

The association between measurement sites of visceral adipose tissue and cardiovascular risk factors after caloric restriction in obese Korean women

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Abstract

Quantities as well as distributions of adipose tissue (AT) are significantly related to cardiovascular disease (CVD) risk factors and can be altered with caloric restriction. This study investigated which cross-sectional slice location of AT is most strongly correlated with changes in CVD risk factors after caloric restriction in obese Korean women. Thirty-three obese pre-menopausal Korean women (32.4 ± 8.5 yrs, BMI 27.1 ± 2.3 kg/m²) participated in a 12 weeks caloric restriction program. Subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) were measured using computed tomography (CT) scans at the sites of L2-L3, L3-L4, and L4-L5. Fasting serum levels of glucose, insulin, triglyceride, total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), leptin and homeostasis model assessment-insulin resistance (HOMA-IR) were observed. Pearson's partial correlation coefficients were used to assess the relationship between AT measurement sites and changes in CVD risk factors after calorie restriction. When calories were reduced by 350 kcal/day for 12 weeks, body weight (-2.7%), body fat mass (-8.2%), and waist circumference (-5.8%) all decreased ($P < 0.05$). In addition, following caloric restriction, serum levels of glucose (-4.6%), TC (-6.2%), LDL-C (-5.3%), leptin (-17.6%) and HOMA-IR (-18.2%) decreased significantly ($P < 0.05$) as well. Changes in VAT at the level of L3-L4 were significantly greater than those at other abdominal sites, and these changes were correlated with changes in TC ($P < 0.05$), LDL-C ($P < 0.001$), SBP ($P < 0.001$) and HOMA-IR ($P < 0.01$). These results show that VAT at L3-L4 had a stronger correlation with CVD risk factors than with other AT measurement sites after caloric restriction.

Key Words: Caloric restriction, CT, measurement site, VAT, CVD risk factors

Introduction

Obesity, particularly abdominal obesity, is strongly associated with insulin resistance and cardiovascular disease (CVD) risk factors such as waist circumference, hypertriglyceridemia, low high-density lipoprotein (HDL)-cholesterol, hyperglycemia, and hypertension [1,2].

Dietary restriction and lifestyle modification are the most effective methods for reducing body weight. Intervention studies have shown that a reduction in excess abdominal fat is associated with a reduced risk of CVD [3]. Ahn *et al.* [4] reported that diabetes mellitus (DM) patients who restrict calories have significant reductions in body weight as well as VAT, and greater improvements in insulin sensitivity.

Many studies use abdominal fat distribution to identify metabolic

syndrome [5-6] and abdominal fat accumulation is influenced by many factors such as age, menopause, stress, smoking, alcohol intake, socioeconomic status, and genetic factors [7-9]. Visceral adipose tissue (VAT), a component of abdominal fat, seems to be more strongly associated with CVD risk factors than subcutaneous adipose tissue (SAT) [10-12].

There still remains controversy surrounding the question of whether VAT is a superior measure of obesity compared to SAT or other anthropometric measures [10,13-15]. Some investigators reported that these questions are best answered by measuring VAT at several sites [16,17]. Recently, several studies questioned whether a single image can predict total VAT and obesity-related health risks [18,19]. Historically, L4-L5 has been selected as a landmark to assess abdominal adiposity. Single images obtained at the L3-L4 or L4-L5 levels are able to predict abdominal

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obesity equally well [20]. Shen *et al.* [21] reported that measuring VAT at the traditional L4-L5 level might not be the best marker of obesity-related health risks in both men and women. Song *et al.* [22] suggested that VAT is more strongly associated with metabolic syndrome than SAT in the Korean population regardless of the measurement site, and an image located in the upper abdomen (L2-L3 or L3-L4) would be a better predictor. Several studies have suggested that L1-L2 or L2-L3 would be a more suitable predictor of metabolic risk than L4-L5 [23]. Kuk *et al.* [24] also reported that VAT measured at L1-L2 is more strongly associated with total VAT volume and CVD risk factors than VAT measured at L4-L5 in Caucasian men.

None of these studies clarified which location of VAT best assesses CVD risk factors after weight reduction through calorie restriction. Thus, this study evaluated the correlation between cross-sectional slice locations and CVD risk factors in obese Korean women who have lost weight via calorie restriction.

Subjects and Methods

Subjects

Thirty-three non-diabetic obese women participated in the study and all subjects successively completed 12 weeks of a calorie restriction program. Subjects were recruited via postings on a notice board in Kyung Hee Medical Center. They were examined at baseline (T0) and after caloric restriction (T1). Women were included if they were premenopausal, had a BMI > 25 kg/m², had a stable weight (< 2 kg) for at least six months prior to the beginning of the study, did not smoke, and had not participated in any structured physical activities during the previous year. Women were excluded if they had an endocrine disease or other secondary causes of obesity, were pregnant or lactating, had evidence of severe hepatic or renal diseases, or used medication that affects body weight, such as estrogen, oral hypoglycemic agents, and beta-blockers. The protocol and consent forms for the study were approved by the ethics review board of the Kyung Hee Medical Center and each subject provided informed written consent before participation.

Anthropometric parameters measures

Anthropometric measurements were taken at baseline and at 12 weeks, with the subjects in light clothing and without shoes. Height and weight were recorded using an automatic height-weight scale, to the nearest 0.1 cm and 0.1 kg. Body mass index (BMI) was calculated by dividing weight (kg) by height squared (m²). Percent body fat and lean body mass were measured by bioimpedance analysis (Inbody 3.0, Biospace, and Seoul, Korea). Mid-upper arm, thigh, waist and hip circumference were measured by a flexible measuring tape. Waist circumference was measured between the costal inferior border and the iliac crest. Hip circum-

ference was measured at the widest point of the hip. Waist-to-hip ratio (WHR) was calculated by dividing waist circumference by hip circumference. Skinfold thickness was measured with a skinfold caliper at the triceps and thigh.

Blood sampling and biochemical assessment

At baseline and at 12 weeks, blood pressure was measured on the left arm with an automated blood pressure monitor after the subjects were at rest for 10-15 min. Blood samples were taken after a 10-h overnight fast and were centrifuged to obtain plasma, which was stored at -70°C until analyzed. Plasma glucose was measured by a glucose oxidase method and total cholesterol, high density lipoprotein cholesterol (HDL-C), triglyceride levels were measured via enzymatic procedures using an auto analyzer (Bayer, USA). Low density lipoprotein cholesterol (LDL-C) was calculated by the Friedewald equation [25]. Insulin was analyzed with a Coat-A-Count Insulin kit (DPC, Diagnostic products Cor.) and leptin was analyzed with a human leptin RIA kit (Linco Research, St Charles, MO, USA). The homeostasis model assessment (HOMA) was used to calculate insulin resistance (IR) as $HOMA-IR = [\text{fasting insulin } (\mu\text{IU/mL}) \times \text{fasting blood glucose (mg/dL)}] / 18 / 22.5$

Measurement of abdominal fat distribution by computed tomography (CT)

A single-slice CT scan taken at the level of L2-L3, L3-L4 and L4-L5 was performed using a PQ6000 scanner (General Electric Medical Systems, Milwaukee, WI) to measure visceral and subcutaneous abdominal adipose tissue areas at baseline and at 12 weeks (Hounsfield units (HU): -190 to -30) (Ryan and Nicklas, 1999). The subcutaneous abdominal adipose tissue (SAT) area was calculated by subtracting the visceral abdominal adipose tissue (VAT) area from the total adipose tissue (TAT) area. The visceral-to-subcutaneous fat ratio (VSR) was calculated by dividing VAT by SAT.

Caloric restriction

Subjects were placed on a low calorie diet of 1,200 kcal/day. A trained dietician met with the subjects during the baseline visit, the 1st week, 2nd week, 4th week, 8th week and 12th week. Subjects were provided with dietary information including the components of a balanced diet, the importance of food choice, and instructions on low-fat cooking methods. All subjects were instructed to complete a three day dietary record (two weekdays and one weekend day) before the start of the study and at the end of the study. Dietary records kept during the study were used to reinforce dietary advice and strengthen compliance. Restriction of alcohol consumption either by reducing the frequency or amount of alcohol intake was strongly recommended to all subjects. A nutrient analysis was quantified using a computer

aided nutritional analysis program (CAN pro, Korean Nutrition Society, and Seoul, Korea).

Statistical analysis

The results were presented as mean \pm SD. Changes in anthropometric measurements, abdominal fat distribution and cardiovascular risk factors before and after the caloric restriction were analyzed by paired t-test. Changes in diet before and after caloric restriction were analyzed by a Student t-test. Correlations among anthropometric measures, abdominal fat distribution, BP and cardiovascular risk factors were analyzed by Pearson's correlation coefficients. The three sites (L2-L3, L3-L4 and L4-L5) were compared by an ANOVA model with a two-sided test level of 0.05 and post-hoc analyses were performed with Duncan's test. Statistical analyses were performed with SAS 9.0 for Windows (SAS institute Inc. Cary, NC). A *P*-value < 0.05 was considered statistically significant.

Results

Baseline characteristics and changes after caloric restriction

Thirty-three obese women enrolled in the study. Basal characteristics and changes in daily intake of nutrients are presented in Table 1. The mean age was 32.4 ± 8.5 yrs (range, 20 to 46 years). All study subjects had a BMI higher than 25 kg/m^2 and the mean BMI of the subjects was $27.1 \pm 2.3 \text{ kg/m}^2$. Weight, BMI and systolic blood pressure decreased significantly after caloric restriction ($P < 0.001$, $P < 0.05$), whereas diastolic blood pressure did not change.

Changes in daily intake of nutrients after caloric restriction

Mean daily energy intake at baseline and after caloric restriction was $1,776.2 \pm 338.6$ and $1,451.5 \pm 139.4$ kcal, respectively. Intake of energy, carbohydrate, fat and cholesterol decreased significantly after caloric restriction ($P < 0.001$, $P < 0.001$, $P < 0.05$ and $P < 0.01$, respectively), whereas protein intake did not change. Daily energy intake decreased by 324.7 ± 438.9 kcal and daily carbohydrate intake decreased by 51.6 ± 47.9 g/d.

Changes in anthropometric measures after caloric restriction

Changes in anthropometric measures and blood parameters are presented in Table 2. Anthropometric measures (body fat mass, waist circumference, hip circumference, waist-to-hip ratio, TSF, MAC, Thigh SF and TICR) decreased significantly after the caloric restriction ($P < 0.001$), whereas lean body mass did not change. Body fat and waist circumference decreased by 2.3 ± 2.5 kg and 5.4 ± 3.3 cm, respectively.

Changes in blood parameters after caloric restriction

Plasma levels of total cholesterol and LDL-cholesterol decreased significantly after caloric restriction ($P < 0.05$), whereas levels of triglyceride, HDL-cholesterol and atherogenic index did not change. Total cholesterol and LDL-cholesterol decreased by 12.6 ± 26.8 mg/dL and 7.7 ± 20.6 mg/dL, respectively. Plasma levels of glucose, insulin, HOMA-IR and leptin also decreased significantly after caloric restriction ($P < 0.01$). Glucose, insulin, HOMA-IR score and leptin decreased by 4.7 ± 8.8 mg/dL, 3.7 ± 10.6 $\mu\text{IU/mL}$, 0.5 ± 1.1 , and 3.1 ± 5.3 ng/mL, respectively.

Changes in abdominal fat at the levels of L2-L3, L3-L4 and L4-L5 after caloric restriction

After caloric restriction, SAT and VAT decreased significantly

Table 1. Changes in subjects' general characteristics and daily intake of nutrients after caloric restriction

	T0	T1	Δ T1-T0	% Δ
General characteristics				
Age (yrs)		32.4 ± 8.5 (20-46)		
Height (cm)		160.9 ± 5.1 (152.7-16.2)		
Weight (kg)	70.2 ± 8.2	$68.15 \pm 6.4^{***}$	-2.0 ± 2.4	-2.7 ± 3.5
BMI (kg/m^2)	27.1 ± 2.3	$26.3 \pm 2.3^{***}$	-0.7 ± 0.9	-2.7 ± 3.6
SBP (mm/Hg)	114.6 ± 14.2	$107.8 \pm 22.9^*$	-3.4 ± 9.6	-5.8 ± 17.3
DBP (mm/Hg)	76.8 ± 11.1	73.3 ± 9.4	-3.5 ± 9.0	-4.6 ± 10.4
Changes in daily intake of nutrients				
Energy (kcal/d)	$1,776.2 \pm 338.6$	$1,451.5 \pm 139.4^{***}$	-324.7 ± 438.9	-15.0 ± 20.6
Carbohydrate (g/d)	261.8 ± 42.2	$211.9 \pm 21.3^{***}$	-51.6 ± 47.9	-17.9 ± 14.3
Protein (g/d)	66.6 ± 18.6	61.8 ± 7.5	-5.1 ± 23.8	-2.2 ± 24.3
Fat (g/d)	52.9 ± 17.9	$41.9 \pm 9.9^*$	-10.9 ± 19.9	-11.4 ± 37.6
Cholesterol (mg/d)	284.7 ± 118.6	$219.8 \pm 55.6^{**}$	-66.3 ± 135.8	-10.2 ± 46.5
CHO: Pro: Fat (%)	$58.7 : 14.7 : 26.6$	$58.0 : 17.0 : 25.0$	-	-

Δ , amount of change; T0, at baseline; T1, after caloric restriction; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure. Significantly different at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ by paired t-test or student's t-test

Table 2. Changes in subjects' anthropometric measures and blood parameters after caloric restriction

	T0	T1	ΔT1-T0	% Δ
Changes in anthropometric measures				
Fat (kg)	26.5 ± 5.1	24.2 ± 4.6***	-2.3 ± 2.5	-8.2 ± 9.6
LBM (kg)	43.6 ± 3.3	43.9 ± 3.4	0.3 ± 1.9	0.92 ± 4.3
Waist (cm)	90.3 ± 5.5	84.9 ± 5.7***	-5.4 ± 3.3	-5.8 ± 3.5
Hip (cm)	101.6 ± 4.5	99.1 ± 4.5***	-2.5 ± 2.0	-2.3 ± 2.0
W/H ratio	0.89 ± 0.05	0.86 ± 0.05***	-0.03 ± 0.03	-3.6 ± 3.0
TSF (mm)	32.0 ± 4.2	26.7 ± 4.1***	-5.3 ± 2.7	-15.7 ± 8.3
MAC (mm)	31.8 ± 2.2	30.6 ± 2.1***	-1.2 ± 1.0	-3.7 ± 3.2
Thigh SF (mm)	38.3 ± 8.7	33.4 ± 7.4***	-4.3 ± 5.1	-11.6 ± 13.7
TCIR (mm)	54.2 ± 3.9	50.8 ± 3.4***	-3.3 ± 2.9	-5.9 ± 4.9
Changes in blood parameters				
TG (mg/dL)	94.8 ± 42.5	88.8 ± 40.4	-6.0 ± 27.0	-4.8 ± 27.9
Total-C (mg/dL)	182.3 ± 33.0	169.7 ± 31.6*	-12.6 ± 26.8	-6.2 ± 14.9
LDL-C (mg/dL)	111.4 ± 29.4	103.7 ± 27.0*	-7.7 ± 20.6	-5.3 ± 18.8
HDL-C (mg/dL)	49.9 ± 8.8	48.2 ± 9.0	-1.4 ± 8.4	-3.2 ± 16.1
Atherogenic index	2.7 ± 0.7	2.5 ± 0.7	-0.12 ± 0.5	-1.89 ± 21.3
Glucose (mg/dL)	90.1 ± 8.3	85.3 ± 7.0**	-4.7 ± 8.8	-4.6 ± 9.9
Insulin (μIU/mL)	9.8 ± 15.9	6.2 ± 6.4**	-3.7 ± 10.6	-17.5 ± 50.4
HOMA-IR score	1.7 ± 1.3	1.1 ± 0.8**	-0.5 ± 1.1	-18.2 ± 52.8
Leptin (ng/mL)	14.6 ± 6.1	11.4 ± 6.0**	-3.1 ± 5.3	-17.6 ± 33.8

Δ, amount of change; T0, at baseline; T1, after caloric restriction; Fat, fat mass; LBM, lean body mass; Waist, waist circumference; Hip, hip circumference; W/H, Waist/Hip; TSF, Triceps skinfold thickness; MAC, Mid-arm circumference; Thigh SF, Thigh skinfold thickness; TCIR, Thigh circumference; TG, triglyceride; Total-C, total cholesterol; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment-insulin resistance; Atherogenic index = total cholesterol - HDL-cholesterol / HDL-cholesterol
Significantly different at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ by paired t-test

at all sites with the exception of VAT at L4-L5 (Table 3). The percent change in VAT at L3-4 was significant compared to other abdominal measurement sites (10% vs. 3-7%).

VSR at L2-L3, L3-L4, and L4-L5 was 0.47 ± 0.21 , 0.43 ± 0.24 and 0.30 ± 0.14 , respectively. VSR did not change after caloric restriction.

Table 4. Partial correlation coefficients adjusted for age describing the association between changes in abdominal fat and CVD risk factors

	ΔWaist	ΔTG	ΔTotal-C	ΔLDL-C	ΔHDL-C	ΔSBP	ΔDPB	ΔHOMA-IR
SAT								
ΔL ₂₋₃	0.2548	-0.1088	-0.2147	-0.0803	-0.3327	-0.0695	0.0439	-0.0900
ΔL ₃₋₄	0.2851	0.0153	-0.2339	-0.1565	-0.3354	-0.1075	0.0927	-0.1716
ΔL ₄₋₅	0.1981	-0.1128	-0.0289	0.0670	-0.3103	0.1604	0.0200	-0.0332
VAT								
ΔL ₂₋₃	-0.1020	0.1625	-0.0909	0.0729	-0.3248	0.5077**	-0.0560	0.0209
ΔL ₃₋₄	0.0095	-0.2555	0.3838*	0.5807***	0.1316	0.5761***	0.2469	0.4928**
ΔL ₄₋₅	0.2220	-0.2079	0.2088	0.1874	0.0043	0.4964**	0.2578	0.1208
VSR								
ΔL ₂₋₃	-0.2388	0.2954	-0.0604	-0.0359	-0.1243	0.3109	-0.0911	-0.0919
ΔL ₃₋₄	-0.0529	-0.2910	0.3743*	0.5222**	0.2680	0.4418**	0.1861	0.5272**
ΔL ₄₋₅	0.1208	0.1471	0.1169	0.0166	0.2074	0.4638**	0.2762	0.0003

Δ, amount of change; T0, at baseline; T1, after caloric restriction; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; VSR, VAT to SAT ratio; ΔL₂₋₃, Change area of lumbar spine 2-3; ΔL₃₋₄, Change area of lumbar spine 3-4; ΔL₄₋₅, Change area of lumbar spine 4-5; TG, triglyceride; Total-C, total cholesterol; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; SBP, systolic blood pressure; DPB, diastolic blood pressure; HOMA-IR, homeostasis model assessment of insulin resistance
Significantly different at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ by Pearson's correlation coefficients.

Table 3. Changes in abdominal fat distribution as measured by computer-assisted tomography after caloric restriction

	T0	T1	ΔT1-T0	% Δ
SAT area (cm ²)				
L ₂₋₃	212.3 ± 40.5 ^c	202.7 ± 45.8 ^{b*}	-9.7 ± 25.1	-4.6 ± 11.9 ^a
L ₃₋₄	242.5 ± 61.8 ^b	231.0 ± 68.2 ^{b*}	-11.6 ± 30.6	-4.8 ± 13.1 ^a
L ₄₋₅	291.1 ± 73.2 ^a	273.6 ± 77.3 ^{a*}	-17.5 ± 39.4	-5.7 ± 15.6 ^a
VAT area (cm ²)				
L ₂₋₃	97.1 ± 44.7 ^{ab}	91.3 ± 41.7 ^{a*}	-5.9 ± 14.7	-7.3 ± 17.7 ^{ab}
L ₃₋₄	99.4 ± 37.5 ^a	86.4 ± 38.6 ^{a***}	-11.2 ± 17.3	-10.3 ± 16.6 ^a
L ₄₋₅	79.6 ± 28.3 ^b	76.9 ± 29.1 ^a	-2.7 ± 12.6	-3.7 ± 17.3 ^b
VSR				
L ₂₋₃	0.47 ± 0.21 ^a	0.47 ± 0.24 ^a	0.00 ± 0.09	-0.89 ± 20.8 ^a
L ₃₋₄	0.43 ± 0.24 ^a	0.41 ± 0.23 ^a	-0.02 ± 0.10	-4.55 ± 23.3 ^a
L ₄₋₅	0.298 ± 0.14 ^b	0.30 ± 0.15 ^b	0.00 ± 0.06	0.71 ± 23.7 ^a

Δ, amount of change; T0, at baseline; T1, after caloric restriction; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; VSR, VAT to SAT ratio; L₂₋₃, Area of lumbar spine 2-3; L₃₋₄, Area of lumbar spine 3-4; L₄₋₅, Area of lumbar spine 4-5

Significantly different at * $P < 0.05$, *** $P < 0.001$ by paired t-test

^{a,b,c} Significantly different between CT sites by ANOVA

Correlation between changes in abdominal fat and CVD risk factors after caloric restriction

Changes in SAT, regardless of the measurement site, were not correlated with changes in CVD risk factors (Table 4). However, changes in VAT measures at L2-L3, L3-L4 and L4-L5 were positively correlated with the changes in SBP. In addition, VAT at L3-L4 was positively correlated with changes in total-cholesterol, LDL-cholesterol and HOMA-IR. The correlation between changes in CVD risk factors and VAT at L3-L4 was stronger than the correlation between changes in CVD risk factors and VAT at L2-L3 and L4-L5. The correlation between changes in VSR and CVD risk factors was similar to that of VAT and CVD risk factors.

Discussion

This study examined the relationship between anthropometric measures, abdominal fat distribution at the levels of L2-L3, L3-L4 and L4-L5, and CVD risk factors. The results show that food intake, anthropometric measures, SBP, % fat, total-C, LDL-C, HOMA-IR, leptin, SAT and VAT decreased significantly after calorie restriction. In addition, after calorie restriction, changes in VAT at L3-L4 were more strongly correlated with CVD risk factors than changes in VAT at L2-L3 and L4-L5. However, changes in SAT, regardless of the measurement site, were not correlated with changes in CVD risk factors.

Weinsier *et al.* [26] and Kelley *et al.* [27] suggested that a low energy diet may rapidly reduce CVD risk factors. Furthermore, Wing *et al.* [28] reported that reducing energy intake by 400 kcal/day improved fasting glucose levels and insulin sensitivity. In this study, the subjects reduced their calorie intake primarily by reducing carbohydrate and fat intake. This caloric restriction was part of a program that included teaching subjects how to eat a balanced diet, make healthy food choices, and use low fat cooking methods. During the study subjects tended to maintain their lean body mass, perhaps due to the consistent intake of protein and a generally well-balanced diet during the period of caloric restriction.

In this study, a reduction in visceral fat was correlated with improvements in insulin resistance and systolic blood pressure. This suggests that a well-balanced, low calorie diet may reduce abdominal obesity, which in turn might improve selected CVD risk factors.

Paré *et al.* [20] showed that the decrease in cross-sectional areas of VAT was higher than a decrease in the SAT area after weight loss. Furthermore, the decrease in VAT at L4-L5 was higher than at L2-L3 (19% vs. 15%). Conversely, Ross and Rissanen [29] reported that the relative loss in the VAT area 15 cm above L4-L5 via diet- and exercise-induced weight reduction was significantly larger than the loss in the VAT area in other abdominal slice areas. In this study, after a 350 kcal/day caloric restriction, SAT decreased by 4-6% and VAT decreased by 4-10% compared to their initial values at L2-L3, L3-L4 and L4-L5. However VSR did not change. The percent change in VAT at L3-L4 was greater than other abdominal sites (10% vs. 3-7%), illustrating that VAT at L3-L4 is strongly influenced by caloric restriction compared to VAT at L2-L3 or L4-L5.

Previous studies have demonstrated that VAT might be more strongly associated with CVD risk factors compared to SAT [23,24]. On the other hand, several investigators have reported that SAT contributes to the development of metabolic syndrome (MS) although the correlation of SAT with MS is inconsistent and varies according to the measurement site [3]. Several studies found that the L4-L5 location is the most frequently used single-slice site to assess abdominal adiposity [19-21] whereas other studies reported that the L2-L3 site might be more appropriate [30,31]. Kuk *et al.* [24] reported that VAT was more

strongly associated with MS than SAT, independent of the measurement site.

In previous studies, changes in VAT and SAT regardless of the measurement site were generally not related to corresponding changes in a number of MS risk factors [5]. In this study, however, changes in VAT at L3-L4 were significantly associated with changes in TC, LDL-C, SBP and HOMA-IR. Changes in VAT at L3-L4 were more strongly correlated with CVD risk factors than those at L2-L3 and L4-L5. VAT based on a single-slice CT scan was somewhat site-specific, and the level of VAT at L3-L4 was more strongly associated with CVD risk factors than L2-L3 and L4-L5 in obese Korean women. These results suggest that an image located in the upper abdominal region L3-L4 rather than the L4-L5 level would be a better predictor of the relationship between VAT and CVD risk factors.

This study has several limitations. First, the sample size was small, which may lessen the significance of the results. The authors attempted to enroll more participants, but this was difficult due to the expensive cost of CT scans. Second, the physical activity levels of the participants were not noted. Therefore, it was not possible to distinguish the additive effects of caloric restriction and increased levels of exercise on the results. Third, the subjects were premenopausal women. Several cross-sectional studies in Western populations have reported that postmenopausal women accumulate more VAT than premenopausal women [6,32] and that VAT increases with age. The results of this study only apply to premenopausal Asian obese women. These limitations should be addressed in future research.

This study found that changes in VAT at the L3-L4 site had a stronger correlation with CVD risk factors than with other AT measurement sites after caloric restriction in obese Korean women.

References

1. Ohkawara K, Nakata Y, Numao S, Sasai H, Katayama Y, Matsuo T, Okura T, Tanaka K. Response of coronary heart disease risk factors to changes in body fat during diet-induced weight reduction in Japanese obese men: a pilot study. *Ann Nutr Metab* 2010; 56:1-8.
2. Després JP, Lemieux I, Bergeron J, Pibarot P, Mathieu P, Larose E, Rodés-Cabau J, Bertrand OF, Poirier P. Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk. *Arterioscler Thromb Vasc Biol* 2008;28:1039-49.
3. Ahn HJ, Cho YO, Kwon HR, Ku YH, Koo BK, Han KA, Min KW. The effects of low-calorie diets on abdominal visceral fat, muscle mass, and dietary quality in obese type 2 diabetic subjects. *Korean Diabetes J* 2009;33:526-36.
4. Ahn HJ, Han KA, Jang JY, Lee JH, Park KS, Min KW. Small rice bowl-based meal plan for energy and macronutrient intake in Korean men with type 2 diabetes: a pilot study. *Diabetes Metab J* 2011;35:273-81.
5. Lee S, Kuk JL, Kim Y, Arslanian SA. Measurement site of visceral adipose tissue and prediction of metabolic syndrome in

- youth. *Pediatr Diabetes* 2011;12:250-7.
6. Goodpaster BH, Krishnaswami S, Harris TB, Katsiaras A, Kritchevsky SB, Simonsick EM, Nevitt M, Holvoet P, Newman AB. Obesity, regional body fat distribution, and the metabolic syndrome in older men and women. *Arch Intern Med* 2005;165:777-83.
 7. Toth MJ, Tchernof A, Sites CK, Poehlman ET. Effect of menopausal status on body composition and abdominal fat distribution. *Int J Obes Relat Metab Disord* 2000;24:226-31.
 8. Kotani K, Tokunaga K, Fujioka S, Kobatake T, Keno Y, Yoshida S, Shimomura I, Tarui S, Matsuzawa Y. Sexual dimorphism of age-related changes in whole-body fat distribution in the obese. *Int J Obes Relat Metab Disord* 1994;18:207-12.
 9. Ferrara CM, Goldberg AP, Nicklas BJ, Sorkin JD, Ryan AS. Sex differences in insulin action and body fat distribution in overweight and obese middle-aged and older men and women. *Appl Physiol Nutr Metab* 2008;33:784-90.
 10. Tulloch-Reid MK, Hanson RL, Sebring NG, Reynolds JC, Premkumar A, Genovese DJ, Sumner AE. Both subcutaneous and visceral adipose tissue correlate highly with insulin resistance in African Americans. *Obes Res* 2004;12:1352-9.
 11. Schautz B, Later W, Heller M, Müller MJ, Bösny-Westphal A. Associations between breast adipose tissue, body fat distribution and cardiometabolic risk in women: cross-sectional data and weight-loss intervention. *Eur J Clin Nutr* 2011;65:784-90.
 12. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, Liu CY, Vasan RS, Murabito JM, Meigs JB, Cupples LA, D'Agostino RB Sr, O'Donnell CJ. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* 2007;116:39-48.
 13. Lovejoy JC, de la Bretonne JA, Klemperer M, Tulley R. Abdominal fat distribution and metabolic risk factors: effects of race. *Metabolism* 1996;45:1119-24.
 14. Abate N, Garg A, Peshock RM, Stray-Gundersen J, Grundy SM. Relationships of generalized and regional adiposity to insulin sensitivity in men. *J Clin Invest* 1995;96:88-98.
 15. Goodpaster BH, Thaete FL, Simoneau JA, Kelley DE. Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. *Diabetes* 1997;46:1579-85.
 16. Ellis KJ, Grund B, Visnegarwala F, Thackeray L, Miller CG, Chesson CE, El-Sadr W, Carr A; Strategies for Management of Anti-Retroviral Therapy (SMART) Study Group. Visceral and subcutaneous adiposity measurements in adults: influence of measurement site. *Obesity (Silver Spring)* 2007;15:1441-7.
 17. Lee S, Janssen I, Ross R. Interindividual variation in abdominal subcutaneous and visceral adipose tissue: influence of measurement site. *J Appl Physiol* 2004;97:948-54.
 18. Kvist H, Sjöström L, Tyén U. Adipose tissue volume determinations in women by computed tomography: technical considerations. *Int J Obes* 1986;10:53-67.
 19. Demerath EW, Shen W, Lee M, Choh AC, Czerwinski SA, Siervogel RM, Towne B. Approximation of total visceral adipose tissue with a single magnetic resonance image. *Am J Clin Nutr* 2007;85:362-8.
 20. Paré A, Dumont M, Lemieux I, Brochu M, Alméras N, Lemieux S, Prud'homme D, Després JP. Is the relationship between adipose tissue and waist girth altered by weight loss in obese men? *Obes Res* 2001;9:526-34.
 21. Shen W, Punyanitya M, Chen J, Gallagher D, Albu J, Pi-Sunyer X, Lewis CE, Grunfeld C, Heymsfield SB, Heshka S. Visceral adipose tissue: relationships between single slice areas at different locations and obesity-related health risks. *Int J Obes (Lond)* 2007;31:763-9.
 22. Song SW, Hwang SS, Shin JH, Kang SG, Cho JH, Nam KM, Kim SH. Relationships between visceral adipose tissue measurement site and the metabolic syndrome in the Korean population. *Obes Res Clin Pract* 2010;4:e253-60.
 23. Bray GA, Jablonski KA, Fujimoto WY, Barrett-Connor E, Haffner S, Hanson RL, Hill JO, Hubbard V, Kriska A, Stamm E, Pi-Sunyer FX; Diabetes Prevention Program Research Group. Relation of central adiposity and body mass index to the development of diabetes in the Diabetes Prevention Program. *Am J Clin Nutr* 2008;87:1212-8.
 24. Kuk JL, Church TS, Blair SN, Ross R. Does measurement site for visceral and abdominal subcutaneous adipose tissue alter associations with the metabolic syndrome? *Diabetes Care* 2006;29:679-84.
 25. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972;18:499-502.
 26. Weinsier RL, James LD, Darnell BE, Wooldridge NH, Birch R, Hunter GR, Bartolucci AA. Lipid and insulin concentrations in obese postmenopausal women: separate effects of energy restriction and weight loss. *Am J Clin Nutr* 1992;56:44-9.
 27. Kelley DE, Wing R, Buonocore C, Sturis J, Polonsky K, Fitzsimmons M. Relative effects of calorie restriction and weight loss in noninsulin-dependent diabetes mellitus. *J Clin Endocrinol Metab* 1993;77:1287-93.
 28. Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN. Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. *Diabetes Care* 1994;17:30-6.
 29. Ross R, Rissanen J. Mobilization of visceral and subcutaneous adipose tissue in response to energy restriction and exercise. *Am J Clin Nutr* 1994;60:695-703.
 30. Han TS, Kelly IE, Walsh K, Greene RM, Lean ME. Relationship between volumes and areas from single transverse scans of intra-abdominal fat measured by magnetic resonance imaging. *Int J Obes Relat Metab Disord* 1997;21:1161-6.
 31. Abate N, Garg A, Coleman R, Grundy SM, Peshock RM. Prediction of total subcutaneous abdominal, intraperitoneal, and retroperitoneal adipose tissue masses in men by a single axial magnetic resonance imaging slice. *Am J Clin Nutr* 1997;65:403-8.
 32. Ross R, Freeman J, Hudson R, Janssen I. Abdominal obesity, muscle composition, and insulin resistance in premenopausal women. *J Clin Endocrinol Metab* 2002;87:5044-51.