Effect of Exercise on Postprandial Lipemia Following a Higher Calorie Meal in Yucatan Miniature Swine

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Exercise has been shown to attenuate the postprandial lipemic (PPL) response to a modest kcal high-fat meal in numerous human studies, but has not been fully examined in swine. In addition, the effects of exercise on a high-fat meal of larger magnitude have not been examined in humans or in swine. Thus, the purpose of this study was to examine the PPL response to a single, high-fat/cholesterol (HFC) meal (~3,000 kcal, 1,300 kcal from fat) and determine if exercise attenuates the PPL response. Sedentary, female Yucatan miniature swine (n = 10) completed 3 PPL trials: (1) pre diet (PRE); (2) post HFC diet (POST); and (3) post HFC diet plus exercise (EX, 45 minutes at 75% heart rate maximum). Blood samples were collected before (0 hour) and at 2, 4, 6, and 8 hours after the single HFC meal for PPL analysis. Postheparin lipoprotein lipase (LPL) activity was assessed at 8 hours. While fasting LPL activity was significantly increased with the HFC diet, the PPL response to the HFC meal did not differ depending on diet. Furthermore, the PPL response was not significantly altered with a single session of exercise, perhaps because of the severity of the HFC meal, the sedentary nature of the swine, or because LPL activity was not elevated after exercise. These findings suggest that administration of a HFC meal of this magnitude (~3,000 kcal, 1,300 kcal from fat) will promote significant elevations in postprandial triglyceride (TG) concentrations, overwhelm the adaptive response to a HFC diet (elevated LPL activity), and attenuate the beneficial effects of a single exercise session on this system.

POSTPRANDIAL LIPEMIA (PPL) is the elevated concentration of plasma triglycerides (TG) that occurs following a meal and is considered a risk factor for cardiovascular disease (CVD).[1] Even those individuals with normal lipid profiles in a fasting state can be in a hypertriglyceridemic state for several hours after a high-fat meal,[1,2] with a peak occurring around 4 to 6 hours.[2,3] During these times of elevated TG concentrations, the vessel wall is exposed to atherogenic postprandial lipoproteins.[4]

Human studies consistently have shown the beneficial effects of aerobic exercise on attenuating the PPL response, lowering it by as much as 51%,[3-9] with these reductions being associated with an increase in lipoprotein lipase (LPL) protein level and enzymatic activity.[10] LPL, an enzyme bound to capillary endothelium, is found predominantly in muscle and adipose tissue.[11] LPL hydrolyzes the TG into free fatty acids (FFAs), which are taken up by muscle and adipose tissue, resulting in reduced plasma TG concentrations.[12]

Yucatan miniature swine have similar lipoprotein profiles and adapt to exercise training in a similar fashion to that of humans, including an increase in stroke volume, $\dot{V}O_{2max}$, muscle oxidative enzymes, and high-density lipoprotein-cholesterol (HDL-C).[13,14] Swine also experience a decreased resting[15,16] and submaximal exercise heart rate,[17] and lowered total cholesterol (TC) with exercise training.[15,18]

Although much of swine physiology has been well investigated, the PPL response to a high-fat meal has not received adequate attention in the swine literature.[19] To further document the value of the swine model in the study of human health and disease, it is important to determine if the swine response is similar to the human in this novel CHD risk factor. In addition, the swine model offers precise control of dietary intake and the ability to examine the PPL response to a meal of much larger magnitude than used previously in human studies.

The purpose of this study was to examine the PPL response of swine fed a single high-fat/cholesterol (HFC) meal (~3,000 kcal) of atherogenic feed. Additionally, after the swine were on the atherogenic diet for 30 to 45 days, we determined if a single session of exercise attenuates the PPL response to this HFC meal. Specific hypotheses included that the HFC meal would significantly elevate TG concentrations, and this elevation would be significantly greater while swine were being fed a low-fat (LF) diet versus a HFC diet. In addition, despite the magnitude of the HFC meal, we hypothesized that the PPL response would be attenuated significantly with the single session of exercise compared with the nonexercise trial, and this attenuation would be associated with an increase in LPL activity.

MATERIALS AND METHODS

Animals

Yucatan female (n = 10) miniature swine (Sinclair Research Center, Columbia, MO) were used. All experimental procedures involving animals were approved by the University of Missouri Animal Care and Use Committee in accordance with the Principles for the utilization and care of vertebrate animals used in testing, research, and training.

All swine (adult, gilts) were approximately 9 months of age when the study began. The average weight of the swine was 37.1 ± 1.1 kg during

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the LF diet testing and 41.1 ± 0.8 kg during the HFC diet testing. Under general anesthesia, a vascular access port (Access Technologies, Skokie, IL) was surgically implanted into each pig via left external jugular vein catheterization similar to previously described methods.[20] Anesthesia was induced with the following drugs administered intramuscularly (in milligrams per kilogram): atropine 0.05, telazol 6.6, and xylazine 2.2; depth of anesthesia was subsequently maintained with isoflurane gas (up to 4%). The vascular access port was located subcutaneously on the left side of the neck and provided a nontraumatic and convenient means of serial venous blood sampling. Prior to the study, the swine's estrous cycles were monitored for a minimum of 3 months. To reduce the effects of cyclic female hormones (17-β estradiol),[21] all PPL trials and baseline blood samples were collected during low estradiol levels.

The pigs were housed in stainless steel cages with Tenderfoot flooring (Tandem Product, Minneapolis, MN). All swine were in separate quarters to ensure controlled feeding of the precise quantity. The facility was maintained on a 12-hour light-dark cycle at a controlled temperature of 68°F to 72°F.

Experimental Design

Each swine completed the following 3 PPL trials and a baseline sample before and after 30 to 45 days on a HFC diet: (1) pre diet (PRE); (2) post diet (POST); and (3) post diet plus exercise (EX). PRE trial was completed while the swine were on a LF diet (8% kcal from fat) for a minimum of 5 months. POST and EX trials were randomized, separated by 7 to 14 days, and completed after the swine had been on an equivalent calorie HFC diet for 30 to 45 days. The HFC diet contained 46% kcal from fat and 2% cholesterol by weight.

A timeline of an individual trial is shown in Fig 1. In all PPL trials, swine were fasted 24 hours and fed a HFC meal (~3,000 kcal-1,300 kcal from fat) to induce PPL. Blood samples were collected prior to HFC meal (0 hour) and at 2, 4, 6, and 8 hours post-HFC meal. A heparin injection (100 U/kg) was administered after 8-hour blood sample, followed by an additional blood collection (8 hours +15 minutes). For the EX trial, the swine completed a single session of aerobic exercise for 45 minutes at 75% of heart rate maximum. A blood sample was collected prior to the exercise session and other blood sampling followed the same timeline as the other PPL trials.

To assess the effects of the atherogenic, HFC diet alone on the lipoprotein profile, a baseline heparinized plasma sample was collected while the swine were on a LF diet and following 30 to 45 days of HFC feeding. The samples were collected following a 24-hour fast.

Exercise Session

During the 30 to 45 days of HFC diet feeding, pigs were acclimated to treadmill running. The habituation consisted of swine being placed on a motor driven treadmill for 3 minutes on 3 separate occasions at an average treadmill speed of 2.5 mph. For the EX trial, each swine performed a single exercise session at approximately 75% of heart rate maximum (60% $\dot{V}O_{2max}$). Heart rates were monitored using a Polar heart rate monitor (Polar CIC, Port Washington, NY). The pigs had an average resting heart rate of 80 ± 2 bpm and 75% heart rate maximum was 208 ± 4 bpm. Since the swine were to remain sedentary, heart rate maximum (272 ± 15 bpm) was determined from our previous work on swine of same age and gender.[22] The single exercise session consisted of a 5-minute warm-up, followed by 45 minutes of target exercise (~4.0 mph treadmill speed), and completed with a 5-minute cool-down. It was determined from previous work in this lab that 45 minutes was the maximal amount of time some swine could endure for their first exercise session.

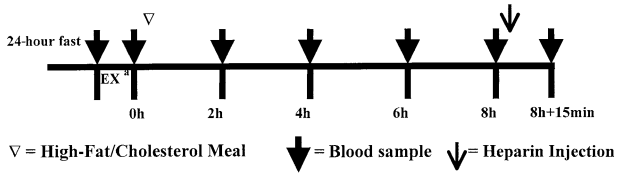

Fig 1. PPL trial timeline. [a]For trial 3 only, exercise session (EX, 45 minutes at 75% heart rate maximum) immediately prior to HFC meal. HFC meal: approximately 3,000 kcal (46% fat) to induce PPL.

Diet

All swine were fed a LF diet for a minimum of 5 months at the beginning of the study. The pig chow for this diet consisted of Purina Lab Mini-Pig Diet Breeder pig chow. By weight, this pig chow contained 16.7% protein, 2.6% fat, and 53.2% total carbohydrate and supplied 3.0 kcal · g^{-1} feed (22% of kcal from protein, 8% from fat, and 70% from carbohydrate). The pigs were fed approximately 1,000 g (~3,000 kcal) of LF diet feed once each day.

All swine were fed a high-fat/cholesterol (HFC) diet for 30 to 45 days. The pig chow for this diet consisted of Purina Lab Mini-Pig Diet Breeder pig chow supplemented with (% = g · 100 g^{-1} by weight) 2.0% cholesterol, 17.1% coconut oil, 2.3% corn oil, and 0.7% sodium cholate. By weight, this pig chow contained 13% protein, 21.3% fat, and 41.3% total carbohydrate and supplied 4.09 kcal · g^{-1} feed (13% of kcal from protein, 46% from fat, and 41% from carbohydrate). The feed was prepared at the Sinclair Research Center. The pigs were fed approximately 700 g (~3,000 kcal) of HFC diet feed once each day.

The HFC meal to induce PPL was the same as one meal of the HFC diet. Water was given ad libitum.

Blood Collection and Analyses

All blood samples were collected via a 21-gauge Huber needle inserted into a vascular access port. All nonheparinized blood samples were collected into 10-mL tubes containing EDTA (anticoagulant and chelating agent). Whole blood was collected into 2 micro hematocrit (Hct) tubes to measure Hct. Heparinized blood was collected into 10-mL heparin tubes. All samples were separated by centrifugation at 4°C for 15 minutes at 2,000 × *g* in a Marathon 22100R centrifuge (Fisher Scientific, Pittsburgh, PA) and plasma stored at −70°C until analyzed. Nonheparinized plasma was used to analyze all variables with the exception of LPL activity and hepatic lipase (HL) activity, which were analyzed using the heparinized plasma samples.

Hct was measured prior to exercise, prior to the HFC meal, and at 2, 4, 6, and 8 hours following the HFC meal. Changes in plasma volume were assessed from Hct as previously described.[23]

For all plasma assays, all samples from each pig were analyzed together to eliminate interassay variability. Plasma TC and TG concentrations were analyzed enzymatically using Sigma Diagnostic kits (INFINITY Reagents, Procedure #402 and #344, St Louis, MO, respectively) and quantitated spectrophotometrically (Beckman model DU-530, Fullerton, CA) at 500 and 520 nm, respectively. The average intra-assay coefficient of variation (CV) was 1.8% and 2.5% for TC and TG, respectively.

TG concentration was measured in each of the nonheparinized blood samples (0, 2, 4, 6, and 8 hours). The magnitude of TG response was quantified as the total area under the TG curve (TG AUCT), the incremental TG AUC (TG AUCI), and the TG peak response. The TG AUCT was calculated using the trapezoidal method described by Tai.[24] The TG AUCI also was calculated using the trapezoidal method, but the baseline TG values were subtracted from each TG value before completing the calculations using the following formula:

$$TG - AUC_T = 0.5*[T_1(t_0 + t_2) + T_2(t_2 + t_4) + T_3(t_4 + t_6) + T_4(t_6 + t_8)]$$

where:

$T_1 = x_2 - x_0$, $T_2 = x_4 - x_2$, $T_3 = x_6 - x_4$, and $T_4 = x_8 - x_6$
x_n = n hour
t_n = the TG concentration at n hours.

The TG peak response was defined as the difference between greatest plasma TG concentration minus the 0 h plasma TG value.

Plasma concentrations of total HDL-C, HDL_2-C, and HDL_3-C were measured while the swine were consuming the LF diet and after 30 to 45 days of HFC feeding (both 24-hour fasted samples). The samples were analyzed using a modified heparin-$MnCl_2$-dextran sulfate method[25] with cholesterol for total HDL-C and HDL_3-C assayed as described above. The average intra-assay CV was 0.8% and 2.3% for HDL-C and HDL_3-C, respectively.

Plasma concentrations of low-density lipoprotein-cholesterol (LDL-C) were calculated using the Friedewald equation.[26] We previously have observed a very high correlation ($r = 0.94$) when comparing LDL-C calculated using the Friedewald equation versus LDL-C determined by ultracentrifugation from swine (n = 62) of same age and gender (unpublished observations).

The LPL assay is the measurement of the rate at which the enzyme catabolizes TG into FFAs.[27] This rate is determined by the degree of activation of LPL and the quantity present in the sample. The liberation of oleic acid from TG is the measured parameter in this assay adapted from Illingworth et al.[27] Specifically, postheparin plasma was incubated with [^{14}C] triolein (American Radiolabeled Chemicals, St Louis, MO) and heat-inactivated serum (a source of apolipoprotein C-II). After incubation with agitation, the reaction was stopped by the addition of organic (methanol/chloroform/heptane) mixture and sodium hydroxide solution. After centrifugation, an aliquot of the upper phase was transferred to a vial and mixed with scintillation cocktail. The liberation of FFAs by LPL and HL was measured in a scintillation counter as total lipase activity. The addition of sodium chloride solution to the substrate emulsion was used to inhibit LPL and thus allowed the measurement of HL activity. LPL activity was calculated as the difference between total and HL activity. All assays were performed in duplicate after incubation at 37°C for 60 minutes using 30 μL plasma per incubation. The average intra-assay CV was 2.1% and 6.2% for total activity and HL activity, respectively.

Statistical Analysis

Plasma TG concentrations before and at 2, 4, 6, and 8 hours after the meal were analyzed using a 2-way analysis of variance (ANOVA) (time × trial) with repeated measures on time and trial. The area under the TG curves and TG peak responses were analyzed by using 1-way ANOVAs to detect differences among the 3 trials. One-way ANOVAs also were used to analyze differences in TC, HDL-C and subfractions, LPL activity, and HL activity. All variables were corrected for plasma volume changes and statistical analyses were repeated. However, no variables differed significantly from the uncorrected values; thus, the original uncorrected data are presented. Significant F ratios ($P < .05$) were followed up using post hoc contrast comparisons with specifically designed error terms. Values are reported as means ± standard error of the mean (SE).

Effect size for power analysis originally was determined on TG AUCI and TG peak response from previous human work since no swine data was available. An N of 5 was calculated to obtain statistical significance for each of the 2 variables in response to exercise.[3] Since we had observed previously that the lipoprotein response to exercise was variable in the swine, we used 10 pigs in the present study.

Table 1. Effects of LF and HFC Diets on Fasting Values of the Lipoprotein Parameters

	LF Diet	HFC Diet
Total cholesterol (mg/dL)	83.8 ± 1.2	460.7 ± 41.1*
Triglycerides (mg/dL)	44.9 ± 2.2	50.5 ± 8.2
LDL-cholesterol (mg/dL)	32.7 ± 1.5	343.2 ± 39.9*
HDL-cholesterol (mg/dL)	42.1 ± 1.9	107.3 ± 5.0*
HDL_2-cholesterol (mg/dL)	33.9 ± 1.3	90.1 ± 6.2*
HDL_3-cholesterol (mg/dL)	8.2 ± 1.1	17.2 ± 1.9*
LPL activity (μmol/mL/hr)	10.9 ± 0.3	11.7 ± 0.4*
HL activity (μmol/mL/hr)	0.8 ± 0.1	0.7 ± 0.1

NOTE. Values are means ± SE.

Abbreviations: LF diet, low-fat diet; HFC diet, high-fat/cholesterol diet.

*Higher than LF diet, $P < .05$.

RESULTS

Table 1 depicts the effects of 30 to 45 days on a HFC diet on fasting values for TC, TG, LDL-C, HDL-C and subfractions, and LPL and HL activity. Each of the variables was collected following a 24-hour fast. As was anticipated, the change from the LF diet to the HFC diet significantly increased all values with the exception of TG concentrations and HL activity.

Plasma concentrations of TG during the postprandial observation period are shown in Fig 2. There were no significant trial ($P = .88$) or trial by time ($P = .11$) interactions. The data for TG peak response are shown in Fig 3. From a mean baseline value of 51 ± 8 mg/dL, PRE versus POST TG peak responses were not significantly different ($P = .10$). Exercise tended to attenuate TG peak responses, POST versus EX, but values did not differ statistically ($P = .07$).

TG AUCI after the HFC meal is shown in Fig 4. PRE versus POST TG AUCI were not significantly different ($P = .23$). POST versus EX comparisons also did not reach statistical significance ($P = .08$). No significant differences were found among the PPL trials for TG AUCT.

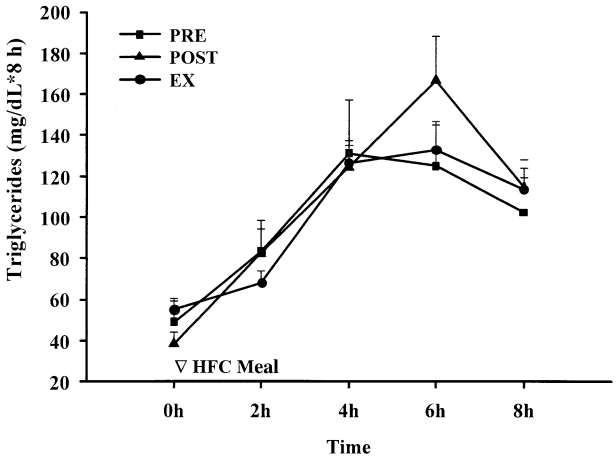

Fig 2. Plasma TG response following a HFC meal. Values are means ± SE. PRE, swine consuming LF diet; POST, swine consuming HFC diet; and EX, swine consuming HFC diet plus exercised for 45 minutes. No significant differences among trials.

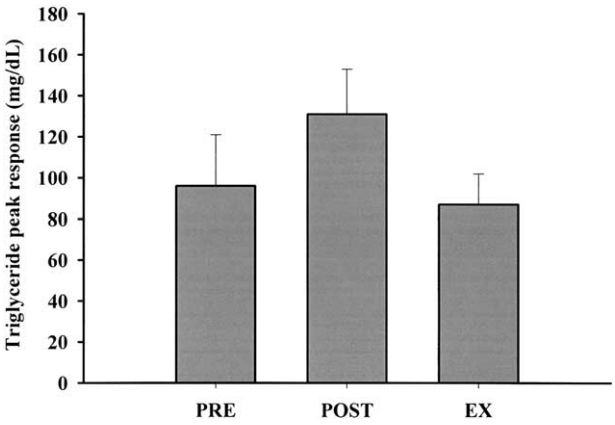

Fig 3. TG peak response following a high-fat/cholesterol (HFC) meal. Values are means ± SE. PRE, swine consuming LF diet; POST, swine consuming HFC diet; and EX, swine consuming HFC diet plus exercised for 45 minutes. No significant differences among trials.

Postprandial LPL and HL activity was assessed 8 hours after a HFC meal. Postprandial LPL activity did not differ significantly ($P = .497$) among the 3 PPL trials (PRE = 11.3 ± 0.3 μmol/mL/h, POST = 11.2 ± 0.3, and EX = 10.9 ± 0.3). In addition, postprandial LPL activity did not differ from fasting values presented in Table 1. Additionally, HL activity did not differ significantly ($P = .709$) among the 3 PPL trials (PRE = 0.7 ± 0.1μmol/mL/h, POST = 0.7 ± 0.1, and EX = 0.7 ± 0.1).

DISCUSSION

To further elucidate the appropriateness of the swine as a model in human disease, the present study examined PPL responses in swine following dietary and exercise manipulations. The similarities between swine and human physiology make Yucatan miniature swine an attractive model for the study of PPL. Furthermore, the ability to control physical activity and administer a meal of larger magnitude avoids issues associated with human studies. The scientific literature on the effects of diet on the lipoprotein profile and PPL response in swine is limited. A few studies have reported that the administration of an atherogenic diet altered the lipoprotein profile of miniature swine.[28-30] Data shown in Table 1 are consistent with previous lipid data collected in our laboratories using the same atherogenic diet.[30-33]

Our study demonstrated that 30 to 45 days of HFC diet feeding resulted in a significant increase in fasting postheparin LPL activity (Table 1), similar to previous reports.[30] Increases in LPL activity following high-fat feeding also have been reported in human literature, with increases attributed to adipose tissue LPL activity.[34] It was not possible from the current study to distinguish between muscle and adipose tissue LPL activities because postheparin plasma LPL has both origins. However, observed increases in the present study did not result in attenuation of the PPL response to a single HFC meal (Figs 3 and 4, PRE *v* POST), which lead to prolonged elevation in TG concentrations.

Results indicated that a single session of exercise did not yield a statistically significant attenuation in the PPL response in sedentary, female Yucatan miniature swine (Figs 2, 3, and 4). The ability of an aerobic exercise session to reduce PPL could be related to the length of time between the exercise session and the meal,[8,35] as well as the length of the exercise session.[3] Several human studies have reported significant reductions in PPL when the exercise session was immediately to several hours prior to the ingestion of a high-fat meal.[3,5-9] Zhang et al[3] reported a 38% reduction in TG AUCI in active human subjects who exercised for 60 minutes at 75% heart rate maximum immediately prior to ingestion of an approximately 1,000 kcal high-fat shake. Using the same timing of the exercise session, the swine in the present study exercised for 45 minutes at 75% heart rate maximum immediately prior to the HFC meal ingestion. Swine only exercised for 45 minutes because we had observed previously that some sedentary swine could not tolerate 60 minutes of exercise. This limited duration affected the energy expenditure associated with the exercise session, which also may play a role in the ability of exercise to reduce the PPL response.[36] Energy expenditure was not measured, but it can be assumed that the 45 minutes that the swine ran did not burn as many calories relatively compared with a 60-minute run or 90-minute walk used in many human studies.

The HFC meal in the present study was a single meal from the atherogenic diet used in our laboratory and contained approximately 3,000 kcal, perhaps representative of a holiday or buffet type meal. This kcal quantity represented 100% of daily caloric intake of the swine, and although energy needs were not assessed directly, the amount was determined previously based on normal growth and weight gain. Though in contrast with most human studies, which have used a high-fat meal consisting only of approximately 1,000 kcal,[2,3,6,7] previous work in our laboratory[19] indicated that swine fed the same HFC meal exhibited TG peak responses similar to those reported in human literature. Furthermore, these swine received approximately 1,300 kcal (~3.5 g/kg) from fat, while humans typically receive approximately 900 kcal (1 to 1.5 g/kg) from fat for the

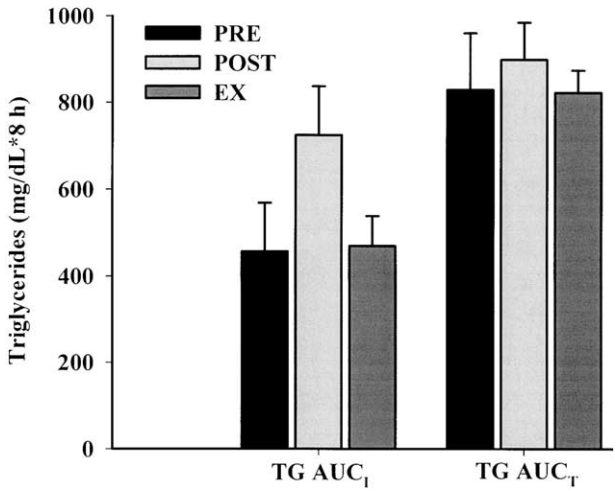

Fig 4. TG AUC (total and incremental) following a HFC meal. Values are means ± SE. PRE, swine consuming LF diet; POST, swine consuming HFC diet; and EX, swine consuming HFC diet plus exercised for 45 minutes. No significant differences among trials.

PPL high-fat meal. Despite the differences in total kcal and fat, TG peak response to the HFC meal were comparable between swine in the present study (~131 mg/dL) and those reported in human literature (~160 mg/dL).[3] However, the larger fat load resulted in a higher TG AUCI in the swine (~593 mg/dL · 8 h) compared with human subjects (~350 mg/dL · 8 h).[3] Recently, Dixon et al[19] fed swine the same 700-g HFC meal once per day and reported TG peak responses similar (126.9 ± 16.9 mg/dL) to the present study, but when the swine were fed a 350-g HFC meal twice per day, not only were TG peak responses lower (71.5 ± 10.2 mg/dL), but TG concentrations also returned to baseline within 4 hours. These data suggest that the HFC meal used may have overwhelmed the swine's ability to effectively metabolize the fat load at the measured time points and may explain why the single session of exercise failed to significantly reduce PPL.

Previous examinations utilizing other animal models have reported significant increases in postheparin and muscle LPL activity following exercise.[37,38] However, this is the first known report that examined postheparin LPL activity following a single session of exercise in Yucatan miniature swine. We found that postheparin LPL activity was not increased following the exercise session in the present study, which could explain the lack of a significant reduction in PPL.

Previous human work also has shown significant increases in LPL activity immediately to 24 hours following exercise,[35,36,39,40] as well as following consumption of a high-fat meal,[7] a finding we did not observe. In the present study, TG concentrations did not peak until 6 hours post HFC meal and remained significantly elevated 8 hours post HFC meal (Fig 2). This finding is in contrast to data from humans, where TG concentrations usually peak around 4 to 6 hours following a high-fat meal, but return to baseline values by 8 hours.[2,3] The extended elevation in TG in the present study could be attributed to the lack of an increase in postheparin LPL activity 8 hours following HFC meal ingestion. It is possible that the combination of an increase in LPL activity both with a high-fat meal and a single session of exercise results in the significant reductions in PPL observed in human research.

Significant attenuations in the PPL response following exercise were not observed in the present study. A post hoc power analysis on TG AUCI using our 10 swine indicated that an N of 25 would have been needed to obtain statistical significance. The PPL response to exercise in swine was highly variable, and although there was a statistical trend for exercise to attenuate the PPL response ($P = .07$), it is unlikely that adding additional swine would have resulted in statistical significance. This variability in PPL response in swine is a novel finding and perhaps an important consideration for future studies. These results differ from previous human observations. This variable response could be attributed to swine being extremely sedentary compared with humans, resulting in a greater range in the stress response to exercise. This variable stress response was illustrated by C-reactive protein elevations following exercise (unpublished observations). Regardless, based on previous human studies, it was surprising that exercise did not consistently attenuate PPL.

In conclusion, this is one of the first published reports of PPL and the effects of an exercise session on the PPL response in swine. Under conditions of the present study, while LPL activity was significantly increased with feeding a HFC diet, the PPL response to a HFC meal did not differ depending on diet. Furthermore, a single session of exercise did not significantly alter the PPL response to the HFC meal. These findings suggest that administration of a HFC meal of this magnitude (~3,000 kcal, 1,300 kcal from fat) will promote significant elevations in postprandial TG concentrations, overwhelm the adaptive response to a HFC diet (elevated LPL activity), and attenuate the beneficial effects of a single exercise session on this system.

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