*Am J Physiol Endocrinol Metab* 282: E657–E663, 2002;
10.1152/ajpendo.00469.2001.

# Abdominal adiposity and insulin resistance in obese men

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Received 17 October 2001; accepted in final form 8 November 2001

**Ross, Robert, James Aru, Jennifer Freeman, Robert Hudson, and Ian Janssen.** Abdominal adiposity and insulin resistance in obese men. *Am J Physiol Endocrinol Metab* 282: E657–E663, 2002; 10.1152/ajpendo.00469.2001.—We examined the independent relationships among various visceral and abdominal subcutaneous adipose tissue (AT) depots, glucose tolerance, and insulin sensitivity in 89 obese men. Measurements included an oral glucose tolerance test (OGTT), glucose disposal by euglycemic clamp, and abdominal and nonabdominal (e.g., peripheral) AT by magnetic resonance imaging (MRI). OGTT glucose and glucose disposal rates were related ($P < 0.05$) to visceral AT ($r = 0.50$ and $-0.41$, respectively). These observations remained significant ($P < 0.05$) after control for nonabdominal and abdominal subcutaneous AT, and maximal $O_2$ consumption ($\dot{V}O_{2\,max}$). Abdominal subcutaneous AT was not a significant correlate ($P > 0.05$) of any metabolic variable after control for nonabdominal and visceral AT and $\dot{V}O_{2\,max}$. Division of abdominal subcutaneous AT into deep and superficial depots and visceral AT into intra- and extraperitoneal AT depots did not alter the observed relationships. Further analysis matched two groups of men for abdominal subcutaneous AT but also for low and high visceral AT. Men with high visceral AT had higher OGTT glucose values and lower glucose disposal rates compared with those with low visceral AT values ($P < 0.05$). A similar analysis performed on two groups of men matched for visceral AT but also for high and low abdominal subcutaneous AT revealed no statistically different values for any metabolic variable ($P > 0.10$). In conclusion, visceral AT alone is a strong correlate of insulin resistance independent of nonabdominal and abdominal subcutaneous AT and cardiovascular fitness. Subdivision of visceral and abdominal subcutaneous AT by MRI did not provide additional insight into the relationship between abdominal obesity and metabolic risk in obese men.

subcutaneous adipose tissue; insulin sensitivity; visceral adipose tissue

DEBATE CONTINUES regarding the independent contribution of abdominal subcutaneous and visceral adipose tissue (AT) toward the etiology of insulin resistance. Whereas some researchers report that visceral AT is the stronger correlate (11, 13, 15, 32), others find that abdominal subcutaneous AT is largely responsible for the established association between abdominal obesity and insulin resistance (1, 2, 19). It has recently been suggested that the discrepancies may be resolved by subdividing abdominal subcutaneous AT according to differences in metabolic characteristics (20, 26, 38, 40). Abdominal subcutaneous AT can be subdivided into superficial and deep compartments by use of the fascia superficialis. The rationale for this division presumes that adipocytes within the deep compartment are more metabolically active compared with superficial adipocytes (12, 25). On the assumption that the liberation of nonesterified fatty acids adversely effects insulin action (29, 37), it follows that the deep compartment would be the stronger predictor of insulin resistance. Indeed, Kelley et al. (20) have shown that deep, but not superficial, subcutaneous AT is strongly related to insulin resistance in a cohort of lean and obese men and women. Toth et al. (40) report similar observations in lean, premenopausal women. These findings confirm the earlier observation of Misra et al. (26), who report that posterior subcutaneous fat (analogous to deep subcutaneous fat in men) is associated with insulin resistance independently of visceral AT.

The subdivision of abdominal subcutaneous AT on the basis of metabolic characteristics is analogous to the partitioning of visceral AT into intraperitoneal and extraperitoneal depots on the basis of anatomical considerations. Subdivision of visceral AT is based on the premise that nonesterified fatty acids from intraperitoneal fat alone (omental and mesenteric adipocytes) are delivered directly to the liver and thus mediate hepatic insulin resistance: the so-called "portal theory" (9, 10). Thus isolation of intraperitoneal AT may improve upon the relationship between visceral AT per se and insulin resistance. To our knowledge, no study has simultaneously examined whether subdivision of abdominal subcutaneous and visceral AT improves the ability of either depot to predict insulin resistance.

Discrepancies regarding the singular importance of abdominal subcutaneous and visceral AT may also be explained by differences in the cohorts studied (11). In some of the reports indicating that abdominal subcutaneous AT independently predicts insulin resistance, the cohort examined combines men and women with wide variation in body composition and insulin sensitivity (19, 20). In other studies, wherein the investiga-

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tors studied homogeneous groups of lean men (2) or lean women (40), the accumulation of visceral AT is below the threshold thought to be associated with distinct elevations in metabolic risk factors (14). By comparison, studies that found visceral AT the stronger predictor of insulin resistance are characterized by homogeneous populations of men or women with marked elevation in visceral AT (11, 13, 15, 32). However, in most of the these studies, abdominal subcutaneous AT was not subdivided into deep and superficial depots (13, 15, 32).

The primary purpose of this study, therefore, was to determine whether subdivision of abdominal subcutaneous and visceral AT strengthens the association between insulin resistance and either subcutaneous or visceral AT alone. To test this objective we studied a cohort of abdominally obese men at increased metabolic risk. We hypothesized that, in this homogenous group of men characterized by marked elevation in visceral obesity, visceral AT would be a strong correlate of insulin resistance independent of abdominal (anterior and posterior) and nonabdominal subcutaneous AT.

## METHODS

*Subjects.* The subjects consisted of 89 males who were initially recruited to participate in two weight loss studies (32, 35). The baseline data from these studies have not been reported previously. Inclusion criteria required that the subjects had a body mass index [BMI (weight in kg/height in $m^2$)] $>27$, a waist-to-hip ratio $>0.95$, a stable weight ($\pm 2$ kg) for 6 mo before the beginning of the study, consumption on average of less than two alcoholic beverages per day, and being nonsmokers and sedentary. All subjects were nondiabetic, as confirmed by plasma glucose levels in a fasting state and 2 h after a 75-g oral glucose tolerance test (OGTT) (4). All subjects gave their fully informed and written consent to participate in the study, which was conducted in accordance with the ethical guidelines set by Queen's University.

*Anthropometric variables.* Body mass was measured on a balance scale to the nearest 0.1 kg with the subjects dressed in light clothing. Standing height was measured to the nearest 0.1 cm with a wall-mounted stadiometer. Circumference measurements were taken, with the subjects in a standing position, at the level of the last rib and hip by means of standard procedures (21).

*Tissue measurement by magnetic resonance imaging.* Whole body (41 images) magnetic resonance imaging (MRI) data were obtained with a GE 1.5-Tesla scanner (GE, Milwaukee, WI) with the use of an established protocol (35). The MRI data were transferred to a stand-alone work station (Silicon Graphics, Mountain View, CA) for analysis using special software (TomoVision, Montreal, QC, Canada) as described elsewhere (27, 35). Total AT (subcutaneous + visceral + intrapelvic + intrathoracic + intermuscular) and skeletal muscle were determined using all 41 images. Total (e.g., volume or mass) visceral and abdominal subcutaneous AT were calculated using the five images extending from 5 cm below to 15 cm above L4-L5. Nonabdominal AT includes all AT other than abdominal subcutaneous and visceral AT (92% of nonabdominal AT is peripheral subcutaneous AT; data not shown). Abdominal subcutaneous AT was divided into anterior and posterior compartments by drawing a perpendicular line along the anterior edge of the vertebral bodies for all five abdominal MRI images (Fig. 1). Visceral AT was subdivided into intraperitoneal and extraperitoneal compartments by use of a method previously described (33, 36) and as illustrated in Fig. 1. AT volume units (l) were converted to mass units (kg) by multiplying the volumes by the assumed constant densities of 0.92 for adipose tissue and 1.04 for skeletal muscle (39).

*Glucose tolerance.* A 2-h, 75-g OGTT was administered the morning after an overnight fast. Blood samples were collected from the antecubital vein at 0, 60, and 120 min. Glucose was measured using an automated glucose analyzer (YSI 2300 Glucose Analyzer, Yellow Springs Instrument, Yellow Springs, OH). Plasma insulin was measured using a radioimmunoassay kit (Intermedico, Toronto, ON, Canada). Areas under the glucose and insulin curves were determined using a trapezoid model (3).

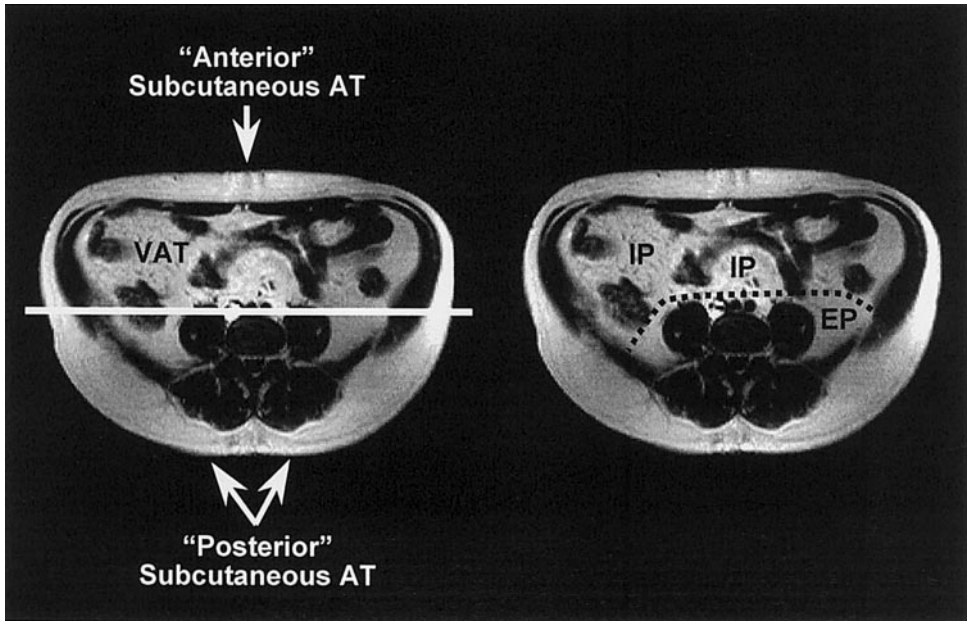


Fig. 1. Subdivision of abdominal subcutaneous adipose tissue (AT) into anterior and posterior depots (*left*) and of visceral AT into intraperitoneal and extraperitoneal depots (*right*). The dashed black line on the image at *right* is the peritoneum, which separates intraperitoneal and extraperitoneal AT. VAT, visceral AT; IP, intraperitoneal AT; EP, extraperitoneal AT.

Table 1. *Subject characteristics*

| | Means ± SD | Range |
|---|---|---|
| Anthropometric data | | |
| Age, yr | 44.0 ± 9.3 | 25–69 |
| Weight, kg | 100.1 ± 11.1 | 71.6–137.2 |
| Body mass index, kg/m$^2$ | 31.9 ± 2.8 | 26.2–40.8 |
| Waist circumference, cm | 108.3 ± 7.1 | 92.0–125.5 |
| MRI variables, kg | | |
| Total AT | 29.9 ± 7.5 | 15.7–53.7 |
| Total abdominal AT | 8.4 ± 1.9 | 4.5–14.6 |
| Abdominal subcutaneous AT | 4.8 ± 1.6 | 2.4–9.6 |
| Anterior subcutaneous AT | 2.3 ± 0.9 | 0.9–5.6 |
| Posterior subcutaneous AT | 2.5 ± 0.8 | 1.3–5.2 |
| Visceral AT | 3.6 ± 1.2 | 0.9–7.2 |
| Intraperitoneal AT | 2.7 ± 0.9 | 0.6–5.6 |
| Extraperitoneal AT | 0.9 ± 0.3 | 0.2–1.6 |
| MRI variables (L4-L5, cm$^2$) | | |
| Total abdominal AT | 485 ± 104 | 290–758 |
| Abdominal subcutaneous AT | 308 ± 97 | 170 ± 607 |
| Anterior subcutaneous AT | 113 ± 42 | 43 ± 242 |
| Posterior subcutaneous AT | 192 ± 55 | 44 ± 373 |
| Visceral AT | 179 ± 66 | 71 ± 428 |
| Intraperitoneal AT | 118 ± 48 | 39 ± 274 |
| Extraperitoneal AT | 52 ± 18 | 21 ± 99 |
| Metabolic variables | | |
| Fasting glucose, mmol/l | 5.4 ± 0.5 | 4.3–6.7 |
| OGTT glucose area, mmol/l × 2 h | 14.5 ± 3.1 | 7.8–20.8 |
| Fasting insulin, pmol/l | 83 ± 71 | 6–363 |
| OGTT insulin area, pmol/l × 2 h | 1031 ± 868 | 123–4828 |
| Glucose disposal, mg·kg muscle$^{-1}$·min$^{-1}$* | 12.5 ± 5.6 | 5.5–29.5 |
| $\dot{V}O_{2max}$, l/min | 3.5 ± 0.7 | 1.6–5.3 |

AT, adipose tissue; $\dot{V}O_{2max}$, maximal oxygen uptake; OGTT, oral glucose tolerance test; MRI, magnetic resonance imaging; *$n = 55$.

*Insulin sensitivity.* Insulin sensitivity data were measured for 55 subjects with the use of a hyperinsulinemic euglycemic clamp. All subjects consumed a weight maintenance diet consisting of ≥200 g of carbohydrate for a minimum of 4 days before measurements of insulin sensitivity and were asked to avoid strenuous physical activity for 3 days preceding the studies. Subjects stayed in the hospital the night before the measurement of insulin sensitivity. All measurements were performed at 8:00 AM after a 12- to 14-h overnight fast. An antecubital vein was catheterized for infusion of insulin plus 20% glucose. An intravenous catheter was inserted in a retrograde fashion in a hand vein, and the hand was placed in a heating pad for sampling of arterialized blood. Insulin was infused at the rate of 40 mU·m$^{-2}$·min$^{-1}$ for 3 h. Plasma glucose was measured using an automated glucose analyzer (YSI 2300 Glucose Analyzer) every 5 min in arterialized blood. Glucose disposal rate was calculated using the average exogenous glucose infusion rate during the final 30 min of euglycemia.

*Statistical analyses.* Data are presented as group means ± SD. Relationships between fat depots and metabolic variables were determined using Pearson product-moment correlation coefficients. Independent correlations were determined using multiple regression stepwise analysis. Paired *t*-tests were used to determine differences between the sizes of the various AT depots at different levels of the abdomen. Unpaired *t*-tests were used to determine differences between groups matched for visceral and abdominal subcutaneous AT. Statistical procedures were performed using SYSTAT (SYSTAT, Evanston, IL).

## RESULTS

*Subject characteristics.* Subjects characteristics are given in Table 1. Despite being obese, as indicated by BMI (31.9 ± 2.8 kg/m$^2$), the cohort was characterized by wide variations in age and total and abdominal adiposity. Table 2 contains the intra- and extraperitoneal AT values and the anterior and posterior abdominal subcutaneous AT values of the five images obtained at different levels of the abdomen. Intraperitoneal AT area (cm$^2$) was greater than extraperitoneal AT area at all levels of the abdomen ($P < 0.01$, Table 2). Anterior subcutaneous AT area was greater ($P < 0.01$) than posterior subcutaneous AT area at the top of the abdomen but less ($P < 0.01$) than posterior subcutaneous AT at the bottom of the abdomen (Table 2). Total visceral AT area was greater ($P < 0.05$) than total abdominal subcutaneous AT area at the top of the abdomen but less ($P < 0.01$) than total abdominal subcutaneous AT area at the bottom of the abdomen (Table 2).

*Relationship between abdominal fat depots and metabolic variables.* Total abdominal AT (visceral + subcutaneous) was significantly ($P < 0.05$) correlated with all metabolic variables with the exception of glucose disposal (Table 3). Separation of total abdominal AT into visceral and subcutaneous AT revealed that visceral AT was significantly ($P < 0.05$) correlated with fasting glucose, OGTT glucose, and glucose disposal by euglycemic clamp, whereas abdominal subcutaneous AT was significantly ($P < 0.05$) correlated with fasting and OGTT insulin values (Table 3). Subdivision of visceral AT into intra- and extraperitoneal depots and abdominal subcutaneous AT into anterior and poste-

Table 2. *Visceral and abdominal subcutaneous AT values for the entire abdominal region (kg) and at different levels of the abdomen (cm$^2$)*

| | Visceral AT | | | Abdominal Subcutaneous AT | | |
|---|---|---|---|---|---|---|
| | Intraperitoneal | Extraperitoneal | Total | Anterior | Posterior | Total |
| Mass | 2.65 ± 0.92 | 0.86 ± 0.31* | 3.59 ± 1.20 | 2.32 ± 0.96 | 2.52 ± 0.75† | 4.78 ± 1.57‡ |
| +15 cm | 164 ± 77 | 4 ± 13* | 174 ± 88 | 94 ± 39 | 56 ± 21† | 151 ± 55‡ |
| +10 cm | 143 ± 54 | 74 ± 36* | 222 ± 88 | 109 ± 44 | 82 ± 33† | 189 ± 71‡ |
| +5 cm | 140 ± 54 | 76 ± 30* | 221 ± 82 | 115 ± 45 | 138 ± 44† | 249 ± 81‡ |
| L4-L5 | 118 ± 48 | 52 ± 18* | 179 ± 66 | 113 ± 42 | 192 ± 55† | 307 ± 91‡ |
| −5 cm | 101 ± 36 | 0 ± 0* | 112 ± 56 | 112 ± 44 | 161 ± 58† | 274 ± 91‡ |

Data are means ± SD. *Significantly ($P < 0.01$) less than intraperitoneal AT at same level; †significantly ($P < 0.01$) different from posterior abdominal subcutaneous AT at same level; ‡significantly ($P < 0.05$) different from total visceral AT at same level.

Table 3. *Correlations among the various AT depots and metabolic variables*

| | Fasting Glucose | Glucose Area | Fasting Insulin | Insulin Area | Glucose Disposal* |
|---|---|---|---|---|---|
| Total AT | | | 0.54 | 0.31 | |
| Total abdominal AT | 0.20 | 0.27 | 0.45 | 0.32 | |
| Visceral AT | 0.24 | 0.50 | | | −0.40 |
| Intraperitoneal AT | 0.24 | 0.51 | | | −0.41 |
| Extraperitoneal AT | | 0.37 | | | −0.31 |
| Abdominal subcutaneous AT | | | 0.46 | 0.27 | |
| Anterior subcutaneous AT | | | 0.51 | 0.25 | |
| Posterior subcutaneous AT | | | 0.43 | 0.29 | |

All listed correlations are significant ($P < 0.05$); *$n = 55$.

rior depots did not alter the magnitude of the correlations with all metabolic variables (Table 3).

Multiple regression analyses revealed that visceral AT remained a significant ($P < 0.05$) correlate of glucose area and glucose disposal after statistical control for abdominal subcutaneous AT, nonabdominal AT, age, and maximal oxygen uptake ($\dot{V}O_{2\,max}$) (Table 4). Abdominal subcutaneous AT did not remain a significant correlate ($P > 0.05$) of insulin values after statistical control for visceral AT, nonabdominal AT, age, and $\dot{V}O_{2\,max}$ (Table 4).

To further explore the relationships among visceral AT, subcutaneous AT, and metabolic risk, the data were also analyzed using a matching strategy. First, we used a percentile approach to identify high (>60th percentile) and low (<40th percentile) levels of visceral AT in our cohort. The matching strategy was then performed on the basis of abdominal subcutaneous AT after having excluded subjects with intermediate levels (e.g., 40th-60th percentile) of visceral AT. The glucose, insulin, and glucose disposal values were compared in two subgroups of 14 subjects matched for abdominal subcutaneous AT but who displayed different levels of visceral AT (Table 5). The groups matched for abdominal subcutaneous AT were also matched for posterior abdominal subcutaneous AT (Table 5). Men with high visceral AT had higher OGTT glucose values and lower glucose disposal values compared with men with low visceral AT ($P < 0.05$, Table 5).

The matching strategy was also performed on the basis of visceral AT by use of the procedures described for abdominal subcutaneous AT. The metabolic variables were compared in two subgroups of 14 subjects matched for visceral AT but who displayed different levels of abdominal subcutaneous AT (Table 5). The glucose, insulin, and glucose disposal values were not different in men with high abdominal subcutaneous AT compared with men with low abdominal subcutaneous AT ($P > 0.1$, Table 5).

Table 4. *Relationship between abdominal AT distribution and metabolic variables*

| | Visceral AT | | Abdominal Subcutaneous AT | |
|---|---|---|---|---|
| | Unadjusted | Adjusted* | Unadjusted | Adjusted† |
| Fasting glucose | 0.24 | | | |
| Glucose area | 0.50 | 0.35 | | |
| Fasting insulin | | | 0.46 | |
| Insulin area | | | 0.27 | |
| Glucose disposal‡ | −0.40 | −0.29 | | |

All listed correlations are significant ($P < 0.05$): *after controlling for nonabdominal AT, abdominal subcutaneous AT, age, and $\dot{V}O_{2max}$; †after controlling for nonabdominal AT, visceral AT, age, and $\dot{V}O_{2max}$; ‡$n = 55$.

*Relationship between abdominal fat area, fat mass, and metabolic variables.* Visceral and subcutaneous AT area ($cm^2$) was measured at five levels through the abdomen. As expected, the visceral and subcutaneous AT areas for each of the five images were significantly ($P < 0.01$) correlated with the respective AT mass (kg) (data not shown). Moreover, visceral and subcutaneous AT area measures were related to the metabolic variables by a similar order of magnitude for each of the five abdominal images (Table 6). Accordingly, the relationship between visceral and abdominal subcutaneous AT mass (kg), derived using all five abdominal images, and the respective metabolic variables was similar to the relationships observed using AT area ($cm^2$) values (Table 6).

## DISCUSSION

In this study, we investigated the independent relationships among various abdominal AT depots with selected measures of insulin and glucose metabolism. The results demonstrate that visceral AT alone was a significant correlate of glucose tolerance and insulin resistance after statistical control for abdominal subcutaneous AT, nonabdominal AT, and cardiovascular fitness. Subdivision of abdominal subcutaneous AT into anterior and posterior compartments and visceral AT into intraperitoneal and extraperitoneal compartments by MRI did not provide additional insight.

The findings of this study suggest that visceral AT is a strong marker of insulin resistance independent of abdominal subcutaneous AT and cardiovascular fitness. That we employed a multislice MRI protocol to determine abdominal and nonabdominal (e.g., peripheral subcutaneous AT) AT depots, measured insulin resistance using the euglycemic clamp method, and performed our measurements in a homogeneous cohort reinforces the importance of visceral AT as a modulator of insulin resistance in abdominally obese men. These observations confirm and strengthen previous observations from others, who reported similar findings in

Table 5. *Characteristics of men matched for abdominal subcutaneous or visceral AT*

| | Visceral AT | | Abdominal Subcutaneous AT | |
|---|---|---|---|---|
| | Low ($n$ = 14) | High ($n$ = 14) | Low ($n$ = 14) | High ($n$ = 14) |
| Visceral AT | 2.4 ± 0.5 | 4.7 ± 1.1* | 3.7 ± 0.8 | 3.7 ± 0.8 |
| Abdominal subcutaneous AT | 4.2 ± 0.8 | 4.2 ± 0.8 | 3.2 ± 0.4 | 5.3 ± 0.9† |
| Posterior subcutaneous AT | 2.3 ± 0.4 | 2.2 ± 0.5 | 1.7 ± 0.2 | 2.8 ± 0.4† |
| Fasting glucose, mmol/l | 5.1 ± 0.6 | 5.5 ± 0.7 | 5.3 ± 0.5 | 5.2 ± 0.5 |
| OGTT glucose area, mmol/l × 2 h | 12.3 ± 2.8 | 16.1 ± 3.2* | 14.9 ± 2.8 | 14.4 ± 3.8 |
| Fasting insulin, pmol/l | 29 ± 15 | 39 ± 22 | 45 ± 22 | 45 ± 24 |
| OGTT insulin area, pmol/l × 2 h | 463 ± 381 | 643 ± 429 | 619 ± 431 | 703 ± 412 |
| Glucose disposal, mg·kg muscle$^{-1}$·min$^{-1}$ | 16.3 ± 7.3 | 11.1 ± 5.2* | 10.5 ± 3.8 | 12.3 ± 5.4 |

Data expressed as group means ± SD. *Significantly different from low visceral AT group ($P < 0.05$); †significantly different from low abdominal subcutaneous AT group ($P < 0.01$).

Caucasian men (28, 32) and women (13, 15, 17, 22, 24), Asian men (5, 18), and African-American men and women (6). However, the findings here contrast with those of others who report that abdominal subcutaneous AT is the stronger correlate of insulin resistance (1, 2, 26, 20, 38, 40). The discrepant findings may be partially explained by differences in the cohorts studied. Several studies implicating abdominal subcutaneous AT as the stronger predictor of insulin resistance are characterized by cohorts that were gender mixed and varied widely in subcutaneous adiposity (19, 20). Because correlation coefficients are a function of the standard deviation of the dependent and independent variables, the greater the variability among observations (e.g., abdominal subcutaneous AT), the greater the correlation coefficient. Another, equally tenable, explanation for the discrepant findings is that, in some of the studies demonstrating that abdominal subcutaneous AT is a strong predictor of insulin resistance (19, 20, 38), the average values for visceral AT are substantially below the value (~130 cm$^2$ at L4-L5) thought to be associated with a marked increase in metabolic risk (14). By contrast, in this study and others (11, 17, 32), wherein a strong, independent relationship between visceral AT and insulin resistance is reported, average values for visceral AT far exceed the 130 cm$^2$ value.

It has also been suggested that discrepancies in the literature regarding the independent relationship between visceral AT, abdominal subcutaneous AT, and insulin resistance may be resolved by subdividing abdominal subcutaneous AT on the basis of metabolic characteristics (18, 36, 38). Abdominal subcutaneous AT can be subdivided into superficial and deep compartments by using the fascia superficialis (23). The rationale for this division derives from animal studies indicating that adipocytes within the deep compartment are more metabolically active than superficial adipocytes (12, 25). With the assumption that the liberation of nonesterified fatty acids adversely affects insulin action (29, 37), it follows that the deep compartment would be the stronger predictor of insulin resistance. Interestingly, recent in vivo evidence suggests that the lipolytic rate of superficial abdominal subcutaneous AT in the anterior abdomen in normal-weight men is higher than that of the deep subcutaneous AT located in the posterior abdominal wall (16). Thus preliminary evidence in human volunteers does not appear to support the hypothesis derived from animal models suggesting that deep abdominal subcutaneous AT is more lipolytically active compared with superficial depots. Despite this observation, several reports observe that deep abdominal subcutaneous AT is a strong correlate of insulin action (20, 38, 40). Findings that confirm the earlier report by Misra et al. (26), that posterior abdominal subcutaneous AT measured by MRI (analogous to deep subcutaneous AT in men) compared with anterior compartment mass, displayed a stronger relationship with glucose disposal. Because we employed an MRI protocol similar to that used by Misra et al., it is unlikely that the equivocal findings are explained by methodological differences. However, although the majority of deep subcutaneous AT is located in the posterior half of the abdomen in men (20, 38), the two compartments (posterior subcutaneous vs.

Table 6. *Correlations between metabolic variables with visceral and abdominal subcutaneous AT mass (kg) and area (cm$^2$) at five different locations in the abdomen*

| | Visceral AT | | | | | Abdominal Subcutaneous AT | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Fasting glucose | Glucose area | Fasting insulin | Insulin area | Glucose disposal* | Fasting glucose | Glucose area | Fasting insulin | Insulin area | Glucose disposal* |
| Mass | 0.24 | 0.50 | | | −0.40 | | | 0.46 | 0.27 | |
| +15 cm | 0.34 | 0.40 | | | | | | 0.43 | 0.30 | |
| +10 cm | 0.32 | 0.47 | | | −0.29 | | | 0.39 | 0.24 | |
| +5 cm | 0.31 | 0.51 | | | −0.37 | | | 0.38 | 0.24 | |
| L5-L5 | 0.25 | 0.46 | | | −0.35 | | | 0.32 | | |
| −5 cm | 0.26 | 0.40 | 0.25 | 0.31 | −0.33 | | | 0.32 | | |

All listed correlations are significant ($P < 0.05$); *$n$ = 55.

deep subcutaneous AT) are not precisely the same; thus differences between our findings and those that employed computed tomography may be explained by differences in the technique used to subdivide abdominal subcutaneous AT.

It is hypothesized that the complications associated with visceral AT may be related to the portally drained fat originating within the peritoneal cavity (intraperitoneal fat) (9, 10). Although access to portal circulation in humans is not practical, data from animal models confirm that intra-abdominal AT is positively related to hepatic insulin resistance (5, 7). Because extraperitoneal AT drains systemically, it has been suggested that removal of extraperitoneal AT would strengthen the relationship between visceral AT and metabolic risk (2). In support of this hypothesis, Abate and colleagues (2, 3) twice reported that intraperitoneal fat is a stronger correlate of insulin resistance compared with extraperitoneal fat. However, in those studies, the authors did not confirm whether intraperitoneal AT was a better predictor of insulin resistance than visceral AT per se (intraperitoneal and extraperitoneal). The observations here are consistent with previous findings from our group indicating that subdivision of visceral AT does not improve on the relationship observed between visceral AT per se and plasma lipid profile (31) and insulin action (30, 33). These observations might be expected, given that intraperitoneal AT comprises ~75% of the total visceral depot (30, 31, 33). Coupled with the fact that intraperitoneal AT is highly correlated with total visceral AT ($r = 0.98$; data not shown), it is not surprising that isolation of intraperitoneal AT does not enhance the relationship between visceral AT per se and insulin metabolism.

In this study, the use of a multislice MRI model revealed that the correlations obtained between subcutaneous AT or visceral AT area ($cm^2$) and insulin resistance were essentially unchanged regardless of which abdominal image was used to quantify AT area. This finding confirms a previous observation from our group (31), wherein the association between visceral AT and plasma lipid profile remained significant independently of the abdominal image used to estimate visceral AT. Together, these observations suggest that the commonly used L4-L5 level for MRI or computed tomography measurement is an accurate measure of abdominal AT distribution and of the relationships between subcutaneous or visceral AT distribution and insulin resistance. This finding is consistent with the observation that the visceral AT area at the level of L4-L5 is highly correlated with both the visceral AT area of adjacent abdominal images (34, 38) and the visceral AT mass derived from multiple images (34).

In summary, the findings of this study reinforce the notion that visceral AT is a robust marker of insulin resistance in abdominally obese men independent of nonabdominal and abdominal subcutaneous AT and cardiovascular fitness. This observation remains true independent of the level of the abdomen at which visceral or abdominal subcutaneous AT was measured. It would also appear that further subdivision of visceral and abdominal subcutaneous AT with the use of the MRI method described here provides no additional insight into the relationship between abdominal obesity and metabolic risk in obese men.

This work was supported by Canadian Institutes of Health Research Grant MT 13448 and Natural Sciences and Engineering Council of Canada Grant OGPIN 030 (to R. Ross). I. Janssen was supported by a Heart and Stroke Foundation of Canada Research Trainee Award.

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