

Effects of exercise and n–3 fatty acids on postprandial lipemia

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Thomas, Tom R., Brian A. Fischer, William B. Kist, Kristen E. Horner, and Richard H. Cox. Effects of exercise and n–3 fatty acids on postprandial lipemia. *J Appl Physiol* 88: 2199–2204, 2000.—Because n–3 fatty acid ingestion and aerobic exercise each has been associated with diminished postprandial lipemia (PPL), the purpose of this study was to evaluate the effect of a combination of these two factors on PPL. Sedentary men underwent a standard dietary preparation, including a 12-h fast before each trial. Six subjects performed a control trial (fat meal, 100 g fat) and an n–3 fatty acid trial (fat meal after 3 wk of n–3 fatty acid supplementation at 4 g/day). In a parallel experiment, six different subjects underwent a control trial and n–3 fatty acid supplementation + 60 min of exercise before ingestion of the fat meal. Supplementation with n–3 fatty acid significantly decreased baseline triglyceride (TG) concentrations but did not significantly affect PPL. The combination of n–3 fatty acid and exercise had no effect on the postprandial TG response. The present study suggests that n–3 fatty acid supplementation lowers resting TG concentrations but inhibits the beneficial effect of aerobic exercise on the postprandial TG response. fish oil; high-density lipoprotein cholesterol; triglycerides

THE CONSISTENT OBSERVATION of the positive relationship between postprandial lipemia (PPL) and cardiovascular disease (CVD) suggests that PPL may be a better predictor of CVD than fasting triglyceride (TG) levels (19). Ziogas et al. (33) showed that exercise training was associated with significantly lowered PPL. Zhang et al. (31) showed that a session of aerobic exercise (1 h of treadmill running) before ingestion of a high-fat meal significantly decreased PPL and elevated high-density-lipoprotein cholesterol (HDL-C) concentrations. Others also have observed a marked attenuating effect of aerobic exercise on PPL (2, 28).

Supplementation with n–3 fatty acid, ingested in the form of fish oils (FO), can also independently lower fasting TG levels and PPL (5, 12, 18, 24). The exercise and n–3 fatty acid treatment effects on PPL may be additive or synergistic, but no previous studies were uncovered that examined the combination of these two TG-lowering treatments on PPL. Therefore, the purpose of this study was to evaluate the effect of a

combination of exercise and n–3 fatty acid ingestion on resting TG and PPL.

METHODS

Subjects were 12 men, ages 19–38 yr. All had been sedentary for the previous 6 mo. No subjects were taking dietary supplements beyond a daily multivitamin. The subjects were randomly assigned to separate experimental trials: FO or exercise + FO (ExFO). There were no significant differences in the characteristics between the two groups of subjects (Table 1), nor were there any statistical differences in dietary intake between the two groups (Table 2).

The subjects provided informed consent, as approved by the Institutional Review Board, and completed a physical activity questionnaire, a health questionnaire, and a 3-day diet record. Subjects with more than one major CVD risk factor or any disease symptom (1) were disqualified.

Before the trials, a maximal O₂ consumption ($\dot{V}O_{2\max}$) test was performed on a treadmill, as previously described (30), to determine the exercise intensity for the subsequent bout of aerobic exercise. Body composition was assessed by hydrostatic weighing, with residual volume measured by the helium dilution technique (27).

Preparatory diet and exercise. Subjects were instructed to maintain their normal diets and activity level during the course of the study. Before each trial, the subjects abstained from exercise for 48 h. On the day before *trial 1*, each subject completed a 1-day diet record and repeated the same diet before *trial 2*. Thus the subjects consumed the same kind of food and drink, quantity and quality, before both trials. The subjects also fasted for 12 h before each trial. Each subject was contacted 48 h before each trial to be reminded of the preparation protocol.

Treatments/trials. In these two parallel experiments, subjects in the FO group performed a control trial (fat meal only) and an experimental trial consisting of FO supplementation, whereas the ExFO group performed the control trial and an experimental trial consisting of exercise + FO supplementation (Fig. 1).

The order in which the subjects performed the control and experimental trials was counterbalanced: the order of trials was random for the first subject in each set, and subsequent order was alternated. When FO supplementation was performed first, a 3-wk washout period was undertaken before the control trial.

During the control trial for each group, the subjects reported to the laboratory after a 12-h fast. A baseline blood sample was taken, and the subjects ingested a high-fat test meal. Additional blood samples were taken at 2, 4, 6, 8, and 24 h after the meal.

In the FO trial, each subject ingested an n–3 fatty acid supplement for 3 wk. After the supplementation period, the FO group reported to the laboratory after a 12-h fast, ingested

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Table 1. *Subject characteristics*

	FO Group	ExFO Group	<i>P</i>
Age	27.5 ± 7.6	25.8 ± 6.8	0.07
Weight, kg	80.0 ± 18.3	81.9 ± 6.7	0.81
Body fat, %	22.4 ± 5.8	25.9 ± 3.5	0.24
$\dot{V}O_{2\max}$, ml · kg ⁻¹ · min ⁻¹	44.3 ± 7.2	39.9 ± 3.5	0.22
HR _{max} , beats/min	191 ± 7	189 ± 9	0.66
RER	1.17 ± 0.07	1.20 ± 0.06	0.38
Cholesterol, mg/dl	190.6 ± 56.9	187.7 ± 28.4	0.92
Triglycerides, mg/dl	122.9 ± 66.7	136.8 ± 72.6	0.74
HDL-C, mg/dl	41.8 ± 6.5	42.9 ± 9.2	0.82

Values are means ± SD. FO, fish oil; ExFO, exercise + FO; $\dot{V}O_{2\max}$, maximal O₂ consumption; HR_{max}, maximal heart rate; RER, respiratory exchange ratio; HDL-C, high-density-lipoprotein cholesterol. There were no significant differences between groups (*P* > 0.05).

the fat meal, and gave blood samples in the same fashion as the control trial.

In the ExFO trial, the group also fasted for 12 h before the 0-h blood sample but reported to the laboratory 1 h earlier to exercise. After a baseline blood sample was obtained, the subjects jogged on a treadmill for 60 min at 60% $\dot{V}O_{2\max}$. After the exercise session, a blood sample was taken, the test meal was given, and additional postmeal blood samples were collected.

Test meal. A standard fat-rich test meal was provided in each trial to magnify the postprandial TG and HDL-C response. The test meal (82.6% fat) was given in a milkshake form and was consumed within 10 min for each trial. The milkshake consisted of a combination of 270 ml of whipping cream and 65 g of specialty ice cream. This was a total of 980 kcal, 100 g of fat, 17 g of carbohydrate, and 3 g of protein.

n-3 Fatty acid FO supplement. The subjects were given n-3 fatty acid supplements (Super EPA-500, Bronson Pharmaceutical, St. Louis, MO) in the form of eight soft gel tablets per day for 3 wk. Each tablet contained 300 mg of eicosapentaenoic acid and 200 mg of docosahexaenoic acid, a total of 500 mg of n-3 fatty acids (4 g/day). The subjects ingested the capsules over the course of the day with their meals.

Standard meal and snack. A standard meal was ingested after the 8-h blood draw, and a standard snack was ingested at 11.5 h after the fat meal (12.5 h before the final blood draw), as previously described (31). Other than the provided meals and prescribed exercise, the subjects were instructed not to consume any other food or drink except water and not to exercise during the treatment periods.

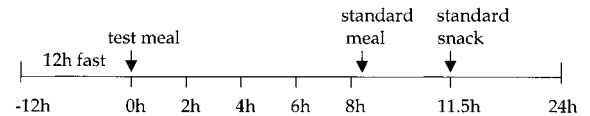
Blood sample collection. All blood samples were collected into EDTA-containing tubes from an antecubital vein after the subject had been seated in an upright position for 3 min. The blood was separated by centrifugation at 4°C for 15 min

Table 2. *Composition of diet from 3-day diet record*

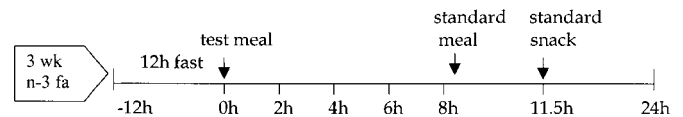
	FO Group	ExFO Group	<i>P</i>	Combined
Protein, %	13.3 ± 5.5	19.1 ± 6.1	0.12	16.3 ± 6.3
Carbohydrate, %	55.7 ± 14.9	43.3 ± 7.4	0.11	49.5 ± 12.9
Fat, %	30.8 ± 12.3	34.3 ± 7.2	0.57	32.6 ± 9.8
Saturated fat, %	10.0 ± 4.0	12.0 ± 3.4	0.38	11.0 ± 3.7
Monounsaturated fat, %	9.0 ± 5.2	9.8 ± 3.2	0.75	9.4 ± 4.1
Polyunsaturated fat, %	6.0 ± 2.5	4.0 ± 1.8	0.10	5.5 ± 3.1
kcal/day	2,240 ± 349	2,420 ± 1,220	0.74	2,330 ± 861

Values are means ± SD. There were no significant differences between groups (*P* > 0.05).

CONTROL TRIAL



n-3 fa FO TRIAL (FO)



EXERCISE PLUS N-3 FA TRIAL (EXFO)

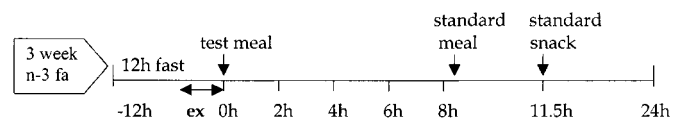


Fig. 1. Time flow chart for trials. Fish oil (FO) group performed control and n-3 fatty acid (n-3 fa) trial; ExFO group performed control and exercise + n-3 fatty acid trial. Blood samples were collected at 0 h (pre) and 2, 4, 6, 8, and 24 h after fat meal.

at 2,000 g (Beckman TJ-6 centrifuge), and the plasma was transferred to a storage vial and stored at -70°C.

TG and HDL-C analyses. Plasma was analyzed for TG and HDL-C. All samples from a given subject were analyzed in a single run. Plasma TG and HDL-C were measured enzymatically using diagnostic kits (Sigma Diagnostics, St. Louis, MO) on a spectrophotometer (model DU-2, Beckman Instruments, Fullerton, CA), as previously described (30). The TG area scores under the TG-time curve, in which the change in TG levels from baseline to each postprandial time point was determined, and the area under the TG curve were determined by the trapezoidal rule. The mean intra-assay coefficient of variation for 10 runs of triplicate or quadruplicate samples was 2.5% for TG and 0.3% for HDL-C.

Fatty acid analysis. Fatty acids were analyzed in the TG and phospholipid fractions of plasma, according to the method of Sun (25), before and after the FO supplementation. This analysis provided a means of confirming the compliance of the subjects in taking the supplement. The mean coefficient of variation for 16 runs of triplicate samples was 3.2%.

Statistics. For within-group analyses, a two-way (trial × time) ANOVA with repeated measures was performed on TG and HDL-C concentrations, and a one-way ANOVA with repeated measures was performed on TG scores. Significant *F* ratios (*P* < 0.05) for time were followed with a Newman-Keuls post hoc test. For between-group comparisons (FO vs. ExFO), one-way ANOVAs were used to compare baseline and peak TG concentrations and TG area scores between the experimental trials.

RESULTS

The results from the maximal testing are given in Table 1. Analysis of individual 20:5 and 22:6 fatty acids indicated that all subjects used the supplement. Al-

Table 3. Fasting triglyceride concentrations

Group	Pre-FO	Post-FO	<i>P</i>
FO	122.9 ± 66.7	80.3 ± 29.2	0.057
ExFO	136.8 ± 72.6	94.7 ± 38.7	0.051
Combined	129.9 ± 66.9	87.5 ± 33.5*	0.003

Values are means ± SD in mg/dl. *Significantly different from Pre-FO.

though no subject had more than trace amounts (<1%) of these fatty acids before the supplementation, all subjects in both experiments had measurable concentrations after supplementation, most of it residing in the phospholipid fraction. For all subjects, the range in the phospholipid fraction for 20:5 was 4.1–7.5% and the range for 22:6 was 2.6–4.9%.

The mean baseline (fasting) TG concentrations were 122.9 ± 66.7 and 136.8 ± 72.6 mg/dl in the FO and ExFO groups, respectively (*P* > 0.05; Table 3). Separately, the fasting TG concentrations in each group before and after FO supplementation were of borderline significance, whereas the combined means were significantly different, with baseline TG 33% lower as a result of the FO ingestion.

In the FO experiment, the FO trial was not significantly different from the control trial (*P* = 0.08). For both trials, the TG concentration was significantly different over time, with the concentration at 2, 4, and 6 h higher than the values at the other time points (Fig. 2). The mean TG area scores were not significantly different between the two trials, whereas the peak TG concentrations approached statistical significance (*P* = 0.07; Fig. 3).

In the ExFO trials, the average O₂ consumption during the submaximal exercise session was 2.08 ± 0.13 l/min, at a mean heart rate of 147 ± 14 beats/min and a respiratory exchange ratio of 0.94 ± 0.06. The

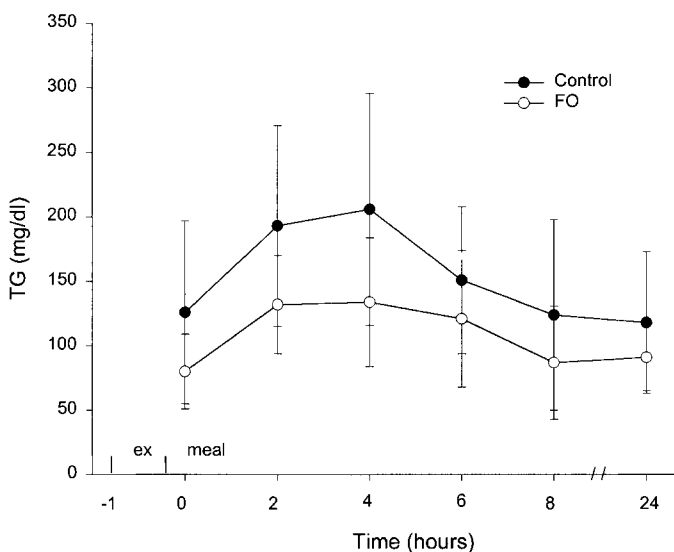


Fig. 2. FO supplementation and triglyceride (TG) concentrations over time. There were no statistically significant differences between trials; for combined trials, values at 2, 4, and 6 h were significantly different from values at 0, 8, and 24 h.

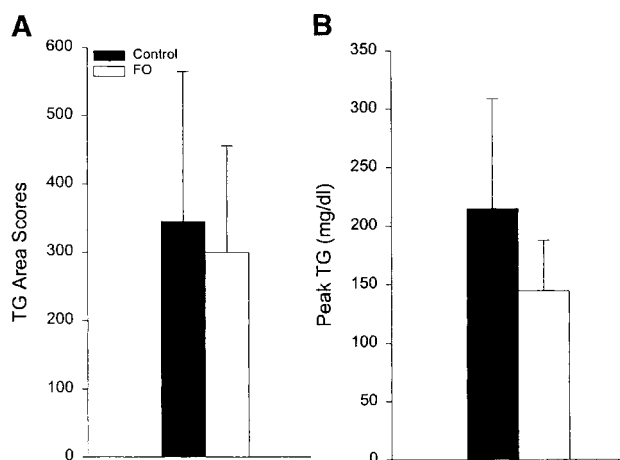


Fig. 3. TG area scores and peak concentrations between trials for FO treatment. A: TG area scores. B: TG peak concentrations. There were no significant differences between trials.

average percent $\dot{V}O_{2\max}$ was 63.0 ± 0.1, and the mean energy expenditure for the 60-min session was 619 ± 38 kcal.

In the ExFO treatment, the TG response in the ExFO trial was not different from the control. However, the TG levels at 2 and 4 h were higher than those at 0, 8, and 24 h (Fig. 4). The TG area score was not different between the ExFO trial and control, nor was the ExFO peak TG concentration significantly different from the control (Fig. 5).

In comparing the TG area scores of the two experimental trials, the FO trial score (299.6 ± 156.0) was not significantly different from the ExFO trial score (289.3 ± 265.6). In addition, the FO trial peak TG concentration (145.0 ± 42.9 mg/dl) was not significantly different from the ExFO trial TG peak (185.2 ± 83.8).

There were no statistical differences in the postprandial HDL-C concentrations between trials for the FO

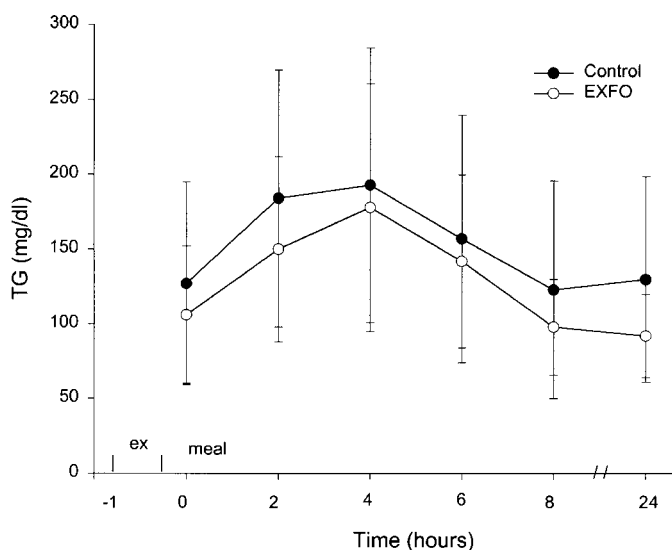


Fig. 4. Exercise + FO supplementation and TG concentrations over time. There were no statistically significant differences between trials, but, for combined trials, values at 2 and 4 h were significantly higher than values at 0, 8, and 24 h.

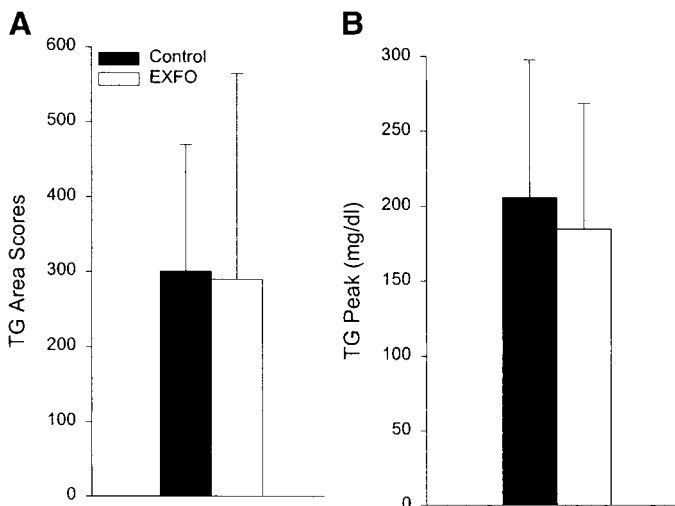


Fig. 5. TG area scores and peak concentrations between trials for ExFO treatment. *A*: TG area scores. *B*: TG peak concentrations. There were no significant differences between trials.

and ExFO treatments (Table 4). A main effect for time did exist for the ExFO treatment, in that the 8-h combined value was significantly higher than the 4-h combined value.

DISCUSSION

Fasting TG levels. The decrease in fasting TG levels after the FO supplementation is similar to many other reported observations (5, 10, 12). Harris and Muzio (12) reported a 32% decrease in plasma TG levels after 4 wk of supplementation compared with the 33% decrease for 3 wk in the present study on about the same n-3 fatty acid dose (~4 g/day). The mechanism for this TG-lowering effect of n-3 fatty acid appears to be the inhibition of TG synthesis and/or secretion in the liver (21).

Supplementation with n-3 fatty acid. The primary objective of this study was to determine whether the effect of FO + exercise on PPL was different from the effect of FO alone. We found that FO alone did not affect the total area under the postprandial TG curve but lowered fasting and tended to lower peak TG concentration. This finding agrees with the results of Marckmann, Bladbjerg, and Jespersen (17), who observed an 18% decline in peak PPL after 4 wk of n-3 fatty acid supplementation.

Table 4. Postprandial HDL-C concentrations

Trial	Time			
	0 h	4 h	8 h	24 h
FO				
Control	41.8 ± 6.5	42.6 ± 6.7	38.5 ± 2.1	40.4 ± 6.3
Experimental	41.7 ± 5.0	42.2 ± 7.4	40.4 ± 4.9	38.9 ± 6.0
ExFO				
Control	42.0 ± 8.6	41.5 ± 10.6	46.4 ± 7.5*	43.0 ± 10.4
Experimental	46.7 ± 13.3	41.8 ± 13.3	48.0 ± 12.9*	45.8 ± 12.8

Values are means ± SD. There were no significant differences between trials but a significant difference across time in ExFO trial. *Significantly different from 4 h ($P < 0.05$).

In contrast to the present findings, other investigators reported a decrease in the TG area score after n-3 fatty acid ingestion (5, 12). However, previous investigations have not controlled for fitness level of the subjects. Ziogas et al. (33), using men and women, demonstrated that aerobic training is associated with a lower postprandial TG response. This finding suggests that fitness status may be an important factor in determining postprandial TG levels and, therefore, should be considered in interpreting results from research on PPL. In the present study, all subjects were sedentary, and thus the results were not confounded by level of previous training.

Combination of n-3 fatty acid and exercise. A session of aerobic exercise, when performed in conjunction with n-3 fatty acid supplementation, did not result in an attenuation of PPL. This was an unexpected result, because aerobic exercise (28, 31) and n-3 supplementation (5, 12) each have been shown to reduce PPL when expressed as TG area scores and peak TG concentrations. The exercise effect has been especially powerful with a 1-h session of aerobic exercise, reducing PPL up to 50% (31). In the present study, the postprandial TG concentration levels were similar after exercise + FO supplementation and the control trial. A logical conclusion from these data is that one treatment interferes with the action of the other. The inhibition of this lipid-lowering effect of FO by exercise has not been reported previously. In a study in which FO was coupled with exercise, Warner et al. (29) found that exercising 3 days/wk for 12 wk in conjunction with FO supplementation can lower fasting TG levels. However, the response was the same in those who exercised and took FO and those who only took FO. Although there did not appear to be a negative interaction between the two treatments in that study, nor was there an additive effect, as would be hypothesized.

Exercise may interfere with the PPL effects of n-3 fatty acid by the following mechanisms: 1) It may block the n-3 fatty acid inhibition of TG synthesis or secretion in the liver. Blood flow to the liver is reduced during exercise (4), which would suggest that exercise, like n-3 fatty acid, causes reduced TG release from liver to blood. A substantial portion of the PPL can originate from the liver (10). However, the exercise interference with the n-3 fatty acid effect occurred after exercise, a time when blood flow to the liver should be normalized. 2) Exercise may inhibit the n-3 fatty acid-induced clearance of TG from plasma. There are few data to support this concept, since exercise is known to stimulate lipoprotein lipase (LPL) activity in muscle (16, 22). In addition, most studies have indicated that n-3 fatty acid supplementation does not alter LPL activity (10, 18). These authors concluded that n-3 fatty acid does not affect clearance of TG via increased LPL activity. Thus an inhibition of LPL activity does not appear to be the mechanism of the exercise interference with n-3 fatty acid.

On the other hand, n-3 fatty acids may interfere with the attenuating effect of aerobic exercise on PPL. At least two possibilities may be postulated: inhibition

of the exercise-induced increase in LPL activity or inhibition of the exercise-induced increase in muscle blood flow. Authors who have reported an exercise-induced attenuation of PPL have attributed the effect to increased activity of LPL (2, 28, 31). Exercise has been shown to induce LPL gene expression in skeletal muscle (22, 23). LPL activity becomes elevated shortly after an exercise session, but the activity does not peak until 18 h after an exercise session and remains elevated for up to 30 h (14, 16). On the other hand, exercise appears to have no effect on the activity of LPL in adipose tissue (22).

The presence of n-3 fatty acid may prevent the exercise effect on PPL by interfering with the function of LPL, perhaps by changing the composition of chylomicrons so that interaction with LPL is diminished or by increasing lipid oxidation (24).

More likely, n-3 fatty acid may have a more direct effect on LPL activity. Muscle and adipose LPL activity have not been differentiated routinely after n-3 fatty acid ingestion. However, at least one study illustrated that n-3 fatty acid supplementation caused an effect similar to exercise, i.e., increased muscle LPL activity but no effect on adipose activity (13). Most investigators have reported no effects on heparin-releasable LPL activity with n-3 fatty acid supplementation (7, 10, 18). However, this finding is not unanimous, since Harris et al. (11) observed increases in endogenous (non-heparin) LPL activity. Thus the evidence suggests that a direct inhibition of muscle LPL by n-3 fatty acid seems unlikely.

n-3 Fatty acid also may inhibit the LPL response via hormone actions. The most likely candidate for this effect is insulin. An exercise session has been shown to have a consistent inhibitory effect on insulin secretion (24), although n-3 fatty acid effects on insulin have been more equivocal (17, 28). However, at least one group of investigators reported an increase in insulin concentration after 2 mo of n-3 fatty acid supplementation (20), and insulin infusion has been shown to cause a decrease in muscle LPL activity and an increase in adipose LPL activity (9). Thus n-3 fatty acid may interfere with the exercise effect on muscle LPL activity via its effect on insulin release.

A second possibility for the antagonistic effect of n-3 fatty acid is related to blood flow. Some authors have speculated that the increased TG clearance related to acute exercise is due to the elevated muscle blood flow during exercise, thus allowing more lipoprotein to come into association with LPL (23). It is possible that n-3 fatty acids inhibit blood flow to muscle during exercise. A decreased endurance exercise performance after n-3 fatty acid supplementation supports this possibility (3). There also is evidence that n-3 fatty acids actually enhance fingernail capillary blood flow (6), which may suggest that peripheral blood flow is enhanced after n-3 fatty acid supplementation and thus diverts blood flow from muscle. In addition, the LPL response to exercise appears to be delayed for several hours (14, 23), a finding that would argue against a blood flow mechanism. Thus the potential for interference of

muscle blood flow by n-3 fatty acid requires further research. The entire hypothesis of an n-3 fatty acid inhibition on exercise effects appears to be a fruitful topic for future investigations.

Another possible explanation for the nonsignificant results was that the number of subjects used did not allow sufficient statistical power. To evaluate this possibility, effect sizes were calculated using a method for calculating η^2 (26). The η^2 indicates the effect size as a function of total variance accounted for by the independent variable. The key comparisons in this study relate to the differences in the TG response between the experimental and control trials in each experiment. In the FO experiment, the η^2 for the trial comparison ($P = 0.07$) for TG concentrations was 0.50, and a sample size of 11 would be needed to achieve a statistically significant difference at a power of 0.80. In the ExFO experiment, a similar calculation for trial ($P = 0.18$) yielded an η^2 of 0.33 and a required sample size of 21.

For the TG area scores in the FO ($P = 0.60$) and ExFO ($P = 0.93$) experiments, the η^2 values were very low, and the calculated required sample sizes would be 150 and infinity, respectively. The TG area scores for the FO vs. the ExFO experimental conditions also were not close to significantly different ($P = 0.94$), with an η^2 of 0.0005, requiring a sample size of infinity to achieve statistical significance with a power of 0.80. In summary, the low number of subjects may have affected the results for TG concentrations over time in the FO experiment. However, for most comparisons and especially for those involving the ExFO experiment, similar findings would be expected even with a very large sample size. Large sample sizes are not practical in experiments in which daily multiple blood sampling is required of the subjects. Although the interference between n-3 fatty acid and exercise needs to be confirmed, these unexpected results do not appear to be a result of small sample size.

The HDL-C concentrations were not different between the control and experimental trials of the FO and ExFO groups. In the ExFO experiment, the 8-h HDL-C level was higher than the 4-h value. Likewise, Zhang et al. (31) reported an elevation of HDL-C 8 h after the test meal in subjects who exercised 12 h before the meal. Most studies have reported that acute exercise increases HDL-C (2, 14, 31). However, none of these studies used exercise in conjunction with n-3 supplementation, which may influence the response of HDL metabolism to exercise.

On the other hand, other investigators have reported that n-3 fatty acid supplementation does not affect baseline HDL-C concentrations (8, 18). This finding was confirmed in the present study. In addition, our data suggest that n-3 fatty acid supplementation for 3 wk does not affect the postprandial HDL-C concentrations after a high-fat meal.

It is possible that changes in plasma volume induced by the aerobic exercise may have affected the interpretation of the TG and HDL-C data. Aerobic exercise has been shown to decrease plasma volume immediately after exercise, and, in recovery, plasma volume has been

shown to expand; this expansion may persist to some degree for several hours after exercise and thus potentially decrease concentration measurements in blood (15). In a previous study (32), we observed only small changes in plasma volume (<5%) caused by moderate-intensity exercise for 60 min. Regardless, the potential hemodilution several hours after an exercise session would cause, at most, small decreases in the TG and HDL-C concentrations in recovery, alterations that would not account for the lack of an exercise-induced TG-lowering effect or the positive exercise effect on HDL-C in recovery.

In conclusion, supplementation with n-3 fatty acid appears to lower fasting and peak postprandial TG concentrations but may interfere with the beneficial effect of acute exercise on PPL. In addition, n-3 fatty acid supplementation does not appear to affect baseline or postprandial HDL-C concentrations.

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