Computed tomography-measured adipose tissue attenuation and area both predict adipocyte size and cardiometabolic risk in women

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Running title: ***Adipose tissue attenuation and adipocyte size***

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ABSTRACT

Objective: To assess the ability of CT-derived measurements including adipose tissue attenuation and area to predict fat cell hypertrophy and related cardiometabolic risk. **Methods:** Abdominal adipose tissue areas and radiologic attenuation were assessed using 4 CT images in 241 women (age: 47 years, BMI: 26.5 kg/m^2). Fat cell weight was measured in paired VAT and SAT samples. Fasting plasma lipids, glucose and insulin levels were measured. **Results**: Adipose tissue attenuation was negatively correlated with SAT (r=-0.46) and VAT (r=-0.67) fat cell weights in the corresponding depot ($p<0.0001$ for both). Women with visceral adipocyte hypertrophy had higher total-, VLDL-, LDL- and HDL-triglyceride and apoB levels as well as a higher cholesterol/HDL-cholesterol ratio, fasting glucose and insulin levels compared to women with smaller visceral adipocytes. Adjustment for VAT area minimized these differences while subsequent adjustment for attenuation eliminated all differences, with the exception of fasting glycaemia. In SAT, adjustment for VAT area and attenuation eliminated all adipocyte hypertrophy-related alterations except for fasting hyperglycaemia. **Conclusion**: CT-derived adipose tissue attenuation and area both contribute to explain variation in the cardiometabolic risk profile associated with the same biological parameter: visceral fat cell hypertrophy.

INTRODUCTION

Visceral obesity is associated with numerous alterations in the cardiometabolic risk profile, which increase the risk of type 2 diabetes and cardiovascular diseases [1]. Under a positive energy imbalance, adipose tissue expansion relies on adipocyte hypertrophy and/or adipose tissue hyperplasia [2]. Arner et al. have shown that subcutaneous adipose tissue (SAT) hypertrophy is associated with an altered lipid profile independent of body fat mass [3]. In addition, we have previously reported that visceral adipose tissue (VAT) hypertrophy is associated with an increase in plasma VLDL-TG levels and with higher total cholesterol/HDL-cholesterol ratio independent of total and regional adiposity [4]. We also found that visceral adipocyte hypertrophy is related to alterations in lipolysis and adipose tissue expression of genes coding for proteins involved in adipocyte metabolism or inflammation, independent of overall adiposity and body fat distribution [5]. Further, obesity is associated with extra-cellular matrix remodelling that often leads to the development of fibrosis in adipose tissue [6 7]. These alterations may partially explain the increased cardiometabolic risk associated with the visceral obesity phenotype [1 8].

Over the past decades, computed tomography (CT) has emerged as the gold-standard technique to measure abdominal body fat distribution [1]. Using a range of attenuation values expressed in Hounsfield units (HUs), this imaging technique is based on the ability of tissues to attenuate x-rays. Using this scale, most soft tissues are characterized by positive HUs while adipose tissue attenuation is located in the negative range [9]. In 1990, Tyrrel et al. [10] compared mean adipose tissue attenuation between patients with and without cirrhosis and found that patients with biopsy-proven cirrhosis were characterized by higher fat attenuation compared to controls. In that study, mean attenuation of mesenteric fat was higher than that of retroperitoneal and

subcutaneous depots [10]. Further, Hu et al. [11] observed higher attenuation values in brown compared to white adipose tissue. More recently, Fox et al. [12] examined associations between SAT and VAT attenuation values and cardiometabolic risk factors. They found that low CT attenuation of both VAT and SAT [13] was associated with an adverse cardiometabolic risk profile, independent of total adiposity. In 2014, Murphy et al. [13] have shown that SAT and VAT attenuation values were good markers of mortality risk in older adults, independent of CRP and IL-6 levels. However, the reason for the relationship between adipose tissue attenuation and cardiometabolic risk is still unclear. To the best of our knowledge, no study has ever examined the association between CT-based measurements and SAT or VAT fat cell size assessed in surgical fat samples, and the extent to which these CT characteristics could explain the risk associated with adipocyte hypertrophy. Our objective was to test the ability of CT-derived measurements to predict adipocyte hypertrophy-related cardiometabolic risk. We hypothesized that the increased cardiometabolic risk associated with visceral adipocyte hypertrophy is largely explained by CT-based measurements of VAT area and radiologic attenuation.

RESULTS

Anthropometric and metabolic characteristics of the women recruited in this study are outlined in Table 1. Mean age of the sample was 47 years. Participants were slightly overweight with a mean body mass index (BMI) of 26.5 kg/m^2 but they covered a large range of adiposity (17.2 - 41.1 kg/m^2). SAT area measured by CT was significantly greater than VAT area ($p<0.0001$). Accordingly, higher adipocyte weight was observed in SAT compared to VAT adipose tissue ($p<0.0001$). Adipose tissue mean attenuation was significantly higher in VAT than in the SAT compartment ($p<0.0001$).

We tested the associations between adipose tissue areas, adipocyte weight and adipose tissue attenuation values in each body fat compartment. As shown in Figure 1, SAT mean attenuation was a significant and negative correlate of SAT area. A significant association was also observed in the visceral fat depot. SAT and VAT areas were positively and significantly associated with adipocyte weight in the corresponding depot. In the SAT depot, adipose tissue attenuation was negatively and significantly correlated with adipocyte weight. The same pattern was observed in the visceral depot.

We investigated whether adipose tissue mean attenuation was related to cardiometabolic risk profile before and after statistical adjustment for VAT area. Supplemental Table 1 shows that markers of cardiometabolic risk, except for fasting glucose levels and the HOMA-IR index, remained associated with attenuation even after adjustment for VAT area, especially in the visceral fat compartment.

To assess whether CT-based measurements explain the increased cardiometabolic risk associated with adipocyte hypertrophy, women were subdivided according to the median of their VAT or SAT adipocyte weights and statistical adjustments for VAT area or for both VAT area and radiologic attenuation were performed. As shown in Figure 2, in the VAT depot, women with high adipocyte weight had higher total and VLDL-TG levels as well as apo B levels compared to women with low adipocyte weight. These differences remained significant after statistical adjustment for VAT area but they were no longer significant after adjustments for both VAT area and radiologic attenuation. The same pattern was observed for the cholesterol/HDL-cholesterol ratio. LDL-TG levels and the HOMA-IR index were also higher in women with high VAT

adipocyte weight. This difference remained significant after adjustment for VAT area but only tended to be significant when adjusted for both VAT area and radiologic attenuation. Women with high VAT fat cell weight were also characterized by higher fasting glucose and insulin levels before and after adjustment for VAT area. When adjusted for both VAT area and radiologic attenuation, only the difference in fasting glucose remained significant. HDL-TG levels were higher in women with high VAT adipocyte weight but this association was not independent of VAT area and radiologic attenuation.

As shown in Figure 3, women with high SAT adipocyte weight had higher total-, VLDL- and LDL-TG levels, a higher total cholesterol/HDL-cholesterol ratio, higher apo B, glucose and insulin levels as well as a higher HOMA-IR index than the subgroup of women with low SAT adipocyte weight. Most of these differences were lost after statistical adjustment for VAT area and for both VAT area and radiologic attenuation, except for total- and VLDL-TG levels. A small but significant difference remained between the 2 groups for glucose levels when adjusted for both VAT area and radiologic attenuation.

Successively excluding participants in each category of hormonal status or adjusting for age had little impact on the association or difference patterns observed in our analyses.

DISCUSSION

The aim of this study was to assess the ability of CT-derived measurements including attenuation and area to predict adipocyte hypertrophy-related cardiometabolic risk factors. Our results first show that SAT and VAT mean attenuation values are inversely significantly correlated with

adipocyte weight or size in the corresponding depot. The fact that adipose tissue area and attenuation both relate to the same biological parameter (adipocyte size) suggest that they perhaps should not be described as opposing aspects of adipose tissue quantification (e.g. adipose tissue quantity vs. quality).

We have previously reported that women with VAT adipocyte hypertrophy have increased VLDL-TG levels and cholesterol/HDL-cholesterol ratio, independent of regional/overall adiposity [4]. Arner et al. [14] also found that obese subjects with VAT adipocyte hypertrophy are characterized by an altered lipid profile. This holds true in the present study, as subjects with high VAT or SAT fat cell weights showed clear alterations in many cardiometabolic variables. A major finding in the present analysis is that CT-derived measurements can largely predict the altered lipid profile associated with VAT adipocyte hypertrophy. Indeed, most metabolic differences related to both VAT and SAT adipocyte weights were lost after adjustment for both VAT area and radiologic attenuation. To the best of our knowledge, our paper is the first to provide evidence that two variables derived from CT analysis of adipose tissue (area and attenuation) both partially capture cardiometabolic risk related to the same biological parameter: visceral adipocyte hypertrophy. Yet, based on the associations between CT-based measurements and adipocyte weight in the corresponding compartment, a portion of the adipocyte weight variance was not explained by attenuation and size of the compartment, suggesting that the latter do not entirely explain interindividual variability in fat cell size.

We found that women with a low VAT mean attenuation were characterized by increased VAT area as well as adipocyte hypertrophy. In the visceral depot, adipose tissue mean attenuation was negatively related to fat cell weight in a linear manner whereas this association appeared to be

curvilinear in the SC depot. We suggest that these observations reflect the propensity of each adipose tissue compartment for adipocyte hypertrophy and hyperplasia. As reflected by adipogenic and lipogenic gene expression, we have previously reported that fat cell hypertrophy occurs in both fat depots while hyperplasia is predominant in the SAT compartment [15]. These results were corroborated by Tchkonia et al. [16] who observed much higher expression levels of two key adipogenic transcription factors: peroxisome proliferator-activated receptor gamma (PPARγ) and CCAAT/enhancer-binding protein alpha (CEBPα) in SAT compared to VAT. Furthermore, preadipocyte replication and lipid accumulation were found to be more extensive in abdominal SAT than in VAT adipocytes. The non-linear relationship observed between SAT mean attenuation and SAT adipocyte weight could be attributable to the capacity of abdominal SAT to recruit new fat cells in this range of adiposity values in women. Conversely, the predominantly hypertrophic nature of the VAT depot could explain the lower mean attenuation observed in VAT adipocytes containing large lipid droplets. Although the present study was not designed to investigate the mechanisms underlying the association between adipose tissue mean attenuation and fat cell weights, we speculate that increased organelle content of small fat cells per surface unit could possibly explain why they show greater mean attenuation. Baba et al. [17] demonstrated that brown adipose tissue mean attenuation increased under activated conditions following a decrease in lipid content. Furthermore, Hu et al. [11] observed that HUs of brown adipose tissue were more positive than those of white adipose tissue and speculated that brown adipocyte characteristics could account for this difference as they contain more non-lipid components and are more vascularized and innervated than white adipocytes. In our study, CT attenuation of VAT was significantly greater than that of SAT. This could result from structural and functional differences between these two abdominal fat depots. Indeed, VAT is more

vascularized with higher blood supply and is more innervated than SAT [18]. As blood CT HUs are located in the positive range, this could partially explain the results observed here. More studies are needed to confirm the physiological significance of adipose tissue attenuation.

As opposed to other markers of cardiometabolic risk, fasting glucose levels remained associated with VAT adipocyte hypertrophy even after adjustment for VAT area and radiologic attenuation. Arner et al. [3] have reported that SAT adipocyte hypertrophy was associated with higher fasting insulin levels and HOMA-IR index independent of BMI. Conversely, Ledoux et al. [19] reported that VAT adipocyte size was more closely related to alterations in indices of plasma glucose-insulin homeostasis in obese individuals than SAT adipocyte size. Our results suggest that factors other than VAT area and attenuation mediate the association between fat cell size and glycaemia. In 2013, Fox et al. [12] demonstrated that in diabetic and insulin resistant women, a 1-SD decrease in VAT attenuation values was associated with an increased risk of having impaired fasting glucose and insulin resistance. The present study extends this finding to nondiabetic women covering a wide range of adiposity and levels of insulin resistance. More recently, Fox et al. has also shown that VAT attenuation was inversely associated with CVD events (when adjusted for age and sex) [20].

This study has some limitations, which should be acknowledged. The cross-sectional design cannot provide information about the directionality of the associations. Therefore, it is not possible to conclude on cause-and-effect relationships between adipose tissue radiologic attenuation or fat cell hypertrophy and metabolic profile variables. Further, this study only included women due to the difficulty of performing similar studies in men. Therefore the findings cannot be extended to men.

In conclusion, our study is the first to provide evidence that CT-derived measurements, including adipose tissue area in conjunction with radiologic attenuation, explain most of the variation in cardiometabolic risk profile associated with fat cell hypertrophy. This finding provides a novel framework by which CT imaging data of adipose tissue may be used as an indirect marker of fat cell size or adipocyte hypertrophy, especially in the visceral compartment.

METHODS AND PROCEDURES

Subjects

Women (n=241), aged 40 to 60 years, were recruited through the elective surgery schedule of the Gynecology Unit of Laval University Medical Research Center from 2001 to 2012. They were scheduled for total (n=229) or subtotal (n=10) abdominal hysterectomies or myomectomy (n=1), sometimes accompanied by salpingo-oophorectomy of one (n=35) or two (n=95) ovaries. Type of surgery was unavailable for one participant. A few weeks before surgery and on the morning of surgery, detailed information was obtained on medication use and reproductive, menstrual, and medical history for each patient. Women using medication affecting metabolic parameters (beta-blockers, ACE inhibitors, fibric acid derivatives, and statins) were not included in the present study. Women reporting use of nonsteroidal anti-inflammatory medication a few weeks before the surgery were also excluded. Menopausal status was assessed by questionnaire (133 premenopausal, 50 perimenopausal and 54 menopausal women). Status was unavailable for 4 women. This project was approved by the medical ethics committee of Laval University Medical Research Center. All women provided written, informed consent before their inclusion in the project.

Body fatness and body fat distribution measurements

These tests were performed on the morning of surgery or a few days before the intervention and in a few cases shortly after the intervention. Body fat percentage, total body fat mass and lean body mass were assessed by dual energy x-ray absorptiometry (DEXA) using a Hologic QDR-2000 densitometer and the enhanced array whole-body software V5.73A (n=73) or a Hologic QDR-4500 densitometer and the whole-body fan V8.26A:3 (n=168) (Hologic Inc., Bedford, MA). Abdominal SAT and VAT data were acquired by CT using a GE Light Speed 1.1 CT scanner (n=233) or the Brightspeed CT scan (General Electric Medical Systems, Milwaukee, WI) (n=8). The scan was performed at the L4-L5 vertebrae level using a scout image of the body to establish the precise scanning position. Four images that were 5 mm-thick and 5 mm apart in the intervertebral space were obtained for each woman. Subjects were examined in the supine position with arms stretched above the head. VAT area was quantified by delineating the intra-abdominal cavity at the internal-most aspect of the abdominal and oblique muscle walls and the posterior aspect of the vertebral body using Image J 1.33u software (National Institutes of Health, USA). The abdominal muscle layer was completely excluded. Adipose tissue areas were computed using an attenuation range of –190 to –30 HUs. Mean SAT and VAT attenuation were computed from the 4 images. All these measurements were performed by the same investigator.

Adipose tissue sampling, adipocyte isolation and cell weight measurements

During surgery, paired SAT and VAT (omental) samples were obtained for each woman at the site of incision and at the distal portion of the greater omentum, respectively. Adipose tissue samples were digested with collagenase type I in Krebs-Ringer-Henseleit (KRH) buffer for up to 45 minutes at 37°C according to a modified version of the Rodbell method [21]. Adipocyte

suspensions were filtered through nylon mesh and washed 3 times with KRH buffer. Adipocyte weight and cell number in the suspensions were calculated using lipid weight, average cell volume and the density of triolein as previously described [5].

Lipid profile and glucose homeostasis

Blood samples were obtained on the morning of surgery after a 12-hour fast. Cholesterol and triglyceride levels were measured in plasma and lipoprotein fractions with a Technicon RA-500 analyzer (Bayer, Etobicoke, ON, Canada) using enzymatic methods or with the Olympus AU400 (Beckman Coulter, Mississauga, Canada). Plasma VLDL were isolated by ultracentrifugation [22] and the HDL fraction was obtained after precipitation of LDL in the infranatant with heparin and $MnCl_2$ [23]. Cholesterol content of the infranatant was measured before and after precipitation and LDL cholesterol concentration was obtained by difference. Apolipoprotein (apo) B and apo A1 concentrations were measured using the rocket immunoelectrophoretic method of Laurell [24] as described previously [25] or using the Siemens Healthcare Diagnostics BN ProsSpect (Siemens Healthcare Diagnostics, Mississauga, Ontario, Canada). Plasma glucose was measured using the glucose oxidase method or with a fully automated Modular P800 system (Roche, Diagnostics, Laval, Canada). Insulin was measured by ELISA (Alpco, Salem, NH, USA and EMD Millipore, Massachusetts, USA) or by radioimmunoassay (Linco Research, St Charles, MO). The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated as fasting insulin (μU/mL) x fasting glucose (mmol/L)/22.5 [26].

Statistical analyses

Paired t tests were used to compare measures performed in the SAT *vs.* VAT depots. Spearman correlation coefficients were computed to test associations between adipose tissue mean attenuation in each fat compartment and body fat distribution as well as adipocyte weight. To investigate the linearity assumption, a generalized additive model (GAM) was performed to appreciate linearity of an unknown smooth function among variables. GAM was also used for inference about these smooth functions. The relationship between adipocyte weight and cardiometabolic profile variables was examined by subdividing women in 2 subgroups according to the median of adipocyte weight in each fat depot (100 low adipocyte weight *vs.* 100 high adipocyte weight in SAT and 100 low adipocyte weight *vs.* 100 high adipocyte weight in VAT). Differences between subgroups were tested using Student's *t*-test. Similar analyses were performed after adjustment for adipose tissue area or adjustment for both adipose tissue area and radiologic attenuation. For these analyses, stratification was based on the residuals of the regressions between adipocyte weight in a given compartment vs. adipose tissue area, or between adipocyte weight in a given compartment vs. adipose tissue area and radiologic attenuation, respectively. We perform all analyses according to menopausal status by successively excluding participants in each category of hormonal status. All statistical analyses were also computed after adjustment for age using multiple regression analysis. Analyses were performed on log10-transformed or Box Cox-transformed values when variables were not normally distributed. P-values lower than 0.05 were considered statistically significant. Statistical analyses were performed using JMP statistical software 10.0.2 (SAS Institute, Cary, NC).

CONFLICTS OF INTEREST STATEMENT AND ACKNOWLEDGMENTS

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411 **FIGURE LEGENDS**

412 **Figure 1.** Correlations between SAT (A) or VAT (B) attenuation and adipose tissue area in the
413 corresponding depot; between SAT (C) or VAT (D) areas and adipocyte weight in the
414 corresponding depot; and between SAT (E) or VAT (F) attenuation and adipocyte weight in the
415 corresponding depot. Spearman correlation coefficients were computed to test associations. P-
416 values lower than 0.05 were considered statistically significant. Linearity testing indicated a
417 significant order 2 for panels A and B, linear relationship for panels C, D and F, and a significant
418 order 3 for panel E.

419

420 **Figure 2.** Cardiometabolic risk profile in subgroups of women defined on the basis of their VAT
421 fat cell weight (low vs. high) using the median value of VAT fat cell weights as cutoff, before
422 (Unadj) and after (Adj) statistical adjustments for adipose tissue area or adipose tissue area and
423 attenuation. Differences between subgroups were tested using Student's *t*-test. Adjustment for
424 VAT area and after adjustment for both VAT area and radiologic attenuation were performed
425 using multiple regression analysis. P-values lower than 0.05 were considered statistically
426 significant. chol: cholesterol

427
428 **Figure 3.** Cardiometabolic risk profile in subgroups of women defined on the basis of their SAT
429 fat cell weight (low vs. high) using the median value of SAT fat cell weights as cutoff, before
430 (Unadj) and after (Adj) statistical adjustments for adipose tissue area or adipose tissue area and
431 attenuation. Differences between subgroups were tested using Student's *t*-test. Adjustment for
432 SAT area and after adjustment for both SAT area and radiologic attenuation were performed
433 using multiple regression analysis. P-values lower than 0.05 were considered statistically
434 significant. chol: cholesterol

435 **Table 1.** Adiposity, body fat distribution and metabolic characteristics of the women

	Mean	±	SD	Range
Age (years)[a]	47.0	±	5.2	35.2-68.3
Height (cm) [a]	161	±	6	145-176
Weight (kg) [a]	68.6	±	12.6	42.8-110
BMI (kg/m^2) [a]	26.5	±	4.6	17.2-41.1
Body fat mass (kg) [a]	25.5	±	8.1	5.9-58.2
Body fat percentage (%) [a]	36.3	±	6.4	14.0-58.2
Lean body mass (kg) [a]	41.2	±	6.4	25.4-58.9
Abdominal adipose tissue areas (cm^2 [a]) and attenuation (HU) [a]				
Total area	403	±	146	70-871
Subcutaneous area	300	±	114	42-735
Visceral area	91	±	44*	21-280
Subcutaneous attenuation	–87.8	±	7.5	–103.2- –72.4
Visceral attenuation	–103.2	±	5.2*	–110.6- –66.2
Adipocyte weight (µg lipid)[b]				
Subcutaneous	0.56	±	0.23	0.04-1.31
Visceral	0.34	±	0.19*	0.02-1.00
Cholesterol content (mmol/L)[c]				
Total	5.03	±	0.91	2.62-7.52
VLDL	0.44	±	0.30	0.03-1.92
LDL	3.14	±	0.84	1.07-5.60
HDL	1.45	±	0.38	0.63-2.75
Total cholesterol/HDL chol	3.68	±	1.07	1.66-9.29
Triglyceride content (mmol/L)[c]				
Total	1.28	±	0.67	0.40-6.0
VLDL	0.75	±	0.52	0.10-3.64
LDL	0.25	±	0.09	0.09-0.58
HDL	0.26	±	0.07	0.13-0.62
Apolipoprotein (mg/dL)[c]				
Apo B	0.9	±	0.2	0.4-1.6
Apo A1	1.4	±	0.3	0.7-2.2
Apo B/ Apo A1	0.7	±	0.2	0.2-1.6
Glucose homeostasis				
Fasting glucose (mmol/L)	5.5	±	0.7	3.8-8.0
Fasting insulin (uU/mL)	8.6	±	5.0	1.5-27.6

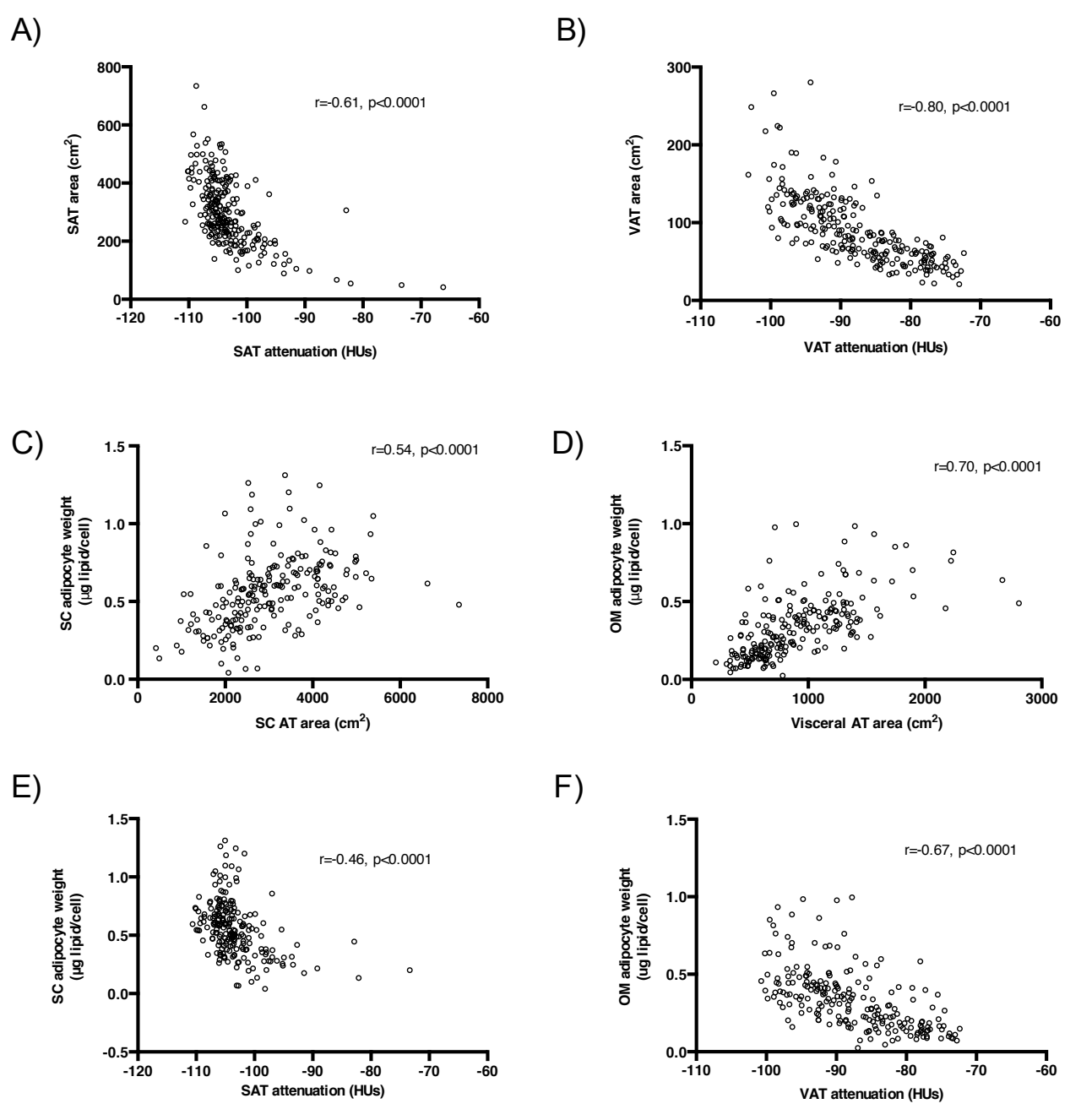
436
437 *p<0.0001 : Subcutaneous vs omental significantly different by paired t test, a : n=241; b : n=217; c : n=238
438
439

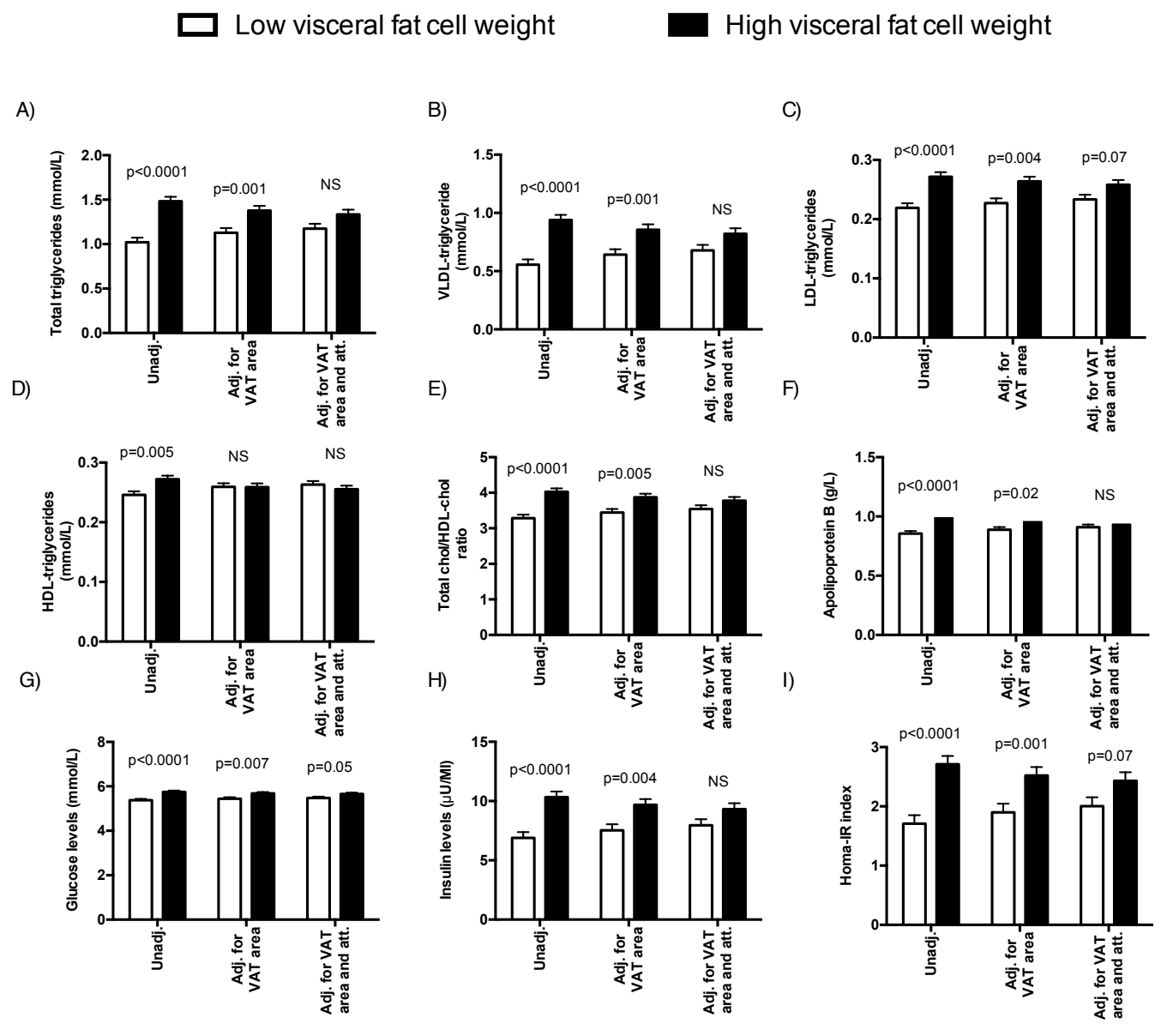
Supplemental Table 1. Spearman correlation coefficients between cardiometabolic profile variables and SAT or VAT mean attenuation before and after statistical adjustment for VAT area.

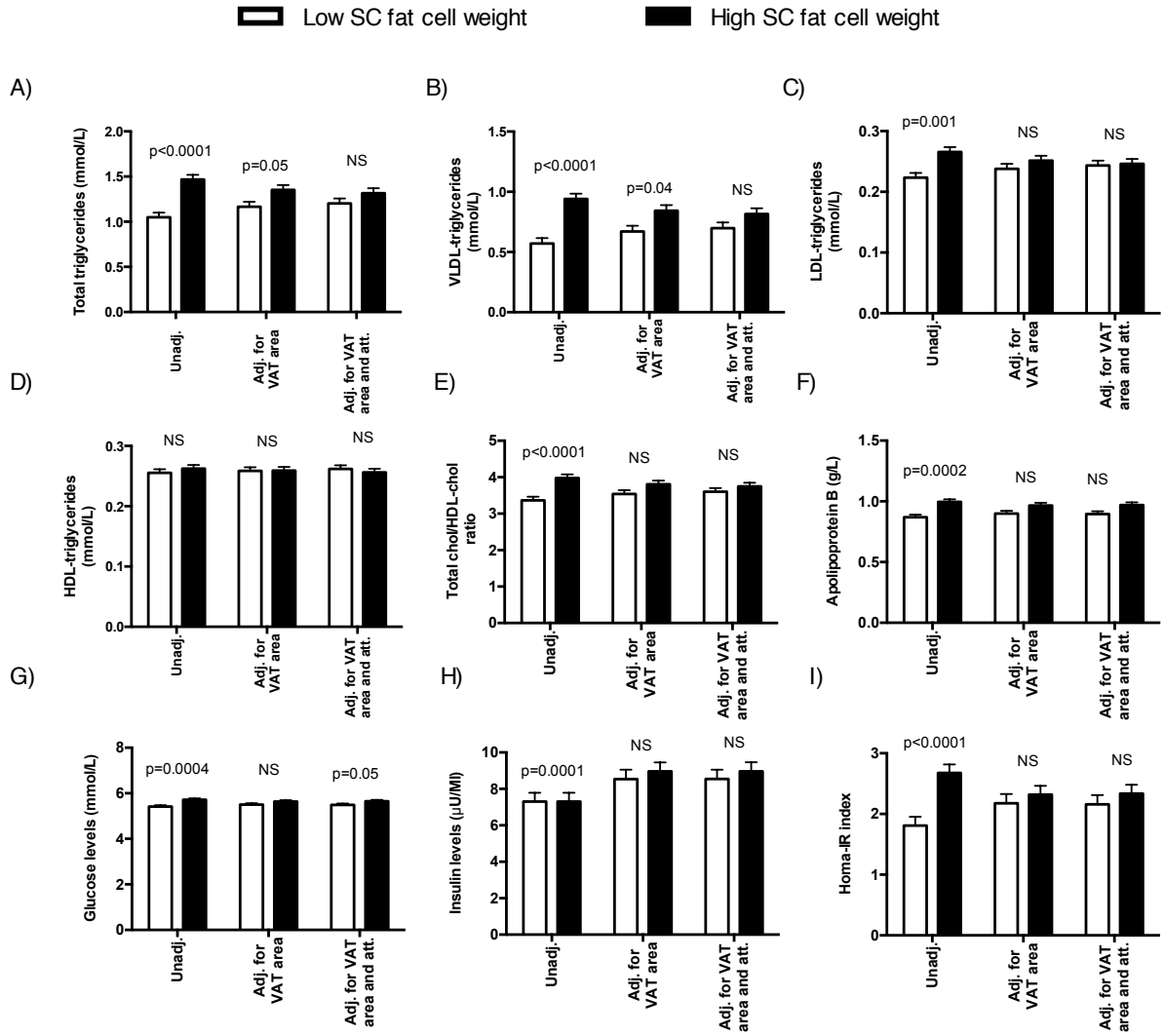
	Adipose tissue attenuation			
	Subcutaneous		Visceral	
	Unadjusted	Adjusted for VAT area	Unadjusted	Adjusted for VAT area
Cholesterol content				
Total	-0.10	0.05	-0.21 **	-0.01
VLDL	-0.25 ***	-0.02	-0.46 ***	-0.22 **
LDL	-0.14 *	0.01	-0.25 ***	-0.04
HDL	0.19 *	0.06	0.35 ***	0.21 **
Cholesterol/HDL-chol	-0.22 **	0.02	-0.46 ***	-0.18 *
Triglyceride content				
Total	-0.20 *	0.04	-0.44 ***	-0.20 *
VLDL	-0.25 ***	-0.01	-0.47 ***	-0.22 **
LDL	-0.16 *	0.01	-0.31 ***	-0.12 †
HDL	-0.07	0.02	-0.18 *	-0.11 †
Apolipoprotein				
Apo B	-0.18 *	0.02	-0.35 ***	-0.09
Apo A1	0.21 **	0.14 *	0.27 ***	0.22 **
Apo B/Apo A1	-0.21 **	-0.01	-0.39 ***	-0.15 *
Glucose homeostasis				
Fasting glucose	-0.21 *	-0.09	-0.31 ***	-0.21 *
Fasting insulin levels	-0.22 **	-0.01	-0.45 ***	-0.23 **
HOMA-IR index	-0.25 *	-0.04	-0.48 ***	-0.27 ***

*** p<0.0001, **p<0.001, *p<0.05, † p<0.08

chol: cholesterol, apo: apolipoprotein, VAT: visceral adipose tissue


A)
r=-0.61, p<0.0001
SAT area (cm2)
SAT attenuation (HUs)
B)
r=-0.80, p<0.0001
VAT area (cm2)
VAT attenuation (HUs)
C)
r=0.54, p<0.0001
SC adipocyte weight (µg lipid/cell)
SC AT area (cm2)
D)
r=0.70, p<0.0001
OM adipocyte weight (µg lipid/cell)
Visceral AT area (cm2)
E)
r=-0.46, p<0.0001
SC adipocyte weight (µg lipid/cell)
SAT attenuation (HUs)
F)
r=-0.67, p<0.0001
OM adipocyte weight (ug lipid/cell)
VAT attenuation (HUs)


Low visceral fat cell weight
High visceral fat cell weight
A)
Total triglycerides (mmol/L)
p<0.0001
p=0.001
NS
B)
VLDL-triglyceride (mmol/L)
p<0.0001
p=0.001
NS
C)
LDL-triglycerides (mmol/L)
p<0.0001
p=0.004
p=0.07
D)
HDL-triglycerides (mmol/L)
p=0.005
NS
NS
E)
Total chol/HDL-chol ratio
p<0.0001
p=0.005
NS
F)
Apolipoprotein B (g/L)
p<0.0001
p=0.02
NS
G)
Glucose levels (mmol/L)
p<0.0001
p=0.007
p=0.05
H)
Insulin levels (µU/Ml)
p<0.0001
p=0.004
NS
I)
Homa-IR index
p<0.0001
p=0.001
p=0.07
Unadj.
Adj. for VAT area
Adj. for VAT area and att.


Low SC fat cell weight
High SC fat cell weight
A)
Total triglycerides (mmol/L)
p<0.0001
p=0.05
NS
B)
VLDL-triglycerides (mmol/L)
p<0.0001
p=0.04
NS
C)
LDL-triglycerides (mmol/L)
p=0.001
NS
NS
D)
HDL-triglycerides (mmol/L)
NS
NS
NS
E)
Total chol/HDL-chol ratio
p<0.0001
NS
NS
F)
Apolipoprotein B (g/L)
p=0.0002
NS
NS
G)
Glucose levels (mmol/L)
p=0.0004
NS
p=0.05
H)
Insulin levels (µU/Ml)
p=0.0001
NS
NS
I)
Homa-IR index
p<0.0001
NS
NS
Unadj.
Adj. for VAT area
Adj. for VAT area and att.