Discussed Posters

SESSION 1:
Body fat distribution and ectopic fat

75

ABDOMINAL OBESITY IN ADOLESCENTS: PREVALENCE AND RISK FACTORS

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Objective: To determine abdominal obesity (AO) prevalence and its risk factors in adolescents in Venezuela. **Method:** The data was obtained from 3140 adolescents, 1371 males and 1769 females, aged 12-19 years, who were randomly selected from high schools, in Maracaibo, Venezuela. **The following information was requested:** The completion of a demographic questionnaire, and the waist circumference (WC) which was measured at the nearest 0.1 cm at the high point of the iliac crest in standing position. Percentiles for WC were calculated by gender and age, and adolescents with WC above 90th percentile were considered as AO. Odds ratios (OR) and 95% CI were calculated using logistic regression to determine AO risks factors. **Results:** The AO prevalence was 23.7 % in all, 23.6 % in males and 23.7 % in females (p: NS). The logistic regression detected that hypertension (OR=5.720; 95% CI=4.130-7.923), smoking (OR=2.860; 95% CI=1.094-7.476), prehypertension (OR=2.365; 95% CI=1.949-2.868), liquor ingestion (OR=1.891; 95% CI=1.335-2.679) and non physical activity (OR=.791; 95% CI=1.418-2.262) were the AO risk factors statistically significant. Likewise, the analysis applied by gender showed that smoking (OR=6.773; 95% CI=1.963-23.364) in males, and alcohol ingestion (OR= 3.097; 95% CI= 1.704-5.629) in females were the main AO risk factors. **Conclusions:** The AO prevalence is high in adolescents in both genders. Hypertension and smoking and alcoholic habits are the most important AO risk factors in adolescents. Thus, it is necessary to identify these risk conditions in adolescents, so that more intensive interventions can be applied in order to reduce the AO.

129

SINGLE SLICE IMAGING FOR ESTIMATING VISCERAL AND SUBCUTANEOUS ADIPOSE TISSUE VOLUME CHANGES FOLLOWING WEIGHT LOSS

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Background: The accuracy of a single slice in measuring visceral and subcutaneous adipose tissue (VAT&SAT) changes during weight loss and how anatomical location of the slice influences accuracy is unknown. **Methods:** VAT and SAT were derived from slice areas taken at 5-cm intervals from magnetic resonance images in 123 overweight and obese subjects [X±SD age: 49.5±12.5 y; BMI: 35.1±3.9 kg/m^2] who participated in a CB1R inverse agonist (Taranabant) mediated weight loss trial. **Results:** VAT areas at 5-10 cm above L4-L5 correlate strongest with VAT volume at baseline and follow up (5-10 cm above (r=0.92-0.95), L4-L5 (r=0.86-0.88)), and for changes (5-10 cm above L4-L5 (r=0.76), L4-L5 (r=0.47)). SAT areas at slice 10 cm below L4-L5 correlate strongest with SAT volume at baseline and follow up (10 cm below (r=0.84-0.90), L4-L5 (r=0.80)), and for changes (10 cm below (r=0.65), L4-L5 (r=0.54)). Studies using VAT or SAT volumes will require 78% and 71% fewer subjects, respectively, than those using slices at L4-L5 and will have equivalent power. Studies using a single slice at the best location will require 62% and 31% fewer subjects, respectively, than those using slices at L4-L5 and will have equivalent power. **Conclusion:** Total volumes of VAT and SAT provide much greater power than single slices in measuring VAT and SAT loss after weight reduction. Single slice areas 5-10 cm above L4-L5 for VAT and 10 cm below L4-L5 for SAT provide greater power for the detection of VAT and SAT volume reduction than do measurements at L4-L5.

478

INCREASING BODY FAT IS ASSOCIATED WITH CARDIAC STEATOSIS IN WOMEN

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Aim: Lipotoxicity has been implicated as a potential common pathway in cardiac myocyte dysfunction and ultimately apoptosis. Fat deposition in the peritoneum, the liver and around vascular structures has been linked to metabolic syndrome and subsequent cardiomyopathy. Therefore this study sought to determine the relationship between excess body fat and intracardiac lipids as assessed by 3T magnetic resonance proton spectroscopy in women. **Methods:** 27 healthy female volunteers with no comorbidities were recruited from the general public according to body mass index - 14 lean (BMI 21.3 ± 2.0), 5 overweight (BMI 27.7 ± 1.7) and 8 obese (BMI 40.5 ± 8.1). All participants were scanned on a 3 Tesla Siemens magnet. Cardiac spectroscopy was performed using a STEAM sequence with water suppression on a septal mid-ventricular voxel (figure 3). A transverse image at the level of the L4 vertebra was used to measure visceral and subcutaneous adipose tissue with manual contouring. All patients fasted for 10 hours prior to their study, and to ensure adequate hydration status water intake was encouraged. **Results:** The normal range for intracardiac lipids in lean women was 0.36% +/- 0.14 of the total water signal, similar to previously reported ranges. Cardiac lipid content was higher in overweight (0.55% +/- 0.31) and in obese women (0.86% +/- 0.57; p 0.01) compared to lean. Intracardiac lipid content correlated strongly with waist circumference, BMI and sagittal abdominal diameter (Fig 1). There was also a significant correlation between ICL and visceral adiposity (r = 0.76, p < 0.001) and total body fat (r = 0.74, p < 0.001). In this healthy population, there was no significant relationship between left ventricular ejection fraction and intracardiac lipids, but there was a significant correlation of lipid content with cardiac mass (r = 0.50, p = 0.01).

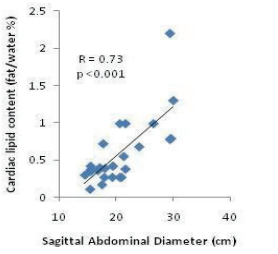

Figure 1. Intracardiac lipids rise in association with increased sagittal abdominal diameter

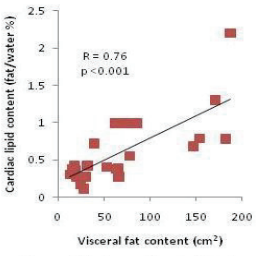

Figure 2. Intracardiac lipids rise in association with visceral abdominal fat, measured at a transverse image at L4

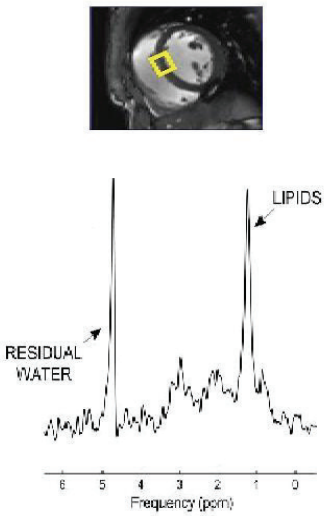

Figure 3. 3 Tesla proton spectroscopy was used to measure intracardiac lipids as a percentage of total water signal in a voxel sited in the interventricular septum (yellow box)

Conclusions: Cardiac lipid content is increased in obese women even in the absence of diabetes and hyperlipidaemia. This may reflect increased lipid deposition within myocytes, and/or altered lipid usage & metabolism.

215

VISCERAL ADIPOSITY/LIVER FAT IN PATIENTS WITH CVD AND/OR TYPE 2 DIABETES AND WITH/WITHOUT STATIN THERAPY: INSPIRE ME IAA RESULTS

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A high waist circumference has been linked to cardiovascular disease (CVD) and type 2 diabetes (T2D). Waist circumference provides only an estimate of the volume of visceral adipose tissue (VAT), which has strong mechanistic links to cardiometabolic risk, CVD and T2D. **Aims:** We sought to assess visceral adiposity and liver fat by computed tomography (CT) and related cardiometabolic risk profile in patients with CVD and T2D classified according to their statin use. **Methods:** A sample of 297 general physicians, diabetologists and cardiologists in 29 countries randomly recruited 4504 subjects to participate in a cross-sectional CT imaging/cardiometabolic study. Data on lifestyle, medical history, medication use, anthropometry, glucose tolerance and fasting lipids were collected. CT imaging was used to measure abdominal VAT and subcutaneous adipose tissue areas (SAT) as well as liver attenuation (as an estimate of liver fat content). Subjects were excluded from the analysis if they were < 39 and >71 years of age for men and < 44 and >71 years for women, had type 1 diabetes, and were < 30 kg or >300kg. We divided our cohort into eight groups according to the presence of T2D, CVD and statin use: healthy (H), healthy +statin use (H+S), T2D only, T2D only +statin use (T2D+S), CVD only, CVD only +statin use (CVD+S), T2D and CVD (T2D+CVD) and T2D and CVD +statin use (T2D+CVD+S). Statistical significance was calculated using a general linear model including age, region, physician's specialty and sex. **Results:** There was a pattern of higher cardiometabolic risk in subjects with T2D and CVD. In addition, statin use was not associated with a better cardiometabolic profile despite lower LDL-cholesterol (Table). **Conclusion:** Patients with T2D and CVD had higher levels of visceral adipose tissue and liver fat and were at increased cardiometabolic risk regardless of statin use.

Table: Subject Characteristics

Unadjusted Mean ± SD	H N=1341 M=43% W=57%	H+S N=413 M=44% W=56%	T2D only N=769 M=52% W=48%	T2D only +S N=539 M=50% W=50%	CVD only N=136 M=60% W=40%	CVD only +S N=341 M=76% W=24%	T2D+CVD N=111 M=58% W=42%	T2D+CVD+S N=373 M=71% W=29%	Group Effect
Age[1] yrs	54.2 ± 7.3	57.7 ± 7.0*	56.5 ± 7.5[A]	58.2 ± 7.2*	58.3 ± 7.3[A]	59.7 ± 6.9*[B]	59.8 ± 6.8[A]	59.7 ± 6.4[B]	<0.0001
BMI kg/m²	27.0 ± 5.0	28.1 ± 4.6	28.1 ± 5.5[A]	29.8 ± 5.2*[B]	27.3 ± 4.6	28.6 ± 4.6	29.9 ± 6.0[A]	30.7 ± 5.2[B]	<0.0001
Waist cir. cm	91.3 ± 13.4	95.2 ± 13.8*	96.2 ± 14.4[A]	100.5 ± 13.6*[B]	96.1 ± 13.3[A]	99.9 ± 12.5	101.2 ± 15.6[A]	105.5 ± 13.3[B]	<0.0001
Liver attenuation HU	56.6 ± 10.5	55.7 ± 9.9	51.0 ± 12.6[A]	48.9 ± 12.6*[B]	56.3 ± 9.7	55.0 ± 10.3	50.8 ± 14.7[A]	49.0 ± 12.8[B]	<0.0001
SAT cm²	255.6 ± 118.7	283.9 ± 106.8*[B]	247.6 ± 119.7	285.3 ± 115.1*[B]	249.0 ± 101.2	266.4 ± 101.3	259.2 ± 118.4	291.6 ± 107.6*[B]	<0.0001
VAT cm²	134.6 ± 60.0	152.4 ± 65.7	168.3 ± 81.2[A]	190.6 ± 82.7*[B]	165.3 ± 78.9[A]	184.5 ± 76.1[B]	200.8 ± 88.5[A]	223.4 ± 86.5[B]	<0.0001
LDL-chol mmol/l	3.25 ± 0.83	2.77 ± 0.81*	3.04 ± 0.84[A]	2.49 ± 0.86*[B]	3.11 ± 0.99	2.33 ± 0.79*[B]	2.90 ± 0.91[A]	2.25 ± 0.74*[B]	<0.0001
HDL-chol mmol/l	1.38 ± 0.41	1.36 ± 0.42	1.26 ± 0.40[A]	1.24 ± 0.34[B]	1.29 ± 0.34	1.18 ± 0.33[B]	1.17 ± 0.34[A]	1.10 ± 0.29[B]	<0.0001

N, total, M, Men; W, Women
[1] Adjusted for region, physician's specialty and sex only
*p<0.05 for H vs H+S, T2D vs T2D+S, CVD vs CVD+S or T2D-CVD vs T2D-CVD+S (no statin use versus statin use)
[A] p<0.05 versus H as reference group
[B] p<0.05 versus H+S as reference group

[Table 1]

144

WAIST CIRCUMFERENCE - A MORE SENSITIVE MARKER THAN BMI IN PREDICTING CARDIOVASCULAR DISEASE

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Purpose: To show by Pulse Wave Velocity (PWV) determination that Waist Circumference (WC) is a more sensitive marker than BMI for predicting cardiovascular disease (CVD). **Method:** PWV was determined with the BPULS apparatus using the left external carotid and left dorsalis pedis arteries as "central" and "peripheral" points respectively. Pulses were picked up by infrared sensors and recorded simultaneously with a single lead ECG. The time difference between the two pulses is measured. A shorter time delay or faster PWV indicates decreased arterial wall elasticity. **Materials:** A total of 957 clinically asymptomatic Filipinos living in rural areas were studied. Males: 447: Females: 510. Age range: 17 - 84 years. Borderline hypertensives: 163; Established hypertensives: 164. Subjects were classified into subgroups acccrding to their BMI and WC. Average PWV time (adjusted for height of the subjects) for the total and each subgroup was noted.The relationship of increased BMI and WC to variations in PWV time was determined. **Results:** Elevated BMI does not significantly influence PWV time in the following; 1) All subjects (p< 0.1397); 2) Females (p< 0.2372), 3) Normotensives (p< 0.0866), and 4) Established hypertensives (p< 0.1548). On the contrary, for every one cm. increase in WC, PWV time correspondingly declines significantly by: 1) 0.000743 sec. (p< 0001) in All subjects; 2) 0.000063 sec. (p< 0.0001) in Females; 3) 0.000759 sec. (p< 0.0001) in Normotensives; and, 4) 0.00035 sec. (p< 0.0001) in Established hypertensives. **Discussion:** Abnormal PWV is an accepted high risk factor for the development of CVD. In the four groups studied above, elevated BMI does not significantly influence PWV. However, in the very same groups, increasing WC significantly affects PWV. This implies that if we rely solely on BMI as an indicator to predict CVD we will miss many cases who are at high risk as shown by increased WC and abnormal PWV. **Conclusion:** This study shows that Waist Circumference is a more sensitive marker than BMI for predicting cardiovascular disease. *Dr. H. Marcoyannopoulou Fojas was a recipient of a grant from the Balik Scientist Program of the Philippine Department of Science and Technology.

187

METABOLIC IMPLICATIONS OF INCREASED WAIST CIRCUMFERENCE IN THE GLUCOSE TOLERANCE GROUPS IDENTIFIED BY OGTT

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Objectives: OGTT correctly detects subjects at risk for type 2 diabetes (DM). Obesity, particularly abdominal adiposity, is increasingly recognized as a cause of elevated cardiometabolic risk. As waist circumference (WC) can be used as a crude estimate of visceral fat accumulation, we verified whether its measurement provides further information above OGTT. **Methods:** We recruited 1527 subjects (695 men) and executed a standard OGTT, measuring fasting and 2h-plasma glucose (ADA 2003), to identify normal glucose tolerance (NGT), pre-diabetes (preDM) and diabetes (DM), as well as insulin to evaluate β-cell function. We measured the WC and calculated the Stumvoll's estimated insulin sensitivity index (EISI) and estimated first phase of insulin secretion (EFPN), and the Gutt insulin sensitivity index (ISI-GUTT). **Results:** NGT was found in 829 (54.28%) subjects (364 men), while 558 subjects (36.54%) (263 men) had preDM and 140 subjects (9.16%) (68 men) presented unknown DM. The NGT, preDM and DM subjects with higher WC were 299 (36%), 305 (54.6%) and 93 (66.4%) respectively. When comparing high WC subjects to normal WC in NGT, pre-DM and unknown DM, a high WC was associated to a significant reduced EISI (p< 0.0001) and ISI-GUTT (p< 0.0001), and an increased EFPN (p< 0.0001). **Conclusions:** A WC indicative of abdominal adiposity, identifies an increased cardiometabolic risk, as well as subjects with a worst metabolic profile in all glucose tolerance groups. Abdominal obesity in NGT and pre-DM subjects can be targeted for pharmacologic intervention in addition to lifestyle changes.

40

52-WEEK TREATMENT WITH DIET AND EXERCISE + TESTOSTERONE IMPROVES NON-ALCOHOLIC FATTY LIVER DISEASE AND CARDIOVASCULAR RISKS IN HYPOGONADAL MEN

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Objectives: Men with the metabolic syndrome (MetS) and type 2 diabetes (T2D) often have low testosterone levels. Elevating low testosterone levels may improve features of the MetS and glycemic control. In this analysis we assessed effects of normalization of circulating testosterone on biomarkers of non-alcoholic fatty liver disease (NAFLD) , and cardiovascular risk. **Methods:** In a single-blind, 52-week clinical trial, 32 hypogonadal men with the MetS and newly diagnosed T2D were randomized to supervised diet and exercise (D&E) alone (n=16) or with additional transdermal testosterone gel (50mg QD;(D&E=T) n=16). The MetS was defined by the Adult Treatment Panel-III and the International Diabetes Federation. Hypogonadism was defined as a total testosterone ≤12.0 mmol/L. Endpoint were baseline adjusted change in biomarkers of NAFLD (GPT, GOT, g-GT, CRP) and cardiovascular risk (homocystein, PAI-1, fibrinogen, Apo(a) and TG). **Results:** 52-weeks of treatment T administration resulted in a significantly larger improvement in all measured biomarkers of NAFLD in T treated patients as compared to supervised D&E alone. Levels of homocystein, PAI-1, fibrinogen, Apo(a) and TG improved significantly in both treated groups, with PAI-1, fibrinogen and TG showing a significantly larger improvement in T treated patients as compared to supervised D&E alone. **Conclusions:** Addition of testosterone to supervised D&E results in greater beneficial effects on biomarkers of NAFLD and cardiovascular risk. Our results invite to consider the significance of diagnosing and, if warranted, treating testosterone deficiency in men with diabetes type 2.

152

REMISSION OF THE METABOLIC SYNDROME THREE YEARS AFTER SCREENING FOR INCREASED WAIST CIRCUMFERENCE

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Objectives: In 2006 we sent a tape measure to 11.862 people not known with hypertension, diabetes or dyslipidemia to detect individuals with the metabolic syndrome (MetS) by letting them measure their waist circumference at home as a first step. Among those with an increased waist circumference (>88/102cm for women/men), 473 new MetS cases (NCEP ATP III-criteria) were detected. They got no more than the advice to contact their general practice. After three years we invited these people for a follow-up measurement, to assess changes in cardiovascular risk factors three years after screening followed by usual care in general practice. **Methods:** From the original group only those still visiting the same general practice (n=432, 91%) were invited for follow-up measurements. We also invited a random selection of 280 individuals who had an increased waist circumference during screening, but did not meet the MetS criteria. **Results:** 197 individuals with and 179 individuals without MetS at screening underwent all follow-up measurements. A significant improvement in all MetS components, except glucose, was seen in the group with screen-detected MetS (table).

	Group with screen detected metabolic syndrome in 2006 (N = 197)		
	2006	2009	P-value
BMI (kg/m2)	30.2 ± 3.6	29.3 ± 4.1	<0.001
Weight (kg)	93.2 ± 15.2	90.8 ± 16.1	<0.001
Waist circumference (cm) Men / Women	109.9 ± 7.4 / 99.6 ± 8.8	106.1 ± 10.0 / 96.1 ± 10.7	<0.001 / <0.001
Blood pressure (mmHg) Systolic / Diastolic	143.7 ± 15.0 / 88.0 ± 7.5	135.5 ± 13.5 / 82.4 ± 7.7	<0.001 / <0.001
Triglycerides (mmol/L)	2.2 ± 1.1	1.9 ± 0.9	0.001
HDL cholesterol (mmol/L) Men / Women	1.1 ± 0.3 / 1.3 ± 0.3	1.2 ± 0.3 / 1.4 ± 0.3	<0.001 / <0.001
Glucose (mmol/L)	5.3 ± 1.2	5.4 ± 0.8	0.02

[Cardiometabolic risk factor levels]

The remission rate was 53%. Significant changes in the group without MetS were a decrease in diastolic blood pressure, an increase in triglyceride level and a decrease in HDL-cholesterol level in women. 15% of the participants in this group had developed the MetS at follow-up. **Conclusions:** Screening for MetS among overweight and obese individuals, followed by care as usual, leads to significant improvements in most MetS components. It might be an attractive option with potential health benefits.

249

SIXTEEN-YEAR LONGITUDINAL TRENDS IN WAIST CIRCUMFERENCE AND THEIR IMPACT ON BLOOD PRESSURE IN CHINA COHORT

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Objective: The aim of this study was to evaluate the association among baseline waist circumference (WC), changes in waist circumference (△WC) and changes blood pressure over a 16-y period. **Methods:** Prospective data from the China Health and Nutrition Survey (CHNS) of 3077 men and women aged 18-45y, with normal blood pressure at baseline (1993) from 9 provinces in China, at least reexamined one time in 4, 7, 11, 13 and 16y later and did not take anti-hypertension drugs. Sex-stratified, controlled baseline age, baseline body mass index (BMI) , changes in BMI and baseline blood pressure, multivariate analysis of variance (MANOVA) for longitudinal, repeated-measures were used to analysis systolic blood pressure (SBP) and diastolic blood pressure (DBP) change in difference baseline WC and △WC groups. The mixed effects linear model was used to analysis the body fat distribution impact on Blood Pressure. All tests of statistical significance were based on two-sided probability. **Results:** In MANOVA analysis, controlled baseline age, BMI, baseline SBP and △WC, SBP increased 8.9mmHg in low WC group and 14.9mmHg in high WC group (P< 0.001) in men. SBP increased 11.3mmHg and 20.6mmHg in low and high WC group (P< 0.001) in women. DBP increased 5.9mmHg in low WC group and 9.9mmHg in high WC group (P< 0.001) in men. DBP increased 5.8mmHg and 11.1mmHg in low and high WC group (P< 0.001) in women. SBP and DBP increased in both high and low △WC gain groups were higher than maintain and loss WC groups, both in men and women. In the mixed effects linear model analysis, we found a substantial association between WC and blood pressure. △WC and baseline WC were all important factors impact on blood pressure both in men and women. **Conclusions:** The result in this study supports an independent effect of the central distribution of body fat and the increase in central body fat on change in SBP and DBP in both men and women. People who increased their waist circumference are increased the risk of hypertension.

3

THE WAIST-HIP-HEIGHT-RATIO (WHHR): AN IMPROVEMENT OF THE WAIST-HIP-RATIO FOR PREDICTING ALL-CAUSE MORTALITY

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Objectives: To examine if adding height to the Waist-Hip-Ratio (WHR) to form the new anthropometric measure Waist-Hip-Height-Ratio (WHHR) improves its predictive ability for all-cause mortality, and to compare WHHR's performance with the other anthropometric measures Body Mass Index (BMI), waist circumference (WC) and Waist-Height-Ratio (WHtR). **Methods:** During the years 1990-99, all men and women in the county of Västmanland, Sweden turning 40 or 50 were invited to a health survey (participation rate=48%). All-cause mortality was followed up until July 1, 2010 for persons with complete anthropometric measures (n=33530, men=48%). WHHR was formed by dividing WHR by height. Cox regression, adjusted for age, smoking, diabetic status, systolic and diastolic blood pressure, heart rate and glucose level, was calculated separately for WHHR, WHR, WHtR, WC and BMI. Each anthropometric measures predictive abilitiy for all-cause mortality was calculated using the C-statistic and pseudo R-square, and compared to WHHR using bootstraped p-values. **Results:** During follow-up, a total of 1640 persons (4.9%; 958 men, 682 women) died. Using either C-statistic or pseudo R-square, WHHR improved the predictive ability for all-cause mortality of WHR, which was close to significant ($p< 0.1$) for men. Also, WHHR had better predictive ability than WHtR, WC and BMI (usually $p< 0.1$). **Conclusions:** Adding height to the Waist-Hip-Ratio may improve its ability to predict all-cause mortality, especially for men.

	C-statistic				Pseudo R-square			
	Men	P-value	Women	P-value	Men	P-value	Women	P-value
BMI	0.7117	0.022	0.6902	0.020	0.4397	0.010	0.4354	0.018
WC	0.7118	0.024	0.6935	0.114	0.4426	0.012	0.4432	0.066
WHtR	0.7128	0.020	0.6946	0.102	0.4456	0.026	0.4468	0.096
WHR	0.7147	0.060	0.6962	0.236	0.4520	0.098	0.4508	0.144
WHHR	0.7165	Ref.	0.6974	Ref.	0.4564	Ref.	0.4550	Ref.

[Predictive ability compared to WHHR]

SESSION 2:
Adipose tissue: from epidemiology to basic studies

586

FIRST-TRIMESTER HYPERTRIGLYCERIDEMIC WAIST PHENOTYPE: A MARKER OF A DETERIORATED METABOLIC PROFILE LATER IN THE PREGNANCY

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Aims: Pregnancy is associated with metabolic perturbations that could result in adverse pregnancy outcomes and influence the postpartum woman's cardiometabolic risk profile. In response to the increasing prevalence of obesity and unfavorable life habits worldwide, these outcomes should become significantly more frequent in the next years. This trend will inevitably compel the development of early, clinically accessible means to screen for metabolic perturbations in order to improve preventive strategies. The "hypertriglyceridemic waist" phenotype (abdominal obesity in combination with hypertriglyceridemia) is a clinical marker of a deteriorated cardiometabolic risk profile. This study aimed to assess the association between the "hypertriglyceridemic waist" phenotype in early pregnancy and the metabolic profile later in the pregnancy. **Methods:** Plasma triglycerides and waist girth were measured at 11-14 weeks' gestation among a sample of 144 Caucasian pregnant women. Lipid profile, insulin, glucose and adiponectin levels were measured at 24-28 weeks' gestation, in the morning, following a 12-hour fast. Glycemia was also measured following a 75g-oral glucose tolerance test (OGTT). **Results:** A waist girth >85 cm in combination with triglycerides ≥1.7 mmol/L in the first trimester was associated with an increased risk of 2-hour glucose ≥7.8 mmol/L following the 75g-OGTT (odds ratio = 6.1; p = 0.002). This risk remained significant even when controlling for maternal age, fasting glucose at first trimester and previous history of gestational diabetes (odds ratio = 4.7; p = 0.02). The combination of first-trimester abdominal obesity and hypertriglyceridemia is also associated with a significant increase of fasting insulinemia and a decrease of plasma adiponectin levels (p< 0.05) measured at the end of the second trimester. **Conclusions:** The measurement of waist girth in combination with triglyceride concentrations in the first trimester of pregnancy could improve the early screening of gestational metabolic perturbations.

222

GASTRIC BYPASS SURGERY RESULTS IN VISCERAL FAT LOSS AND NORMALIZATION OF ADIPOSE TISSUE INSULIN SENSITIVITY

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Objective: We hypothesized that individuals who have undergone gastric bypass have greater insulin sensitivity than obese subjects but less compared to lean. **Research design and methods:** We measured free fatty acid (FFA) and glucose kinetics at steady state during a two-step [low dose insulin (LDI) and high dose insulin (HDI)], hyperinsulinemic, euglycemic clamp in non-diabetic subjects who were 38±5 months post-gastric bypass surgery (gastric bypass, N=15), in lean subjects (lean, N=15), and in obese subjects (obese, N=16) subjects. Body composition was measured using DEXA and single-slice (L2-L3) CT scans. **Results:** Total FFA and palmitate concentrations were not significantly different between the three study groups at baseline. The rate of appearance (μmol·min^{-1}) of palmitate was significantly lower in lean (28.7±4.0) than both obese (66.4±7.6) and gastric bypass (51.6±5.0) at LDI but at HDI it was greater (P< 0.05) in obese (33.8±4.6) than both lean (13.5±2.2) and gastric bypass (19.9±1.6) which were not significantly different. The effective insulin concentration (mU/L) resulting in half-maximal suppression of FFA from baseline levels (EC_{50}) was similar between lean (6.1±0.5) and gastric bypass (7.3±0.7) and significantly less (P< 0.05) in both groups compared with obese (19.5±2.3). Glucose infusion rates (mmol·kg^{-1}·min^{-1}) during LDI were not significantly different in gastric bypass (8±1) compared with either lean (13±2) or obese (5±1) but during HDI, glucose infusion rates were greater in gastric bypass (36±3) than in obese (23±3) and lower than in lean (50±2), both P< 0.05. Total body fat (%) was similar between gastric bypass (42.9±1.3) and obese (49.6±2.0) compared with lean (26.7±2.2) but visceral fat (cm^2) was higher (P< 0.05) in obese (148±15) than both gastric bypass (56±9) and lean (34±5). **Conclusions:** Gastric bypass patients have near-normal visceral fat, and their adipose tissue insulin sensitivity is more similar to lean individuals than obese controls. Considering that total body fat was similar in gastric bypass patients and obese subjects, these results support the concept that visceral fat is a significant contributor to systemic lipolysis during hyperinsulinemia. Normalization of this fat depot may be a major contributor to improvement in systemic insulin sensitivity after gastric bypass surgery.

593

HYPERTRIGLYCERIDEMIC WAIST IDENTIFIES MEN AND WOMEN AT INCREASED. CARDIOMETABOLIC RISK IN A COHORT OF 2322 HIV PATIENTS

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Objective: Screening for increased waist circumference (WC) and hypertriglyceridemia (the hypertriglyceridemic waist phenotype: HTGW) is an inexpensive approach to identify patients at risk of coronary artery disease in apparently healthy individuals who may be at increased risk due to an excess accumulation of intra-abdominal (visceral) fat. We examined the relationship between the HTGW phenotype and selected cardiometabolic risk factors in HIV individuals. **Methods:** The HTGW phenotype was defined as a WC of 90 cm or more and a triglyceride (TG) level of 2.0 mmol/L or more in men, and a WC of 85 cm or more and a TG level of 1.5 mmol/L or more in women. Using these threshold values a total of 2322 patients (841 women and 1481 men) with HIV aged 18-75 years were divided into 4 groups: Low TG/Low WC, High TG/Low WC, Low TG/High WC, High TG/High WC. Continuous variables were analyzed using ANOVA or Kruskal-Wallis test where appropriate; categorical variables were compared using X^2-test. The relationship between HTGW and cardiometabolic risk assessed with Framingham risk score (FRS) was analyzed using multivariable logistic regression analyses. **Results:** Compared with patients who had a WC and TG level below the threshold values, those with the HTGW phenotype had higher visceral adipose tissue (P< 0.001), higher prevalence of hypertension and the metabolic syndrome (P< 0.001), higher levels of total and LDL-cholesterol (P< 0.001), lower levels of HDL-cholesterol (P< 0.001), and higher values of HOMA-insulin resistance (P< 0.001). The FRS (median 10, range 5-16) was also highest in those with the HTGW phenotype (P< 0.001). These observations were true independent of gender and remained significant after statistical control for illicit drug use, insulin resistance, antiretroviral therapy exposure, leg fat and proteinuria. **Conclusions:** Among HIV patients from an Italian monocentric cohort, the HTGW phenotype was associated with a deteriorated cardiometabolic risk profile and an increased FRS. It is suggested that the simultaneous measurement and interpretation of WC and fasting TG could also be used among HIV patients as an inexpensive tool to identify patients with excess visceral fat and with related cardiometabolic abnormalities.

237

NOT ALL OBESE SUBJECTS OF MULTIETHNIC ORIGIN ARE AT SIMILAR RISK FOR DEVELOPMENT OF HYPERTENSION AND TYPE 2 DIABETES

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Objective: To evaluate in a group of moderately-to-severely obese Brazilians in what extension the degree in insulin sensitivity and obesity contribute to the prevalence of hypertension and type 2 diabetes (DM2). **Methods:** This was a cross-sectional study. After clinical and laboratory evaluation, those subjects with DM2, stage 2 hypertension, secondary forms of hypertension and with any evidence of complication of cardiovascular disease were excluded. The study sample comprised 118 untreated individuals (34men and 84women). The insulin resistance status was assessed by HOMA-IR index. **Results:**

Risck Factor	Tertile 1 2.7±0.8 (n=39)	Tertile 2 4.8±0.7 (n=40)	Tertile 3 (9.1±2.4) (n=39)	P Value For trend
BMI (kg/m2)	36.1±5.6	38.8±8.6	40.8±8.7	0.03
Waist circumference (cm)	105.3±10.0	113.6±15.8	117.6±14.3	0.01
HDL-Cholesterol (mg/dL)	48.1±11.6	46.5±10.5	42.2±8.0	<0.05
Triglyceride (mg/dL)	124.1±76.4	145.4±61.2	158.6±71.5	0.09
Fasting plasma glucose (mg/dL)	93.6±12.1	98.1±12.7	100.0±11.0	<0.05
Systolic blood pressure (mmHg)	123.9±17.0	130.2±15.6	136.9±17.0	<0.01
Diastolic blood pressure (mmHg)	79.6±10.5	82.8±11.1	88.4±10.5	<0.01
Plasma adiponectin (µg/mL)	7.8±3.3	7.0±2.8	6.3±6.5	0.02
Serum Insulin (um/l)	11.7±3.2	20.3±3.9	37.2±8.2	<0.0001

[Clinical Characteristics of the obese subjects.]

Patients were divided into tertiles according to their HOMA-IR. The mean HOMA-IR in tertile 3 was 3-fold higher than the average of the most insulin-sensitive group (tertile1). Mean arterial pressure showed a linear and significant variation across HOMA tertiles. The disparity in risk of hypertension and DM2 across HOMA tertiles was even more evident when analyzed on the basis of categorical variables. A multiple linear regression analysis showed that only HOMA-IR and age independently affected the risk for increased systolic blood pressure (β= 0.364; 0.228, respectively, p< 0.01). **Conclusion:** In this group of obese subjects of multiethnic origin differences in insulin sensitivity, more than the degree of obesity, contributed to the prevalence of risk factors associated with cardiovascular disease.

426

ARE THERE ETHNIC DIFFERENCES IN THE ASSOCIATIONS BETWEEN BODY FAT DISTRIBUTION/VISCERAL ADIPOSITY AND LIVER FAT CONTENT? THE INSPIRE ME IAA STUDY

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Aims: The lack of ethnicity-specific anthropometric and metabolic markers to define cardiometabolic risk in specific populations has been previously reported. The objective of this study was to identify, in both men and women, potential ethnic differences in abdominal visceral/subcutaneous adiposity, in liver fat content and in their inter-relationship. **Methods:** The INternational Study of Prediction of Intra-abdominal adiposity and its RElationship with cardioMEtabolic risk / Intra-Abdominal Adiposity (INSPIRE ME IAA) recruited 4504 patients followed by 297 primary care physicians, cardiologists, endocrinologists and diabetologists from 29 countries. 4097 subjects (men and women) for whom ethnicity was documented were included in the present analyses: 2011 Caucasians, 166 Blacks, 381 Hispanics, 1192 East Asians, 347 South Asians. Abdominal fat compartments (Abdominal total (TAT), visceral (VAT) and subcutaneous (SAT)) and liver fat content (LFC, estimated by liver attenuation) were assessed by computed tomography. Other measurements included body mass index (BMI), waist circumference, systolic blood pressure (SBP), plasma lipids, adiponectin, CRP and HOMA-IR. **Results:** In both genders, there were significant differences among ethnicities for BMI, waist circumference, TAT, VAT, SAT, VAT/SAT ratio and LFC (Table 1), as well as for HDL-cholesterol, adiponectin, HOMA-IR and CRP. In both genders, East Asians had the lowest TAT, VAT and SAT levels compared to other ethnicities but the highest VAT/SAT ratio. In all ethnic groups, VAT was significantly correlated to BMI and SAT, with significant differences among ethnicities regarding the slopes of the regression lines. Whereas LFC was positively correlated to VAT and to VAT/SAT ratio (women), these relationships were not different among ethnic groups. **Conclusion:** Despite lower overall and visceral adiposity values, East Asians are exposed to a more deleterious VAT/SAT ratio i.e. larger relative VAT accumulation, and a related higher LFC. However, in all ethnic groups, the relation of VAT to LFC was similar.

Ethnicity	Caucasians mean±sd	Blacks mean±sd	Hispanics mean±sd	East Asians mean±sd	South Asians mean±sd	Ethnicity effect p value
n (% of men)	2011(55%)	166(34%)	381(44%)	1192(53%)	347 (52%)	<0.0001
Age (y)	57.5±7.4	56.1±6.9	55.3±7.8	56.8±7.7	54.6±7.5	<0.0001
BMI (kg/m2)	30.2±5.3 h,ea,sa	30.5±5.5 h,ea,sa	29.2±5.1 c,b,ea,sa	25.0±3.7 c,b,h,sa	27.5±4.3 c,b,h,ea	<0.0001
Waist circumference (cm)	102.5±13.9 b,h,ea,sa	100.4±13.3 c,h,ea,sa	97.5±13.4 c,b,ea,sa	87.1±10.7 c,b,h,sa	93.1±11.8 c,b,h,ea	<0.0001
Visceral Adipose Tissue, VAT (cm2)	192±87 b,ea,sa	148±67 c,h,ea	163±71 c,b,ea	134±57 c,b,h,sa	156±64 c,ea	<0.0001
Subcutaneous Adipose Tissue, SAT (cm2)	304±113 ea,sa	318±134 ea,sa	306±118 ea,sa	194±81 c,b,h,sa	261±102 c,b,h,ea	<0.0001
VAT/SAT ratio	0.68±0.35 b,h,ea	0.52±0.29 c,h,ea,sa	0.60±0.33 c,b,ea,sa	0.77±0.33 c,b,h,sa	0.65±0.27 b,h,ea	<0.0001
Liver fat content, LFC (liver attenuation)	52.4±12.8 b,ea,sa	57.2±11.2 c,h,sa	51.5±12.8 b,sa	55.2±10.0 c,h,sa	50.6±12.3 c,b,ea	<0.0001
c p<0.05 compared to Caucasians,b p<0.05 compared to Blacks,h p<0.05 compared to Hispanics,ea p<0.05 compared to East Asians,sa p<0.05 compared to South Asians,Adjusted for age and practitioner speciality						

[Table 1: Ethnic group characteristics]

234

FRUCTOSE OVERLOAD IN RATS WOULD INCREASE CORTICOSTERONE PRODUCTION MODIFYING NADPH METABOLISM IN EPIDIDYMAL WHITE ADIPOSE TISSUE

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Fructose-overload (FO) in rats resembles the human metabolic syndrome characterized by insulin resistance (IR), dislypidemia and hypertension. NAPDH metabolism links the pentose phosphate pathway with the antioxidant enzymes network and glucocorticoids synthesis. Altered glutathione peroxidase (GPx) and glucose-6-phosphate dehydrogenase (G6PD) activities have been associated to IR and lipid dysregulation. Moreover, corticosterone (Cort) administration produces increased adiposity, IR, and elevated plasma leptin, insulin and triglycerides (TG). **Objectives:** Characterize metabolic parameters and enzymatic activities involved in NADPH metabolization in epididymal white adipose tissue (EWAT) from rats subjected to FO. **Methods:** Male Sprague-Dawley rats received fructose (F, 10% w/v tap water, n=8) by 7 weeks or tap water as control group (C, water, n=8). Plasma TG and glucose were determined by enzymatic assay and Cort by HPLC-UV. Enzymatic activities were measured spectrophotometrically in homogenates of EWAT: GPx by the GR-coupled method, GR monitoring NADPH loss in the presence of GSSG, CAT following H_2O_2 loss, SOD by cytochrome C method, and G6PD by monitoring NADPH production in the presence of G6P. **Results:** FO produced hypertriglyceridemia (mg/dl: 173±4 vs. 79±16; p< 0.01) without significant changes in glucemia. CAT, SOD and GR activities were unchanged by the treatment. GPx activity decreased in FO (nmoles/min. mg prot: 52±5 vs. 87±10; p< 0.05). On the contrary, G6PD activity increased in FO (nmoles of reduced NADP/min.mg prot: 4.7±0.5 vs. 3.0±0.2; p< 0.03). The endogenous content of NADPH in EWAT remained unchanged by FO treatment, while plasma Cort was significantly increased in FO rats (pg/ml: 190±22 vs. 90±12; p< 0.01). **Conclusion:** As a result of the increase in G6PD and the decrease in GPx activities there should be a higher NADPH supply for Cort production by 11b-HSD1 in FO rats. In this way, EWAT could be one of the sources of the higher Cort levels detected in FO plasma respect to C rats.

459

ENHANCED VISCERAL ADIPOSE TISSUE INFLAMMATION IN A MURINE MODEL OF ATHEROSCLEROSIS AND INSULIN RESISTANCE THAT IS NOT ASSOCIATED WITH OBESITY

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Objectives: Obesity-linked diabetes and dyslipidemia are known to be associated with chronic low-grade inflammation that involves the recruitment of macrophage in the enlarged visceral fat depot. However, the respective contribution of each metabolic disorder to the inflammatory state of visceral adipose tissue remains unknown. **Methods:** We have characterized wild-type (WT) and two murine models of dyslipidemia and diabetes. Mice with a genetic deletion for both the LDL receptor and apolipoprotein B48 (LDLr/ApoB^{48} dKO) were used and were crossbred with mice overexpressing insulin growth factor II in pancreatic β-cells (LDLr/ApoB^{48} dKO x IGF-II $^{+/-}$) to promote T2D. All mouse models were either fed with a standard diet (SD) or a diet rich in fat (55%) (HF) for 24 weeks. We assessed glucose tolerance and insulin sensitivity using intraperitoneal glucose tolerance tests (IPGTT) and hyperinsulinemic-isoglycemic clamps. Adipose tissue inflammation was evaluated by measuring a panel of cytokines and chemokines by Luminex® in lysates of epididymal fat and by assessing macrophage recruitment by immunocytochemical detection of the macrophage marker F4/80. **Results:** LDLr/ApoB^{48} dKO and LDLr/ApoB^{48} dKO x IGF-II $^{+/-}$ mice had increased cholesterol and triglyceride plasma content and developed insulin resistance. Marked glucose intolerance was observed in LDLr/ApoB^{48} dKO x IGF-II $^{+/-}$ mice fed with the HF diet. Adipose tissue of LDLr/ApoB^{48} dKO mice had a similar cytokine production profile compared to WT mice. In contrast, we found a significantly increased (p< 0.05) proinflammatory profile in adipose tissue of LDLr/ApoB^{48} dKO x IGF-II $^{+/-}$ mice compared with LDLr/ApoB^{48} dKO or WT animals on either diets, as revealed by augmented levels of 17 different cytokines/chemokines in the former group. This difference of cytokine production was not associated with significant changes of adipocytes size or body mass between the respective genotypes. Increased macrophage infiltration was also observed in adipose tissue of LDLr/ApoB^{48} dKO (29%) and LDLr/ApoB^{48} dKO x IGF-II $^{+/-}$ (65%) mice compared with WT. **Conclusions:** Our results suggest that the IGF-II $^{+/-}$ x LDLrKO mice have increased adipose tissue inflammation independently of obesity. This mouse model may therefore be an interesting tool in order to assess the links between diabetes and adipose tissue inflammation.

412

EXCESS VISCERAL FAT ACCUMULATION IS AN INDICATOR OF ADIPOSE TISSUE MACROPHAGE INFILTRATION IN WOMEN

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Objective: Obesity is associated with a chronic, low-grade inflammatory state and macrophage infiltration in adipose tissues. We tested the hypothesis that visceral obesity would be the best predictor of omental adipose tissue macrophage infiltration in women. **Methods:** Omental and subcutaneous fat samples were surgically-obtained in 40 women (age: 47.0±4.0 years, BMI: 28.4±5.8 kg/m^2). Adipocyte diameter was measured in cell suspensions of collagenase-digested tissues. Body composition and fat distribution were measured by DEXA and computed tomography. A detailed lipid profile was obtained. CD68+ macrophages were identified in adipose tissue using fluorescence immunohistochemistry. Analyses were performed using the number of CD68+ cells per 100 adipocytes. **Results:** Mean CD68+ cell percentage tended to be higher in subcutaneous (18.30%) compared to omental adipose tissue (15.49%) (p=0.07). CD68+ cell percentage in the subcutaneous depot was positively associated with BMI (r=0.31), waist circumference (r=0.37), total abdominal adipose tissue area (r=0.32), visceral adipose tissue area (r=0.40), subcutaneous adipocyte diameter (r=0.39) and fasting glucose (r=0.31, p≤0.05 for all). After adjustment for total body fat mass, these associations were no longer significant. CD68+ cell percentage in omental adipose tissue was positively correlated with BMI (r=0.44), waist circumference (r=0.47), total abdominal adipose tissue area (r=0.35), visceral adipose tissue area (r=0.52), fasting insulin (r=0.31) and HOMA-IR (r=0.33, p≤0.05 for all). The association between omental CD68+ cell percentage and visceral adipose tissue area remained significant after adjustment for total body fat mass (p=0.02). Visceral adipose tissue area was the best predictor of CD68+ cell percentage in both the omental and subcutaneous depots, explaining respectively 20% and 12% of the variance in models also including subcutaneous adipose tissue area, adipocyte sizes and total body fat mass. CD68+ cell percentage in omental adipose tissue was inversely associated with serum concentrations of HDL-cholesterol, HDL_2-triglycerides and HDL-apolipoprotein A1 (-0.39≤r≤-0.45, p≤0.05 for all). The association between CD68+ cell percentage in the omental compartment and HDL-cholesterol remained significant after adjustment for total body fat mass (p=0.03), but not visceral adipose tissue area. **Conclusion:** Visceral adipose tissue accumulation is an indicator of macrophage infiltration in both the subcutaneous and omental fat compartment of lean to moderately obese women.

87

ROLE OF MESENTERIC FAT, IL6, AND SPHINGOMYELIN IN HEPATIC STEATOSIS AND INSULIN RESISTANCE

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The associations between hepatic lipid accumulation, dyslipidemia and insulin resistance has been well characterized in several dietary models of chronic metabolic diseases. Evidence is mounting that inflammation is a primary factor in the pathogenesis of insulin resistance and dyslipidemia. In addition, lipid species other than triglycerides (TG), such as sphingolipids, have been identified as key players in the development of the hallmark metabolic disturbances associated with insulin resistance. Our laboratory has characterized a dietary model of insulin resistance and dyslipidemia using a high fat, high fructose, high cholesterol (FFC) diet. We have investigated the link between mesenteric fat accumulation and hepatic inflammation/steasis using the FFC hamster and rat models. In this study, a 10 day time course comparing FFC to chow was completed in 60 rats (n=6 per group per day). Hepatic lipids, as measured biochemically and visually with oil-red o staining, were significantly increased (p< 0.05) in as little as 2 days of FFC feeding. FFC treated rats had significantly elevated plasma TG (p< 0.05), with the greatest difference in TG (2.3 fold) achieved by Day 10. Plasma and liver sphingolipids were analyzed by LC/MS/MS. Rats treated consuming FFC had significantly decreased liver and plasma sphingomyelin following 6 and 10 days. Additionally, sphingomyelin correlated strongly (r=0.88, p< 0.05) with ORO staining. Several gene targets were evaluated by qPCR and rat specific primers. IL-6 mRNA levels were significantly elevated at Day 6 and Day 10 (p< 0.05), but not Day 2 while TNFα and MCP-1 were not changed during the same time period. Although tissue ceramide levels were not increased, mRNA levels of serine palmitoyl transferase was significantly increased at Day 2 and Day 6 (p< 0.05). Sphingomyelin synthase mRNA was increased throughout the time course (Day 2, 6, and 10), while acid sphingomyelinase was increased at Day 2 only (p< 0.05). Taken together, this early examination of the development of dyslipidemia indicates a particular role for IL-6 related inflammation and suggests that there is an interaction between tissue sphingomyelin and the development of hepatic steatosis.

206

MACROPHAGE GENE EXPRESSION IN BOTH SUBCUTANEOUS AND VISCERAL FAT IS RELATED TO OBESITY AND METABOLIC SYNDROME

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Objectives: Our goal was to identify a set of human adipose tissue macrophage (ATM)-specific markers in order to investigate whether their gene expression in subcutaneous adipose tissue (SAT) as well as in visceral adipose tissue (VAT) was related to obesity and to the occurrence of metabolic syndrome (MS). **Methods:** ATM-specific markers were identified by DNA microarray analysis of AT cell types isolated from SAT of lean and obese subjects. Then, gene expression of these markers was analyzed by reverse transcription-qPCR in paired samples of SAT and VAT of 53 women stratified into 4 groups (lean, overweight, obese and obese with MS). Anthropometric measurements, euglycemic hyperinsulinemic clamp, blood analysis and computed tomography scans were performed. **Results:** A panel of 24 genes was selected as ATM-specific markers based on over expression in ATM compared to other AT cell types. In both, SAT and VAT, gene expression of ATM markers was the lowest in lean and the highest in MS group. mRNA levels in the 2 fat depots were negatively correlated with glucose disposal rate and positively associated with indices of adiposity and MS. **Conclusions:** In humans, expression of ATM-specific genes increases with the degree of adiposity and correlates with markers of insulin resistance and MS similarly in SAT and in VAT. This work was supported by grants IGA NS 10519-3-2009, MSM 0021620814, Inserm, Région Midi-Pyrénées, Integrated Project HEPADIP, (www.hepadip.org) and Collaborative Project ADAPT (www.adapt-eu.net).