Effects of Linoleate and Arachidonate Deficiencies on Reproduction and Spermatogenesis in the Cat[1]

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ABSTRACT The inability of the cat to convert significant quantities of linoleate [18:2(9,12)] to arachidonate [20:4(5,8,11,14)] in the liver makes the cat a useful model for studying the specific physiological roles of these two fatty acids. In these studies, cats were fed purified diets that were either deficient in essential fatty acids (EFAs) or that provided linoleate with or without arachidonate. Male cats that were fed the EFA-deficient diet for approximately 2 years exhibited extensive degeneration of the testes, and the fatty acid composition of testes changed in a manner consistent with EFA deficiency. Linoleate prevented testis degeneration. Levels of arachidonate, 22:4n6, and 22:5n6 were higher in testis phospholipids of cats supplied with linoleate than in the deficient cats, indicating that the testis of the cat has the capacity to desaturate and elongate linoleate. In contrast, female cats that were fed diets lacking arachidonate were unable to bear live kittens, whether linoleate was provided in the diet or not. Arachidonate, supplied by oral supplements of ethyl arachidonate or by animal fat in the diet, significantly improved reproduction. Thus, linoleate appears to meet the requirements for spermatogenesis in males, but dietary arachidonate is essential for adequate reproduction in female cats. J. Nutr. *114*: 719–726, 1984.

INDEXING KEY WORDS linoleate • arachidonate • essential fatty acids • reproduction • testis • cat

The EFA requirement of most mammals can be met by either linoleate or arachidonate (1, 2). In the liver, linoleate is converted to arachidonate by alternating desaturation and elongation (3). These two observations have led some authors to propose that arachidonate is the physiologically essential fatty acid, and that linoleate is simply a readily available precursor.

In cat liver, there is negligible conversion of linoleate to arachidonate (4, 5). Nevertheless, linoleate prevents several signs of EFA deficiency, including scaly skin, increased transepidermal water loss and enlarged fatty livers (5). Thus, linoleate has a specific role as an essential fatty acid, independent of arachidonate synthesis.

The requirements of the cat for essential fatty acids for reproduction have not been assessed previously. Rivers and co-workers (6, 7) reported reproductive failure in female cats that were fed purified diets containing 25% safflower seed oil, which provided a high level of linoleate but no arachidonate. However, they did not show whether the failure to reproduce was a result of a deficiency of essential fatty acids or of some other nutrient in the purified diet. McLean (8) reported adequate reproduction in cats fed a diet containing 5%

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safflower seed oil for 3 years, but the composition of the diet was not published.

The purpose of the present study was to determine whether linoleate meets the EFA requirements of the cat for reproduction and spermatogenesis. Our results show that linoleate may meet the requirement for spermatogenesis in male cats, but that dietary arachidonate is essential for adequate reproduction in female cats.

METHODS

Specific pathogen–free cats were housed as described previously (5). To determine the effects of linoleate deficiency on spermatogenesis, male cats were fed purified diets containing different sources of fat (by weight) as follows: EFAD, 35% hydrogenated beef tallow; SSO, 5% safflower seed oil + 30% hydrogenated beef tallow; SSO/TO, 5% safflower seed oil + 0.2% tuna oil + 29.8% hydrogenated beef tallow; and SSO/CHF, 5% safflower seed oil + 5% chicken fat + 15% hydrogenated coconut oil. Reproductive performance was assessed in female cats fed either the EFAD, SSO, SSO/TO or SSO/CHF diets or a diet (SSO/TLW) containing 2.5% safflower seed oil + 25% unbleached tallow (Florin Tallow Co., Dixon, CA).

The fatty acid compositions of hydrogenated beef tallow, safflower seed oil and tuna oil were given previously (5). The fatty acid composition of chicken fat was (in percent of fatty acids by weight): 16:0, 18.9%; 16:1, 5.1%; 18:0, 5.1%; 18:1, 48.6%; 18:2, 19.8%; 18:3n3, 0.4%; 20:1n9, 0.4%; 20:2n6, 0.2%; and 20:4n6, 0.1%. The fatty acid composition of unbleached tallow was: 16:0, 26%; 16:1, 4.2%; 17:0, 1.7%; 18:0, 20.6%; 18:1, 41.3%; 18:2, 3.0%; 20:0, 0.1%; 18:3n3, 0.6%; 20:1n9, 0.7%; 21:0, 0.3%; 20:2n6, 0.2%; 20:3n6, 0.1%; 20:4n6, 0.1%; 22:1, 0.1%; 24:0, 0.04%; 22:4n6, 0.06%; 22:5n6, 0.04%; 22:5n3, 0.1%; and 22:6n3, 0.1%. The experimental diets provided different levels of dietary linoleate and arachidonate as shown in table 1.

All the diets met or exceeded the NRC requirements for protein, vitamins and minerals (9). The composition of the EFAD, SSO and SSO/TO diets was described previously (5); when the reproduction trials were begun, at the third year of the study, the level of vitamin-free casein was increased to 40% by weight. The SSO/CHF diet contained 27% vitamin-free casein, 0.5% L-methionine, 1% L-arginine hydrochloride, 5% mineral mix, 1.3% vitamin mix, 0.38% sodium acetate, 20% sucrose, 10.2% cerelose and 10% cornstarch. The SSO/TLW diet contained 36% isolated soy protein (Procon, A. E. Staley Mfg. Co., Decatur, IL), 10% vitamin-free casein, 0.5% L-methionine, 5% mineral mix, 1.2% vitamin mix, 10% sucrose and 9.5% cornstarch. The compositions of the vitamin and mineral mixes were described previously (5).

TABLE 1

Levels of linoleate and arachidonate in the experimental diets[1]

Diet	Linoleate	Arachidonate
	% of dietary energy	
EFAD	0.3	0
SSO	6.7	0
SSO/TO	6.7	0.01
SSO/CHF	8.9	0.01
SSO/TLW	4.8	0.04

[1]EFAD, 35% hydrogenated beef tallow; SSO, 5% safflower seed oil + 30% hydrogenated beef tallow; SSO/TO, 5% safflower seed oil + 0.2% tuna oil + 29.8% hydrogenated beef tallow; SSO/CHF, 5% safflower seed oil + 5% chicken fat + 15% hydrogenated coconut oil; SSO/TLW, 2.5% safflower seed oil + 25% unbleached tallow.

Male cats fed the EFAD, SSO, SSO/TO or SSO/CHF diets were castrated after approximately 2 years. Three cats, one fed the EFAD diet and two fed the SSO diet, had their right testis removed, and were then supplemented orally with ethyl arachidonate in gelatin capsules (100 mg/day, 5 times/week for 8 weeks). The left testis was removed from each supplemented cat 10 weeks after supplementation ended.

A portion of each testis was fixed in Zenker's formalin, sectioned transversely, and stained with hematoxylin/azure-eosinate for histological examination by light microscopy. For each testis, at least 100 tubules were classified into stages of the cell cycle, by using a classification described by Clermont (10). Partial degeneration of tubules was defined as the absence of one or

more groups of spermatogenic cells within the tubules. Complete degeneration was defined as the absence of all the spermatogenic cells within the tubules. The remaining portion of each testis was extracted twice with chloroform/methanol (1:1, vol/vol). Phospholipids were separated from total lipids by thin-layer chromatography, and fatty acids of total lipids and phospholipids were analyzed by gas-liquid chromatography as described previously (5).

Female cats that had been fed the experimental diets for 2 years were mated with a proven male breeder that had been fed a variety of commercial cat foods. Groups of females were kept with the male during alternate weeks for 4 months, or until pregnancy occurred. After parturition, the cats fed the SSO diet were supplemented with ethyl arachidonate (92% pure, Hoffmann-La Roche Inc., Nutley, NJ) to determine whether arachidonate would improve reproduction. The purity of the ethyl arachidonate was verified by gas-liquid chromatography. Arachidonate was given in oral supplements of 250 mg/day for 16 days. To determine the effect of arachidonate supplements on the fatty acid composition of plasma and erythrocyte lipids, blood samples were taken 4 weeks after the start of supplementation. Ethyl arachidonate was then incorporated into the diet at 0.03% (wt/wt) by mixing it with safflower seed oil before the diet was made. When estrus occurred, the supplemented cats were mated with a normal male. The last kittens were weaned 3 years after the study began.

RESULTS AND DISCUSSION

Fatty acid composition of testis lipids

The fatty acid composition of testis lipids is shown in table 2. Cat testes contained higher levels of 18:2n6 and 22:4n6, and lower levels of 20:4n6 and 22:5n6, than do the testes of the rat (11). The level of the n3 polyunsaturated fatty acids was low unless these were included in the diet (SSO/TO). The phospholipids of cat testes were enriched in 20:4n6 and 22:5n6 compared to total lipid, whereas the neutral lipids apparently were enriched in linoleate. A similar pattern occurs in the lipids of rat testes (12).

The testis fatty acids of the cats fed the EFAD diet showed changes, which were qualitatively, but not quantitatively similar to EFA deficiency in other species. The levels of 16:1, 18:1, 20:1n9 and 20:3(5,8,11) were significantly higher, and the levels of

TABLE 2

Fatty acid composition of testis phospholipid and total lipid from cats fed the experimental diets[1,2]

Fatty acid	Phospholipid				Pooled	Total lipid				Pooled
	EFAD	SSO	SSO/TO	SSO/CHF	SEM	EFAD	SSO	SSO/TO	SSO/CHF	SEM
16:0	24.9[a]	24.4[a]	24.5[a]	25.5[a]	0.42	22.5[a]	20.1[a]	19.5[a]	23.6[a]	0.83
16:1	2.49[a]	1.03[b]	1.20[b]	0.92[b]	0.18	4.77[a]	1.60[b]	1.75[b]	1.99[b]	0.48
18:0	11.8[a]	14.7[b]	14.1[ab]	13.0[ab]	0.49	14.2[a]	15.4[a]	16.1[a]	11.9[a]	0.59
18:1	21.4[a]	11.8[b]	11.5[b]	11.4[b]	1.2	31.0[a]	15.0[b]	15.2[b]	18.6[b]	2.0
18:2(9,12)	3.16[a]	9.40[b]	9.72[b]	11.9[b]	1.0	3.90[a]	17.8[b]	18.1[b]	21.8[b]	2.3
18:3(6,9,12)	0.10[a]	0.13[a]	0.09[a]	0.08[a]	0.02	0.03[a]	0.14[b]	0.08[ab]	0.05[a]	0.02
20:0	0.11[a]	0.17[a]	0.11[a]	0.11[a]	0.02	0.12[a]	0.21[b]	0.17[ab]	0.14[ab]	0.02
18:3(9,12,15)	ND	ND	ND	ND		0.08[a]	0.19[b]	0.03[a]	0.11[ab]	0.02
20:1(11)	1.13[a]	0.51[b]	0.52[b]	0.58[b]	0.08	1.93[a]	0.70[b]	0.68[b]	0.75[b]	0.18
20:2(11,14)	0.30[a]	1.61[b]	1.51[b]	1.79[b]	0.19	0.38[a]	2.15[a]	1.98[a]	1.89[a]	0.34
20:3(5,8,11)	4.28[a]	0.15[b]	0.33[b]	0.21[b]	0.56	2.04	ND	ND	ND	0.56
20:3(5,11,14)	0.56[a]	0.70[a]	0.49[a]	1.08[b]	0.07	0.29[a]	0.40[a]	0.27[a]	0.57[a]	0.06
20:3(8,11,14)	3.24[a]	3.79[ab]	4.40[b]	4.18[ab]	0.17	1.58[a]	3.40[a]	3.47[a]	2.24[a]	0.42
20:4(5,8,11,14)	10.6[a]	14.7[b]	14.7[b]	12.8[b]	0.5	4.82[a]	8.06[a]	7.63[a]	5.77[a]	0.94
22:4(7,10,13,16)	2.92[a]	4.62[b]	4.15[b]	3.75[ab]	0.23	4.72[a]	6.46[a]	6.27[a]	3.68[a]	0.92
22:5(4,7,10,13,16)	9.72[a]	11.5[a]	9.38[a]	12.0[a]	0.44	4.97[a]	7.39[a]	5.48[a]	6.24[a]	0.85
22:5(7,10,13,16,19)	0.34[a]	0.13[b]	0.30[a]	ND	0.03	0.43[a]	0.18[a]	0.42[a]	0.07[a]	0.06
22:6(4,7,10,13,16,19)	0.94[a]	0.18[a]	2.51[b]	0.15[a]	0.26	0.47[a]	0.24[a]	2.11[b]	0.42[a]	0.22

[1]Values are means. Number of cats in each group: EFAD, $n = 4$; SSO, $n = 6$; SSO/TO, $n = 3$; SSO/CHF, $n = 2$. Fatty acids are described as number of carbons:number of double bonds. Diet abbreviations, table 1, footnote 1; ND, not detectable. [2]Means not sharing a common superscript are significantly different, $P < 0.05$, by Duncan's multiple-range test.

18:2n6, 20:4, 22:4n6 and 22:5n6 were lower, than in cats fed a source of linoleate. The decreases in 20:4n6 and 22:5n6 were less marked in the EFA-deficient cat than in other species such as the rat (13), rabbit (14) or pig (15). This may be because of the presence of some linoleate in the EFAD diet. However, because the level of 18:2n6 in the testes of EFA-deficient cats was not higher than in some other EFA-deficient mammals (14, 15) the retention of these fatty acids may result from a difference in their metabolism in the cat.

When linoleate was included in the diet (SSO diet), the levels of linoleate, arachidonate, 20:2n6 and 22:4n6 in phospholipids and in total lipid were significantly higher than the levels of these fatty acids in cats fed the EFAD diet. The level of 22:5n6 was also higher in testis lipids of cats fed the SSO diet than in those fed the EFAD diet, although this difference was not statistically significant.

An unusual fatty acid, 20:3(5,11,14), was present at low levels in testis lipids. This fatty acid also occurs in liver, plasma and erythrocyte lipids of the cat (5, 16). The synthesis of 20:3(5,11,14) from 20:2(11,14) is evidence that cat testes have the capacity to desaturate at the Δ^5 position, as does cat liver (16).

The accumulation of arachidonate in the testis of cats provided with linoleate as the only EFA suggests that the testis has the capacity to synthesize arachidonate from linoleate. In other mammals, the testis has a full complement of enzymes for arachidonate synthesis (17), including a Δ^6 desaturase (18). Therefore, arachidonate could be synthesized from linoleate by one of two pathways: desaturation of 18:2 at the Δ^6 position to form 18:3(6,9,12), followed by elongation to 20:3(8,11,14) which is desaturated at the Δ^5 position to form arachidonate; or, elongation of linoleate to 20:2(11,14), which can then be desaturated at the Δ^8 position to form 20:3(8,11,14). Direct measurements of desaturase activities would be necessary in order to discern which of these pathways are active in cat testes.

In cats fed the SSO/TO diet, 22:6n3 appeared to quantitatively replace 22:5n6 in testis phospholipids, and testes from these cats showed extensive degeneration (discussed below). The level of 22:5n6 in the SSO/TO group was even lower than in the EFAD group. Thus, 22:5n6 may be an important fatty acid for testis function in the cat, as in many other mammals. In the rat, 22:5n6 accumulates in the testis at the time of maturation of the spermatids (19); the level of 22:5n6 is higher in spermatids than in spermatocytes or Sertoli cells (20, 21); and treatments that lower fertility always result in decreased levels of this fatty acid (22).

Effects of experimental diets on spermatogenesis in male cats

The effects of the experimental diets on spermatogenesis are given in table 3. In cats fed the linoleate-deficient diet (EFAD) for 2 years, 75% of the cats exhibited extensive degeneration of the testes and there were few sperm present in the epididymides (fig. 1). This effect of EFA deficiency also occurs in rats (23). In cats fed the EFAD diet, the distribution of tubules at various stages of the cell cycle was altered: fewer tubules were at stages II and III of the cycle, and more tubules were at stage VI compared to cats fed the diets containing linoleate. However, these differences were not statistically significant. In one cat that had its right testis removed, only Sertoli cells were present in 46% of the tubules. After this cat was supplemented with arachidonate (see methods section), the remaining testis showed marked regeneration. There were no completely degenerated tubules, and the number of partially degenerated tubules remained at 15%. In addition, the distribution of cell types shifted, such that 40% of the tubules were at stage III of the cycle. This suggests that arachidonate may be able to reverse the testis degeneration caused by EFA deficiency.

Compared to the cats fed the EFAD diet, spermatogenesis was significantly improved by the addition of safflower seed oil to the diet. In the SSO group, the testes appeared essentially normal at histological examination. Only one cat exhibited partial degeneration of more than 5% of the tubules, and none of the cats had completely degenerated tubules. The epididymides contained moderate to large numbers of

TABLE 3

Results of histological examination of testes from cats fed the experimental diets[1]

Diet	Distribution of tubules at different stages of the cycle						Incidence of degeneration[2]	Avg % of tubules degenerated		Description of epididymides
	I	II	III	IV	V	VI		Partial	Complete	
	% of total tubules counted						*% of cats*			
EFAD	33.2	13.0	13.0	9.9	20.8	10.1	75	8.8	11.6	Few sperm. Many spermatogenic cells in lumen. Some intraepithelial cysts.
SSO	28.8	19.5	16.6	12.4	16.7	6.1	17	5.6	0	Moderate to large numbers of sperm. Few immature cells.
SSO/TO	32.6	19.7	15.7	11.8	13.0	7.2	66	5.6	10.4	
SSO/CHF	37.4	23.1	19.6	7.9	7.2	4.4	0	2.0	0	Few intraepithelial cysts.
Pooled SEM	2.0	1.8	1.8	0.9	2.7	0.9	—	1.8	3.7	

[1]Number of cats in each group: EFAD, $n = 4$; SSO, $n = 6$; SSO/TO, $n = 3$; SSO/CHF, $n = 2$. Diet abbreviations, table 1, footnote 1. [2]Degeneration was counted when more than 5 out of 100 tubules were degenerated.

sperm. Two cats, in which 3–4% of the tubules in the right testis were partially degenerated, were supplemented with ethyl arachidonate and then fully castrated. None of the parameters changed with supplementation. We concluded that the partial degeneration of less than 5% of the tubules is not abnormal in cats of this age. Although the fertility of these cats was not tested, the fact that spermatogenesis was essentially normal in cats fed the SSO diet provides evidence that linoleate alone may meet the requirements for reproduction in male cats.

In cats fed the SSO/CHF diet, the testes were normal; there was no degeneration, and there were large numbers of sperm in

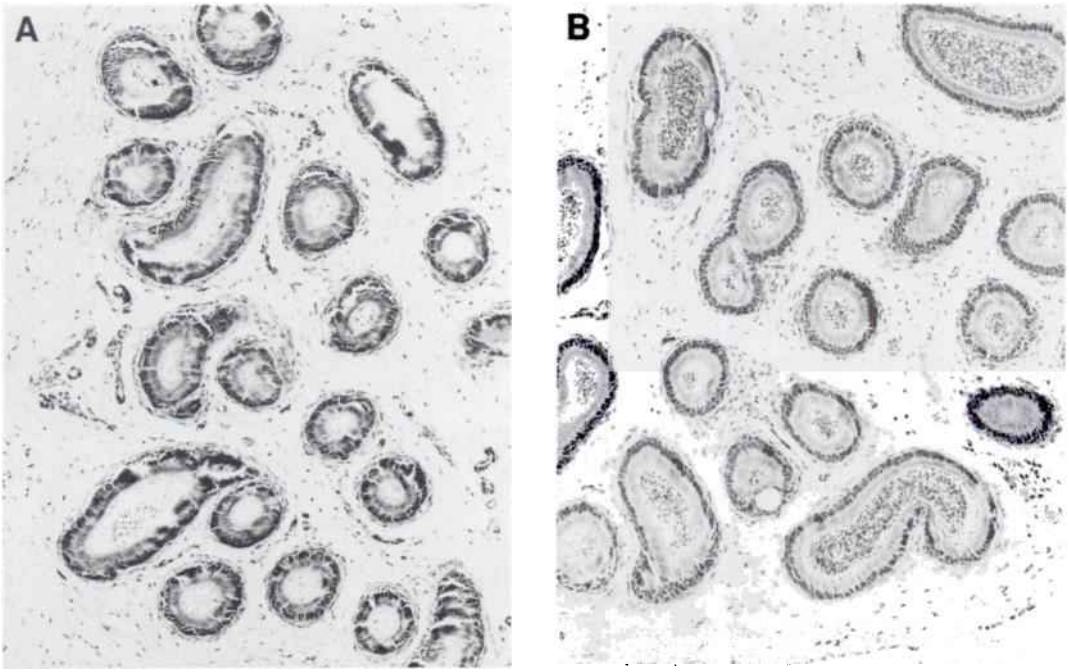

Fig. 1*A* Epididymis of a cat fed a diet deficient in linoleate (EFAD diet), showing the paucity of sperm. 1*B* Large numbers of sperm in the epididymis of a cat fed the diet containing linoleate (SSO diet). × 79.

the epididymides. Similar results were obtained with cats fed a purified diet containing 25% chicken fat (24).

In cats fed the diet containing tuna oil (SSO/TO) the condition of the testes was poorer than in the cats fed the diet containing safflower seed oil alone. Although there were moderate to large numbers of sperm in the epididymides, the incidence of degeneration was high. This may result from the presence of 22:6n3 in tuna oil. As discussed above, this fatty acid apparently replaced 22:5n6 in phospholipids of the testis. Although the mechanism by which long-chain polyunsaturated fatty acids maintain spermatogenesis are not understood, some authors have suggested that 22:6n3 may function as well as 22:5n6. Ayala et al. (25) observed that the addition of 3% fish oil to a diet containing sunflower seed oil did not impair the development of rat testes, even though 22:6n3 appeared to replace 22:5n6 in testes lipids. However, the rats were examined after only 7–9 weeks of age. Cox et al. (26) examined male rats reared on an EFAD diet. When the diet was supplemented with linolenate (18:3n3), the testis degeneration at 35–48 weeks of age was more severe than in rats fed the EFAD diet without linolenate. The level of 22:6n3 in testes lipid was elevated in rats supplemented with linolenate (27). Cox and coworkers (26) concluded that n3 fatty acids may not provide for normal spermatogenesis.

Reproductive performance of female cats fed the experimental diets

The reproductive performance of female cats fed the purified diets is shown in table 4. Cats fed the EFAD diet were unable to reproduce adequately. Of the four cats fed the EFAD diet, only one became pregnant after 4 months of mating. This cat died during parturition with a ruptured uterus, and two of her kittens were never expelled.

Linoleate, supplied by safflower seed oil, did not appear to improve reproductive performance. In the SSO group, two abortions occurred out of four pregnancies. Of the two pregnancies that went to term, all of the kittens were born dead and were underdeveloped, with an average weight of only 45 g.

To determine whether arachidonate would improve reproduction, the cats previously fed the SSO diet were supplemented with ethyl arachidonate. Supplementation with arachidonate resulted in a doubling in the level of arachidonate in plasma and erythrocyte lipids (table 5). In addition, reproduction was improved significantly (table 4). The average litter size and birth weights were doubled, and viability was increased from 0 (before supplementation) to 75% (after supplementation). These results provide evidence that dietary arachidonate is essential for adequate reproduction in female cats.

In cats fed any of the diets containing

TABLE 4

Reproductive performance of female cats fed the experimental diets[1,2]

Diet	No. of cats	No. of litters	Avg litter size	Avg kitten wt	Neonatal viability[3]	% weaned of kittens born
			kittens/litter	*g*	*%*	
EFAD	4	1	3.0	71.5^{abc}	0^{a}	0
SSO	5	2	2.0	44.9^{a}	0^{a}	0
SSO + arachidonate	4	3	4.3	79.8^{b}	75^{b}	39
SSO/TO	4	3	4.3	67.7^{ab}	62^{b}	7
SSO/TLW	6	8	3.6	108^{c}	89^{b}	82
SSO/CHF	2	1	3.0	102^{c}	100^{b}	100
Pooled SEM	—	—	0.3	3.5	9	10

[1]Diet abbreviations: see table 1, footnote 1. [2]Means, within a column, not sharing a common superscript are significantly different, $P < 0.05$, by Duncan's multiple-range test. [3]No. of kittens alive 1 day after birth, as a percentage of kittens born.

TABLE 5

Effect of supplementation with ethyl arachidonate on concentrations of linoleate and arachidonate in plasma and erythrocytes of female cats[1]

Diet	Linoleate		Arachidonate	
	Plasma	Erythrocytes	Plasma	Erythrocytes
	% of total fatty acids by wt			
SSO	43.8 ± 1.4	33.9 ± 1.4	1.48 ± 0.31	5.69 ± 0.81
SSO + arachidonate	48.6 ± 1.3	32.7 ± 0.7	4.22 ± 1.38*	11.9 ± 0.72*

[1]Values are means ± SEM, $n = 4$. SSO, 5% safflower seed oil + 30% hydrogenated beef tallow. *Significantly different from SSO value by paired-t statistic, $P < 0.05$.

arachidonate (SSO/TO, SSO/CHF or SSO/TLW) reproductive performance was better than in cats fed the SSO diet. Cats fed the SSO/TLW or SSO/CHF diets were able to reproduce normally. Birth weights were relatively high; mortality was low, and most of the kittens survived to weaning. This is the first documentation of adequate reproduction in cats fed purified diets.

In the SSO/TO group, most of the kittens were born alive, but few survived to weaning. Those which died during the first weeks had low birth weights. In three kittens, the ductus arteriosus was structurally patent, and functional patency was suspected but not confirmed. Although the level of arachidonate in the SSO/TO diet was the same as in the SSO/CHF diet, the tuna oil contained n3 polyunsaturated fatty acids, which may have interfered with the utilization of arachidonate.

Conclusions

Unlike other mammals, the cat is unable to convert linoleate to arachidonate in the liver (4, 5). Nevertheless, linoleate prevents some signs of EFA deficiency, including poor growth, scaly skin, enlarged fatty livers and excessive water loss through the skin (5). However, if arachidonate itself or the production of eicosanoids from arachidonate is important for some physiological functions, it is expected that arachidonate would be a dietary essential for the cat. The present studies show that female cats were unable to bear live kittens when the diet was deficient in arachidonate, whether or not linoleate was supplied in the diet. When arachidonate was supplied, either as pure arachidonate or by animal fat, reproductive performance was greatly improved. Thus, dietary arachidonate is specifically required for normal reproduction in female cats.

Although linoleate is not converted to arachidonate in cat liver, our results suggest that this conversion occurs in the testes. As in other mammals, dietary linoleate (SSO) prevented testis degeneration in cats. Although spermatogenesis was essentially normal in cats fed the SSO diet, further studies would be needed to determine whether male cats fed arachidonate-deficient diets are able to mate and fertilize normal female cats.

Although arachidonate synthesis apparently occurs in cat testes, the arachidonate synthesized in the testes is not released into the bloodstream. The level of arachidonate in plasma and erythrocytes of cats fed the SSO diet was not higher in males than in females (data not shown). Therefore, dietary arachidonate may also be essential for male cats for functions other than reproduction.

The arachidonate requirement of the cat appears to be quite low. Reproduction was essentially normal in female cats fed the SSO/TLW diet, which provided arachidonate at 0.04% of energy. However, the arachidonate requirement may be increased when n3 fatty acids are present at high levels (as in the SSO/TO diet).

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