Nutritive Value of Rapeseed Oils

Effects on growth

There is the general agreement in the literature that rapeseed oil retards the growth of young rats in proportion to the amount in the diet (JACQUOT et al., 1969). This growth retardation becomes important at levels of rapeseed oil equal to or exceeding 15 % by weight of the feed which corresponds to 30 % of total dietary calories. These effects have also been found in mice and hamsters (THOMASSON et al., 1970), guinea pigs (VLES & ABDELLATIF, 1970), ducklings (ABDELLATIF & VLES, 1970 b), rabbits (ABDELLATIF & VLES, 1971 b), pigs (ROINE et al., 1960) and turkeys and chicks (SALMON, 1969 a; SHEPPARD et al., 1971; VOGTMANN et al., 1973; WALKER et al., 1970). In the past, the poor growth performances have been mainly related to decreased feed consumption. It has now become clear that the poor appetite of rats fed large amounts of rapeseed oil is only partly responsible for the retarded growth. The preference of the rat for oils and fats other than rapeseed oil becomes only of importance at dietary levels of 40 % of total calories and more (20 % by weight). From an impressive series of experiments performed in rats (CHENITI et al., 1967 a; DEUEL et al., 1948; HORNSTRA, 1972; PIPY et al., 1972; ROCQUELIN & LECLERCQ, 1969; THOMASSON, 1956; ZIEMLANSKI et al., 1972), pigs (McDONALD, 1972; ROINE et al., 1960; THORON, 1969), rabbits (ABDELLATIF & VLES, 1971 b) and birds (SALMON, 1969 b; SALMON, 1970; SHEPPARD et al., 1971; WALKER et al., 1970) it may be concluded that poor digestibility, lower rate of intestinal absorption and inefficient energetic utilization contribute to the lower nutritive value of rapeseed oil in comparison with other oils and fats and that erucic acid is the main factor responsible for these phenomena.

Low levels of saturated fatty acids or palmitic acid also characterize the fatty acid composition of rapeseed oil. The possibility that the growth retardation would further be due to the unbalanced ratio of unsaturated to saturated fatty acids or to the low level of palmitic acid, stimulated a series of experiments in various animal species. It appeared indeed to be possible to improve the growth of rapeseed oil fed animals by adding palmitic acid to the diet (ABDELLATIF & VLES, 1971 a; ABDELLATIF et al., 1972; BEARE et al., 1963 b; THOMASSON et al., 1970). ROCQUELIN et al. (1970) demonstrated this in rats only when the linoleic acid level of the fat mixtures was equal or lower than 10 % of the total fatty acid content. In ducklings fed constant amounts of erucic acid, an improvement in the growth was observed when the content of palmitic acid in the diet was increased at the expense of the unsaturated fatty acids (VLES & ABDELLATIF, 1970). These beneficial effects of palmitic acid are not related to any correction of the ratio saturated to mono-unsaturated fatty acids since low-erucic rapeseed oils, which are also unbalanced in this respect, do not impair the growth of rats and ducklings (ABDELLATIF & VLES, 1973 b; ROCQUELIN & CLUZAN, 1968; ROCQUELIN et al., 1970) and the addition of saturated fatty acids to these low erucic acid oils does not improve weight gains

(CRAIG & BEARE, 1968). The beneficial action of palmitic acid is due to a synergistic effect of erucic and palmitic acids on their digestibilities. When mixed in the diet, palmitic and erucic acid both show higher digestibilities than when fed singly (LALL & SLINGER, 1973; SALMON, 1970).

Longevity

Undernutrition or restriction of feed intake and feed utilization may increase the life-span of experimental animals. At dietary levels of 50 cal% rapeseed oil, the feed intake of rats distinctly decreases, so the life-span may be expected to be prolonged. In accordance with this hypothesis, THOMASSON (1955) observed indeed a longer life-span in rats fed rapeseed oil than in those fed butterfat. However, in later experiments, no relationship between fat content of type of fat on the one hand and the life-span on the other hand could be found (THOMASSON et al., 1970). Similarly, we fed rats large amounts of different fats during their life-term. We did not find significant differences either in mortality during the experiment, or in the average life-span between groups of 24 male Wistar rats fed 60 cal% coconut oil, butterfat, corn oil, soyabean oil, rapeseed oil or whale oil. In ducklings, however, we found impressive differences in mortality between the animals fed rapeseed oil, low-erucic rapeseed oil or sunflowerseed oil for 3 months. High mortality was found in the group fed rapeseed oil and no mortality at all was noted in the ducklings fed low-erucic rapeseed oil (ABDELLATIF & VLES, 1973 b). If it is true that, at least under ad libitum feeding conditions, high levels of rapeseed oil do not seem to shorten the life-span of rats, it should be emphasized again that in ducklings, a lowering of the erucic acid content of rapeseed oil included in the diet, has impressive effects on their survival.

Metabolism of erucic acid

The lack of information on the fate of erucic acid in mammalian tissue stimulated further research. Two approaches have been used to clarify the metabolic pathway of erucic acid. First, the studies on the incorporation of erucic acid into different tissue lipids and secondly those which traced the in vivo transformation and distribution of labelled erucic acid and measured its oxidation rate.

The results of incorporation studies show that the deposition of erucic acid in tissue other than adrenals and the heart is limited. In rats and pigs (CRAIG et al., 1963; HORNSTRA, 1972; KRAMER et al., 1973; ROCQUELIN et al., 1971; WALKER, 1972 a, b) the incorporation of erucic acid seldom exceeds 7 % of total fatty acids even when a diet high in erucic acid is fed. In young animals, the depot fat may show somewhat higher incorporation of erucic and eicosenoic acids (CRAIG & BEARE, 1968). In turkeys and chickens (SALMON, 1969 b, 1970) larger amounts of very-long-chain fatty acids may have been incorporated on body fat.

In animals, CARREAU et al. (1968) proved the in vivo conversion of erucic acid into oleic acid. So, erucic acid was considered to be neutralized after conversion into oleic acid, an endogenous fatty acid normally metabolized in the body. But the demonstration of the existence of this conversion did not give any idea about the rate of oxidation of erucic acid in comparison with other fatty acids. From the work of an impressive number of investigators (BACH et al., 1969; BOUCROT & BEZARD, 1973; CARROLL, 1962 b, 1966; CHRISTOPHERSEN & BREMER, 1972; KETEVI et al., 1973; LAPOUS et al., 1970; LEMARCHAL et al., 1972; MARTINELLI et al., 1973; PINSON & PADIEU, 1973; SWARTTOUW, 1974) it may be concluded that erucic acid is metabolized more slowly than oleic acid and palmitic acid, that after a single administration, erucic acid disappears quickly from various organs except from spleen and heart and that erucic acid is converted not only into oleic acid but also into shorter and longer mono-unsaturates.

Physiopathological effects of rapeseed oils

In the fifties, changes in the reproductive organs and in the adrenals were reported in rats fed erucic acid (CARROLL, 1951; CARROLL & NOBLE, 1957). There is conclusive evidence (ABDELLATIF & VLES, 1971 b; BEARE et al., 1959; CHENITI et al., 1967 b; ROCQUELIN & CLUZAN, 1968; THORON, 1969) that the testicular degeneration was in fact due to the lack of essential fatty acids in the experimental diet and not to a specific effect of erucic acid or rapeseed oil. On the other hand, the changes observed in the adrenals were more pertinent. Biochemical analyses of the adrenals revealed large amounts of cholesteryl esters with a high erucic acid content (CARROLL, 1962 a). The functional consequences of these biochemical findings seem to be far reaching since recent studies (BEARE et al., 1963 a; BEARE-ROGERS, 1972; CARNEY et al., 1972; WALKER & CARNEY, 1971) indicate that these changes could well account for the low growth performances and high mortality of rats fed rapeseed oil and submitted to a cold stress of 4° C. This susceptibility to stress appeared to be clearly related to the presence of erucic acid in the diet.

The first systematic study of the histopathological effects of feeding rapeseed oil in rats and pigs is that of ROINE et al. in 1960. Their results in pigs are not conclusive owing to the similarity of the lesions found in test and control groups. In rats, however, feeding large amounts of rapeseed oil induced changes in the heart which did not occur in the control animals. In 1968, ROCQUELIN & CLUZAN confirmed these findings and in 1970 we showed that these lesions were preceded by severe intracellular lipid deposition (Fig. 2) which explains the creamy colour of the heart on autopsy after a 3 day's period of feeding (ABDELLATIF & VLES, 1970 a). These phenomena have been confirmed in the following animal species; mice, gerbils, hamsters, guinea pigs, rabbits, pigs, ducklings, chickens and squirrel monkeys (BEARE-ROGERS & NERA, 1972; THOMASSON et al., 1970). Biochemical studies (BEARE-ROGERS, 1970; HOUTSMULLER et al.,

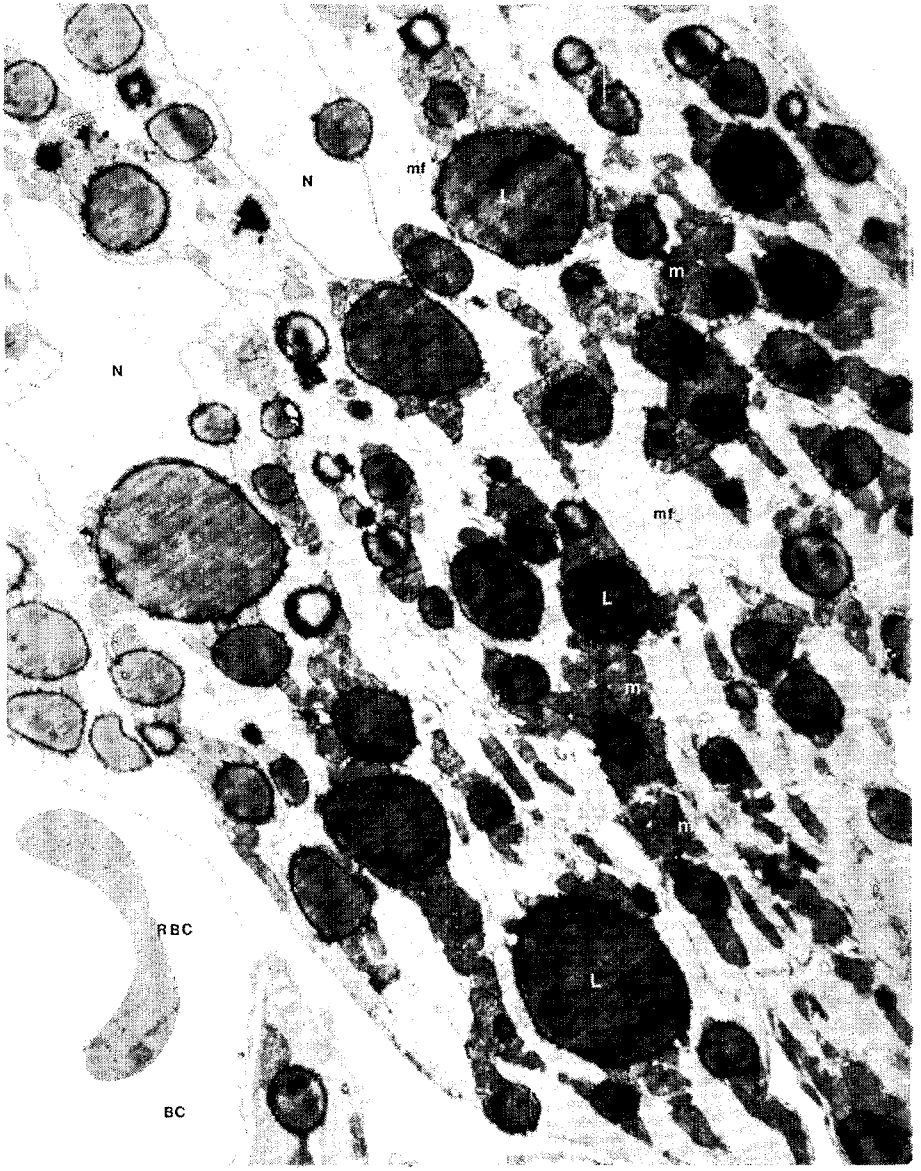

Fig. 2: Rat heartmuscle (initial magnification 9.000x). Fat deposition after feeding 50 cal% rapeseed oil for 3 days. (Courtesy of Dr.F.C.Jager.)

L	= Lipid droplet	RBC	= Red Blood Cell
N	= Nucleus	m	= mitochondrion
BC	= Blood Capillary	mf	= myofibril

1970; ROCQUELIN, 1970, 1973; ROCQUELIN et al., 1973) have largely confirmed these histopathological findings and have shown that the fatty deposition is not permanent; it reaches its maximum after 3 to 6 days after which it decreases, the free fatty acid fraction remaining relatively large during the feeding period (HOUTSMULLER et al., 1970; STRUIJK, 1972). Isomers of erucic acid also induce fatty deposition and experiments with rats fed very-long-chain fatty acids from various natural and synthetic sources (BEARE-ROGERS et al., 1971, 1972 a; BEARE-ROGERS, 1972 b; HOUTSMULLER et al., 1972) have shown that the dietary intake of docosenoic acids (22 carbon atoms and one double bond) is the most important factor in the accumulation of cardiac triglycerides.

The decrease of heart triglycerides during continued feeding of rapeseed oil is unfortunately followed by the development of degenerative changes in the heart muscle (necrosis and fibrosis). If it is clear that erucic acid and its isomers play an essential role in the genesis of the lipid accumulation, the assumption that they are also fully responsible for the longterm degenerative changes is still under discussion, since lesions have been reported in male fed low-erucic rapeseed oils for longer periods (BEARE-ROGERS, 1974; KRAMER et al.; 1973; ROCQUELIN & CLUZAN, 1968; ROCQUELIN et al., 1973). In order to clarify this problem, it may be useful to explain the mechanism of lipid accumulation and of its decrease. Electron microscopical studies of fatty hearts (Fig. 2) revealed the presence of many fat droplets in the close vicinity of mitochondria, strongly suggesting that something is going wrong at the mitochondrial site. These intracellular organelles synthesize ATP - adenosine triphosphate - which is the main source of cellular energy. The explosive decomposition of ATP is directly responsible for muscular contraction. HOUTSMULLER et al. (1970, 1972) studied in vitro heart mitochondria isolated from rats fed rapeseed oil. They showed that the rate of ATP synthesis decreased when the amount of rapeseed oil or erucic acid in the diet increased. But this functional alteration of the mitochondria appeared to be the consequence rather than the cause of the lipid accumulation. It points to the mitochondrial impact or site of action of very-long-chain fatty acids and Fig. 3 may help to explain the mechanism of fat deposition and the ensuing cellular changes.

Intracellular fat accumulation occurs when there is an imbalance between the influx of free ionized fatty acids (FFA) into the heart muscle cell and the rate of utilization of this fuel by the intracellular mitochondria. Adipose tissue, the chylomicrons originating from the intestine during digestion and the very-low-density lipoproteins (VLDL) synthesized in the liver are the carries and suppliers of the fatty acids. Lipolytic enzymes liberate these fatty acids from their esterified forms. The albumine in the blood binds transports the free fatty acids to the cellular site. An equilibrium exists between albumin-bound and uncomplexed fatty acids. The unbound (ionized) fatty acids are taken up by the cell and the binding capacity of albumin for a fatty acid may influence the influx of the latter. In the cell, there are three major steps in the utilization process of fatty acids by the mitochondria; activation, transport of activated fatty acid through a double mitochondrial membrane system and intramitochondrial β-oxidation.

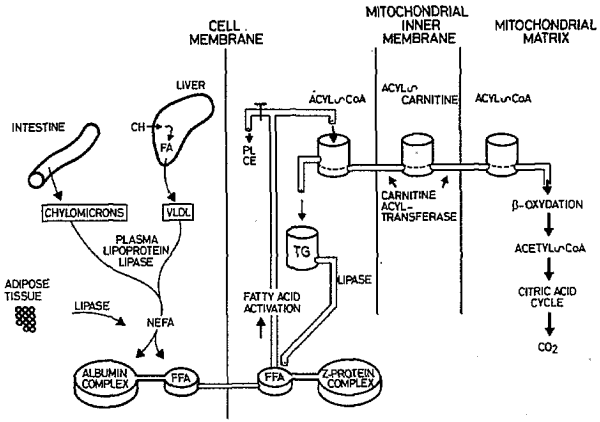

Fig. 3: Metabolism of fatty acids in the heart (courtesy of Dr. U. M. T. Houtsmuller).

CH	= Carbohydrates	FFA	= Free (ionized) Fatty Acids
FA	= Fatty Acids	PL	= Phospholipids
VLDL	= Very Low Density Lypoproteins	CE	= Cholesteryl Esters
		TG	= Triglycerides
NEFA	= Non Esterified Fatty Acids		

Erucic acid may induce an increased influx as a result of 2 factors:

1. A relatively high erucic acid level in the plasmatic free fatty acids. This shift in free fatty acid composition of the plasma towards erucic acid remains fairly constant during the whole rapeseed oil feeding period.
2. The low binding capacity of albumin for this fatty acid.

Once in the cell, erucic acid will be submitted to the three metabolic steps; activation, transfer, oxidation. In vitro, it appears that all three steps show a weak activity with erucic acid, the most limiting step being in vivo the transport through the mitochondrial membrane (GUMPEN & NORUM, 1973; HOUTSMULLER et al., 1972; SWARTTOUW, 1974). If the mitochondrial penetration is reduced, the acyl ∿ CoA (activated fatty acids) in excess will be re-esterified and will deposit as triglycerides (TG) in the cell. This deposition may be considered as the witness of a defence mechanism. In time, the cell loses these unusual foreign molecules with the aid of lipolytic enzyme systems which will activate the hydrolysis of the accumulated triglycerides and the liberated free fatty acids will join and increase their pool resulting in re-esterification and re-hydrolysis.

Intracellularly, the Z-binding proteins (fatty acid binding protein) may have lower binding capacity for erucic acid. When the concentration of these fat-

ty acids exceeds the binding capacity of the proteins (plasmatic albumin and cellular Z-protein) they may become toxic and induce cellular lesions. In this context, it is conceivable that long-term feeding of low levels of very-long-chain fatty acids in a fat-rich diet might in a susceptible animal like the male rat, lead to some cumulative irritation and subsequently to degenerative changes. There are some arguments and experimental facts in favour of this hypothesis. The comparison of the long-term effects of high and low erucic acid rapeseed oils shows that incidence and severity are less in animals fed low-erucic rapeseed oil (ABDELLATIF & VLES, 1973 a). The changes after feeding low-erucic rapeseed oil are, when present, clearly delayed. Table II summarizes the results of an experiment in which the lesions which were found were minor lesions. Only after 6 months were they no longer neglibible in 2 out of the 6 animals fed 2.8 cal% erucic acid.

Table II: Incidence of cardiac changes in male wistar rats fed 40 cal% low erucic rapeseed oil and 20 cal% sunflowerseed oil (LER containing 7 % erucic acid), 60 cal% groundnut (GNO) or olive oil (OLO) for various periods of time (n=6)

	Experimental weeks											
	12			16			20			24		
Dietary groups	LER	GNO	OLO	LER	GNO	OLO	LER	GNO	OLO	LER	GNO	OLO
No lesions*	5	5	6	4	6	2	2	2	2	4	6	5
One minor lesion	1	1	0	2	0	4	4	4	4	0	0	1
Two or more lesions	0	0	0	0	0	0	0	0	0	2	0	0

* In transversal sections sampled at 5 different myocardial levels.

We have interpreted these residual lesions observed in male rats as the result of the long-chain fatty acids still present in the low-erucic oils. This assumption is supported by the results of a dose-response study with Sprague-Dawley rats (ABDELLATIF & VLES, 1973 a) in which no level of rapeseed oil containing 50 % erucic acid appeared to be really safe. Even the lowest dose in this fat-rich diet seemed to increase the frequency of changes which were also found in controls. Moreover, recent data (BEARE-ROGERS, 1974) indicate that it is improbable that any technological manipulation (extraction, refining, deodorizing, washing the seeds) may be held responsible for the residual changes. Neither saturated fatty acid content, sulphur content nor insaponifiable fraction seem to play any definite role

(ABDELLATIF & VLES, 1973 a). In fact, the specificity of the long-term lesions in the rat is still under discussion and one should be careful to draw definite conclusions.

There is no unanimity about the long-term effects observed in the heart of pigs fed rapeseed oils (ROINE et al., 1960; SVAAR, 1974; VODOVAR et al., 1973). It has been suggested that high fat diets as such might not be tolerated well by pigs and this important point needs urgent clarification. On the other hand, in ducklings (ABDELLATIF & VLES, 1973 b) which are highly susceptible to erucic acid, no lesions at all were found when large amounts of low-erucic rapeseed oil had been fed for a long time. Amoung all uncertainties, there is one certainty; lowering the very-long-chain fatty acid content of experimental diets lowers the incidence of cardiac lesions, and considering all facts studied up to now such as growth performances, feed efficiency, life-span, mortality rate, resistance to stress, heart muscle hypertrophy, effects of erucic acid in vivo and in vitro and comparative anatomical studies, one may state that the development of low-erucic acid rapeseed strains can be regarded as a major step towards improving the nutritional status of the oil. The residual effects found in the heart of male rats of some strains when feeding low rapeseed oils need clarification. Further systematic studies will enable proper identification of the cardiac changes and better understanding of their pathogenesis.

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