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THE COMPARATIVE EFFECTS OF LOW ENERGETIC
ACID RAPESEED OIL AND SOYBEAN OIL ON
ENERGY METABOLISM IN YOUNG ADULT MEN

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SHARON PARKER

A dissertation submitted to the Faculty of Graduate Studies of
the University of Manitoba in partial fulfillment of the requirements
of the degree of

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ABSTRACT

The comparative effects of low erucic acid rapeseed oil (RSO) and soybean oil (SO) on energy metabolism were investigated in 8 male subjects. Energy metabolism was assessed by monitoring both respiratory (oxygen consumption, ventilation rate, heart rate and respiratory quotient) and blood parameters (serum glucose, plasma lactate and pyruvate, serum free fatty acids, serum glycerol and the calculated lactate/pyruvate ratio). The 32-day metabolic study consisted of four experimental periods of 8 days each. Experimental period I served as a stabilization period during which time a mixed fat diet was fed. During experimental periods II and III the diet contained either RSO or SO as the sole dietary fat. The diets were fed in a cross-over experimental design. During experimental period IV, the subjects again received a mixed fat diet. On the 7th day of each experimental period all subjects cycled at a uniform work load of 70% VO_2 max. On the 8th day of each experimental period subjects cycled at an adjusted work load of either 60 or 80% VO_2 max. Each 30 minute exercise protocol consisted of a 5 minute resting period, followed by a 15 minute exercise period and a 10 minute recovery period. Respiratory measurements were monitored continuously throughout the exercise sessions at both uniform and adjusted work loads. Blood samples were taken during exercise sessions at a uniform work load during rest, with 30 seconds following exercise and at the end of the recovery period. The diet consisted of ordinary foods with minimal fat content to ensure that the test fats comprised 95% of the total dietary fat. Diet had no significant effect on respiratory parameters at uniform or adjusted work loads, with the exception of heart rate. Mean heart rate for subjects fed the RSO diet was higher during exercise and recovery at adjusted work loads of 60 and 80% VO_2 max, than the mean heart rate for subjects fed the SO diet. There was significant interaction between the RSO and SO diets and adjusted work loads with respect to oxygen consumption, as well as significant interaction between the RSO and SO diets and rest and recovery, with respect to heart rate for subjects exercised at adjusted work loads. Diet had no significant effect on any of the blood parameters measured. The mean lactate/pyruvate ratios for subjects were comparable during rest, exercise and recovery irrespective of diet, suggesting that the oxidation: reduction potential of the muscular tissues was not altered by substitution of the SO or RSO diets for the mixed fat diet. Results indicated that the ingestion of a diet containing low erucic acid RSO as the sole source of dietary fat by 8 young adult men for 8 days had effects on energy metabolism which were similar to those when the diet contained SO.

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INTRODUCTION

Rapeseed was first grown commercially in Canada in 1943 as a war measure to supply oil for marine engines. Since then rapeseed has become Canada's major oilseed crop and third most important grain crop.

Prior to 1973 rapeseed produced in Canada was characterized by a high erucic acid content--approximately 38% of total fatty acid content. As of December 1, 1973 edible oil processors at the request of National Health and Welfare switched the production of rapeseed oil to the low erucic acid type. The shift to low erucic acid varieties stemmed from observations that long chain monoenoic acids, such as erucic acid, caused pathological changes in tissues, predominantly those dependent on fat for energy, namely the heart and skeletal muscle (Abdellatif and Vles, 1970; Beare-Rogers and Nera, 1972). Subsequently, low erucic acid rapeseed oil has been associated with necrosis and fibrosis in the myocardium of experimental animals (Rocquelin and Cluzan, 1968; Beare-Rogers et al., 1974).

There have been conflicting reports on the effects of rapeseed oil on energy production. Houtsmuller et al. (1970) reported decreased oxygen consumption and ATP synthesis for isolated heart mitochondria from rats fed high erucic acid rapeseed oil. Trémolières et al. (1971) reported decreased oxygen consumption during exercise for subjects fed a single dose of high erucic acid rapeseed oil, as compared to peanut

oil, as well as decreased respiratory quotients during rest, which were attributed to preferential oxidation of fatty acids. Lake (1975) reported that the ingestion of 126 grams of low erucic acid rapeseed oil daily for 10 days had no deleterious effect on energy metabolism of subjects at rest or during exercise.

The present study was designed to compare the effects of low erucic acid rapeseed oil (Brassica Napus cultivar Tower) and soybean oil on energy metabolism of adult male subjects both at rest and during exercise. Energy metabolism was assessed by monitoring both respiratory (oxygen consumption, ventilation rate, heart rate and respiratory quotient) and blood parameters (serum glucose, plasma lactate and pyruvate concentrations, lactate/pyruvate ratios, serum free fatty acids and serum glycerol).

REVIEW OF LITERATURE

Two varieties of the Brassica species of rapeseed are grown in Canada, Brassica Napus and Brassica Campestris. Oils from the Brassica seed are characterized by a significant content of the long chain monoenoic fatty acids, eicosenoic (C_{20:1}) and erucic (C_{22:1}), a relatively low level of linoleic acid and the saturated fatty acids, palmitic and stearic, and moderate amounts of linolenic acid. Rapeseed oil (RSO) differs from other vegetables oils, both in its significant content of long chain monoenoic acids, and its relatively low saturated fatty acid content (Downey et al., 1975).

Early varieties of RSO characterized by an erucic acid content as high as 45%, were associated with depressed growth and food intake in rats (Boer et al., 1947; Deuel et al., 1948; Beare et al., 1957) as well as severe myocardial abnormalities, including interstitial inflammatory changes, foci of histiocyte infiltration, lipidosis, necrosis and fibrosis (Roine et al., 1960; Craig and Beare, 1967; Rocquelin and Cluzan, 1968; Beare-Rogers et al., 1972). Maximum fat accumulation in the myocardium due, mainly to an increase of triglycerides containing large amounts of erucic acid, was reported to occur in 3 to 6 days, and to decrease to near normal levels after approximately 16 weeks (Beare-Rogers, 1970; Abdellatif and Vles, 1973). With the introduction of low erucic acid RSO with an erucic acid content of 5% or less, rats no longer showed depressed growth and food intake

(Craig and Beare, 1967; Rocquelin and Cluzan, 1968). However, studies on the effects of low erucic acid RSO on changes in the myocardium have produced conflicting results. Low erucic acid RSO is reported not to be associated with cardiac lipidoses (Craig and Beare, 1967; Beare-Rogers, 1970; Beare-Rogers et al., 1971; Rocquelin et al., 1973), but has been associated with necrosis and fibrosis of the myocardium (Rocquelin and Cluzan, 1968; Kramer et al., 1973; Beare-Rogers et al., 1974).

Metabolism of Erucic Acid

Evidence that rats fed RSO incorporated into tissues a relatively small proportion of dietary erucic acid, but produced an unusually high proportion of oleic acid suggested that erucic acid underwent β -oxidation (Craig et al., 1963 a,b; Beare et al., 1963) which was later verified by Craig and Beare (1967).

The accumulation of triglycerides in the myocardium of experimental animals fed high erucic acid RSO has been attributed to an inhibition of β -oxidation (Christophersen and Bremer, 1972), reduced activity of fatty acid oxidation enzymes (Kramer et al., 1973; Swarttouw, 1974), an increased uptake of fatty acids (Gumpen et al., 1973), and impaired respiratory activity (Houtsmuller et al., 1970).

Christophersen and Bremer (1972) have reported that the presence of erucylcarnitine caused a significant inhibition of the mitochondrial oxidation of palmitylcarnitine--

the inhibition being significantly more pronounced in the heart than in the liver mitochondria. The oxygen uptake of liver mitochondria however, was reported to be more inhibited by erucylcarnitine in the presence of malonate than malate, suggesting an inhibition of B-oxidation of palmitate. It is suggested that erucic acid inhibits the oxidation of other fatty acids, causing them to accumulate and be channelled into other pathways which are uninhibited or relatively less inhibited, such as triglyceride synthesis.

Erucic acid may undergo a relatively slow catabolism as compared to other fatty acids due to a reduced activity of the enzymes of fatty acid activation and B-oxidation for erucic acid. Swarttouw (1974) in a study on isolated rat heart mitochondria, reported a slower conversion rate for erucate than for palmitate in all enzymic reactions occurring in the oxidation of fatty acids. In addition to reduced activity of the enzymes of fatty acid activation and B-oxidation for erucic acid, Kramer et al. (1973) reported reduced levels of triglyceride lipase may be associated with the accumulation of fat in tissues of animals fed high erucic acid RSO.

Jaillard et al. (1973) have suggested that erucic acid may be more slowly catabolized than other fatty acids, because it must first undergo shortening of the chain in an extra-mitochondrial process before B-oxidation can occur in the mitochondria.

Swarttouw (1974) however, observed a decreased affinity of albumin for erucic acid, and has not ruled out the possibility that weaker binding to carrier proteins may result in differences in the transport of erucic acid to the site of B-oxidation.

RSO high in erucic acid has been reported to increase fatty acid uptake of fat utilizing tissues. Gumpen and Nørum et al. (1973) reported that the fractional amount of long chain acyl-carnitines of the rat heart remained unchanged after a RSO diet, while high levels of acyl-carnitines accumulated in the liver and brown adipose tissues. It was suggested that the latter reflected increased uptake of fatty acids and high oxidation rates, whereas the unchanged acyl-carnitines in the heart reflected unaltered myocardial fat oxidation, despite increased uptake of fatty acids, due to an inhibition of B-oxidation. A decrease in triglyceride content of the heart could then be attributed to decreased fatty acid uptake rather than to an increased ability of heart mitochondria to oxidize incoming fatty acids.

The rapid decrease in cardiac lipids after the first week of a RSO diet has been attributed to an enzymic adaptation at either the extra or intra-mitochondrial level (Jaillard et al., 1973). Struijk et al. (1973) have also reported a high erucic acid RSO diet increased postheparin lipoprotein lipase activity of the plasma after a feeding period of 3 to 6 days which would coincide with the rapid

decrease in triglyceride content of the heart in experimental animals fed RSO.

Houtsmuller et al. (1970) reported that isolated heart mitochondria of rats fed high erucic acid RSO oxidized glutamate and other substrates including α -oxoglutarate, caprylate and succinate at a reduced rate, and that there was an inverse relationship between the decrease in oxygen uptake and the erucic content of the heart. Other investigators (Kramer et al., 1973) have reported a decrease in palmityl-carnitine oxidation in heart mitochondria of rats fed high erucic acid RSO, but in contrast to Houtsmuller et al. (1970) observed no marked difference in oxygen uptake or energy production. The differences between these results may have been due to improper isolation of mitochondria (Kramer et al., 1973).

Studies on the utilization of RSO for energy production in the human are limited. Trémolières et al., (1971) investigated the effect of a single dose of a high erucic acid RSO in man at rest and during mild exercise at work loads of 120 and 240 kpm/min. They observed a decrease in respiratory quotient (RQ) followed by a rise during exercise. Peanut oil did not produce a similar decrease in RQ during rest, and produced a smaller increase in RQ during exercise. The decrease in RQ during rest was interpreted as preferential oxidation of fatty acids after the ingestion of RSO. No significant differences in plasma

concentrations of lactate and pyruvate were observed between the two diets, suggesting that mitochondrial function was not altered. However, decreased oxygen consumption was observed during exercise after the ingestion of RSO. No significant differences in serum free fatty acids were observed at rest or during exercise after the ingestion of either RSO or peanut oil.

Lake (1975) compared the effect of low erucic acid RSO and soybean oil on energy metabolism in 4 male subjects, both at rest and during exercise at a standardized work load of 950 kpm/min. In contrast to the work of Tremolieres et al., (1971) on high erucic acid RSO, results did not indicate preferential oxidation of fatty acids. Respiratory measurements and blood samples were taken during exercise sessions both after a single meal and after prolonged feeding of the test fats. No significant differences were found in oxygen consumption, ventilation rate, or RQ during rest or exercise on either the soybean or low erucic acid RSO. Similarly, no significant differences in serum glucose, serum free glycerol, plasma lactate and pyruvate concentrations or the lactate/pyruvate ratio were observed between the test fats.

Whether the differences in metabolism of RSO are related to the erucic acid content or some other factor is not known at the present time. In order to further elucidate the effect of low erucic acid RSO on energy utilization in man, a study similar to the one reported by Lake (1975) was

conducted involving 8 subjects and graded work loads of 60,
70 and 80% VO_2 max.

Energy Metabolism

Energy requirements of the body are met by the breakdown of adenosine triphosphate (ATP)---a high energy phosphate compound to adenosine diphosphate (ADP) and phosphate (P). The supply of ATP is derived from energy produced by the oxidation of foodstuffs in a process called aerobic metabolism. When the energy requirements of the body are increased during exercise, there is an increased turnover rate of ATP.

During moderate exercise the restoration of ATP may continue to depend on energy derived from aerobic metabolism as reflected by an increased oxygen consumption. Carbohydrate in the form of a molecule of glucose or glycogen is metabolized to pyruvate with the formation of 2 moles of ATP. Pyruvate is then oxidized by way of the tricarboxylic acid cycle and respiratory chain to carbon dioxide and water.

If the maximal rate of oxygen consumption does not supply adequate oxygen to restore the ATP as rapidly as it is broken down, the energy needs are provided by anaerobic metabolism. Pyruvate is reduced to lactic acid instead of being oxidized to carbon dioxide and water. This results in the production of lactic acid and a low yield of ATP, only 2 moles of ATP per molecule of glucose, as compared to 38 moles of ATP produced during aerobic metabolism.

Oxygen Consumption

At the beginning of exercise there exists a greater need for oxygen than the cardiorespiratory processes can provide. During this transitional unsteady state, oxygen consumption will not reflect total energy needs. It is during this initial lag period, before the supply of oxygen has been increased by adjustments in ventilation and circulation, that anaerobic metabolism and the utilization of phosphagen stores (ATP and creatinine phosphate) of the muscle play an indispensable role in meeting energy requirements.

Oxygen uptake at the beginning of work is thought to depend on oxygen supply and perhaps ultimately on blood flow through the working muscles (Craig, 1972). Thereafter, oxygen consumption is reported to increase exponentially with time, the maximum level attained being unrelated to the intensity of exercise except in exercise of very high intensity and short duration (Margaria et al., 1963). However, the time to steady state has been reported to increase with the intensity of exercise. Wasserman et al. (1967) reported that oxygen consumption rose for a longer period of time when the subject exercised at heavy work rates as compared to more moderate work rates. Whipp and Wasserman (1972) reported that a steady state was progressively delayed at higher work rates, the difference between oxygen consumption at 3 and 6 minutes at each work level being increasingly greater, the higher the work rate. Di Prampero et al. (1970) however, reported