

SOME NUTRITIONAL STUDIES ON RAPESEED OILS

J. L. Beare-Rogers, E. A. Nera and H. A. Heggtveit

The extent of the early lipidosis in rats fed rapeseed oil was shown to be proportional to the intake of erucic acid (BEARE-ROGERS et al., 1971). The phase of triglyceride accumulation, although temporary, was a period when a stress such as cold exposure was associated with a high mortality. Under such a condition, rats fed low-erucic rapeseed oil survived well (BEARE-ROGERS and NERA, 1974).

Long-term lesions in rats fed either high-erucic or low-erucic rapeseed oils have been described (ROCQUELIN and CLUZAN, 1968; ROCQUELIN et al., 1973; KRAMER et al., 1973). Our first evidence of necrosis or fibrosis with canbra oil (rapeseed oil containing less than 5 % erucic acid) was obtained in an experiment in which 3/12 hearts were affected. The controls then appeared to be unaffected while 9/11 of those fed a high-erucic oil had lesions. The level of sphingomyelin, in  $\mu\text{g}/\text{heart}$ , was  $26 \pm 3$  in the controls,  $70 \pm 11$  in rats fed canbra oil containing 1.4 % erucic acid and  $145 \pm 8$  in those fed rapeseed oil containing 38.1 % erucic acid.

Canbra oil was again studied in a larger experiment (Table 1). Rapeseed

Table 1: High and low erucic oil and oil mixtures and their effect on the incidence of cardiac lesions after 16 weeks

<u>Dietary fat</u>	<u>Cardiac lesions</u>
Lard: CO <sup>a</sup> (3:1)	0/17
20 % LRSO <sup>b</sup>	15/15
20 % HRSO <sup>c</sup>	10/17
20 % LCBO <sup>d</sup>	10/16
10 % LCBO, 10 % OO <sup>e</sup>	4/17
18 % LCBO, 2 % PO <sup>f</sup>	4/15
18 % LCBO, 1 % CO, 1 % PO	7/17
18 % HCBO <sup>g</sup> , 2 % CO	7/17
16 % HCBO, 4 % CO	6/13

<sup>a</sup>CO, corn oil

<sup>b</sup>LRSO, liquid rapeseed oil (38.1 % 22:1)

<sup>c</sup>HRSO, hydrogenated rapeseed oil (I. V. 78; 33.1 % 22:1)

<sup>d</sup>LCBO, liquid canbra oil (2:9 % 22:1)

<sup>e</sup>OO, olive oil

<sup>f</sup>PO, palm oil

<sup>g</sup>HCBO, hydrogenated canbra oil (I. V. 78; 2.9 % 22:1)

oil containing 38.1 % erucic acid, partially hydrogenated rapeseed oil containing 33.1 % or liquid canbra oil containing 2.9 % were tested as the only source of fat in the diet. This canbra oil was tested in combination with 10 % olive oil, 2 % palm oil or a mixture of corn oil and palm oil (1:1); partially hydrogenated canbra oil was tested with 2 or 4 % corn oil. All rats fed the high liquid rapeseed oil had cardiac lesions. Similar frequencies of lesions were observed with hydrogenated rapeseed oil and with liquid canbra oil. There appeared to be an advantage in diluting the latter with 10 % olive oil or just 2 % palm oil, although any benefit from the small addition of corn oil or palm oil appeared questionable, as was the effect of hydrogenation of this canbra oil. The hydrogenated rapeseed oil fed without additional linoleic acid was expected to produce an essential fatty acid deficiency, but did not make the myocardium more susceptible to lesions.

An oil from a low-erucic *Brassica napus*, Oro, containing 0.6 % C 22:1 was fed as 0, 5, 10, 15 or 20 % of the diet. The 3:1 mixture of lard and corn oil was used to make each diet 20 % fat. An additional group of rats received 18 % Oro oil 2 % palm oil. The experiment was replicated in time, once with Charles River rats and once with rats obtained from Bio Breeding Ltd., Ottawa. Half of the rats were killed at 16 weeks and the rest at 26 weeks.

Sixteen weeks later there were background lesions and little evidence of a dose response (Table 2). A similar experiment with span oil, a *Brassica campestris* oil with 2.7 % erucic acid produced a dose response with the highest incidence of lesions associated with the highest dietary levels (Table 3).

**Table 2:** Varying levels of dietary Oro Oil

Oro Oil %	Cardiac Lesions					
	CR <sup>a</sup>	16 wk BB <sup>b</sup>	Total	CR <sup>a</sup>	26 wk BB <sup>b</sup>	Total
0	3/10	1/10	4/20	1/9	2/9	3/18
5	5/10	1/10	6/20	0/10	2/10	2/20
10	1/10	1/10	2/20	1/9	2/10	3/19
15	5/10	6/9	11/19	4/10	2/9	6/19
20	7/10	2/10	9/20	1/9	3/10	4/19
18 + 2PO <sup>c</sup>	6/10	4/10	10/20	1/8	2/10	3/18

<sup>a</sup>CR - Charles River rats

<sup>b</sup>BB - Bio Breeding rats

<sup>c</sup>PO - Palm Oil

**Table 3:** Varying levels of dietary Span Oil

Span Oil %	Cardiac Lesions					
	CR <sup>a</sup>	16 wk BB <sup>b</sup>	Total	CR <sup>a</sup>	26 wk BB <sup>b</sup>	Total
0	1/10	2/10	3/20	0/9	0/9	0/18
5	1/10	1/10	2/20	0/9	1/10	1/19
10	4/10	3/10	7/20	4/10	3/10	7/20
15	4/10	5/10	9/20	5/9	4/10	9/19
20	5/10	6/10	11/20	6/10	7/10	13/20
18 + 2PO <sup>c</sup>	5/10	8/10	13/20	5/9	2/9	7/18

<sup>a</sup>CR - Charles River rats

<sup>b</sup>BB - Bio Breeding rats

<sup>c</sup>PO - Palm Oil

A pressed oil containing 2.1 % C22:1 was tested at the 10 and 20 % level (Table 4). The results for this oil were similar to those for the span oil, and indicated that the method of extraction of the oil had little effect when the test oil was the only dietary fat fed.

**Table 4:** Two levels of protein (20 and 25 %) with low erucic oil

Dietary Fat (20 % of Diet)	Cardiac Lesions			
	20 % protein	25 % protein	Total	
Lard: Corn oil (control)	1/12	1/12	2/12	4 * /36
10 % CBO + 10 % Control	0/12	3/12	4/11	7 * /35
20 % CBO	4/12	6/12	5/12	15 /36
20 % Span 0	5/12	6/12	7/12	18 /36

\* Minor lesions

Other Oro, Span and rapeseed oils tested were similar to those studied by KRAMER et al. (1973). The crude Span oil was compared with the corresponding refined oil which had also been tested for the previously described dose-response.

All of the Brassica oils shown in Table 5 were significantly different from the controls in their effect on cardiac lesions. To determine if a factor arising from the processing of the oil might be promoting lesions, crude Span oil was compared with refined oil, and another crude canbra oil with the corresponding refined oil. No processing step of the crude oil appeared to aggravate the cardiac lesions. The last oil, a commercial vegetable oil, contained 23 % erucic acid and was associated with relatively high incidence of cardiac lesions.

**Table 5:** Brassica oils of different levels of erucic acid

Dietary fat (20 % of diet)		Cardiac lesions	
		Total	%
	22 : 1 %		
Lard: corn oil (3:1)	0	4/56	7
SBO	0	4/27	15
Oro oil	1.9	10/27	37
Span oil	3.4	16/27	59
RSO	23.3	21/29	73
Crude Span Oil	2.5	10/18	56
Refined Span Oil	2.7	11/19	58
Crude CBO	2.5	11/20	55
Refined CBO	2.5	7/20	37
Comm. Veg. oil	23.2	14/19	74

Crude Zephyr oil, alkali refined and bleached oil, salad oil, and partially hydrogenated oil (I. V. 70) mixed with corn oil (3:1 w/w) had some differences in fatty acid composition (Table 6). These oils, crude Bronowski oil and a deodorizer condensate from a *B. campestris* oil, diluted in olive oil

**Table 6:** Zephyr oils, Bronowski oil and "deodorizer condensate" in olive oil

Dietary fat	% 22:1	Cardiac Lesions		
		I	II	Total
1. Lard: CO (3:1)	0	2/10	2/10	4/20
2. Zephyr O., Crude	0.3	6/10	5/10	11/20
3. Zephyr O., Alk. Ref. & Bl.	0.5	7/10	8/10	15/20
4. Zephyr O., Salad	0.7	2/10	5/10	7/20
5. Zephyr O., Hydrog., :CO	0.3	2/10	2/10	4/19
6. Bronowski O., Crude	16.4	6/10	8/10	14/20
7. Olive O.	0	0/20	1/10	1/20
8. <i>Campestris</i> -Deod. Cond.: OO	1.4	4/10	7/10	11/20

(1:4 w/w), were also tested. The crude oil and the alkaline-refined and bleached oil were associated with significantly more lesions than appeared in the hearts of rats fed the control diet. The salad oil which had also been deodorized appeared somewhat less cardiopathogenic and the partially hydrogenated oil mixed with corn oil, perhaps even less so. The latter oil was similar to the control.

Bronowski oil from seed lacking glucosinolates also caused a high incidence of lesions. When a "deodorizer condensate" in olive oil was fed, lesions were also present. Whether the low level of erucic acid per se or some other material in this fraction was responsible is not known. Thin layer chromatography of the material has shown it to be rich in sterol esters.

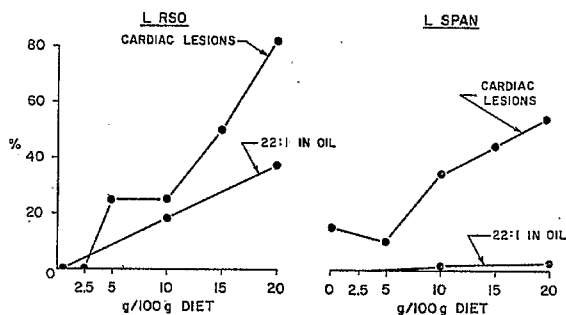
Hearts from rats fed mustard oil containing 33 % erucic acid had a high incidence of lesions (Table 7). Those in rats fed a new Span oil containing 0.2 % erucic acid were not statistically different in frequency from the controls, but 3 hearts had extensive pathology. Triglyceride fractions prepared by molecular distillation did contain small amounts of sterols. The proportions of erucic acid were 3.8 % in D<sub>5</sub>, 3.4 % in D<sub>6</sub> and 10.6 % in D<sub>7a</sub>. All of these fractions were associated with cardiac lesions, as was a rapeseed oil containing 18.7 % erucic acid.

**Table 7:** Mustard oil, Span oil, molecular distillates and rapeseed oil

Dietary Oil	Cardiac Lesions				
	Grade				Total
	1	2	3	4	
Lard:CO	2		1		3/20
OO	2	1		1	4/20
Mustard O	4	6	3	4	17/20
Span O (0.2 % 22:1)	4	1	3		8/20
Distillate D5	4	6		1	11/20
D6	8	4	6	1	17/20
D7a	5	7	4	1	17/20
RSO	5	2	6	3	16/20

ABDELLATIF and VLES (1973) provided convincing evidence that erucic acid per se was cardiopathogenic. The cause of lesion in rats from the feeding of canbra oil is not yet determined. A comparison of the dose-response obtained with a high-erucic rapeseed oil (BEARE-ROGERS and NERA, 1972) and with Span oil (Fig. 1) indicates that the frequency of lesions is related to the level of test oil and not to its content of erucic acid. If another factor is involved, it is hoped that it can be readily removed.

**Figure 1:** Relationship between frequency of cardiac lesions, the level of test oil in the diet and its proportion of erucic acid. On the left, liquid rapeseed oil (7); on the right, liquid span oil



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