NUTRITIONAL ASPECTS OF LONG-CHAIN FATTY ACIDS

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Cardiac changes have been demonstrated in rats fed liquid rapeseed oil. In short-term experiments, fatty deposits accumulated in the tissue, and in longer experiments, necrotic lesions developed (1, 2, 3, 4).

To determine the extent of fat deposition in the heart with different dietary sources of long-chain fatty acids, 320 Charles River rats, half of each sex, were fed a purified basal diet to which the test fat was added as 20% by weight or 40% of total calories. The dietary fats were a 3:1 mixture of lard and corn oil, liquid rapeseed oil, partially hydrogenated rapeseed oil and partially hydrogenated herring oil. The latter three were kindly supplied by Canada Packers Research and Development Laboratories. The fatty acid composition of these oils is shown in Table I. The control fat contained mostly C_{16} and C_{18} fatty acids; the other dietary fats contained also C_{20} and C_{22} fatty acids. In the partially hydrogenated rapeseed oil the C_{22} fraction was still mostly in the monoenoic form. The principal C_{22} monoenoic acid in herring oil prior to hydrogenation was a positional isomer of erucic acid having its double bond two carbons closer to the carboxyl group(5). Upon partial hydrogenation the rapeseed and herring oils contained elevated levels of saturated fatty acids.

Rats fed these dietary fats were killed at 3, 7, 14 and 28 days. At each of these intervals 12 rats from each group were designated for histopathological examination and 8 for lipid analyses. In Fig. 1, the total fatty acids, expressed as mg/heart, are shown for each diet and time interval. Each bar represents data from 6 rats (3 randomly chosen from each sex). The controls, plain bar, were lower than all of the others. That is, the diets containing the long-chain fatty acids produced a significantly increased level of cardiac lipids. The peak of deposition in rats fed liquid rapeseed oil was reached at one week and thereafter decreased, but by four weeks had not returned to the control level. Histopathologically, fat droplets were observed in the muscle of all hearts from rats fed the long-chain fatty acids for 3, 7 and 14 days. At the last time interval studied in this experiment the cardiac tissue from all rats fed liquid rapeseed still showed

TABLE I

FATTY ACID COMPOSITION OF DIETARY OILS

Fatty Acid	PERCENT OF TOTAL FATTY ACIDS			
	Lard:CO	LRSO	HRSO	HHO
14:0	2.6	0.2	0.3	5.3
14:1	-	-	-	0.4
15:0	-	-	-	0.5
15:1	-	-	-	0.4
16:0	21.7	3.8	3.4	12.6
16:1	3.1	0.3	0.5	10.1
16:2	-	-	-	1.0
17:0	-	-	-	0.3
18:0	10.2	1.8	8.0	3.2
18:1	37.4	22.6	35.6	13.2
18:2	22.8	17.8	7.4	1.0
18:3	-	6.0	-	-
20:0	1.6	1.1	2.3	2.25
20:1	-	12.9	11.0	18.4
20:2	0.7	0.6	-	2.3
20:3	-	-	-	1.5
20:3	-	-	-	0.9
22:0	-	-	4.7	2.5
22:1	-	32.9	26.3	24.1
T O T A L	36.1	6.9	19.3	26.7

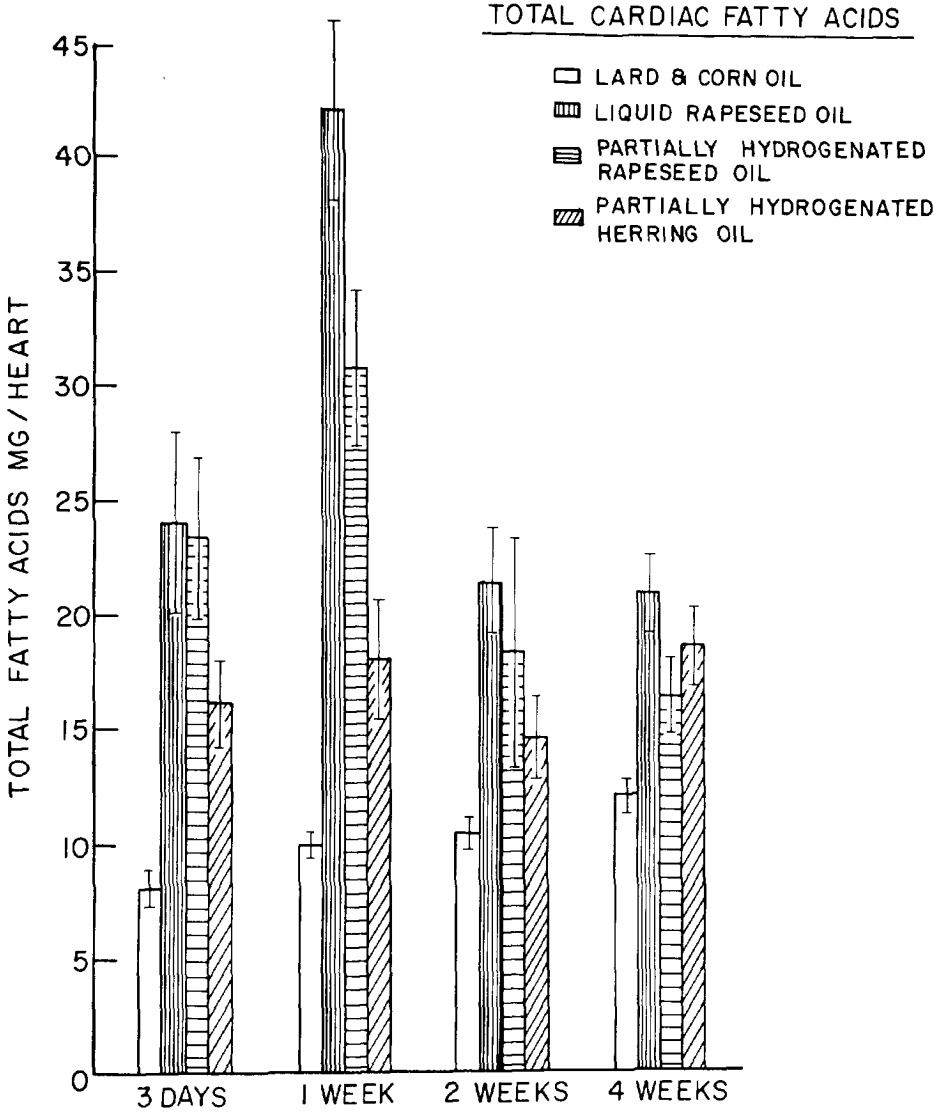

Fig. 1.

The total cardiac fatty acids of young rats fed different dietary fats.

fat deposition whereas fewer than half of those fed the partially hydrogenated oils did.

The proportion of C_{22} monoenoic acid in the cardiac fatty acids is shown in Fig. 2. The retention of this fatty acid was greatest in the group fed liquid rapeseed oil, although the partially hydrogenated product had contained similar amounts of the long-chain fatty acids. The two hydrogenated oils were associated with similar proportions of the $C_{22:1}$. In Fig. 3, the C_{22} monoenoic acids are shown plotted as mg/heart. The correlation between the total fatty acids and their content of $C_{22:1}$ decreased as the experimental time increased. It appeared, therefore, that this type of fatty acid in the cardiac tissue was closely related to the level of fat deposition only at the early stages.

The one week interval, the time of peak deposition, was used to determine the no-effect level of liquid rapeseed oil. Table II contains the levels fed of rapeseed oil containing 29% erucic acid and of the fatty acids deposited in cardiac tissue. Histopathologically, the zero-effect level was found to be 5% of this rapeseed oil in the diet, the first effect appearing at the 10% level of rapeseed oil or 3% of erucic acid. The total fatty acids deposited with the two lower levels of rapeseed were similar in amount to those obtained with canbra oil. This oil appeared to induce no histopathological effect after one week when rapeseed oil was associated with peak cardiac lipid deposition. Above 10% of the liquid rapeseed oil, the deposition of total fatty acids and of erucic acid increased rapidly. These aspects are illustrated, Fig. 4, in which the total fatty acids in mg/heart are plotted against the level of liquid rapeseed oil in the diet. Again, it is emphasized that the first histopathological effects were observed at the 10% level. The amounts of $C_{20:1}$ and $C_{22:1}$ acids are shown in Fig. 5. There was a striking increase in the deposition of $C_{22:1}$. At the highest level of rapeseed oil intake the relative amounts of these two acids approximated the ratio in the diet. In Fig. 6 are some other cardiac fatty acids found with varying levels of rapeseed oil in the diet. The C_{18} monoenoic acid increased markedly. There was an increase in $C_{18:2}$, also found in rapeseed oil, but not in $C_{20:4}$ which does not occur in the oil. The cardiac tissue was therefore accumulating the dietary fatty acids, found to be deposited primarily as triglycerides.

It is emphasized that these results were obtained with weanling rats. To determine the effect in older rats, they were maintained on standard fox cubes for two months before receiving liquid rapeseed oil as the 20% by weight of the diet for one week. The results are shown in the Table III where a comparison is made of the cardiac fatty acids in the young and older rat. The

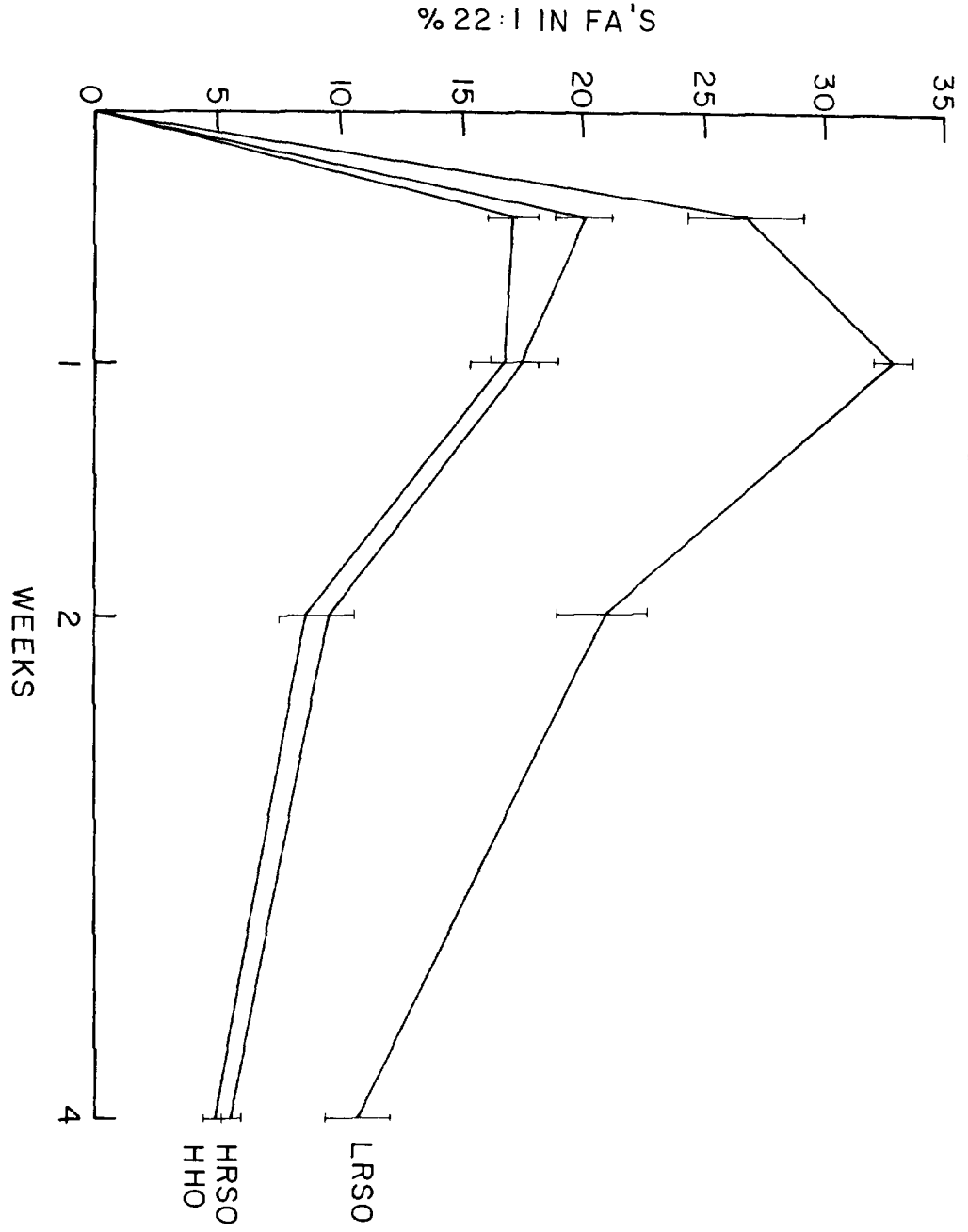

Fig. 2.

The proporation of docosenoic acid in the cardiac fatty acids of rats fed liquid rapeseed oil (LRSO), partially hydrogenated rapeseed oil (HRSO) or partially hydrogenated herring oil (HHO).

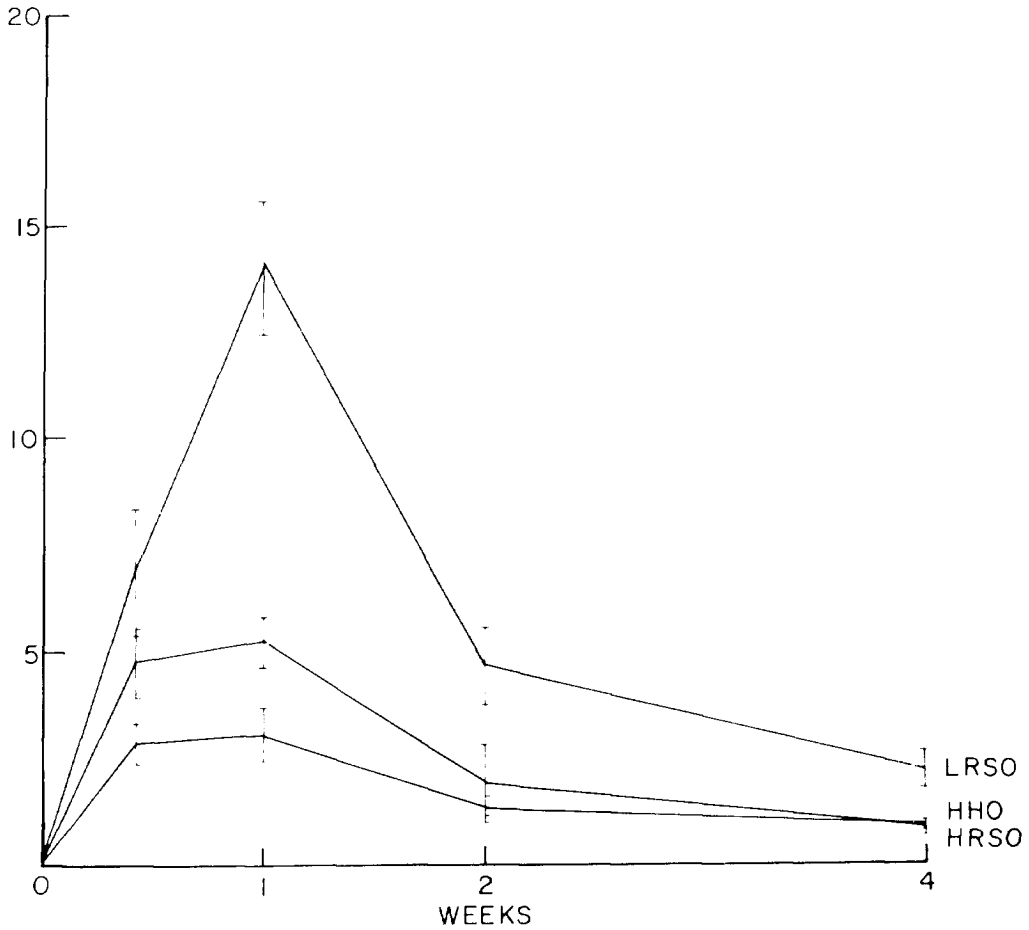

Fig. 3

The total amount of docosenoic acid deposited in cardiac tissue of rats fed liquid rapeseed oil (LRSO), partially hydrogenated rapeseed oil (HRSO) or partially hydrogenated herring oil (HHO).

TABLE II

CARDIAC FATTY ACIDS IN RATS FED VARIOUS LEVELS OF RAPESEED OIL (RSO) OR CANBRA OIL FOR ONE WEEK

	TOTAL FATTY ACIDS 22:1		
	mg/g Heart	mg/Heart	Percent of F.A.'s
PERCENT RSO IN DIET			
0	17.3 ± 3.1	7.4 ± 1.1	- -
2.5	15.7 ± 0.8	6.3 ± 0.2	0.6 ± 0.3
5	18.1 ± 0.9	8.3 ± 0.3	3.2 ± 0.4
10	24.5 ± 2.7	9.7 ± 1.0	7.7 ± 1.4
15	40.6 ± 7.9	17.1 ± 2.5	18.5 ± 0.5
20	55.1 ± 7.4	26.1 ± 5.0	26.3 ± 0.5
PERCENT CANBRA IN DIET			
10	14.6 ± 1.6	6.2 ± 0.9	- -
20	19.2 ± 0.4	7.1 ± 0.3	1.8 ± 0.3

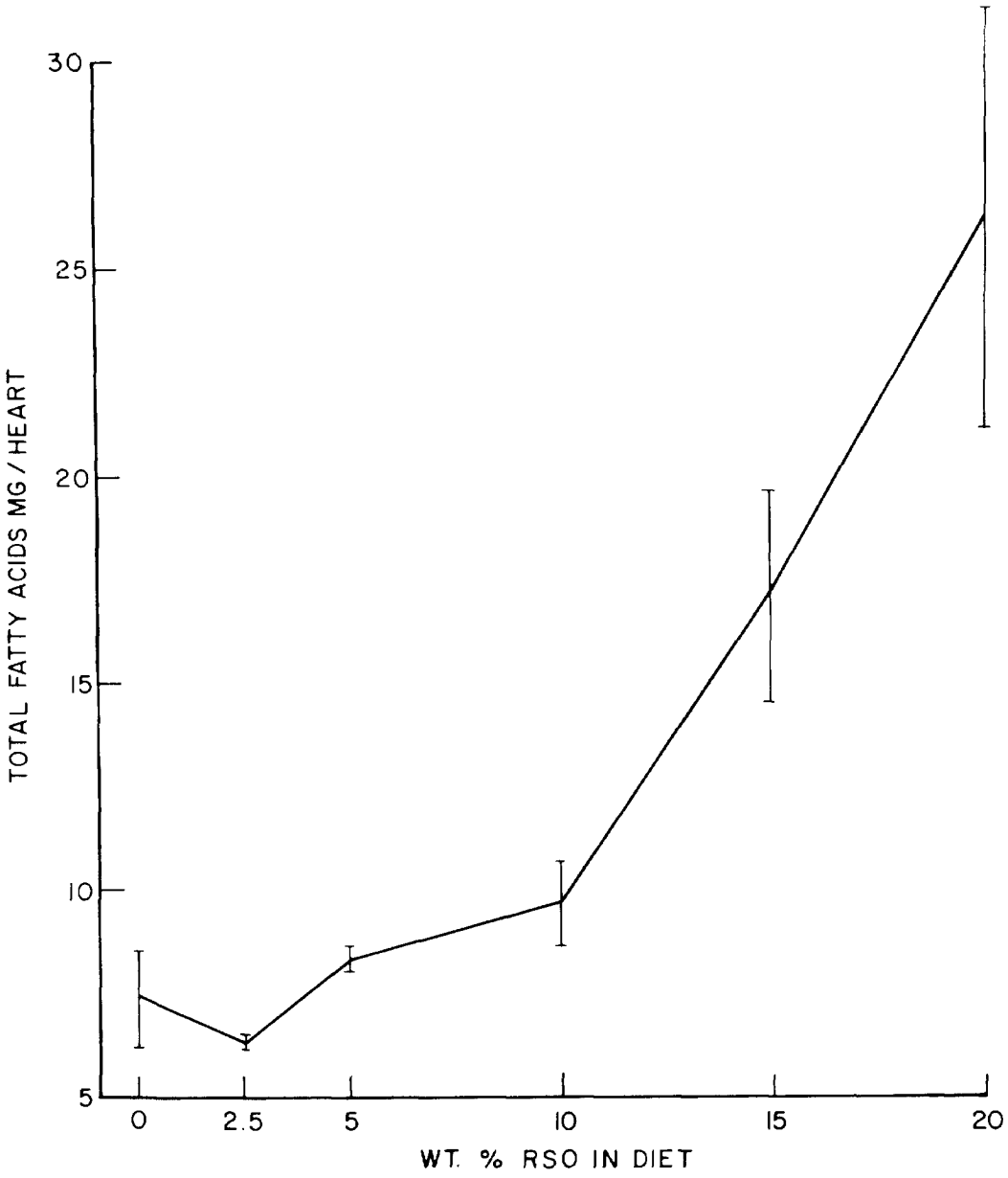

Fig. 4.

Total cardiac fatty acids of rats fed different levels of liquid rapeseed in a 20% fat diet.

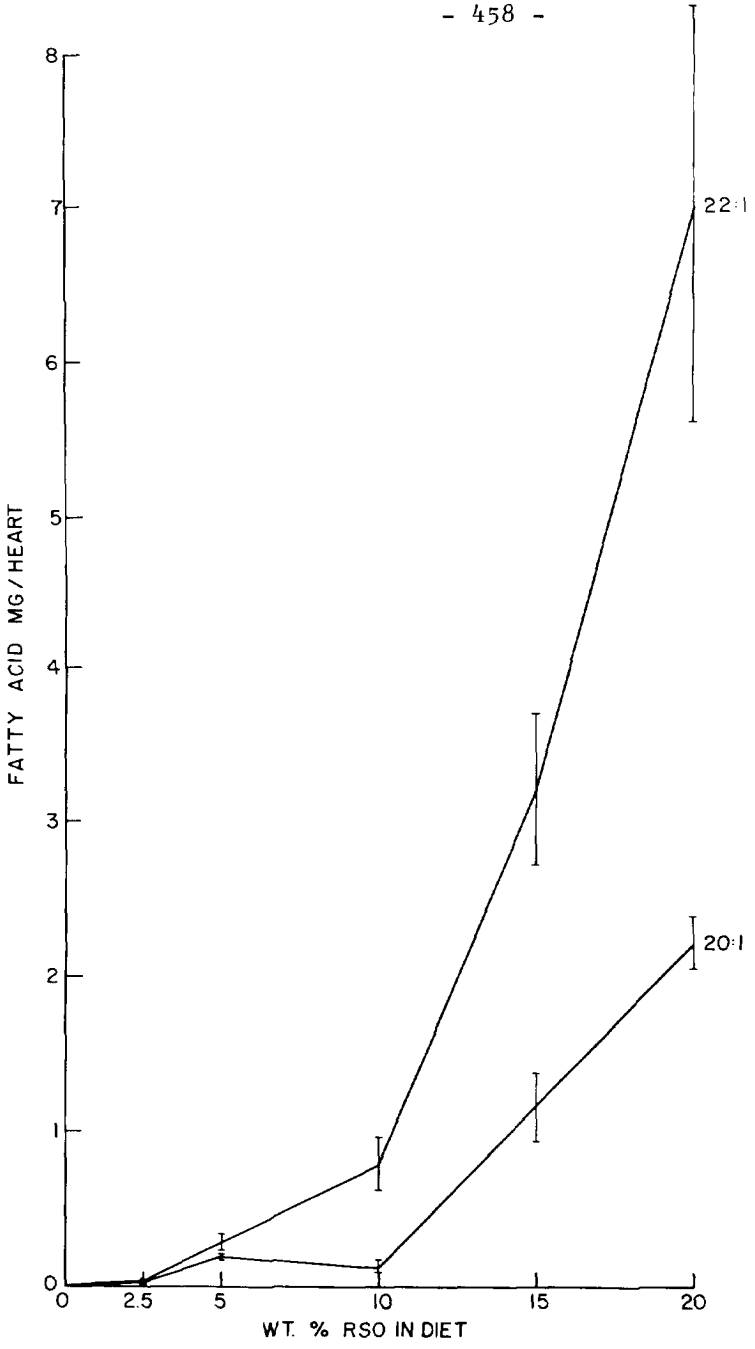

Fig. 5.

Deposition of eicosenoic acid (20:1) and erucic acid (22:1) in the cardiac tissue of rats fed different levels of liquid rapeseed oil.

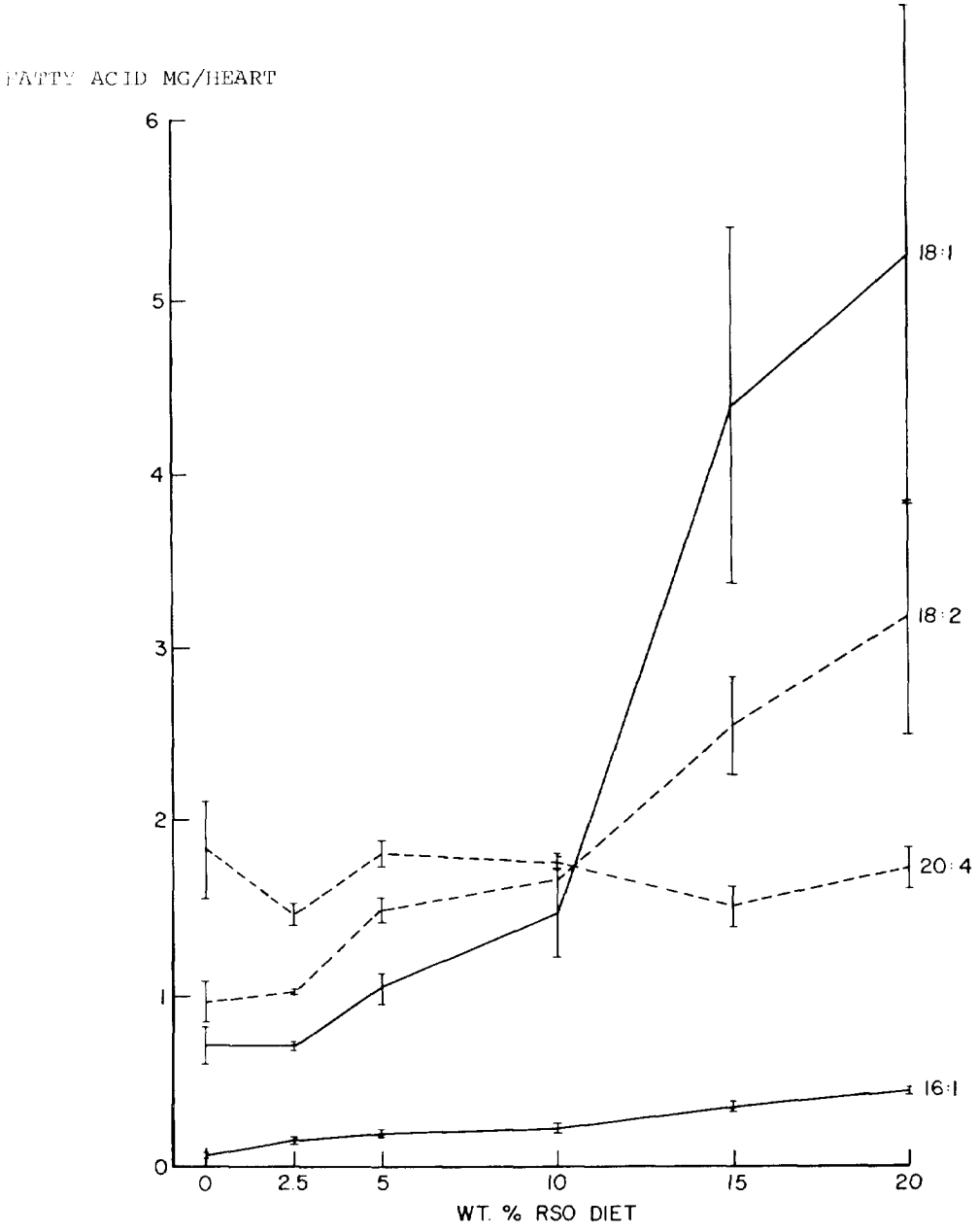

Fig. 6.

Deposition of palmitoleic acid (16:1), oleic acid (18:1), linoleic acid (18:2) and arachidonic acid (20:4) in the cardiac tissue of rats fed different levels of liquid rapeseed oil.

TABLE III

EFFECT OF AGE ON THE ERUCIC ACID DEPOSITION IN THE HEART OF RATS FED A 20% FAT DIET FOR ONE WEEK

Age of Rat(a)	Sex	No. of Rats	Diet	Wt. of Heart	Total Fatty Acids	Percent 22:1 in Fatty Acids
Wk				mg	mg/Heart	
4	M	5	Control	451 ± 17	7.4 ± 1.1	0
		5	RSO	465 ± 41	26.1 ± 5.0	26.3 ± 0.5
12	F	3	Control	850 ± 38	19.8 ± 4.2	0
		3	RSO	886 ± 54	23.4 ± 3.9	11.9 ± 1.8
	M	4	Control	1215 ± 44	22.6 ± 2.5	0
		4	RSO	1211 ± 119	30.9 ± 5.7	12.2 ± 3.3

(a) - Age at termination of feeding period.

concentration of $C_{22:1}$ in these lipids was appreciably lower in the older animal.

To determine if saturated fat might influence the amount of fat deposition, Table IV, weanling rats were fed 15% liquid rapeseed oil without added fat or supplemented with 5% hydrogenated coconut oil. This source of saturated fat was mixed with lard for the control group. On the basis of the amount of food consumed, the second group appeared to have received as much rapeseed oil, but the fatty acids deposited in the heart, while still appreciable, were significantly lower than those obtained with rapeseed oil alone. Is this apparent beneficial result from added saturated fatty acids similar to the effect observed after partial hydrogenation of the oil? It is known that saturated fatty acids added to rapeseed oil alter the non-cardiac responses to this oil which is high in $C_{22:1}$ and low in saturates(6). At this point it is interesting to speculate that saturated fatty acids reduce the cardiac lipid deposition associated with an intake of long-chain fatty acids.

The necrotic lesions reported for long-term feeding of liquid rapeseed oil have also been observed (Table V). After 20 weeks of receiving 20% of this oil, the level of erucic acid in the cardiac lipids was low. One of the rats fed olive oil showed cardiac myocytolysis, but necrotic lesions were observed in 12/14 of the rats fed liquid rapeseed oil. The lesions appeared to be at various stages of development.

To ascertain if necrosis was the result of early fat deposition, rats were fed 20% by weight of liquid rapeseed oil to produce the peak deposition of cardiac lipids and then fed a control mixture of lard and corn oil for 9 weeks. At that time histopathological examination of the hearts failed to distinguish them from those of rats which had at no time received rapeseed oil. Under these conditions the necrotic lesions appeared not to develop from the early fat deposition, but rather from the continuous exposure to a particular mixture of fatty acids containing C_{22} monoenes.

Besides the rat, the mini-pig and the squirrel monkey have been fed rapeseed oil for one week to determine the deposition of cardiac lipids. In Table VI are some data from 12 mini-pigs 10 days past being weaned. They were fed 0, 10 or 20% liquid rapeseed oil in which all diets were made to contain 20% fat through the addition of the lard and corn oil mixture. The levels of fatty acids deposited were not significantly different. Unlike the rat, the mini-pig deposited as much $C_{20:1}$ as $C_{22:1}$.

The highest proportion of $C_{22:1}$ deposited was but 1.9% of the total fatty acids. The age of the animal may have had some

TABLE IV

CARDIAC FATTY ACIDS IN RATS FED RAPESEED OIL (RSO) AND HYDROGENATED COCONUT OIL (HCCO)

Diet	Food Intake	Total Fatty Acids		22:1	
	g/wk	mg/g Heart	mg/Heart	mg/Heart	Percent of F.A.'s
15% RSO	55.7 ± 1.2	49.6 ± 2.7	20.4 ± 0.4	4.2 ± 0.3	20.3 ± 0.9
15% RSO + 5% HCCO	57.7 ± 3.8	34.6 ± 1.4	14.2 ± 1.5	2.4 ± 0.6	16.2 ± 0.3
15% lard + 5% HCCO	63.7 ± 2.3	19.1 ± 1.4	8.3 ± 0.7	- -	- -

TABLE V

CARDIAC CHANGES IN RATS FED 20% FAT DIETS FOR 20 WEEKS

Dietary Fat	CARDIAC LIPIDS		Positive H.P.S.
	Percent 22:1	Positive Fat Stain	
20% Olive Oil	-	0/11	1/11
20% Rapeseed Oil	3.8	7/14	12/14

TABLE VI

CARDIAC FATTY ACIDS IN MINI-PIGS FED 20% FAT DIETS FOR ONE WEEK

Percent RSO in Diet	TOTAL FATTY ACIDS	20:1	22:1
	mg/g Heart	Percent of F.A.'s	Percent of F.A.'s
0	12.3 ± 2.0	0.6 ± 0.1	- -
10	21.0 ± 6.2	1.3 ± 0.1	0.9 ± 0.3
20	24.0 ± 6.3	2.4 ± 0.3	1.9 ± 0.5

influence on its ability to metabolize long-chain fatty acids, but at no time was such a low level observed in the rat.

The results from similar levels of rapeseed oil fed to the squirrel monkey for one week are shown in Table VII. Here there were but three animals killed per group, but it is interesting that none of them produced the type of cardiac lipid deposition observed in the rat. Histopathologically, cardiac fat droplets appeared in all groups, but chemically the amount of fat appeared not to be excessive.

It is apparent that the cardiac tissue of the rat, particularly the young rat, is unable to metabolize readily long-chain fatty acids, and that the effects obtained with liquid rapeseed oil are reduced by partial hydrogenation or the addition of saturated fat. To further the studies in this susceptible species, the specificity of the fatty acids which cause changes in cardiac lipids will be investigated with simulated oils being prepared by Dr. B.M. Craig in Saskatoon. It is hoped that through various biochemical approaches we can learn more about the cardiac metabolism of long-chain fatty acids and the factors which modify their action.

TABLE VII

CARDIAC FATTY ACIDS IN SQUIRREL MONKEYS FED 20% FAT DIETS FOR ONE WEEK

Percent RSO in Diet	TOTAL FATTY ACIDS mg/g Heart	20:1 Percent of F.A.'s	22:1 Percent of F.A.'s
0	34.9 ± 8.1	0.5 ± 0.1	- -
10	44.0 ± 8.5	1.6 ± 0.4	2.0 ± 0.4
20	25.8 ± 5.0	3.0 ± 0.6	4.9 ± 0.7

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QUESTIONS AND ANSWERS — SESSION VI — ON NUTRITIONAL PROPERTIES OF RAPESEED OIL

1) QUESTION: I would like to ask some practical questions directed at Dr. Abdellatif and Dr. Vles. Were they laboratory prepared oils or plant produced oils which you used in your experiments? Secondly, the procedure, was it pressed or pre-pressed extracted, refined bleached, deodorized oils that you were using? In the case it was plant prepared oil, could anything have come into the oil from the plant? Have you analyzed for that? Thirdly, have you made any tests with partially hydrogenated rapeseed oil and what is your opinion about that compared to what Dr. Beare-Rogers said in her last speech? Fourth question, did you add any lecithin or phosphatides to the diet, also vitamins and minerals? This is interesting in connection to what was presented before, namely that crude oil and refined oil had different effects on hens.

ANSWER: (Dr. R.O. Vles)

The rapeseed oil that we used in all our experiments were all indeed plant-refined oils from different origin. There were Polish, there were French, Swedish. As to the last question, we added vitamins and also extra choline to the diets. With regard to partially hydrogenated oils, we obtained the same figures as Dr. Rogers.

2) QUESTION: I think that I have asked this question before of Dr. Abdellatif. What was the cholesterol content of the guinea pig erythrocytes?

ANSWER: (Dr. A.M.M. Abdellatif)

We have some data. The most important feature was that there was no difference in serum cholesterol content between the three groups.

3) COMMENT OF Dr. B.L. Walker

Can I make just one other comment? In a way I would like to come a little bit to the defense of rapeseed oil. There have been a number of comparisons made of tissue lipids, feeding rapeseed oil and various other oils. You can draw any conclusion you want from this depending on what you choose as your control. You have to remember that there is linolenic acid in there, I think that Dr. Rocquelin was using palm oil which is

3) COMMENT OF Dr. B.L. Walker (Cont'd.)

very low in linolenic, and when going to peanut oil one really influence the content of polyunsaturated fatty acids, and what you get out of there. If, for instance, you use olive oil, you can get surprisingly similar results with rapeseed zero-erucic and olive oil, as far as fatty acid composition is concerned. Not in all tissues, with tissues where you are getting high levels of erucic acid going in, such as the adrenal, you do upset the balance. But in the majority of tissues you get quite a similar pattern, it is quite surprising, at least as far as the major polyunsaturates, the arachidonic are concerned. You do get lower levels of the 22:5 and 22:4, fortunately. But I think the influence here is the linolenic, and not the erucic acid that is doing an awful lot of damage.

COMMENT of Dr. R.O. Vles

Yes, I would like to comment, and I would indeed like to find something to defend rapeseed oil. But for 10 years we have been studying the pathology of an enormous amount of material of animal tissues fed all kinds of oils and fats, and the long-term effects of feeding butterfat or feeding soybean oil or feeding corn oil, or feeding coconut oil, or olive oil. We have some data now for Canbra oil. I would like to extend this in other animal species but really we never found at short-term such fatty infiltration. There is no question. Even linseed oil, 50 cal.percent of linseed oil fed for 3 months gave no change.

4) QUESTION: If my figures are correct, Poland is consuming 18 kilos per capita per year of which 35 percent, or 4½ kilos is rapeseed oil. Now, my question is, how is it that Poland is one of the countries having the lowest death rate from heart diseases?

ANSWER: (Professor A. Rutkowski)

In our country there are no significant results about the correlation between heart diseases and consumption of erucic acid or consumption of rapeseed oil.

COMMENT by J.A. Campbell

Well, Mr. Chairman, this question of correlating or relating rat data to humans is a very difficult problem particularly in this case. Now, what we normally do in toxicological studies, is to express the intake on a per kilogram basis because of the different rates of

COMMENTS by J.A. Campbell (Cont'd.)

metabolism of say rats and humans. When we do that we end up, and I will not go through the full details, we end up with a safety factor of about 20 in the intake of rapeseed oil we feel that humans consume. It can also be calculated that the average intake, I believe, amounts to about 7 grams per day of rapeseed oil which would be 63 calories, which would then be 3 percent of the caloric intake. Now the situation is, that it is really difficult to say which one of these or which approach you should take in this case.

I might just make a couple more comments. For those of you who may be interested in how we arrived, when we arrived at this decision, I would just like to very briefly go back over the various events that have come up. As you know, in the Food & Drug Directorate we have been interested in this for a long time. In about April of this year we became particularly concerned that we are getting results which had rather important and rather serious implications. On June 25th, we called in experts, a pathologist from the University of Ottawa, we had the opportunity of having Dr. Vergroesen from Vlaardingen in Holland and others to discuss this whole matter interdepartmentally. This brought the other Departments into the situation. On July 28th, we contacted the rapeseed oil industry in Canada and briefly briefed them on the situation and indicated our position to them at that time. Now as you know, and I notice you have copies there of the statement which was made by our Minister of Health on August 12th, which describes the situation. I might also read to you a statement which we have prepared before this meeting which in essence says the same thing as that, but to bring you up-to-date, this is our feeling at this time. Evidence obtained in the research laboratories of the Food and Drug Directorate indicates that rapeseed oil containing 30 percent erucic acid when fed to young rats at levels in excess of 5 percent by weight in the diet results in a transient accumulation of fat in the heart. You have seen all the rest of the data this morning. The magnitude of this accumulation is directly related to the amount of rapeseed oil fed and is reduced by the presence of saturated fats. Prolonged feeding of rapeseed oil results in necrotic changes in the heart. The effects observed appear to be related largely to the erucic acid content. There is evidence from other laboratories which you have heard, which confirms these results and indicates that erucic acid containing rapeseed oil interferes with several enzyme systems and has other harmful effects in other animal species. No harmful effects on humans have been attributed to the consumption of this oil.

COMMENTS by J.A. Campbell (Cont'd.)

Canbra oil, containing essentially no erucic acid, does not cause fatty accumulation in the heart. Since rapeseed oil is never used as the sole source of that in the Canadian diet, and is consumed at levels lower than those fed to rats, the likelihood of harm resulting from its use is extremely small. Nevertheless, the potential hazard cannot be overlooked, and it is considered prudent as a sound public health measure to replace erucic acid containing rapeseed oil with Canbra oil as soon as practical. Now, as soon as practical means in essence what it says, you will note that I have not specified any dates, I have not specified any amounts or restrictions on the type of oils to be used or the levels which can be used. And I think that from the results that have been heard here this morning, I would not see any need to change this policy at this time.

5) QUESTION: I would like to ask Mr. Salmon if the digestibility and absorption of fat in the turkey is considered to be that which is classically expected for the monogastric animal?

ANSWER: (Mr. R.E. Salmon)

I really haven't looked at the data for other monogastric animals to be able to answer that question, properly. Digestibility of these fats for the turkey as compared with the chicken differs somewhat in relation to figures I have seen. At two weeks in particular the digestibility of total fatty acids and also individual fatty acids seem to be very much lower in the turkey than in the chicken, although I have to rework this data because the fat content of the excreta was so high that we had some difficulty in handling these samples.

QUESTIONER: Yet, you reported a very high digestibility of erucic acid, which is quite contrary to what we were seeing with pigs, we are seeing a somewhat lower digestibility of erucic acid or apparent digestibility of erucic acid, which coincides with the generalization made by our Chairman of the nutrition section of 40 some years ago, in which one bond of unsaturation would be equivalent to about shortening of the chain by 6 carbons In other words you would expect erucic acid to have some what the same digestibility of palmitic, and we are in fact, finding this precisely. We are not finding anything like 90-95%.

At two weeks it is true, that digestibility of erucic acid was down to around 50-60 percent but it seems to be very much more absorbed at the time the birds were six weeks old.

6) QUESTION: I would like to ask Dr. Walker, I realize that there is some variability in their data, but it seems to me that the rapeseed oil is really improving the digestibility of tallow although he continued to refer to it into the opposite direction.

ANSWER: (Dr. S.J. Slinger)

Actually, you are both right. In our calculations the synergism we got when we mixed tallow and rapeseed oil was so great that we had to have some improvement in the digestibility from both oils. Incidentally, in partial answer to Dr. McDonald, we did find a somewhat higher metabolizability of the energy in rapeseed oils for the turkey than for the chicks. And again, we found this synergism going right across the board but the turkey does seem to be able to utilize rapeseed oil, particularly the undegummed rapeseed oil very very well. I take it that the gum in the oil is something that needs more attention. We certainly got very definite growth depression due to the refining, and we are now getting different oils refined, following each step in the refining procedure to find out exactly what this is due to. There maybe something deleterious about this refining procedure that needs further study.

7) QUESTION: I would like to return to the possibility of potential hazard of rapeseed oil in the human diet. I am interested in the effect of the dietary status of the animal on the pathological effect of toxic compounds. My interest stems from some work which we have been conducting over the years. In the first place I would mention that some time ago there was concern with respect to the possible toxicity of fish oils produced in certain ways and from different sources. And we found that for poultry at least, the reported toxicity in the literature was actually due to induction of various deficencies of vitamin B complex in the diet. Secondly, more recent work, not just in our laboratory but in other laboratories, has indicated that the protein level particularly of the diets that are fed has a very important bearing on the response to potentially toxic compounds, and in the case of the rat where they are testing for toxicity, provided the primary compound introduced into the diet is the toxic one and not a secondary metabolate of this compound. If the protein level of the diet is raised, they find that they get a much lesser degree of toxicity evidence by the inclusion of a given level of a toxic compound. Also, in rats that are fed toxic substances the disastrous effect of toxic compounds is very much greater in the case of animals that are fed purified and semi-purified diets.

7) QUESTION: (Cont'd.)

I would like to ask Dr. Vles, if he has any further comments to make on his results of his experiments in which he showed a lessening of the toxicity when he raised the protein level of the diet?

ANSWER: (Dr. R.O. Vles)

I would be very happy to give some comments to this important and interesting question. We have asked too, whether we could influence this type of lesion with different factors?

With reference to your fish oils, it is known that unhardened fish oil increases the need of Vitamin E and that the first thing we did with ducklings was to give to those ducklings ten-fold high Vitamin E levels and we found just the same degree of fatty infiltration. This is as far as Vitamin E is concerned. We investigated the levels of Vitamin B complex in all our diets. They were according to the general data about this question optimal. As far as the protein level is concerned, we used 26 calorie percent of protein which is optimal according to Hegstedt for rats. We used also lower protein diets in rats, and we did not find any quantitative difference in the score of fatty infiltration. In ducklings, as you saw from this data, we increased the level of protein up to 30 percent protein, which is optimal for ducklings. We even studied the soy protein, and we found qualitatively the same effect. Quantatively you saw the results. There was a little decrease of fatty infiltration, but still the fatty infiltration was present.

8) QUESTION: Did you ever add the oil to a commercial diet as opposed to synthetic diets, and are the results influenced by that?

ANSWER: (Dr. R.O. Vles)

You mean to add the oils to commercial diets? The problem is, that when you are adding fats to commercial diets you get relatively too low a protein level. It is rather dangerous to manipulate commercial diets, which are balanced. When you are giving 20-30-40 caloric percent fat, you get a shift in the proportions of the carbohydrates, the fats and proteins. Therfore, we do not like to use this system.