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Impact of different dietary fatty acids on plasma and liver lipids is influenced by dietary cholesterol in rats

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The purpose of this study was to determine blood and liver lipid responses in rats to stepwise replacement of palm oil with high-stearate and high-oleate lipids with and without dietary cholesterol. Two 28-day experiments were conducted in which 12% fat (wt/wt) was fed to rats in diets containing 0 (EXP1) or 0.4% cholesterol (EXP2). Nine diets were compared in each experiment: 12% palm oil was replaced in 3% increments by a high-stearate lipid (five treatments); 6% palm oil combined with 6% high-oleate safflower oil, 12% high-oleate safflower oil, 12% tallow, and 12% corn oil. Gain:consumption ratios were lower when rats were fed high-stearate fats. Plasma and liver cholesterol were greater when rats were fed 0.4% cholesterol (EXP2 compared with EXP1). The tallow diet produced the lowest plasma cholesterol in EXP1, but the highest level in EXP2. Similar plasma cholesterol levels occurred for groups fed 12% palm oil, corn oil, and safflower oil, with or without dietary cholesterol. In EXP1, dietary treatments had little effect on liver cholesterol. In EXP2, liver cholesterol decreased as consumption of the high-stearate fat increased, and was highest with 12% safflower oil. Proportions of hepatic fatty acids generally reflected intake; however, greater (16:1 and 18:1) and lower (18:0 and 20:4) occurred in cholesterol-supplemented rats (EXP2 compared to EXP1). In conclusion, rat plasma and liver lipid responses to different dietary fats are affected by dietary cholesterol, the presence of which greatly increases liver cholesterol, liver total fat, and tallow-induced hypercholesterolemia. (J. Nutr. Biochem. 7:142–149, 1996.)

Keywords: rats; liver; plasma; cholesterol; fatty acids; lipids

Introduction

Hegsted et al.[1] concluded that degree of fatty acid saturation and cholesterol are the primary determinants of serum cholesterol in humans. Reducing total fat intake may not lower serum cholesterol unless saturated fat intake is reduced.[2,3] Individual fatty acids also exhibit different cholesterolemic effects. Lauric (12:0) and myristic (14:0) acids, for example, are more hypercholesterolemic than palmitic acid (16:0), which is hypercholesterolemic compared with stearic acid (18:0) and unsaturated fatty acids.[4–7] Neutral and cholesterol-lowering effects of 18:0 have also been observed.[8–11] High-oleate (18:1) containing lipids have lowered plasma cholesterol when compared with 16:0;[12] however, not all high-oleate lipids are hypocholesterolemic.[13]

Combining oils with different fatty acid compositions has been used to evaluate effects of reciprocal changes in dietary fatty acids. Simultaneously decreasing dietary palmitate and increasing oleate did not affect serum cholesterol of hamsters;[14] however, with 2% dietary cholesterol, serum cholesterol increased in palm oil fed animals, but not in those fed olive or safflower oil. Thus, dietary cholesterol appears to influence cholesterolemic effects of specific fatty acids. The purpose of this study was to determine blood and liver lipid responses in rats to stepwise replacement of palm oil with high-stearate and high-oleate lipids in diets with and without dietary cholesterol.

Methods and materials

Seventy-two male Sprague-Dawley rats (Harlan Sprague-Dawley, Indianapolis, IN, USA about 4 weeks old) were used for each of

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two experiments differentiated by the absence (EXP1) or presence (EXP2) of dietary cholesterol. Rats were housed individually in a ventilated room with a 12-hr light/dark cycle. Animals adapted to their environment for 25 days with ad libitum access to water and AIN-76 diet (United States Biochemical, Cleveland, OH, USA). Rats were randomly assigned to one of nine treatments based on source of dietary fat with eight animals per treatment.

Mean initial body weights calculated across treatments were 177.8 ± 0.2 g (SEM) for EXP1 and 169.8 ± 0.3 g for EXP2. Diet compositions are described in *Table 1*. Dietary fat was 12% (wt/wt) in both experiments. In EXP1, diets were not cholesterol supplemented; in EXP2, diets contained 0.4% (wt/wt) cholesterol. All diet ingredients were purchased from United States Biochemical (Cleveland, OH, USA) except for cholesterol, which was purchased from Sigma Chemical Co. (St. Louis, MO, USA # C-8667). Dietary fats used in formulating the diets are described in *Table 2*. Five combinations of palm oil and a high-stearate oil, two combinations of palm oil and high-oleate safflower oil, tallow alone, and corn oil alone were compared. Tallow was included because weight percentages of 16:0, 18:0, and 18:1 were similar to those of several of the vegetable oil blends (*Table 3*), but of markedly different origin. Corn oil was included because of its common use in previous studies, and its use allowed comparison with the two non-blended vegetable oils (P12 and O12).

The high-stearate fat was produced by interesterification of high-oleate safflower oil with hydrogenated soybean oil as previously described[8] (provided by Anderson Clayton/Humku Products, Memphis, TN, USA). Palm oil was purchased from Agro Ingredients, Inc. (Des Plains, IL, USA). High-oleate safflower oil was purchased from Wilchem, Inc. (Brea, CA, USA). Corn oil was purchased locally (Mazola), and tallow was prepared by the University of Wyoming Meats Laboratory. Corn oil was stabilized by addition of 0.2 g of tertiary butylhydroquinone per 100 g of oil. Oil blends were combined and then mixed as the last ingredient of each diet. Diets were mixed for 2 hr using a Hobart mixer.

All diets were provided as a dry powder for ad libitum consumption. Rats were weighed weekly, and food consumption measured every 4 days, by weighing food containers. After 28 days of feeding and following a 12-hr fast, rats were killed by ether anesthesia. Cardiac puncture was used to obtain blood from which plasma was harvested and stored at −20°C. Livers were weighed after removal of blood and fat and then stored at −20°C.

Plasma cholesterol and triglyceride concentrations were determined using assay kits (Stanbio Laboratory, San Antonio, TX, USA). Entire livers were freeze dried, ground, and total lipids extracted in duplicate from 50-mg samples in 1:2:0.8 chloroform: methanol:water (vol:vol:vol).[15] Lipid extracts were saponified[16] and cholesterol determined in the non-saponified fraction.[17] Fatty acid analyses of the saponified lipids and total lipids of dietary fats were accomplished using GLC.[18] Cholesterol and cholesteryl esters were separated by thin-layer chromatography using plates coated with silica gel-G (250 μm, Analtech, Newark, DE, USA) and developed in petroleum ether, diethyl ether, and acetic acid (90:10:1) (vol:vol:vol). Cholesterol and cholesteryl ester fractions were recovered, dissolved in chloroform, and total cholesterol determined by the colorimetric method of Rudel and Morris.[17]

Data were analyzed by analysis of variance for the completely randomized design.[19] When the overall analysis indicated significant treatment effects ($P < 0.05$), Duncan's New Multiple Range test was used to locate significantly different treatment means.

Table 1 Diet composition[a]

Item	− Cholesterol (EXP1)	+ Cholesterol (EXP2)
Sucrose	43.0	42.6
Corn starch	15.0	15.0
Casein	20.0	20.0
DL-Methionine	0.3	0.3
Fat	12.0	12.0
Cholesterol	0.0	0.4
Cellulose	5.0	5.0
Mineral mixture[b]	3.5	3.5
Vitamin mixture[c]	1.0	1.0
Choline bitartrate	0.2	0.2

[a]Composition is percentage by weight, as fed.
[b,c]Mineral and vitamin mixtures were AIN Mineral Mixture 76[R] and AIN Vitamin Mixture 76[R] (AIN, 1977).

Table 2 Description of dietary fats

Diet[a]	Source of dietary fat: Palm oil	High-stearate oil	High-oleate safflower oil	Beef tallow	Corn oil
	%, by weight of diet				
P12	12	0	0	0	0
P9S3	9	3	0	0	0
P6S6	6	6	0	0	0
P3S9	3	9	0	0	0
S12	0	12	0	0	0
P6O6	6	0	6	0	0
O12	0	0	12	0	0
TA	0	0	0	12	0
CO	0	0	0	0	12

[a]P = palm oil; S = high-stearate oil; O = high-oleate safflower oil; TA = tallow; CO = corn oil.

Results and discussion

Composition of experimental fats

Fatty acid compositions of dietary fats are given in *Table 3*. Stepwise replacement of palm oil with high-stearate fat resulted in reciprocal changes in proportions of 16:0 and 18:0 with nearly constant levels of 18:1 and linoleate (18:2). The stepwise replacement of palm oil with high-oleate safflower oil resulted in reciprocal changes in 16:0 and 18:1 with a

Table 3 Fatty acid weight percentages of dietary fats[a]

Diet	Fatty acid[b]: 16:0	18:0	18:1	18:2
P12	40.9	4.2	42.1	10.4
P9S3	33.8	12.4	41.6	9.9
P6S6	27.4	20.8	40.9	9.5
P3S9	18.4	29.9	41.1	9.3
S12	8.9	38.6	42.5	9.0
P6O6	24.4	3.4	58.0	12.4
O12	5.5	2.3	74.6	15.5
TA	25.4	10.8	46.0	1.9
CO	11.9	2.0	26.0	58.6

[a]From lipids extracted from prepared diets; dietary lipid comprised 25.7 en% of fat, as fed.
[b]Carbons: double bonds; 16:0, palmitate; 18:0, stearate; 18:1, oleate; 18:2, linoleate.

constant level of 18:0, but 18:2 was less constant (compare P12, P6O6, and O12).

Food consumption and growth

Food consumption and growth data are illustrated in *Figures 1* and *2* for EXP1 and EXP2, respectively. Food consumption was greatest for S12 in EXP1 ($P < 0.05$) and for P3S9 in EXP2 ($P < 0.05$). Body weight gain was lowest for S12 ($P < 0.05$) in EXP2, but not in EXP1. Gain:consumption ratios were lowest for rats fed high-stearate fats, suggesting less efficient digestion/absorption of these diets.

Poor digestibility of high-stearate fats has been previously reported in rats[20,21] and humans.[22] Rat fecal fatty acids have reflected major dietary fatty acids, which has been most pronounced with 18:0.[23] Total fatty acids of EXP1 fecal samples from rats fed P12 and S12 contained 80% 16:0 and 18:0, respectively, whereas 18:1 in fecal total fatty acids was 62% in rats fed O12 (data not shown, no fecal samples were collected in EXP2), indicating the possibility of lower absorbability of these fatty acids. Bonanome and Grundy,[8] however, reported efficient absorption of 16:0 (98.9%), 18:0 (97.4%), and 18.1 (99.9%) in humans who consumed palm oil, high-stearate fat (similar to that used in this study), and high-oleic acid safflower oil, respectively.

Plasma lipids

Plasma cholesterol and triglyceride values are presented in *Figure 3*. In EXP1, rats fed tallow exhibited the lowest plasma cholesterol ($P < 0.05$) compared to those fed corn oil, O12, P12, P9S3, and P3S9; values were similar for corn, safflower, and palm oils ($P > 0.05$). Plasma triglycerides were similar ($P > 0.05$) for all treatments except P6S6, which exhibited the lowest triglyceride concentration.

In EXP2, rats fed P6S6 had lower plasma cholesterol ($P < 0.05$) than all other groups except P3S9, S12, and corn oil. Rats fed tallow had significantly greater plasma cholesterol than rats fed corn oil, P6S6, P3S9, or S12 ($P < 0.05$). Plasma triglycerides did not vary consistently with the reciprocal changes in 16:0 and 18:0. Rats fed S12, however, had significantly higher ($P < 0.05$) triglycerides than rats fed P12, any combination of palm oil and high-stearate oil, tallow, and corn oil. With the non-blended vegetable oils (P12, O12, corn oil), no differences in plasma cholesterol were observed but triglycerides were greatest ($P < 0.05$) for rats fed O12, and similar for those fed palm or corn oils.

Although some reports have indicated little change in blood cholesterol of rats in response to consumption of different dietary fatty acids,[21,24–26] others have reported greater plasma cholesterol in animals fed saturated compared to unsaturated fatty acids and in animals fed lipids that contained 12:0 and 14:0 compared to those containing 16:0 or 18:0.[20,27,28] In this study, saturated fatty acid-induced changes in plasma cholesterol were dependent on the presence of supplemental cholesterol. Moreover, with 0.4% dietary cholesterol, mean plasma cholesterol (for all treatments) was approximately 120 mg/ 100 mL compared to 90 mg/100 mL without supplemental cholesterol.

In the Cebus monkey, plasma cholesterol was not influenced by dietary 16:0 until the diet contained 0.3% cholesterol, which resulted in a hypercholesterolemic effect of 16:0 compared with 18:0.[29] In hamsters, 12:0, 14:0, and 16:0 were hypercholesterolemic compared with 18:0.[23] With 0.3% dietary cholesterol, however, consumption of the different fatty acids resulted in similar blood cholesterol levels, which were higher than those when diets were devoid of supplemental cholesterol. Also in hamsters, 2% dietary cholesterol was needed to demonstrate a hypercholesterolemic effect of palm oil compared with high-oleate safflower oil.[14]

In the present study, the interaction of dietary cholesterol with dietary fatty acids was most extreme with the tallow diet; plasma cholesterol was 72 mg/100 mL without dietary cholesterol and 140 mg/100 mL with 0.4% dietary cholesterol. Wistar rats that consumed 10% tallow and 1% cholesterol had over threefold higher blood cholesterol than rats fed either safflower oil or perilla oil, which contained about 60% α-linolenic acid (18:3).[28] In humans, lean beef consumption caused baseline plasma cholesterol concentrations to decrease, and when beef fat was added, plasma cholesterol increased to levels that were still below those at baseline.[30] The interaction of dietary cholesterol with fat may not be restricted to tallow because consumption of either butter fat[31] or lard[32] with cholesterol resulted in large plasma cholesterol increases in rats and guinea pigs, respectively. The fat-cholesterol interaction may be partially related to reduced cholesterol absorption associated with consumption of plant sterols in vegetable oils.[33]

Liver cholesterol and cholesteryl esters

Liver cholesterol and cholesteryl ester values are shown in *Figure 4*. In EXP1, rats fed S12 had lower ($P < 0.05$) liver cholesterol than did corn oil fed rats with all others intermediate. However, a more pronounced decrease in percentage cholesteryl ester with increased dietary high-stearate was observed compared to the change observed in EXP2. In EXP2, liver cholesterol decreased in association with the sequential replacement of palm oil with high-stearate fat ($P < 0.05$). The highest liver cholesterol concentrations (P6O6, O12, tallow, and P12) were greater ($P < 0.05$) than that of the corn oil-fed group. Liver cholesterol of EXP2 rats was about fourfold greater than that of EXP1 rats. Moreover, percentage cholesteryl ester was over 3 fold greater in rats fed cholesterol (EXP2).

Previous studies also demonstrated stearate-induced decreases in liver cholesterol. Mani et al.[21] reported a negative relationship between dietary stearate and liver cholesterol levels in rats fed a diet that contained 0.4% cholesterol. Kritchevsky et al.[20] reported lower liver cholesterol in rats fed cocoa butter (high in 18:0) than in those fed either corn, palm kernel, or coconut oils.

In a summary of previous work, Beynen[34] concluded that consumption of monounsaturated fatty acids results in increased liver cholesterol. Olive oil consumption by mice caused greater liver cholesterol than did animal fat, corn oil, or cocoa fat.[26] Results of the present study (i.e., significantly higher liver cholesterol in P6O6 and O12 groups compared to corn oil and stearate-fed groups in EXP2) and of others[27,28] corroborate this conclusion, but the presence

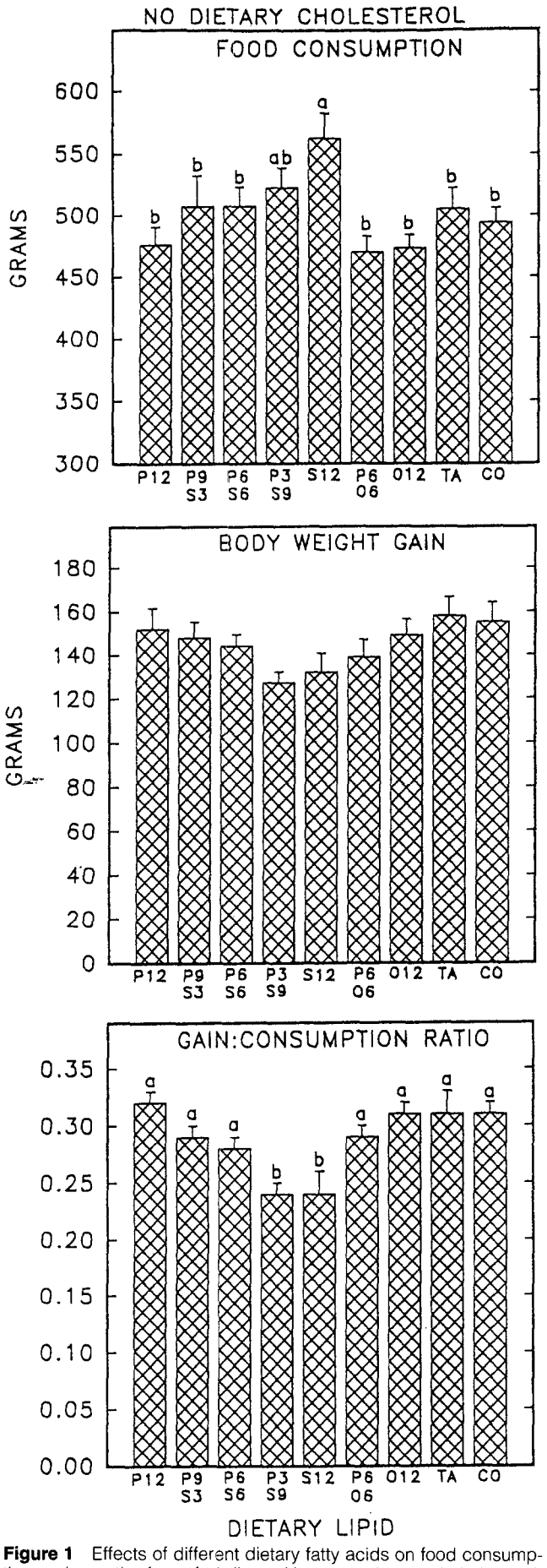

Figure 1 Effects of different dietary fatty acids on food consumption and growth of rats fed diets without supplemental cholesterol. Bars are means ± SEM (eight rats per treatment). Different superscripts indicate significant differences ($P < 0.05$). Dietary lipids are described in *Table 2*. TA is beef tallow and CO is corn oil.

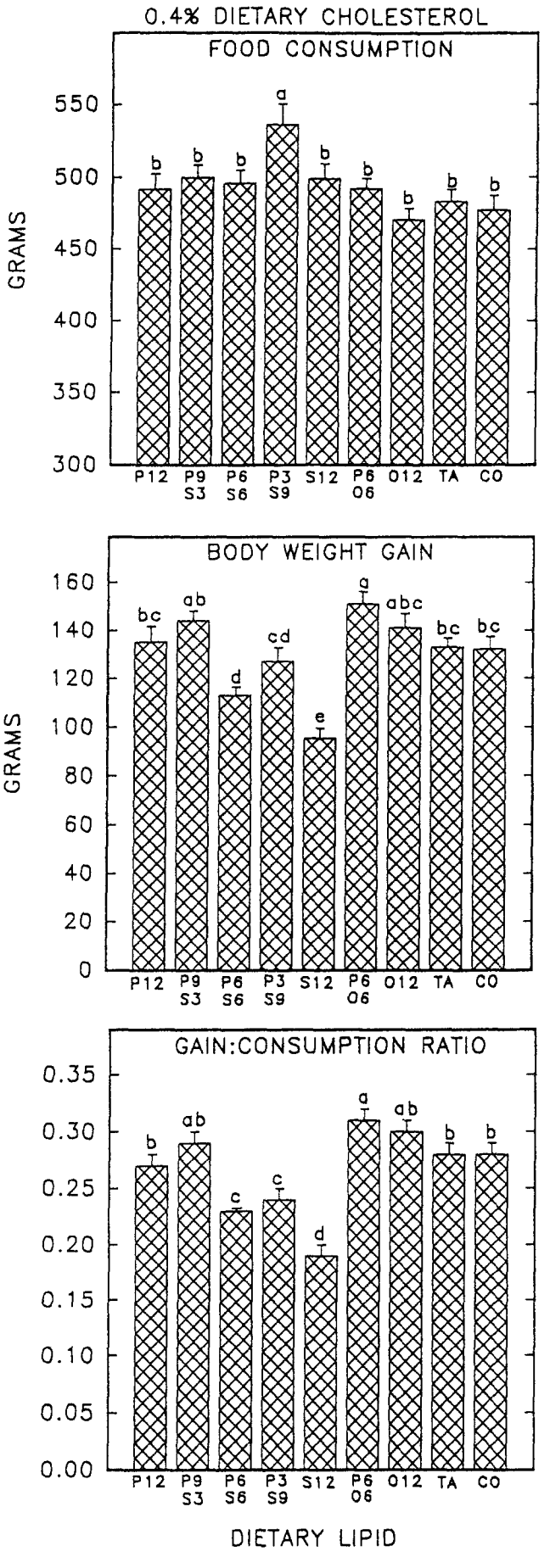

Figure 2 Effects of different dietary fatty acids on food consumption and growth of rats fed diets containing 0.4% cholesterol. Bars are means ± SEM (eight rats per treatment). Different superscripts indicate significant differences ($P < 0.05$). Dietary lipids are described in *Table 2*. TA is beef tallow and CO is corn oil.

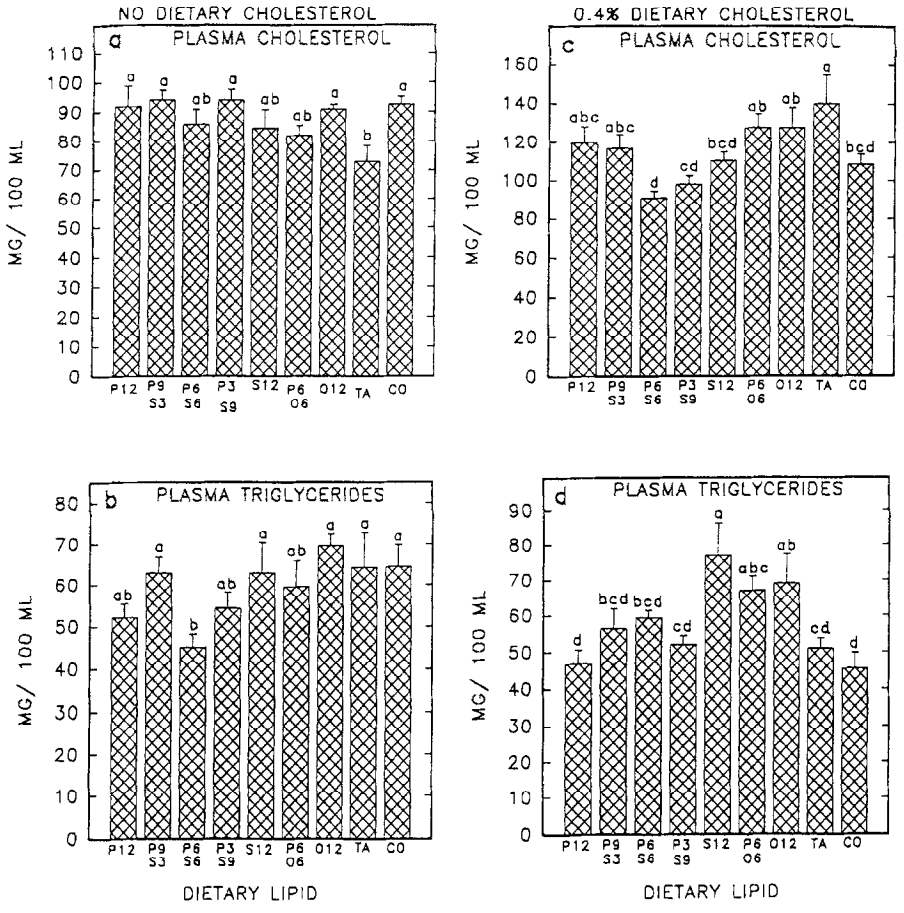

Figure 3 Effects of different dietary fatty acids on plasma cholesterol and triglycerides in rats fed diets that contained either no (*Figure 3a, 3b*) or 0.4% supplemental cholesterol (*Figure 3c, 3d*). Bars are means ± SEM (eight rats per treatment). Different superscripts indicate significant differences ($P < 0.05$). Dietary lipids are described in *Table 2*. TA is beef tallow and CO is corn oil.

of dietary cholesterol was necessary to elicit this response whereas the mice used by Beynen et al.[26] were not fed cholesterol. Moreover, the large cholesterol-induced increase in liver cholesterol (3 fold) and cholesteryl ester (4 fold), which occurred in the present study corroborates findings of others.[23,32,35]

In the present study, when no dietary cholesterol was provided, consumption of fats high in monounsaturated fatty acids tended to result in greater proportions of cholesteryl esters whereas reduced proportions occurred with ingestion of the high-stearate fat. However, with 0.4% dietary cholesterol, cholesteryl ester formation was much greater regardless of the dietary fat, likely from an effort by hepatocytes to sequester the large influx of cholesterol.

Liver fatty acids

Fatty acid weight percentages of liver lipid extracts are shown in *Table 4*. With or without dietary cholesterol supplementation, weight percentages of 16:0 were partially reflective of dietary 16:0 concentration. However, magnitude of treatment differences in hepatic 16:0 was considerably less than 16:0 level differences among dietary fats. This suggests that hepatic 16:0 metabolism may not have been influenced greatly by the various dietary fats. Similar responses to dietary fat type were observed for 16:1. This fatty acid arises from desaturation of 16:0 and, with the exception of liver lipids of tallow-fed rats, greater intake of 16:0 resulted in a general increase in 16:1. Consumption of 16:1 was highest in rats fed tallow because bovine adipose tissue contains from 3 to 6% 16:1.[36]

With or without added dietary cholesterol, hepatic 18:0 increased ($P < 0.05$) in parallel with the level of high-stearate fat ingested (*Table 4*), and in both cases reached a maximum weight percentage with the P3S9 diet. Moreover, compared with hepatic decreases in 16:0 when palm oil was sequentially replaced with high-stearate oil, greater relative increases in 18:0 occurred suggesting more rapid incorporation. These findings suggest that absorption of the high-stearate oil was sufficient to lead to accumulation of this fatty acid. Smaller, yet significant ($P < 0.05$), treatment differences in liver 18:0 were observed in EXP1 than EXP2. In EXP2, rats fed diets that were lowest in 18:0 generally had the lowest hepatic 18:0.

Few differences in liver 18:1 occurred in EXP1, and no differences ($P > 0.05$) in 18:1 occurred for the palm oil and high-stearate oil-combinations. Rats fed O12 did not have the greatest proportion of 18:1 in liver lipids, which was the

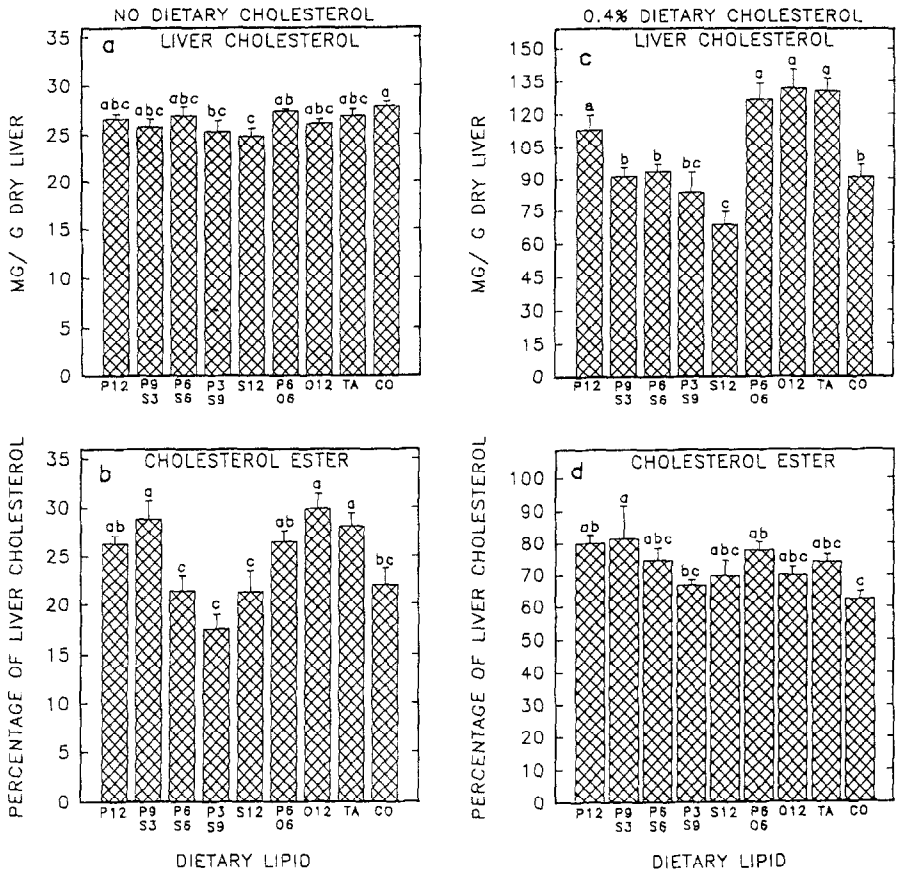

Figure 4 Effects of different dietary fatty acids on liver cholesterol concentration and percentage cholesteryl ester in rats fed diets that contained either no (*Figure 4a, 4b*) or 0.4% supplemental cholesterol (*Figure 4c, 4d*). Bars are means ± SEM (eight rats per treatment). Different superscripts indicate significant differences ($P < 0.05$). Dietary lipids are described in *Table 2*. TA is beef tallow and CO is corn oil.

case in EXP2. In both experiments, corn oil-fed rats had the lowest hepatic 18:1, which paralleled the dietary level of this fatty acid.

Weight percentages of hepatic 18:1 were greatest ($P < 0.05$) for cholesterol-fed rats (EXP2) fed P6O6, O12, and tallow (*Table 4*). As palm oil was sequentially replaced with the high-stearate fat, hepatic 18:1 decreased, and was significantly lower ($P < 0.05$) with P3S9 than with the other palm oil and high-stearate fat combinations. All palm oil and high-stearate fat combinations provided essentially the same proportion of 18:1 in the diet, but as the dietary proportion of 16:0 increased and that of 18:0 decreased in EXP2, hepatic 18:1 increased at a rate similar to the increase in hepatic 16:0. The relevance of this observation is not clear, but it likely involves interaction of dietary fatty acid changes with dietary cholesterol because similar events did not occur in EXP1.

Changes in liver 18:2 closely followed changes in dietary concentration of this fatty acid in both experiments; however, hepatic 18:2 was lower in EXP1 than in EXP2. Weight percentages of arachidonic acid (20:4) (*Table 4*) varied somewhat with treatment, with tallow-fed rats exhibiting the lowest level in both experiments, but changes were not consistent with the stepwise changes in dietary fatty acids.

The greatest differences in response to supplemental cholesterol (EXP1 compared to EXP2) occurred for 16:1, 18:0, 18:1, and 20:4. Liver 16:1 and 18:1 were higher in EXP2. Conversely, hepatic 18:0 and 20:4 were greater in EXP1. In a previously reported study, rats fed 0.4% cholesterol had high levels of monounsaturated fatty acids in hepatic cholesteryl esters.[20] In the present study, 16:1 and 18:1 probably were esterified to cholesterol to a greater extent than the other fatty acids; greater levels of cholesteryl ester associated with cholesterol feeding could explain differences in levels of these fatty acids between experiments, as well as the effect of dietary cholesterol on hepatic 18:2 of corn oil-fed rats. In addition, cholesterol-fed rats may have exhibited a greater hepatic conversion of 18:0 to 18:1, thus partially accounting for the decreased 18:0 and increased 18:1. The dramatic difference in hepatic 20:4 between the two experiments is difficult to explain, especially in light of the marked similarity among experiments in levels of 18:2, a key precursor for 20:4 synthesis.

Rat hepatic fatty acids generally reflect dietary fatty acids.[20,21,37] Similar diet effects occurred for plasma triglyceride fatty acids of rats[38] and humans[5]; however, serum total fatty acids of rats fed cocoa butter did not have greater 18:0 than rats fed corn, palm kernel, or coconut oils.[20] Few studies have reported on the interaction between dietary cholesterol and fatty acids with respect to liver fatty acid composition. Feeding rats 10% olive oil with or without 0.5% cholesterol did not influence cholesteryl ester 18:1, but 0.5% dietary cholesterol caused a significant increase in total liver lipid.[35]

Table 4 Fatty acid weight percentages of liver lipid extracts of rats fed various fats and no or 0.4% dietary cholesterol[a]

Diet[b]	Fatty acid[c] 16:0	16:1	18:0	18:1	18:2	20:4
	---------- no dietary cholesterol (EXP1) ----------					
P12	22.5^{d}	2.5^{de}	17.4^{g}	25.8^{de}	9.4^{ef}	22.3^{f}
P9S3	22.0^{de}	2.0^{efg}	18.5^{g}	25.8^{de}	10.0^{ef}	21.6^{f}
P6S6	22.2^{de}	2.1^{ef}	21.1^{ef}	22.7^{ef}	9.4^{ef}	22.6^{ef}
P3S9	22.1^{de}	2.0^{efg}	25.3^{d}	23.8^{ef}	8.7^{efg}	18.1^{g}
S12	19.4^{f}	1.8^{fgh}	23.2^{de}	23.9^{ef}	8.6^{fg}	23.1^{ef}
P6O6	21.5^{de}	1.5^{gh}	18.9^{fg}	22.4^{f}	10.1^{e}	25.7^{de}
O12	17.7^{g}	1.3^{hi}	19.1^{fg}	24.6^{ef}	10.1^{e}	27.3^{d}
TA	21.8^{de}	3.1^{d}	18.0^{g}	28.5^{d}	7.6^{g}	20.9^{fg}
CO	20.9^{e}	0.9^{i}	18.4^{g}	10.4^{g}	22.9^{d}	26.5^{d}
SEM[j]	0.6	0.2	0.9	1.2	0.5	1.2
	---------- 0.4% dietary cholesterol (EXP2) ----------					
P12	23.0^{d}	4.2^{e}	12.5^{g}	36.1^{efg}	11.3^{e}	12.9^{ef}
P9S3	22.3^{de}	4.0^{ef}	14.7^{fg}	34.8^{fg}	9.4^{fg}	14.8^{de}
P6S6	21.3^{f}	2.3^{h}	19.0^{e}	33.2^{gh}	10.5^{ef}	15.8^{d}
P3S9	19.3^{g}	2.5^{gh}	23.9^{d}	29.4^{i}	8.8^{g}	14.0^{def}
S12	19.4^{g}	2.4^{h}	23.0^{d}	31.6^{hi}	9.9^{fg}	13.6^{ef}
P6O6	22.8^{d}	3.3^{fg}	14.3^{fg}	36.7^{ef}	9.9^{fg}	12.9^{ef}
O12	19.9^{g}	3.6^{ef}	13.2^{g}	42.1^{d}	9.1^{g}	12.0^{fg}
TA	22.8^{d}	6.0^{d}	16.2^{f}	39.1^{de}	7.2^{h}	8.7^{h}
CO	21.6^{ef}	2.1^{h}	14.7^{fg}	20.9^{j}	30.4^{d}	10.2^{gh}
SEM[j]	.4	.3	.9	1.2	.4	.7

[a]Values were adjusted to 100%; values presented represent about 97% of peak areas.
[b]See *Table 2* for description of diet codes.
[c]Carbons: double bonds.
[d,e,f,g,h,i]Means within the same column with different superscripts are different ($P < 0.05$).
[j]Pooled standard error of the mean.

Conclusion

In summary, fatty acid-induced alterations in rat plasma and liver cholesterol required supplemental dietary cholesterol, and may have been partially due to reduced absorption of stearic acid. The propensity of high-stearate fat to lower and of tallow to raise plasma and liver cholesterol was evident only when 0.4% cholesterol was included in the diet. This suggests that tallow, and animal fats in general, may interact with dietary cholesterol to affect plasma cholesterol, possibly mediated via alteration in lipid absorption. The exact nature of this potential interaction requires further study. Although hepatic fatty acids generally reflected intake, greater hepatic 16:1 and 18:1 and lower 18:0 and 20:4 were observed in cholesterol-supplemented compared to non-cholesterol supplemented rats.

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