences in other dietary constituents, such as taurine concentrations in the diet or the amount of hydrogenated coconut oil in the diet, may have accounted for the differences in these aforementioned studies.

More recently, it has been reported⁶ that both Δ 5- and Δ 6-desaturase are active in cats, but the amount of activity does not appear to be adequate for maintaining tissue stores of LCPUFAs. Linoleate and α -linolenate are desaturated and elongated to LCPUFAs in low concentrations. In 1 report,³ cats fed linoleate-rich diets for 10 months had a marked decrease in arachidonate and an increase in 18:2(n-6) and 20:2(n-6), along with a 6-fold increase in 20:3(n-6) (ie, 5,11,14-20:3). Most likely, this fatty acid was produced from the elongation of linoleic acid and a subsequent Δ 5-desaturase. In other tissues of cats fed an EFA-deficient diet, an unknown fatty acid was detected, isolated, and characterized as 5,8,11-20:3 (20:3[n-9]), which is a classic indicator of EFA deficiency.⁷

Deficiency of EFAs induces fatty livers in cats⁸ and other animals.^{7,9} Deficiency of EFAs also affects lipoprotein lipase, lipoprotein transport from the liver, lecithin cholesterol acyltransferase, and fatty acid synthetase activities¹⁰; alterations of any of these may contribute to the development of hepatic lipidosis in cats. On the basis of reported observations, it is plausible that cats during weight loss are more susceptible to developing LCPUFA deficiency, which may be associ-

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ated with accumulation of lipids in the liver and the pathogenesis of hepatic lipidosis.

In cats, $\Delta 6$ -desaturase is minimally active.⁶ Dietary protein influences $\Delta 6$ -desaturase activity. In growing rats, plant protein reduces desaturase activity, whereas casein (CA) increases it.¹¹ In another study,¹² there was significantly higher $\Delta 6$ -desaturase activity in rats fed CA, compared with activity in those fed soy. Cats that develop hepatic lipidosis have decreased concentrations of long-chain fatty acids following long-term starvation,¹³ and feeding a high-quality protein partially ameliorates hepatic lipidosis in cats.¹⁴

The study reported here is part of a larger set of experiments that was conducted to determine the role of protein quality and LCPUFAs in the development or prevention of hepatic lipidosis in cats. Data from the experiments have been reported elsewhere.^{15,16} In the study reported here, we specifically attempted to determine the effects of feeding high-quality protein on maintenance of the LCPUFA composition of storage tissues during weight loss and whether feeding LCPUFAs such as arachidonic, eicosapentaenoic, and docosahexaenoic acids will bypass the rate-limiting $\Delta 6$ -desaturase step and increase the content of these fatty acids in tissues of cats undergoing weight reduction.

Materials and Methods

Animals—Twenty-four domestic shorthair female cats between 2 and 5 years of age were procured from a commercial vendor,^a as described for other studies^{15,16} conducted by our laboratory group. Cats were housed separately in an Association for Assessment and Accreditation of Laboratory Animal Care International-accredited facility and maintained on a 12-hours light:12-hours dark cycle (mean room temperature, 25°C). Cats were allowed 1 week to acclimate to the environment. Body weight of each cat was recorded, and cats then were anesthetized by administration of ketamine hydrochloride, acepromazine maleate, and isoflurane and ovariectomized. Blood samples (10 mL) were obtained 2 days prior to ovariectomy, and a wedge liver biopsy specimen (approx 1 g) was obtained during that surgery. All procedures were carefully monitored to assure that the cats had minimal amounts of pain. All surgical procedures and the experimental protocol were performed in accordance with established guidelines¹⁷ and approved by an institutional animal care and use committee.

Prior to surgery, cats were provided ad libitum access to water and a commercial diet.^b After surgery, cats were provided ad libitum access to water and a high-quality diet that consisted of a combination of 2 commercially available preparations.^{c,d} Food was provided ad libitum, because it was believed that this feeding practice represented the feeding practice used in most households. Cats were fed the high-quality diet until they weighed a minimum of 30% more than their ideal weight (ideal weight was considered 2.5 to 2.9 kg as determined on the basis of information provided by the vendor). Once the cats attained a minimum weight gain of 30% more than their ideal weight, they were randomly assigned to 1 of 4 weight-reduction treatment diets (6 cats/treatment diet) in staggered intervals (4 cats in each treatment diet each week). Diets consisted of Casein (CA), oil blend (OB), corn oil (CO), and corn gluten meal (CG; Appendices 1 and 2). The weight reduction

protocol was similar to that for weight loss programs in humans that use low-calorie dietary intakes of approximately 25% of normal caloric intake. Cats were maintained on the various weight-reduction diets until they achieved a body weight similar to that of their starting body weight. All of the experimental diets were formulated as described elsewhere.¹⁵

Protein efficiency ratios (grams of weight gain per grams of protein intake) of the diets fed to the cats during the weight-reduction period were determined from data obtained in chickens. Values for each diet were as follows: CA–OB, 2.3; CA–CO, 3.0; CG–OB, 1.1; and CG–CO, 1.4. Lysine and tryptophan are limiting amino acids in CG.

Collection of data—Body weight of each cat was recorded weekly. As described in another study¹⁵ conducted by our laboratory group, liver biopsy specimens were obtained during ovariectomy (ie, baseline), after cats attained a minimum of 30% more than their ideal weight (ie, obese), and after cats lost weight and returned to their body weight at the start of the study. Cats were anesthetized for the ovariectomy and each biopsy procedure. Samples of subcutaneous, perirenal, and abdominal fat were collected at the time of each liver biopsy procedure.

Analysis of fatty acid composition of liver and adipose tissue—All samples were spiked with a 15:0 fatty acid internal standard. Samples then were homogenized, and lipids were extracted by use of hexane:isopropanol (3:2 [vol:vol]) containing 0.05% butylated hydroxy-toluene.¹⁸ Total lipids were transmethylated by use of 14% boron trifluoride-methanol. Methylated samples were injected into a gas chromatograph^c equipped with a flame ionization detector and a capillary column¹ (length, 30 m; inside diameter, 0.32 × 0.25 mm). Nitrogen flow was maintained at 1.0 mL/min by use of an electronic pressure control. Temperature program for the column was an initial temperature of 160°C that was increased at a rate of 2°C/min up to 250°C and then increased at a rate of 5°C/min up to 310°C, which was maintained for 10 minutes. Peaks were identified on the basis of standards (ie, fatty acids) included with each batch of samples. Peaks were further verified by comparison with standards on a 10-m capillary column⁸ (inside diameter, 0.2 mm; film thickness, 0.33 μ m).

Statistical analysis—Data were analyzed by use of a 2-way analysis of covariance (the covariate was obesity) followed by analysis of least-squares means to assess significant differences among treatment groups. Differences between means were considered significant at $P \leq 0.05$.

Results

Changes in body weight—Cats readily consumed all food provided to them and, in general, appeared to be healthy throughout the study. After ovariectomy, cats gained approximately 4.7 g/d for 80 days. Cats did not gain additional weight between days 80 and 108 after surgery.

Cats appeared lively and playful and did not have signs of discomfort during weight loss. Irrespective of the diet fed to induce weight loss, all cats lost weight at a comparable rate (4.51 to 5.00 g/d/kg) averaged over the entire weight loss period; the greatest rate of weight loss was detected during the first week of feeding the treatment diets. We did not detect significant differences in weight loss between treatment diets. As has been reported elsewhere,¹⁵ obese cats lost about 7 to 10% of their body weight during the first week, 3 to

5% during the second week, and 2 to 4% during each week thereafter for the remainder of the weight loss period.

Hepatic lipidosis—As reported elsewhere,¹⁵ 3 cats developed hepatic lipidosis. These cats had sub-clinical hepatic lipidosis as determined on the basis of liver lipid scores of 5 to 6 (scale, 1 to 6), which were derived from histologic examination of sections of liver; these scores indicated lipid accumulation and severe lipidosis. In addition, gravimetric measurements of lipid extracts from wedge biopsy specimens were indicative of hepatic lipidosis. Furthermore, many of the lipid droplets in the liver of each of these 3 cats with hepatic lipidosis were heterogeneous in size, electron-dense, contained inclusion bodies, and had fine structural changes

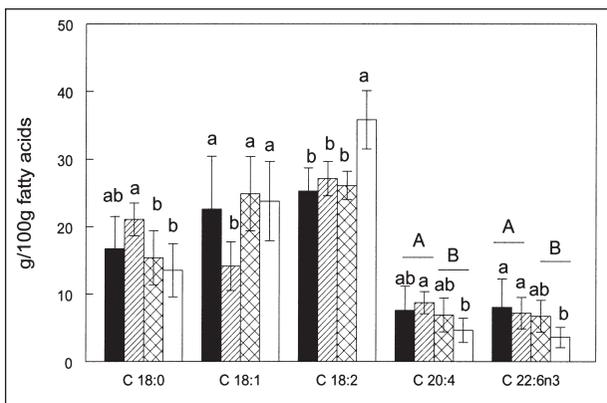


Figure 1—Mean \pm SD values for fatty acid composition of liver biopsy specimens obtained after rapid weight loss from cats on various weight-reduction diets. Overweight cats were randomly assigned to receive 1 of 4 weight-reduction diets: casein and oil blend (CA-OB; black bars), casein and corn oil (CA-CO; diagonal striped bars), corn gluten meal and oil blend (CG-OB; cross-hatched bars), and corn gluten meal and corn oil (CG-CO; white bars). Cats were maintained on the diets until they lost at least 30% of their body weight. ^{ab}Bars with different letters differ significantly ($P < 0.05$) between treatment diets. ^{AB}Values with different letters indicate significant ($P < 0.05$) differences between casein and corn gluten meal.

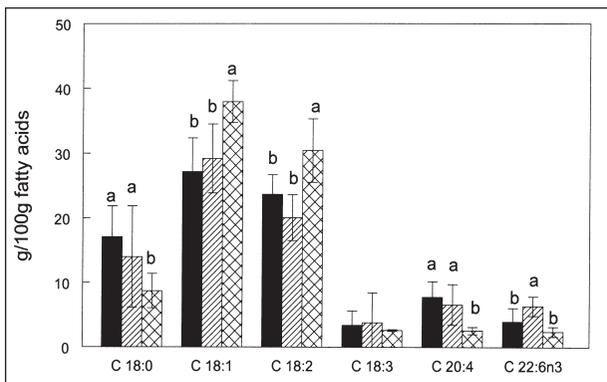


Figure 2—Mean \pm SD values for fatty acid composition of liver biopsy specimens obtained from cats at time of ovariectomy (baseline; black bars) and after cats gained weight and attained a minimum of 30% more than their ideal weight (overweight; diagonal striped bars) as well as specimens obtained from 3 cats that developed hepatic lipidosis (cross-hatched bars). ^{ab}For each fatty acid, bars with different letters differ significantly ($P < 0.05$).

suggestive of an alteration in lipid and protein metabolism.

Fatty acid composition of liver—Following weight loss resulting from consumption of the various dietary regimens, there were significant differences for several of the fatty acids in hepatic tissues (Fig 1). Although dietary protein significantly affected hepatic content of 20:4(n-6) and 22:6(n-3), dietary lipid did not have a significant effect. Cats fed CA had significantly higher concentrations of 20:4(n-6) and 22:6(n-3), compared with concentrations in cats fed CG. Concentrations of arachidonic acid (20:4[n-6]) and docosahexaenoic acid (22:6[n-3]) were significantly lower in cats fed the CG-CO diet, compared with concentrations in cats fed the CA-CO diet. Also, docosahexaenoic acid concentration was significantly lower in cats fed the CG-CO diet, compared with concentrations for cats fed the CA-OB diet. The percentage of 18:0 was significantly higher in cats fed the CA-CO diet, compared with values for cats fed CG-OB or CG-CO diets. Concentration of

Table 1—Fatty acid composition of subcutaneous adipose tissues after weight reduction in overweight cats consuming various weight-reduction diets*

Fatty acid	CA-OB	CG-OB	CA-CO	CG-CO
C14:0	0.38 \pm 0.15	0.36 \pm 0.19	0.27 \pm 0.09	0.39 \pm 0.11
C16:0	13.06 \pm 1.18	13.90 \pm 1.93	12.78 \pm 1.12	13.78 \pm 1.41
C16:1	1.68 \pm 0.06	1.91 \pm 0.75	1.46 \pm 0.26	1.62 \pm 0.43
C18:0	10.62 \pm 0.34	10.22 \pm 1.44	10.29 \pm 0.75	10.42 \pm 1.02
C18:1(n-9)	47.60 \pm 0.30	49.42 \pm 2.17	48.34 \pm 0.84	47.79 \pm 1.87
C18:2(n-6)	16.30 \pm 0.37 ^a	15.90 \pm 0.40 ^b	17.03 \pm 0.71 ^a	18.2 \pm 1.09 ^a
C18:3(n-3)	2.61 \pm 0.20	2.57 \pm 0.14	2.46 \pm 0.08	2.69 \pm 0.20
C20:4(n-6)	0.31 \pm 0.08 ^a	0.03 \pm 0.07 ^b	0.16 \pm 0.15 ^c	0.09 \pm 0.13 ^{ab}
C20:0	0.30 \pm 0.07	0.28 \pm 0.23	0.35 \pm 0.06	0.28 \pm 0.09
C20:1	1.81 \pm 0.21	1.49 \pm 0.82	2.08 \pm 0.48	1.60 \pm 0.31
C22:5(n-3)	0.61 \pm 0.17 ^a	0.15 \pm 0.24 ^b	0.42 \pm 0.25 ^c	0.02 \pm 0.05 ^b
C22:6(n-3)	0.86 \pm 0.22 ^a	0.20 \pm 0.24 ^b	0.55 \pm 0.29 ^a	0.22 \pm 0.23 ^b
C22:1	0.55 \pm 0.45	0.57 \pm 0.37	0.62 \pm 0.30	0.47 \pm 0.04

Values reported represent mean \pm SD number of grams per 100 grams of fatty acids.

*Overweight cats were fed diets at a rate of approximately 25% of normal caloric intake until they lost at least 30% of their body weight.

^{ab}Within a row, values with different superscript letters differ significantly ($P < 0.05$). CG-CO = Corn gluten meal-corn oil.

CG-OB = Corn gluten meal-oil blend. CA-CO = Casein-corn oil. CA-OB = Casein-oil blend.

Table 2—Fatty acid composition of abdominal adipose tissues after weight reduction in overweight cats consuming various weight-reduction diets*

Fatty acid	CA-OB	CG-OB	CA-CO	CG-CO
C14:0	0.24 \pm 0.06	0.31 \pm 0.04	0.26 \pm 0.06	0.27 \pm 0.06
C16:0	12.97 \pm 0.98	14.20 \pm 1.12	14.00 \pm 0.53	13.51 \pm 1.71
C16:1	1.45 \pm 0.20	1.90 \pm 0.48	1.71 \pm 0.28	1.49 \pm 0.29
C18:0	10.71 \pm 1.07	10.16 \pm 0.77	9.89 \pm 0.47	9.42 \pm 1.10
C18:1	47.68 \pm 1.41	48.68 \pm 1.15	47.3 \pm 1.41	49.98 \pm 3.46
C18:2(n-6)	16.77 \pm 0.27	16.72 \pm 0.72	17.86 \pm 1.02	18.31 \pm 1.75
C18:3(n-3)	2.61 \pm 0.18	2.59 \pm 0.18	2.45 \pm 0.22	2.44 \pm 0.21
C20:4(n-6)	0.20 \pm 0.24	0.15 \pm 0.16	0.22 \pm 0.28	0.06 \pm 0.14
C20:0	0.24 \pm 0.16 ^b	0.25 \pm 0.13 ^b	1.70 \pm 0.19 ^a	0.23 \pm 0.13 ^b
C20:1	2.06 \pm 0.39	1.60 \pm 0.25	1.39 \pm 0.18	1.48 \pm 0.20
C22:5(n-3)	0.61 \pm 0.35 ^b	0.22 \pm 0.22 ^{ab}	0.43 \pm 0.16 ^b	0.10 \pm 0.12 ^a
C22:6(n-3)	0.77 \pm 0.20 ^a	0.38 \pm 0.23 ^{ab}	0.30 \pm 0.08 ^{bc}	0.19 \pm 0.25 ^c
C22:1	0.82 \pm 0.39	0.53 \pm 0.11	0.47 \pm 0.35	0.53 \pm 0.21

Values reported represent mean \pm SD number of grams per 100 grams of fatty acids. ^{ab}Within a row, values with different superscript letters differ significantly ($P < 0.05$). See Table 1 for key.

Table 3—Fatty acid composition of perirenal adipose tissues after weight reduction in overweight cats consuming various weight-reduction diets*

Fatty acid	CA-OB	CG-OB	CA-CO	CG-CO
C14:0	0.34 ± 0.29	0.43 ± 0.07	0.40 ± 0.07	0.52 ± 0.0
C16:0	14.80 ± 1.77	15.98 ± 1.89	15.35 ± 0.50	17.63 ± 1.77
C16:1	1.62 ± 0.53	1.96 ± 0.39	1.79 ± 0.22	2.34 ± 0.43
C18:0	10.44 ± 1.25	10.16 ± 0.76	9.56 ± 0.18	9.14 ± 1.40
C18:1(n-9)	47.10 ± 0.66	48.67 ± 2.09	46.87 ± 0.93	45.20 ± 0.43
C18:2(n-6)	17.05 ± 1.30 ^{ab}	16.38 ± 0.73 ^b	17.42 ± 0.85 ^{ab}	18.84 ± 0.31 ^a
C18:3(n-3)	2.34 ± 0.20	2.09 ± 0.22	2.30 ± 0.12	2.46 ± 0.30
C20:4(n-6)	ND	ND	0.05 ± 0.11	ND
C20:0	0.13 ± 0.23	ND	ND	ND
C20:1	2.17 ± 0.47	1.71 ± 0.99	1.96 ± 0.29	1.89 ± 0.74
C22:5(n-3)	ND	0.06 ± 0.13	0.25 ± 0.35	ND
C22:6(n-3)	0.57 ± 0.32 ^a	0.09 ± 0.20 ^b	0.34 ± 0.34 ^a	ND
C22:1	1.14 ± 0.62	0.93 ± 0.73	1.00 ± 0.27	0.60 ± 0.09

Values reported represent mean ± SD number of grams per 100 grams of fatty acids.
 ND = Not detectable.
 See Table 1 for remainder of key.

oleic acid (18:1) was significantly decreased in cats fed the CA-CO diet, compared with concentrations for cats fed all other diets. Concentration of 18:2(n-6) was significantly greater for cats fed the CG-CO diet, compared with concentrations for cats fed all other diets.

Weight gain, weight loss, and hepatic lipidosis in cats significantly affected fatty acid composition of the liver (Fig 2). The only fatty acid whose concentration differed significantly between baseline and obese in these cats was 22:6(n-3), which was significantly greater in cats that were obese. However, in cats that developed hepatic lipidosis, concentrations of 18:0, 20:4(n-6), and 22:6(n-3) were significantly lower than concentrations in cats at baseline or obese stages. Additionally, concentrations of 18:1 and 18:2(n-6) were significantly greater in cats with hepatic lipidosis than in cats at baseline or obese stages.

Fatty acid composition of adipose tissue—Dietary protein significantly affected concentrations of LCPUFAs in adipose tissue. The percentage of 20:4(n-6), 22:5(n-3), and 22:6(n-3) was significantly lower in the subcutaneous fat stores of cats fed CG than in cats fed CA (Table 1). Similar trends were evident for fatty acid composition of abdominal and perirenal adipose tissues (Tables 2 and 3). Also, dietary lipid significantly affected concentrations of 22:6(n-3) in the abdominal and perirenal fat stores. The percentage of 22:6(n-3) was significantly higher in the abdominal and perirenal adipose tissues of cats fed OB than in cats fed CO. Compared to subcutaneous and abdominal adipose tissues, perirenal adipose tissue was nearly devoid of 20:4(n-6) and 22:5(n-3) fatty acids. In subcutaneous adipose tissue, the percentage of 18:2(n-6) was significantly higher in cats fed the CG-CO diet than in cats fed the CG-OB diet.

Discussion

In general, analysis of data for the study reported here revealed that concentrations of LCPUFAs in tissues of cats after weight loss were affected significant-

ly by dietary protein. Cats fed CA had significantly higher amounts of 20:4(n-6) and 22:6(n-3) in the liver, compared with the amounts in cats fed CG. Also, significant effects of dietary protein (ie, CA greater than CG) were detected for the percentage of arachidonic and docosahexaenoic acids in adipose tissue stores. Cats fed CG-CO had the lowest concentrations of LCPUFAs, suggesting that dietary lipids and dietary protein quality influence fatty acid composition of tissues. Interestingly, the adipose tissue stores differed in the percentage of LCPUFAs that they accumulated. For example, subcutaneous and abdominal adipose tissues contained substantial amounts of 20:4(n-6), whereas perirenal adipose tissue was practically devoid of that fatty acid. The reasons for these differences are unclear, but they may reflect more localized use of stored fatty acids after release. It has been reported¹⁹ that a diet high in CA increases the Δ 5- and Δ 6-desaturase enzymes in rats. In another study,²⁰ feeding a low-protein diet to lactating rats strongly decreased activity of Δ 5- and Δ 6-desaturase enzymes. Although data from rats cannot be extrapolated to other species, analysis of data from the study reported here suggests that the higher concentrations of LCPUFAs detected in liver and adipose tissues of cats fed CA, compared with CG, may be related to alterations in activity of desaturase enzymes. However, activity of desaturase enzymes was not directly measured in this study, and further investigations will be needed to determine whether activity of desaturase enzymes is directly altered. As mentioned previously, Δ 6-desaturase activity is low or totally lacking in cats.^{5,6}

Availability of LCPUFAs contained in the OB diets, compared with the CO diets, also revealed that the availability of these fatty acids influences tissue stores. Although dietary LCPUFAs enhanced the amount of these fatty acids in the tissues assessed, significant differences were only detected for 22:6(n-3) in the abdominal and perirenal fat stores. Although 5,8,11-20:3(n-9) has reportedly been identified in cats,² this fatty acid appeared only when an EFA-deficient diet (ie, 18:2[n-6], 18:3[n-3]) was fed. Our weight-reduction diets contained 25 to 63% of the total fatty acids in the form of 18:2(n-6); therefore, inhibition of 18:1(n-9) desaturation most likely occurred and, thus, prevented the accumulation of the 5,8,11-20:3(n-9) product.

Hepatic lipidosis is a disease that often affects obese cats that have been placed on a weight-reduction diet. Only by understanding the pathogenic mechanisms involved can we hope to prevent or cure the disease. For reasons that remain unclear, lipids accumulate in the liver of affected cats, frequently leading to the death of the animal. Several mechanisms could alter this buildup of fat, such as an increase in triglyceride synthesis, a decrease in fatty acid oxidation, or a decrease in lipid transport (ie, very-low-density lipoprotein [VLDL]) from the liver. In 1 study,¹⁶ neither increased triglyceride synthesis nor decreased VLDL transport from the liver was altered in cats undergoing weight loss.

Because the LCPUFAs are significantly reduced in

cats with hepatic lipidosis,¹³ the lack of LCPUFAs as a result of their availability or decreased synthesis (which is unlikely, because the cats are in a catabolic state) may contribute to the pathogenesis of hepatic lipidosis. We speculated that deficiency of essential LCPUFAs, which may develop during weight loss in cats, may contribute to the pathogenesis of hepatic lipidosis. The 20:4(n-6) and 22:6(n-3) are required as components of specific phospholipids for formation of intracellular organelle membranes. Alteration of the formation of mitochondrial or peroxisomal membranes may result in dysfunctional β -oxidation of fatty acids in peroxisomes and mitochondria. In cats with hepatic lipidosis, liver peroxisomes are greatly diminished or totally lacking^{15,21}; the decrease in peroxisome numbers appears to be related to the duration of the withholding of food.²² Furthermore, mitochondrial alterations have been documented in cats with hepatic lipidosis.²¹ Although increased accumulation of very-long-chain saturated and monounsaturated fatty acids could be predicted for cats with hepatic lipidosis as a result of this apparent peroxisomal defect, the minimal adipose stores of these very-long-chain fatty acids observed in the cats fed the various weight-reduction diets in our study (Tables 1–3) and data reported in another study,¹³ that suggest that these fatty acids do not accumulate during rapid weight loss, probably because they are in negligible amounts, not mobilized from adipose stores, or not synthesized. Although it seems unlikely, another possibility is that the number of peroxisomes and mitochondrial aberrations detected in cats with hepatic lipidosis does not markedly alter β -oxidation. Additional studies are needed to elucidate the mechanisms involved.

In the study reported here, fatty acid composition of tissues during weight loss in cats was primarily affected by dietary protein quality. Cats consuming CA had significantly higher percentages of 20:4(n-6) and 22:6(n-3), compared with percentages in cats consuming CG. It was apparent that the most substantial decreases were in cats fed the CG–CO diet, suggesting that lack of LCPUFAs coupled with protein quality influence tissue composition of fatty acids. Although we did not directly measure the activity of Δ 5- or Δ 6-desaturase, the effect of protein quality on tissue composition of fatty acids suggests altered activity of Δ 5- or Δ 6-desaturase or both. Because concentrations of LCPUFAs are significantly reduced in cats with hepatic lipidosis, the lack of LCPUFAs as a result of their availability or decreased synthesis may contribute to the pathogenesis of hepatic lipidosis in cats. Therefore, it appears that dietary LCPUFAs and protein quality each play an important role in maintaining tissue stores of LCPUFAs during weight loss.

[†]HsdCpb:CADS, Harlan Sprague Dawley, Indianapolis, Ind.

[‡]Harlan Teklad Cat Diet, Harlan Teklad, Madison, Wis.

[§]Nutritional Recovery Formula, The Iams Co, Dayton, Ohio.

[¶]Ocean Fish Formula Cat Food, The Iams Co, Dayton, Ohio.

^{‡‡}Shimadzu GC 9A, Shimadzu American, Columbia, Md.

^{§§}DB-5ms, Supelco, St Louis, Mo.

^{¶¶}FSAP, Hewlett Packard, Palo Alto, Calif.

Appendix 1

Composition of diets fed to obese cats to induce weight reduction*

Ingredients	CA–OB	CG–OB	CA–CO	CG–CO
Corn gluten meal (%)	NI	68.30	NI	68.30
Casein (%)	52.00	NI	52.00	NI
Corn oil (%)	3.90	NI	18.70	13.00
Poultry fat (%)	13.50	11.70	NI	NI
Corn starch (%)	19.60	5.00	19.60	5.00
Calcium carbonate (%)	NI	3.70	0.02	3.70
Dried beet pulp (%)	3.00	3.00	3.00	3.00
Monosodium phosphate (%)	0.49	2.40	0.49	2.40
Choline chloride (%)	1.80	1.50	1.80	1.50
Minerals (%)†	1.20	1.20	1.20	1.20
Vitamins (%)‡	1.20	1.20	1.20	1.20
Sodium chloride (%)	1.00	NI	1.00	NI
D-L-methionine (%)	0.32	NI	0.32	NI
Fish oil (%)	0.90	0.90	NI	NI
Borage oil (%)	0.15	0.15	NI	NI
Ground flax (%)	0.15	0.15	NI	NI
Taurine (%)	0.15	0.15	0.15	0.15
Potassium chloride (%)	0.61	0.49	0.54	0.49
Fatty acids				
Saturated (% of total)	19.69	19.82	7.64	7.70
Polyunsaturated (% of total)	33.59	30.52	63.27	60.10
Monounsaturated (% of total)	45.47	46.05	27.89	27.05
20:4(n-6) (% of total)	0.29	0.28	NI	NI
22:6(n-3) (% of total)	0.87	1.11	NI	NI
Protein (16.7 kJ/g)	41.94	44.37	42.97	45.09
Moisture (%)	10.85	7.62	10.28	5.81
Ash (%)	7.06	7.98	7.36	7.87
Fat (37.6 kJ/g)	18.51	17.29	17.1	17.18
Crude fiber (%)	1.59	2.34	2.28	2.22
Nitrogen-free extracts (16.7 kJ/g)	19.97	20.4	19.79	21.84
Calculated energy (kJ/kg)	17,313	17,330	16,925	17,652

*Overweight cats were fed diets at a rate of approximately 25% of normal caloric intake until they lost at least 30% of their body weight. †Minerals were added to the weight-reduction diets in amounts that were at least 2 times the requirements established by the Association of American Feed Control Officials (AAFCO) within the following ranges: calcium, 1.43 to 1.64% on a dry-matter (DM) basis; phosphorus, 1.00 to 1.09% on a DM basis; magnesium, 688 to 709 mg/kg; copper, 36 to 47 mg/kg; iron, 78 to 201 mg/kg; manganese, 177 to 184 mg/kg; potassium, 7,740 to 9,065 mg/kg; sodium, 5,017 to 6,184 mg/kg; and zinc, 688 to 755 mg/kg. ‡Vitamins were added to the weight-reduction diets in amounts that were at least 4 times the requirements established by AAFCO within the following ranges: vitamin A, 1,442,000 to 1,448,000 IU/kg; vitamin D, 9,232 to 9,268 IU/kg; vitamin E, 601 to 653 IU/kg; thiamine, 89 to 90 mg/kg; riboflavin, 24 to 25 mg/kg; pantothenic acid, 104 to 105 mg/kg; niacin, 348 to 403 mg/kg; pyridoxine, 76.7 to 76.8 mg/kg; folic acid, 10 to 162 mg/kg; biotin, 2.2 to 2.3 mg/kg; vitamin B₁₂, 0.75 mg/kg; and choline, 9,537 to 9,561 mg/kg. CA–OB = Casein–oil blend. CG–OB = Corn gluten meal–oil blend. CA–CO = Casein–corn oil. CG–CO = Corn gluten meal–corn oil. NI = Not included.

Appendix 2

Fatty acid composition* of diets fed to cats during weight gain and weight loss

Fatty acid	Weight gain		Weight loss			
	Recovery diet ^c	Ocean fish diet ^d	CA–OB	CG–OB	CA–CO	CG–CO
C16:0	14.25	20.15	12.11	13.16	4.76	4.73
C16:1	3.29	4.38	2.50	2.91	0.00	0.00
C18:0	7.90	9.72	6.34	6.66	2.24	2.36
C18:1(n-9)	46.15	36.07	43.35	43.85	28.62	28.21
C18:2(n-6)	19.08	13.56	29.85	25.87	63.62	63.75
C18:3(n-3)	3.14	2.13	2.20	3.31	0.00	0.00
C20:4(n-6)	1.11	1.84	0.28	0.29	0.00	0.00
C20:5(n-3)	1.46	3.18	0.95	1.06	0.00	0.00
C20:0	0.00	0.00	0.25	0.13	0.55	0.64
C20:1	1.08	4.01	0.69	0.85	0.21	0.31
C22:5(n-3)	0.67	0.00	0.38	0.39	0.00	0.00
C22:6(n-3)	1.87	4.94	0.87	1.11	0.00	0.00

*Values reported represent the number of grams per 100 grams of fatty acids. See Appendix 1 for key.

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