Dietary Fatty Acid Profile Affects Endurance in Rats

Kerry J. Ayre* and A.J. Hulbert

Department of Biological Sciences, University of Wollongong, Wollongong N.S.W. 2522, Australia

ABSTRACT: Typically, athletes are advised to increase their consumption of carbohydrates for energy and, along with the general population, to reduce consumption of saturated fats. It is now recognized that fats are not identical in their influence on metabolism, and we argue that the composition of the polyunsaturated fat component should not be ignored. The aim of this study was to manipulate the dietary fatty acid profile in a high-carbohydrate diet in order to investigate the effect of dietary polyunsaturates on submaximal endurance performance in rats. Rats were fed one of three isoenergetic diets containing 22 energy percentage (E%) fat for 9 wk. The diets comprised an essential fatty acid-deficient diet (containing mainly saturated fatty acids); a diet high in n-6 fatty acids, High n-6; and a diet enriched with n-3 fatty acids, High n-3. Submaximal endurance in rats fed the High n-3 diet was 44% less than in rats fed the High n-6 diet ($P < 0.02$). All rats were then fed a standard commercial laboratory diet for a 6-wk recovery period, and their performances were reevaluated. Although endurance in all groups was lower than at 9 wk, it was again significantly 50% lower in the High n-3 group than the High n-6 group ($P < 0.005$). Although n-3 fats are considered beneficial for cardiovascular health, they appear to reduce endurance times, and their side effects need to be further investigated.
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A vast literature (and indeed a vast industry) advises athletes to consume high-carbohydrate diets in order to maximize physical performance. In doing so, athletes have often been advised to reduce their consumption of fats. This seems to stem from studies of cardiovascular disease where advice is that patients should reduce their total fat consumption while increasing their consumption of n-3 fats, but these recommendations for athletes have little comprehensive research support. However, fats are at least as physiologically important and their metabolism is as complex as carbohydrates. Clearly, the consequences and side effects of this changed dietary profile in humans need to be investigated.

*To whom correspondence should be addressed at Department of Biological Sciences, University of Wollongong, Northfields Ave.,Wollongong N.S.W. 2522 Australia. E-mail: kerry_ayre@uow.edu.au.

Abbreviations: ANOVA, analysis of variance; EDL, extensor digitorum longus; EFAD, essential fatty acid-deficient; E%, energy percentage; High n-6, diet high in n-6 fatty acids; High n-3, diet high in n-3 fatty acids; PUFA, polyunsaturated fatty acid; S,M,P_{n-6},P_{n-3}, ratio of saturated to monounsaturated to n-6 PUFA to n-3 PUFA; $\dot{V}O_2$max, maximal rate of oxygen consumption.

Although classical studies have led to the concept of carbohydrate loading for optimal physical performance (1), at least six articles published since 1983 have shown that performance in rats (2–4), dogs (5), and humans (6,7) may actually be improved when a high-fat diet is consumed for a short time. Surprisingly, the effects on endurance of different types of fats (whether saturated or unsaturated) have not been investigated, even though they have clearly been shown to have a multitude of physiological effects. The few studies which have investigated the effects of dietary fat manipulation on physical performance (8–10) have shown that altered levels of n-3 fatty acids do not affect maximal oxygen consumption, but none of these studies has investigated possible changes in endurance. Although the current dogma is that the diets of endurance athletes should be high in carbohydrates, we argue that the fat component should not be ignored. Specifically, the classes of fatty acids should not be treated identically because (i) different fatty acids are oxidized at different rates (11) and (ii) we have recently shown that a diet rich in saturated fats is detrimental to endurance performance compared to diets rich in unsaturated fats or carbohydrates (12).

This study was a part of a larger study examining the effects of changes in dietary essential polyunsaturated fats on muscle phospholipid composition (13), isolated muscle performance *in vitro* (14), and physical performance of whole animals. For 9 wk, rats were fed one of three isoenergetic test diets which differed only in their fatty acid composition. The diets were (i) deficient in the essential fatty acids, EFAD; (ii) high in n-6 fatty acids, High n-6; and (iii) enriched with n-3 fatty acids, High n-3. Following this, the rats were fed a commercial stock diet in order to determine the reversibility of any dietary effects on endurance.

We now report the effects of these same dietary fatty acid manipulations on the endurance of rats, as well as the extent to which these dietary effects were reversed after returning animals to the stock diet.

MATERIALS AND METHODS

Animals and diets. All experiments were approved by the University of Wollongong's Animal Experimentation Ethics Committee. Male weanling Wistar rats, bred at the University of Wollongong (housed individually at 22 ± 2°C and 57 ± 2% relative humidity, age 21–23 d, mean weight of 54 ± 1 g) were

randomly assigned, one per litter, to each of three dietary groups. Groups of eight rats were maintained on each test diet for 9 wk. Endurance was then assessed using a graded submaximal treadmill running test. Following this test, all rats were fed a common rat commercial diet [Allied Rat and Mouse Cubes (mean energy composition 12 E% fat, 64 E% carbohydrate, and 24 E% protein), Fielders' Agricultural Products, Tamworth, Australia)] for a 6-wk "recovery" period. At all times, rats were given free access to food and water, and food intake and body mass were recorded throughout the study.

All test diets were identical except for their lipid composition (22 E% fat, 56 E% carbohydrate, 22 E% protein). They contained (g/kg) sucrose 560, protein (casein) 220, oil 100, water 50, mineral mix 50, vitamin mix 10, cellulose 10. Details of the mineral mix and vitamin mix as well as of the methods of lipid analysis used are given in Ayre and Hulbert (13). The EFAD diet contained 100 g/kg coconut oil (ETA Food Services, Wollongong, Australia) as the only source of fat and was therefore lacking both n-6 and n-3 essential polyunsaturated fatty acids (PUFA) (the ratio of saturated to monounsaturated to PUFA, S,M,P_{n-6},P_{n-3} ratio, was 95:4:1:0). The High n-6 diet contained 100 g/kg sesame oil (Meadowlea Foods, Sydney, Australia) which is high in n-6 fatty acids; in this diet the S,M,P_{n-6},P_{n-3} ratio was 16:30:50:4. The High n-3 diet contained 70 g/kg sesame oil and 30 g/kg Max EPA oil (R.P. Scherer, Melbourne, Australia) containing 30% n-3 fatty acids. It was therefore enriched with n-3 PUFA and in this diet the S,M,P_{n-6},P_{n-3} ratio was 21:25:35:16. The S,M,P_{n-6},P_{n-3} ratio of the rat commercial diet was 33:32:30:2. The specific fatty acid content of the test diets and the stock diet is given in Ayre and Hulbert (13). In another group of rats fed the same diets, the fatty acid composition of phospholipids extracted from both the soleus and extensor digitorum longus (EDL) muscles was determined. The methods are presented in Reference 13.

Testing program. We assigned a cryptic code number to each rat, and endurance tests were performed so that the dietary group of each animal was concealed from the tester. Rats were fasted overnight and all littermates were tested in random order on the same day toward the end of the light cycle. Submaximal endurance was assessed while rats ran on a single-lane motor-driven treadmill. In the week prior to each testing period (i.e., after both 8 wk on the test diets and 5 wk on the commercial diet), rats underwent a habituation program (15). Each rat was given a 5-min exercise period with treadmill speed and incline increasing daily to 25.2 m/min and 10°, respectively. Endurance tests were then performed both after 9 wk on the three test diets and following 6 wk recovery on the commercial stock diet, following the protocol of Baldwin *et al.* (15). Rats commenced running at 12 m/min speed, 0° incline for 3 min, after which the treadmill was raised 2° and speed was increased to 27 m/min and held constant. Thereafter, the angle of incline was increased by 2° every 10 min. We measured time to exhaustion during treadmill running in order to provide an estimate of endurance. The length of time each rat could keep running at this workload was recorded. To ensure that rats ran to exhaustion, we routinely stimulated them with jets of compressed air while they were running free on the treadmill. A rat was considered to have reached exhaustion when it positioned itself at the back of the chamber for 10 s or more and no longer responded to repeated jets of compressed air. The amount of work performed by each rat was calculated as the product of body mass (i.e., force in kiloponds), distance traveled, and percentage grade (i.e., the product of the sine of the treadmill angle and 100), as described by Powers and Howley (16).

Statistical methods. All values are expressed as mean ± SEM. The *a priori* alpha level was 0.05. A one-way analysis of variance (ANOVA) was used to test for heterogeneity of food intake and rat weights. To test for significant differences in endurance and work between the dietary groups, we used the statistical package, SAS (SAS Institute, Cary, NC: 1979) to perform Model lll two-factor ANOVA without replication; the fixed factor was diet and the random factor was litter and/or day of testing. Since only one rat per litter was allocated to each treatment and each litter was tested on a different day, it is not possible to determine whether significant effects of this factor are litter effects as such, or the product of day-to-day variation in experimental conditions. However, the design ensures that variation among litters and days of testing does not obscure the effects of the diets. Two separate ANOVA were performed: after 9 wk on the test diets and after 6 wk on the common diet. Wherever ANOVA revealed significant effects of diet, we used Tukey's studentized range test to determine which dietary groups produced significantly different responses (17).

RESULTS

There was no significant effect of diet on body mass, either after the 9-wk experimental period (EFAD = 363 ± 20 g; High n-6 = 387 ± 14 g; High n-3 = 405 ± 5 g), or after 6 wk on the common diet (EFAD = 411 ± 24 g; High n-6 = 445 ± 14 g; High n-3 = 463 ± 7 g). There were significant effects of the diets on the fatty acid composition of muscle membranes (see Table 1). The percentage of total unsaturates in membranes was not influenced by diet, but the relative content of the different types of unsaturates was dramatically influenced by diet. For example, the different diets resulted in a 10-fold range in the n-3 polyunsaturate content of muscle phospholipids. The influence of diet was similar for both the fast (EDL) and the slow (soleus) muscle types.

Almost all rats ran willingly and there was surprisingly little variation within dietary groups (Fig. 1). The overall endurance performance for all groups decreased between the two endurance tests by approximately 50%. Dietary manipulation had a highly significant effect on exercise performance (Fig. 1). The endurance of both the High n-3 and EFAD groups was markedly lower than that of the High n-6 group both after 9 wk on the test diets and after 6 wk on the common diet. After the 9-wk experimental period, rats on the

TABLE 1
Effects of Dietary Fatty Acid Profile on Phospholipid Fatty Acid Composition (g/100 g fatty acids) of Soleus and Extensor Digitorum Longus Muscles of Wistar Rats[a]

Fatty acid	Soleus[b]			Extensor digitorum longus[b]		
	EFAD	High n-6	High n-3	EFAD	High n-6	High n-3
16:0	10.2 ± 1.8	13.2 ± 0.6	14.2 ± 1.0	23.9 ± 0.7	21.8 ± 1.0	21.6 ± 2.2
16:1n-9	5.3 ± 0.5^{a}	0.9 ± 0.4^{b}	0.7 ± 0.4^{b}	6.4 ± 0.6^{a}	0.2 ± 0.2^{b}	0.3 ± 0.2^{b}
18:0	21.9 ± 1.8	19.4 ± 1.6	20.0 ± 1.3	13.6 ± 0.7^{a}	15.9 ± 0.3^{a,b}	17.6 ± 1.0^{b}
18:1n-9	15.6 ± 1.4^{a}	8.4 ± 1.7^{b}	6.0 ± 0.5^{b}	21.6 ± 0.5^{a}	5.9 ± 0.3^{b}	17.6 ± 1.0^{b}
18:1n-7	4.2 ± 0.7	3.1 ± 0.2	2.9 ± 0.3	3.3 ± 0.1^{a}	2.1 ± 0.1^{b}	2.2 ± 0.2^{b}
18:2n-6	9.2 ± 1.0^{c}	20.1 ± 1.4^{a}	15.4 ± 1.0^{b}	6.8 ± 0.5^{a}	19.1 ± 1.3^{b}	16.3 ± 0.6^{b}
20:3n-9	9.6 ± 0.3^{a}	0.1 ± 0.1^{b}	—^{b}	6.1 ± 0.7^{a}	0.1 ± 0.0^{b}	—^{b}
20:3n-6	0.9 ± 0.1	0.4 ± 0.2	0.5 ± 0.2	0.5 ± 0.2^{a}	0.2 ± 0.2^{a,b}	—^{b}
20:4n-6	17.7 ± 2.1^{b}	23.0 ± 1.2^{a}	10.4 ± 0.4^{c}	11.3 ± 0.7^{a}	19.9 ± 2.0^{b}	7.2 ± 0.4^{c}
20:5n-3	—^{b}	—^{b}	1.1 ± 0.4^{a}	0.3 ± 0.2	0.1 ± 0.1	0.3 ± 0.2
22:4n-6	1.1 ± 0.2^{a,b}	2.4 ± 0.6^{a}	—^{b}	0.9 ± 0.1^{a,b}	2.0 ± 0.6^{a}	—^{b}
22:5n-6	1.2 ± 0.7^{b}	3.8 ± 0.5^{a}	—^{b}	2.0 ± 0.1^{a,b}	2.8 ± 0.8^{a}	—^{b}
22:5n-3	0.2 ± 0.1^{b}	1.6 ± 0.9^{a,b}	3.5 ± 0.2^{a}	0.5 ± 0.0^{a}	1.2 ± 0.3^{a}	3.1 ± 0.2^{b}
22:6n-3	2.6 ± 0.5^{b}	3.7 ± 0.3^{b}	25.5 ± 1.7^{a}	2.3 ± 0.2^{a}	7.2 ± 3.9^{a}	24.5 ± 1.3^{b}
∑ Saturated	32.1 ± 1.9	32.6 ± 1.3	34.2 ± 0.5	37.5 ± 1.1	37.7 ± 0.7	39.1 ± 1.3
∑ Unsaturated						
∑ n-9	30.5 ± 1.6^{a}	9.4 ± 2.0^{b}	6.6 ± 0.9^{b}	34.0 ± 1.4^{a}	6.1 ± 0.5^{b}	6.5 ± 0.5^{b}
∑ n-6	30.0 ± 2.5^{b}	49.6 ± 0.8^{a}	26.3 ± 1.1^{b}	21.5 ± 0.4^{a}	43.9 ± 4.5^{b}	23.5 ± 0.4^{a}
∑ n-3	2.9 ± 0.5^{b}	5.4 ± 0.9^{b}	30.1 ± 1.6^{a}	3.7 ± 0.3^{a}	10.2 ± 4.1^{a}	28.7 ± 1.7^{b}
n-3/n-6	0.1 ± 0.0^{b}	0.1 ± 0.0^{b}	1.2 ± 0.1^{a}	0.2 ± 0.0^{a}	0.2 ± 0.1^{a}	1.2 ± 0.9^{b}

[a]—, Not detected; phospholipid fatty acid composition of soleus and extensor digitorum longus muscles after 9 wk on the three test diets, High n-6, EFAD, and High n-3. Only fatty acids detected at levels greater than 0.1 g/100 g of total fatty acids are listed.
[b]Values are means ± SEM, $n = 4$. The statistical significance of variation in mean phospholipid fatty acid levels among dietary treatments was assessed by one-way analysis of variance. Significantly different treatment means are denoted by different superscript roman letters. Abbreviation: EFAD, essential fatty acid-deficient.

High n-3 diet ran only 56% as long as those on the High n-6 diet ($P < 0.02$). The endurance of the EFAD group was also relatively low, being only 73% of that of the High n-6 group, although this difference was nonsignificant. Similarly, the amount of work done was significantly lower in both the High n-3 and EFAD groups [46 and 52% of the work performed by the High n-6 group ($P < 0.005$), respectively]. Very similar relationships persisted following recovery, where the High n-3 group ran for only 50% of the High n-6 group's mean endurance time ($P < 0.005$), and the High n-3 and EFAD rats did only 25 and 42% as much work as the High n-6 group ($P < 0.01$), respectively. Mean endurance time decreased by 49–55% for the three groups during the recovery period, and mean amount of work performed decreased by 73–85%.

We detected several litter effects throughout this study. For example, there were significant litter effects on endurance both after 9 wk on the test diet ($P < 0.02$) and after the 6 wk recovery period ($P < 0.05$). Although several rats needed encouragement to run, there were few that refused. Those that did were not included in the dataset. One rat that refused to run at 9 wk also refused following recovery. Also, his two littermates, which ran well after the 9-wk test diet, refused to run after the 6-wk recovery diet. These litter effects may represent real effects of variation among litters, or show day-to-day variation in experimental conditions. However, since all littermates were tested on the same day, and litter was one of the two factors considered in the ANOVA, these effects do not "interfere" with the description of the effects of dietary treatments.

DISCUSSION

Our study demonstrates that manipulation of the composition of dietary fatty acids can affect whole-animal physical performance in rats. We found three important effects: (i) that treadmill endurance (both "time to exhaustion" and "work performed") was influenced by the relative composition of n-6 and n-3 PUFA in the diet, (ii) that this effect may not be easily reversed, in that the differences between dietary groups persisted even after all rats were fed a common stock diet for 6 wk after being fed the test diets, and (iii) that treadmill endurance decreased in all groups when they were fed the same common diet after the experimental period.

Endurance and work were both dramatically reduced by consumption of the High n-3 diet, compared with those fed the High n-6 diet. Rats fed the High n-3 diet ran for only 56% as long as the High n-6 rats. These diets had the same total fat content, but the n-6 PUFA content in the High n-6 diet was partly replaced with n-3 fatty acids in the High n-3 diet. To our knowledge, this is the first study to report a substantial influence of the type of dietary polyunsaturated fat on whole-animal performance. These results were part of a larger study examining the role of dietary polyunsaturates (using the same three diets) on (i) composition of muscle phospholipids (13)

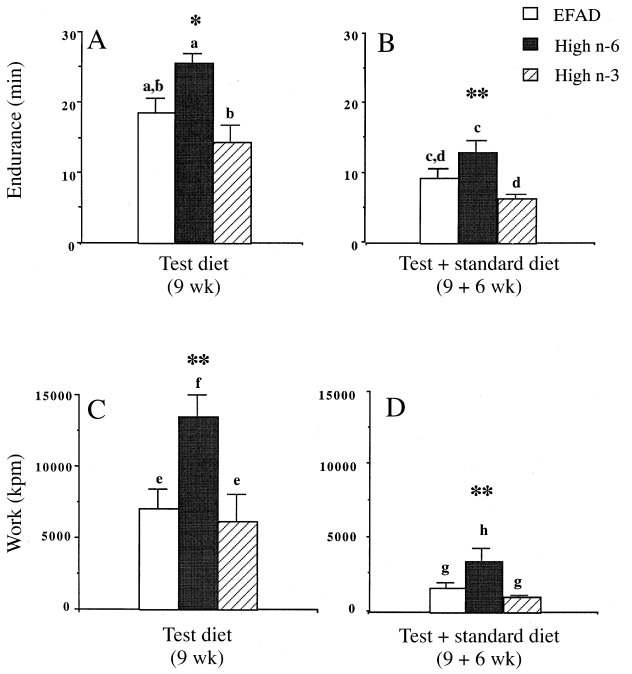

FIG. 1. The influence of diets of differing fatty acid composition on the submaximal treadmill endurance of rats. Figures A and B show the effect on endurance time whereas Figures C and D show the effect on work performed. Figures A and C show the effect after 9 wk on the test diets, and Figures B and D show the results obtained after a further 6 wk on a stock laboratory diet for all groups. Columns with different letters are significantly different from each other. $^{*}P < 0.05$; $^{**}P < 0.01$. Abbreviations: EFAD, essential fatty acid-deficient; High n-6, diet high in n-6 fatty acids; High n-3, diet high in n-3 fatty acids.

and (ii) contractile properties of isolated muscles (14), and whole-animal performance in rats. Recently, we have also shown that a diet rich in saturated fats is detrimental to endurance performance of rats when compared to diets either low in fat (i.e., carbohydrate-rich) or containing mainly n-6 polyunsaturated fats (12).

Whole-animal endurance during treadmill running is possibly influenced by a wide and complex range of physiological parameters. For example, limiting factors may sometimes occur in the respiratory and/or cardiovascular systems rather than the muscles themselves and similarly, the role of motivational differences is not easy to determine. In this study, we were particularly careful in trying to negate motivational influences between experimental groups by concealing the dietary treatment of each rat from the tester.

We have no direct knowledge of the possible mechanistic basis of the effects we have observed, but our previous studies can give some insight. The fact that the current dietary manipulations do not have any substantial influence on the *in vitro* contractile properties (including time to fatigue) of either isolated fast or slow muscles of rats (14) implies that the current dietary effects are not related to changes in the contractile properties of the muscles *per se*.

Limitation in substrate supply is a potential mechanism relating the type of dietary fat to treadmill endurance. A recent examination of the substrate and oxygen pathways during exercise at 40% maximal rate of oxygen consumption ($\dot{V}O_2$) max in both the highly aerobic dog and less aerobic goat has shown that about 70% of the energy requirements are provided from fat, and that at 60% $\dot{V}O_2$ max, its relative contribution is decreased to approximately half of total energy turnover (18). About 65–70% of the fat metabolized in these cases is from intramuscular triglyceride stores, which illustrates the relative importance of these stores during submaximal exercise. Whether the current diets resulted in different intramuscular triglyceride reserves in the rat is unknown. It is of interest that dietary n-3 fats increase the insulin sensitivity of muscle in rats (19). In humans, there is an inverse relationship between muscle insulin sensitivity and muscle triglyceride content (20). If this is also the case in rats, then the difference in treadmill endurance between rats fed the High n-6 and High n-3 diets may in turn be related to differences in intramuscular triglyceride stores. This possibility requires further investigation.

Dietary n-3 fats restore tissue insulin sensitivity in rats that are insulin-resistant, and thus they increase glucose entry and utilization in rat muscles (21). However, such effects may not be important in explaining the current results since oxidation of circulating glucose is only responsible for about 10–20% of total aerobic metabolism during treadmill exercise of dogs and goats (22). If, however, insulin-regulated glucose entry into muscle is important in determining treadmill endurance of rats, then we would expect that dietary n-3 enrichment would result in an increased endurance whereas we observed the opposite effect, a decrease in treadmill endurance.

Different types of fatty acids are metabolized *in vivo* at different rates. In rats, n-3 linolenic acid is metabolized more rapidly than n-6 linoleic acid (11). Furthermore, the rate of oxidation of n-3 linolenic acid is known to be influenced by the nature of the other fats in the diet, with linolenic acid being metabolized most rapidly when the other dietary fats are saturated and slowest when the other fats are predominantly n-6 polyunsaturates (19). Whether such differences are related to the endurance difference observed in the current study is unknown.

Dietary n-3 fats have been shown to have substantial influences on cardiovascular function and most of these are regarded as beneficial (23). If reduced cardiovascular function during exercise is the mechanism behind our dietary effect, then increased dietary intake of n-3 fats may not be so beneficial as generally thought. Using the same treadmill protocol, Baldwin *et al*. (15) showed that rats with (drug-induced) enlarged hearts have only a slightly greater endurance ($P < 0.1$) than normal rats.

Both n-6 and n-3 fatty acids are intimately involved in many aspects of physiological regulation in that they are both important membrane constituents and are also precursors of the eicosanoids, which are important chemical messengers (24). Whether the different diets resulted in different levels of eicosanoid production is unknown, but it is possible that the effects we report here are the result of such changes in eicosanoid messenger levels.

The second important finding of this study was that, although all rats were transferred to a common stock diet for 6 wk following the 9-wk test diet period, the relative differences between the experimental groups remained. This may indicate that the diet-induced differences are long-term and at least partly irreversible. Using the same diets, we have shown that in both slow (soleus) and fast (EDL) muscles the relative n-3 and n-6 composition of muscle phospholipids is related to their relative dietary presence (13; and see Table 1) and that even after 6 wk on the common diet the relative n-3 and n-6 composition of muscle phospholipids was little changed from what it was on the test diets. As we reported previously (13), the levels of n-3 fatty acids in the phospholipids of both soleus and EDL muscles were still significantly higher in rats from the High n-3 group than the other two groups after 6-wk recovery. This may be related to the persistent decrease in endurance seen in these rats after the recovery period.

All groups showed the same relative decrease in endurance when transferred to the common stock diet. Since all rats were 6 wk older and 50–60 g heavier, it may be that general aging was the cause. In a separate group of rats fed the same diets, there were no significant decreases in muscle mass or length following the common diet (14). This suggests that muscle atrophy is not an important component of the decline in treadmill endurance. Another explanation could be that the test diets were all 22E% fat, whereas the stock diet contained only 12E% fat. A number of studies have shown that an increase in the fat content of the diet can result in improved exercise performance in rats (2–4), dogs (5), and humans (6,7). Thus, it may be that a decrease in the fat content of the stock diet, compared to all three test diets, is the common factor that explains the decreased endurance in all groups. However, the important point is that the pattern of results was almost identical to the results after 9 wk on the test diets.

We have shown that rats fed diets with the same fat content but different lipid compositions show different endurances during submaximal running on a treadmill. The most surprising result was that the balance of n-3 and n-6 polyunsaturated in the diet has a highly significant influence on exercise performance of rats. We have used sedentary rats in the current study. Whether the same is true for trained rats or for humans is yet to be determined. Similarly, knowledge of the underlying mechanisms awaits further research.

In general, recommendations for weight reduction and amelioration of coronary artery disease have lead to increased consumption of PUFA containing high levels of n-6 fatty acids (25) and thus an increase in the dietary n-6/n-3 ratio. However, since the n-3 PUFA have been associated with decreased coronary artery disease, decreasing the dietary n-6/n-3 ratio will reduce the risks from deleterious eicosanoid production. Owing to publicity surrounding the findings that n-3 fatty acids are beneficial for the cardiovascular system and neural development, there has been an increase in human consumption of foods such as canola-based products and fish. Clearly however, the side effects of such increased consumption on physical performance in humans need to be investigated.

In conclusion, dietary fatty acids clearly exert significant effects on whole-animal performance. What determines submaximal treadmill running endurance is not known and difficult to ascertain. Effects on whole animals may reflect changes in muscles themselves or in the levels of eicosanoids, which are known to exert effects on secretory, cardiovascular, and immune functions (24). At present, diets containing a high level of PUFA relative to saturated fatty acids (and particularly the n-3 fatty acids) are recommended owing to their obvious benefits for the cardiovascular system (26). However, our results show a striking difference in effect for two diets that differ only in their ratio of the n-6 and n-3 fatty acids and point to the need for further research on the effects of such diets.

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