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Body composition and cardiometabolic disease risk factors in captive baboons (*Papio hamadryas* Sp.): sexual dimorphism

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Abstract

Baboons (*Papio hamadryas* Sp.) exhibit significant sexual dimorphism in body size. Sexual dimorphism is also exhibited in a number of circulating factors associated with risk of cardiometabolic disease. We investigated whether sexual dimorphism in body size and composition underlie these differences. We examined data from 28 male and 24 female outdoor group-housed young adult baboons enrolled in a longitudinal observational study of cardiometabolic disease risk factors. Animals were sedated with ketamine HCl (10mg/Kg) before undergoing venous blood draws, basic body measurements, and dual-energy X-ray absorptiometry (DXA) body composition scans. Percentage glycated hemoglobin (%HbA_{1C}) was measured in whole blood. Serum samples were analyzed for glucose, insulin, C-peptide, HDL-, and triglyceride concentrations. Males were heavier and had greater body length and lean tissue mass than females. Females had a greater body fat percentage relative to males (10.8 ±6.4 vs. 6.9 ±4.0, P=0.01). Although C-peptide, fasting glucose, and %HbA_{1C} did not differ between the sexes, females had greater fasting insulin and triglyceride compared to their male counterparts. Insulin and percentage body fat were significantly correlated in males (r=0.61, P=0.001) and to a lesser extent in females (r=0.43, P=0.04). Overall, relations between adiposity and fasting insulin and fasting triglyceride were stronger in males. After accounting for differences in percentage body fat, fasting insulin and triglyceride were no longer statistically different between males and females. Despite stronger correlations between relative adiposity and insulin and triglyceride in males, the higher fasting insulin and triglyceride of female baboons may be underlain by their greater relative body fat masses.

Keywords

sex differences; baboon; body fat; triglyceride; insulin

The baboon (*Papio* Sp.) has been used as a model to study several different human diseases over the years (VandeBerg, 2009). Recently, the baboon model has been evaluated for its utility in the study of common human metabolic disorders such as obesity, the cardiometabolic syndrome, and type 2 diabetes (Comuzzie et al., 2003; Chavez et al., 2008). Baboons exposed to similar dietary environments exhibit significant variation in body weight and fat mass (Comuzzie et al., 2003). Moreover, the relations between adiposity, whole body insulin sensitivity, and insulin signaling defects in muscle and adipose tissue in baboons are consistent with those observed in humans (Chavez et al., 2008). In addition, there have been multiple cases of adult-onset diabetes documented in baboons (Guardado-Mendoza et al., 2009). We have recently demonstrated that, when exposed to a diet high in sugar and saturated fatty acids, adult male baboons accrue fat mass and develop metabolic abnormalities in keeping with emerging cardiometabolic syndrome (Higgins et al., 2010). Combined, these findings demonstrate the potential utility of the baboon for studying and ultimately understanding the etiology of common human metabolic disease.

Adult baboons manifest considerable sexual dimorphism in body size (Glassman et al., 1984; Mahaney et al., 1993). Males have on average 60–70% greater body mass than their female counterparts, arising from a more rapid male growth rate during the pubertal transition (Glassman et al., 1984). Greater body mass in human males relative to females is also well documented, although the magnitude of the difference is less than that observed in baboons. Body composition assessments have shown that male baboons have greater fat free mass and lesser relative body fat mass than females. These findings are also consistent with reports in normal healthy human populations (Power and Schulkin, 2008). Several metabolic disease risk factors such as fasting insulin, cholesterol, and triglyceride are also reported to be higher in female baboons (Chavez et al., 2008; Harewood et al., 1999; Chavez et al., 2009). However, these results are inconsistent with the less adverse lipid profile found in human females (Wang et al., 2011).

The potential contribution of body size and composition to the sex differences in baboons has not been investigated. Toward this end, we further assessed sex differences in these traits and determined whether the association among measures of body composition and metabolic disease risk factors are similar in both sexes. We also hypothesized that sex differences in circulating metabolic traits were related to differences in body size or body composition, or both.

METHODS

Animals

Twenty-eight male and twenty-four female adult baboons (*Papio hamadryas* Sp.) from the Southwest National Primate Research Center colony located at Texas Biomedical Research Institute, San Antonio, Texas were studied. All animals were group housed in 95m², 9m-high covered outdoor cages according to established National Research Council guidelines. A veterinarian performed standard health assessments, including blood chemistry and hematology profiles, on all animals before their inclusion in the study. Animals had no significant history of health problems. Study procedures were approved by the Institutional Animal Care and Use Committee of the Texas Biomedical Research Institute, San Antonio,

TX. This research was undertaken in compliance with National Guidelines and with American Society of Primatologists Principles for the Ethical Treatment of Non-Human Primates.

Design

At the time of measurement, all animals were consuming the commercially available Teklad Primate Diet (Teklad 15% Monkey Diet, Harlan Teklad, Indianapolis, IN) and normal water *ad libitum*—a diet low in simple carbohydrates and fat. A standard allotment of approximately 550g per animal of the Teklad Primate Diet was provided daily. This diet is formulated specifically to meet the nutritional requirements of Old World monkey species and is produced in the form of an extruded biscuit. By mass, this diet was composed of 15% protein, 2.6% fat, 62.7% carbohydrate, 9% fiber, 6.4% ash, and 3.3% minerals. The diet contained 3.38Kcal per gram with protein, fat, and carbohydrate representing 17.7%, 6.9%, and 75.4% of total energy, respectively. The ingredients of the diet were ground corn, wheat middlings, soybean meal, ground wheat, corn gluten, alfalfa meal, dried whey, fish meal, sucrose, dried beet pulp, di-calcium phosphate, calcium carbonate, soybean oil, porcine fat, brewer's yeast, choline chloride, and iodized salt, with added vitamins and minerals. Following a 12-hour overnight fast, animals were sedated with ketamine HCl (10mg/Kg i.m.) before undergoing baseline body composition assessment and blood sample collection. All blood samples were collected from the femoral vein via venipuncture.

Body composition

Body weight was measured on an electronic scale (GSE 665, Texas Scales). Body length was measured using a calibrated measuring tape to the nearest centimeter: crown to rump length was measured first and rump to base length was then measured. Dual energy X-ray absorptiometry (DXA) body composition scans were undertaken using a Lunar Prodigy densitometer (GE Healthcare, Madison, WI). Animals were placed in the supine position on the DXA bed and extremities were positioned within the scanning region. Scans were analyzed using encore2007 software (v.11.40.004) GE Healthcare, Madison, WI). The coefficients of variation for total fat mass and total lean mass for two replicate scans in 3 baboons were found to be 2.2 and 2.3%, respectively

Glucose, hemoglobin A_{1c}, and lipid analyses

Serum concentrations of glucose, triglyceride, and HDL-cholesterol were determined using an ACE[®] chemistry analyzer (Alfa Wasserman Diagnostic Technologies, LLC; West Caldwell, NJ). LDL-cholesterol was estimated using the Friedewald formula (Friedewald et al., 1972). Since blood samples were taken after an overnight fast, the triglyceride concentrations reported were likely to reflect triglyceride carried in very low density lipoprotein (VLDL). Total hemoglobin and percentage hemoglobin A_{1c} (%HbA_{1c}) were determined from whole blood using the ACE hemoglobin A1c reagent kit.

Plasma insulin and C-Peptide

Serum insulin and C-peptide were determined using a DPC Immulite 1000 Analyzer® (Diagnostics Products Corporation; Los Angeles, CA). The Immulite is an automated chemiluminescent peptide and steroid hormone analyzer.

Statistical analyses

Group means were compared using independent samples *t*-tests. Pearson correlation coefficients were used to determine relations between body composition and circulating metabolic variables in each sex. When necessary, variables were log transformed to correct for non-normal distributions in the comparisons of means and correlation analyses. General linear models with maximum likelihood estimation of model parameters were used to assess the contribution of body size and composition to sex differences in the circulating metabolic variables; sex was included as a fixed effect in all models. Statistical significance was set at $P < 0.05$. Statistical analysis was undertaken using SYSTAT v.12 (SYSTAT Inc., Chicago, IL).

RESULTS

Males had significantly greater lean tissue mass and lesser percentage body fat mass than females (**Table 1a**). Females had greater total insulin and triglyceride concentrations (**Table 1b**). Of note, despite large differences in insulin concentrations, glucose, %HbA_{1c}, and C-peptide were not significantly different between the groups.

In the males, fasting triglyceride and insulin concentrations were significantly and positively correlated with measures of adiposity (**Table 2a**). Although the correlations were of moderately similar magnitude and direction, the correlation between percentage fat and insulin only reached statistical significance in the females (**Table 2b**). Body length was correlated with insulin in females and showed a strong trend for correlation in the male group (**Table 2**). C-peptide was more strongly correlated with insulin in females ($r=0.81$, $P<0.001$) than in males ($r=0.58$, $P=0.002$). Fasting insulin and fasting triglyceride were not significantly correlated within each group, but when data from males and females were combined a statistically significant correlation was present ($r=0.40$, $p=0.003$).

Sexual dimorphism in insulin and triglyceride concentrations were investigated further using general linear models. For fasting insulin, percentage body fat and body length were entered into the model as covariates and sex was entered as a fixed effect. Only percentage body fat was found to be associated with insulin in this model (Table 3a). In a second model, we found no evidence for an interaction between sex and percentage body fat (Table 3b). The same set of covariates was modeled with fasting triglyceride as the dependent variable. Similarly, after adjusting for percentage body fat, sex was no longer associated with fasting triglyceride (Table 4a) and there was no interaction between sex and percentage body fat in the model (Table 4b).

DISCUSSION

Recent studies have established the baboon as an important model for the study of obesity, the cardiometabolic syndrome, and type 2 diabetes (Comuzzie et al., 2003; Chavez et al., 2008; Guardado-Mendoza et al., 2009; Higgins et al., 2010; Chavez et al., 2009; Tejero et al., 2004; Bose et al., 2009; Kemnitz et al., 2002; Banks et al., 2003). Herein we have corroborated earlier data describing the sexual dimorphism in body composition and important circulating metabolic risk factors in a sample of young adult baboons matched for age, consuming the same diet, and housed in the same environment. As previously described, female baboons weigh less than their male counterparts; this is attributable to lesser lean mass. Hence, despite similar total fat masses, females have significantly greater relative or percentage fat mass than males. Baboons also manifest sex differences in several important metabolic disease risk factors, i.e., fasting insulin and triglyceride concentrations. Our objectives were to do the following: 1) determine whether the relations between adiposity and cardiometabolic disease risk factors were sex dependent; and 2) determine whether sex differences in insulin and triglyceride concentrations were associated with differences in body size and/or composition. We found that adiposity was strongly correlated with fasting insulin and triglyceride concentrations in males. In the female group, only insulin was significantly correlated with percentage body fat. After accounting for percentage body fat in our general linear models, we found that sex was no longer associated with fasting insulin and triglyceride concentrations.

Baboons manifest considerable variation in body mass and adiposity (Comuzzie et al., 2003). Overt late-onset diabetes, consistent with human type 2 diabetes, has been documented to occur in baboons (Chavez et al., 2008). Increases in adiposity and alterations in related biomarkers can also be induced in baboons by exposing them to a Westernized diet (Higgins et al., 2010). Furthermore, recent work demonstrated that aged baboons are insulin resistant and share many of the molecular and cellular hallmarks of human skeletal muscle and adipose tissue insulin resistance (Chavez et al., 2008). In addition, pancreatic islet lesions in late onset diabetic baboons bear remarkable similarity to those observed in humans and in other animal models (Guardado-Mendoza et al., 2009). Data from these studies combined with the advantages of working with a large, long-lived, and tractable non-human primate, highlight the utility of the baboon as a model for the study of obesity-associated metabolic diseases.

In agreement with earlier studies (Chavez et al., 2008, Comuzzie et al., 2003, Glassman et al., 1984), we report significant sex differences in baboon body composition. As expected, the greater body mass of males compared to females is attributable to greater lean body mass. Total fat mass was found to be similar in the two groups and females had greater relative or percentage fat mass compared to males. The magnitude of the sex difference in relative adiposity reported here is in accord with observations in aged baboons (Chavez et al., 2008) and, as in humans, is likely to be caused by genetic, endocrine, and metabolic differences (Power and Schulkin, 2008). The levels of adiposity in these baboons would be considered very low in most human populations (Pineau et al., 2009); however, these body fat percentages are normal for baboons consuming a low-fat diet in an open housing environment (Comuzzie et al., 2003). While this environment differs from that of modern

sedentary humans, it permits the assessment of sex differences without the potential confounding effects of an obesogenic environment. In addition, in line with studies of modern hunter-gatherer populations, this environment is likely to better approximate that of early humans (Barnicot et al., 1972; Truswell and Hansen, 1968). As a result the model may also provide insight into human sex differences in body composition and metabolism.

To our knowledge, this is the first report of sex differences in the strength of the relations between adiposity and metabolic risk factors in baboons. In humans, women have less metabolic dysregulation for a given level of adiposity than men (Power and Schulkin, 2008; Magkos et al., 2010; Karastergiou et al., 2012). Adiposity was strongly correlated with fasting insulin and triglyceride among male baboons. In contrast, only fasting insulin was significantly correlated with percentage of body fat in female baboons. Therefore, despite sex differences in risk factors in humans being opposite in direction to those of baboons (Wang et al., 2011, Sugiyama and Agellon, 2012), the weaker associations between adiposity and fasting insulin and triglyceride in female versus male baboons are consistent with human data.

The less metabolically deleterious effect of adiposity in women is not fully understood but may be caused in part by differences in the distribution of body fat. In humans, differences in the distribution of body fat into upper body/central/visceral depots versus lower body/peripheral/subcutaneous depots are considered to explain some portion of the apparent attenuated dysmetabolic effects of adiposity in women (Power and Shulkin, 2008, Lemieux et al., 2002, Kautzky-Willer and Handisurya, 2009). Central visceral body fat deposition is considered a risk factor for cardiometabolic disease and its association with insulin resistance is long established (Roust and Jensen, 1993).

Interestingly, our DXA data demonstrate that the female baboons had greater trunk body fat percentages than males, while males had greater leg fat mass, and both groups had similar leg percentage fat values. This suggests that female baboons do not preferentially distribute body fat in the lower body, as is also the case in a large subset of human women (Meigs et al., 2003, Jensen, 2008, Karastergiou et al., 2012, Nellemann B et al., 2012), and consequently manifest a centralized body fat distribution. However, in the female baboons, the strength of the associations between trunk fat and the risk factors were not much different than those between total body fat and the risk factors. As our DXA scanner and software did not distinguish between upper body visceral and upper body subcutaneous adipose tissue deposition, we could not determine whether the female baboons had greater central subcutaneous fat deposition or greater central visceral fat deposition. Higher resolution imaging techniques should be undertaken to more accurately determine body fat distribution and its relationship to cardiometabolic risk factors in baboons.

Sex differences in fasting insulin are not typically observed in human populations (Magkos et al., 2010). We observed two-fold greater fasting insulin concentrations in female baboons than in male baboons, but we found no differences in fasting C-peptide concentrations. Discord between insulin and C-peptide concentrations is typically taken to reflect hepatic insulin extraction and clearance: insulin and C-peptide are released from the β -cell in equivalent molar quantities but, unlike C-peptide, a large proportion of insulin is taken-up

and metabolized as it passes through the hepatic vasculature (Castillo et al., 1994). If basal insulin release were higher in female baboons then fasting C-peptide concentrations would also be higher. Hence, one possible explanation for our observations is that female baboons have lower hepatic insulin clearance relative to their male counterparts. In support of this, we found a stronger correlation between fasting insulin and fasting C-peptide in females than in males: greater concordance between the two peptides would be likely to result from less insulin being cleared by the liver. Higher fasting insulin in female baboons compared to males with no difference in fasting C-peptide concentrations was also reported in an earlier study (Chavez et al., 2008). It will be important to determine whether sex differences in insulin are also present following a nutrient challenge in baboons. A more direct assessment of insulin clearance is required to confirm the presence of lesser hepatic insulin clearance in female baboons.

Results from our general linear model analyses suggest that the greater relative adiposity of females is the principal factor underlying their greater fasting insulin and fasting triglyceride. Inclusion of percentage body fat resulted in the exclusion of sex and body length from both the insulin and the triglyceride models. Lack of interaction between sex and percentage body fat suggests that the percentage body fat effect is not mediated by sex. In humans, there is strong support for the theory that increases in body fat, particularly visceral fat, lead to insulin resistance. Insulin resistance can increase the availability free fatty acids (from increased adipose tissue lipolysis) for hepatic VLDL triglyceride production and can attenuate the suppression of *de novo* lipogenesis, resulting in elevated VLDL triglyceride concentrations in the presence of fasting hyperinsulinemia (Nielsen and Karpe, 2012). This is in keeping with the clustering of relatively higher percentage body fat, fasting insulin, and fasting triglyceride in female baboons that we describe here.

In the study by Chavez et al., aged female and male baboons did not differ in insulin clamp-derived insulin sensitivity, fasting glucose, or %HbA_{1c}. However, females did have greater percentage body fat and greater concentrations of fasting insulin, fasting triglyceride, and free fatty acids (Chavez et al., 2008). As indicated by Chavez et al., these findings suggest that female baboons have greater degree of hepatic insulin resistance but are not more insulin resistant in skeletal muscle. Indeed, the greater discordance between insulin and C-peptide in male versus female baboons lends further support to this contention: lower hepatic insulin clearance is a potential manifestation of hepatic insulin resistance (Castillo et al., 1994). Taken together, the data suggest that the greater percentage body fat of female baboons may result in lower insulin sensitivity—primarily in the liver and perhaps in adipose tissue—that causes greater hepatic VLDL-triglyceride production and a lesser hepatic insulin clearance. This would manifest as higher fasting concentrations of triglyceride and insulin.

Increased hepatic VLDL triglyceride production has been shown to occur in obese versus lean men despite similar rates of hepatic glucose production in the two groups (Sorenson et al., 2011). If a similar phenomenon were to occur in baboons, it could explain why female and male baboons have similar fasting glucose despite significant differences in fasting triglyceride. To gain a clearer understanding, further studies using tracer methodology are

needed to specifically test whether insulin sensitivity differs by sex in the liver, adipose tissue, and skeletal muscle of young baboons.

Body length was negatively correlated with fasting insulin in the females and the negative association approached significance in the male group ($p=0.07$). Nevertheless, length was not independently associated with fasting insulin and fasting triglyceride in our general linear models, and we conclude that baboon sex differences in fasting insulin and fasting triglyceride are unlikely to be attributable to differences in body size. Our conclusions regarding sex differences in insulin and triglyceride will be further scrutinized in longitudinal analyses of this cohort.

Additional limitations of our study warrant mention. Unfortunately, the outdoor housing environment did not enable individual assessments of energy intake or physical activity to be undertaken and we cannot rule out the possibility that differences in these variables could have contributed, in part, to the sex differences in risk factors. In addition, we did not have measures of circulating sex hormones. It is unlikely that sex hormone variation would have explained the sex differences observed (Wang et al., 2011), but variation in sex hormone concentrations within the female group could have influenced the strength of the correlations between body composition and the risk factors, and warrants further investigation.

We describe significant sexual dimorphism in baboon body composition and cardiometabolic disease risk factors. These findings provide important considerations for future studies that will use the baboon to model metabolic diseases. In addition, the study of sexual dimorphism in baboon metabolic phenotypes has the potential to contribute to our understanding of human sex differences in glucose and lipid metabolism. In conclusion, despite stronger correlations between relative adiposity and fasting insulin and fasting triglyceride in male compared to female baboons, the greater fasting insulin and triglyceride concentrations of female baboons may be underlain by their greater relative body fat masses.

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Table 1a

Age and body composition of male and female baboons

	Male (n=28)	Female (n=24)	P
Age (yrs.)	8.3 $\pm$ 0.4	8.5 $\pm$ 0.4	0.020
Weight (Kg)	28.8 $\pm$ 3.6	17.6 $\pm$ 2.8	<0.001
Length (cm)	106.4 $\pm$ 5.6	92.0 $\pm$ 4.1	<0.001
Fat mass (g)	2020 $\pm$ 1274	2207 $\pm$ 1762	0.569
% Fat Mass	6.9 $\pm$ 4.0	10.8 $\pm$ 6.4	0.010
Lean mass (g)	25500 $\pm$ 3151	13845 $\pm$ 3088	<0.001
Trunk fat mass (g)	1211 $\pm$ 941	1579 $\pm$ 1376	0.260
% Trunk fat mass	8.3 $\pm$ 5.6	15.3 $\pm$ 10.7	0.004
Leg fat mass (g)	482 $\pm$ 222	350 $\pm$ 230	0.040
% Leg fat mass	8.1 $\pm$ 4.3	9.3 $\pm$ 6.0	0.375
BMC (g)	1256 $\pm$ 26	769 $\pm$ 15	<0.001

Data are mean $\pm$ SD.

Table 1b

Circulating metabolic variables in male and female baboons

	Male(N=28)	Female (n=24)	P
Total Cholesterol (mg/dL)	95.1 ±14.6	108.6 ±25.3	0.020
LDL-cholesterol (mg/dL)	28.3 ±6.2	39.0 ±13.1	<0.001
HDL-cholesterol (mg/dL)	58.6 ±10.9	58.3 ±16.0	0.842
Triglyceride (mg/dL)	41.0 ±11.6	57.5 ±19.3	0.001
Glucose (mg/dl)	77.6 ±8.2	79.1 ±9.4	0.490
%HbA _{1c}	4.4 ±2.3	4.1 ±1.8	0.421
Insulin (μIU/mL)	13.8 ±8.7	27.1 ±16.7	0.001
C-peptide (ng/mL)	2.42 ±1.38	2.68 ±1.41	0.226

Data are mean ±SD.

Table 2a

Pearson correlation coefficients between body composition and risk factors (males)

	Weight	Length	Fat mass	Lean mass	% Fat mass	Trunk fat mass	% Trunk fat mass
Glucose	-0.01 (0.98)	0.14 (0.49)	0.10 (0.60)	-0.05 (0.89)	0.14 (0.48)	0.07 (0.67)	0.06 (0.75)
HDL	0.04 (0.85)	0.25 (0.23)	0.02 (0.94)	0.06 (0.75)	-0.01 (0.93)	-0.04 (0.85)	-0.15 (0.42)
LDL	0.01 (0.95)	0.17 (0.42)	-0.05 (0.79)	0.03 (0.87)	-0.09 (0.67)	-0.05 (0.81)	-0.12 (0.53)
%HbA _{1c}	0.30 (0.12)	0.13 (0.53)	0.07 (0.71)	0.32 (0.10)	0.00 (0.99)	0.14 (0.49)	0.33 (0.13)
Insulin	0.14 (0.49)	-0.37 (0.07)	0.62 (<0.001)	-0.09 (0.64)	0.61 (0.001)	0.58 (0.001)	0.55 (0.003)
C-peptide	-0.11 (0.60)	-0.40 (0.06)	0.30 (0.14)	0.10 (0.60)	0.34 (0.08)	0.22 (0.29)	0.29 (0.137)
Triglyceride	-0.04 (0.83)	-0.38 (0.06)	0.44 (0.02)	-0.21 (0.30)	0.48 (0.01)	0.39 (0.04)	0.346 (0.07)

P values are in parentheses. LDL: LDL-cholesterol; HDL: HDL-cholesterol.

Table 2b
Pearson correlation coefficients between body composition and risk factors (females).

	Weight	Length	Fat mass	Lean mass	% Fat mass	Trunk fat mass	% Trunk fat mass
Glucose	-0.10 (0.65)	-0.01 (0.98)	0.02 (0.91)	0.25 (0.23)	-0.11 (0.60)	0.02 (0.93)	0.05 (0.81)
HDL	-0.03 (0.90)	0.20 (0.42)	-0.24 (0.25)	0.02 (0.92)	-0.27 (0.21)	-0.25 (0.23)	-0.25 (0.24)
LDL	-0.37 (0.08)	-0.02 (0.92)	-0.42 (0.04)	0.16 (0.46)	-0.51 (0.01)	-0.44 (0.04)	-0.40 (0.06)
%HbA _{1c}	0.35 (0.10)	0.49 (0.03)	0.18 (0.41)	0.13 (0.56)	0.03 (0.90)	0.17 (0.42)	0.06 (0.79)
Insulin	0.09 (0.71)	-0.51 (0.03)	0.37 (0.09)	-0.29 (0.20)	0.43 (0.04)	0.37 (0.09)	0.39 (0.07)
C-peptide	-0.01 (0.97)	-0.45 (0.07)	0.17 (0.44)	-0.13 (0.58)	0.21 (0.35)	0.17 (0.46)	0.34 (0.15)
Triglyceride	0.32 (0.12)	-0.07 (0.77)	0.30 (0.16)	0.01 (0.99)	0.31 (0.15)	0.29 (0.18)	0.23 (0.29)

P values are in parentheses. LDL: LDL-cholesterol; HDL: HDL-cholesterol.

Table 3a

General linear model for the dependent variable **fasting insulin**, $R^2=0.39$, $P<0.01$.

	Type III SS	F	P
Model	1.780	9.79	<0.001
Intercept	0.247	4.01	0.050
Sex	0.005	0.09	0.765
Length	0.207	3.41	0.072
%FAT	0.495	8.17	0.007

Table 3b

General linear model for the dependent variable **fasting insulin**, including sex $\times$ %FAT interaction term, $R^2=0.30$, $P<0.01$.

	Type III SS	F	P
Model	1.701	7.99	<0.001
Intercept	4.978	70.2	<0.001
Sex	0.001	0.01	0.925
%FAT	0.485	8.17	0.012
Sex $\times$ %FAT	0.078	1.10	0.299

Table 4a

General linear model for the dependent variable **fasting triglyceride**, $R^2=0.27$, $P<0.05$

	Type III SS	F	P
Model	0.314	6.36	0.001
Intercept	0.036	2.18	0.148
Sex	0.004	0.27	0.605
Length	0.009	0.57	0.456
%FAT	0.100	6.08	0.018

Table 4b

General linear model for the dependent variable **fasting triglyceride** including the sex $\times$ %FAT interaction term, $R^2=0.25$, $P<0.05$.

	Type III SS	F	P
Model	0.357	7.59	<0.001
Intercept	0.035	2.08	<0.001
Sex	0.00003	0.00	0.990
%FAT	0.090	5.39	0.026
Sex $\times$ %FAT	0.0006	0.03	0.854