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DXA-Determined Regional Adiposity Relates to Insulin Resistance in a Young Adult Population with Overweight and Obesity

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Abstract

Obesity is a well-established risk factor for insulin resistance and type 2 diabetes mellitus, and body fat distribution has important implications for this metabolic risk. In this cross-sectional study, we used dual X-ray absorptiometry body composition data from 123 young adult participants with overweight or obesity, and correlated with 2 indices of insulin resistance calculated from oral glucose tolerance tests. Participants were 70% women, with mean (standard error) age 30.1 (0.6) yr, body mass index (BMI) 34.0 (0.6) kg/m², homeostatic model assessment of insulin resistance (HOMA-IR) of 2.1 (0.2), and Matsuda insulin sensitivity index (Matsuda ISI) of 5.8 (0.4). In women, the strongest correlations were observed with the android-to-gynoid ratio ($r = 0.52$, $p < 0.001$ for HOMA-IR; $r = 0.46$, $p < 0.001$ for Matsuda ISI), and these correlations remained significant after adjustment for BMI. For men, the strongest correlations were with android fat mass ($r = 0.40$, $p = 0.01$ for HOMA-IR; $r = 0.37$, $p = 0.02$ for Matsuda ISI). Visceral adipose tissue was correlated with HOMA-IR and Matsuda ISI in women, and only with Matsuda ISI in men. BMI correlated with HOMA-IR and with Matsuda ISI in both women and men. Regional adiposity determined by dual X-ray absorptiometry correlates with indices of insulin resistance in sedentary young adults with overweight and obesity.

Keywords

Body composition; DXA; HOMA-IR; insulin resistance; Matsuda index

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Introduction

Obesity is a well-established risk factor for insulin resistance and type 2 diabetes mellitus. However, body fat distribution within an individual with obesity may have a greater influence on risk than total adiposity. In particular, increased abdominal adiposity has been associated with increased glucose intolerance and hyperinsulinemia (1), increased hemoglobin A1c (2), and increased risk of diabetes mellitus (3).

Dual X-ray absorptiometry (DXA) is increasingly used in clinical and research settings to measure body composition, including quantification of abdominal adipose tissues. DXA measurement of abdominal adiposity correlated with measurement by computed tomography (CT) and magnetic resonance imaging (MRI) (4–6). A recent development in DXA allows for the calculation of visceral adipose tissue (VAT) (7), which is the component of abdominal adipose tissue believed to have the most metabolic impact (measures of total abdominal adipose tissue also include the adipose tissue in the subcutaneous compartment). VAT determined by DXA correlates with CT-determined VAT (7–9) and MRI-determined VAT (10). In addition, DXA is widely available and offers advantages over CT or MRI for this use, including relatively low cost, low radiation exposure, quick scanning time, and immediate results that can be shared with patients. Thus, DXA measurement of VAT is a useful tool that improves upon previous methods, and that may be used increasingly in the future.

Previous work demonstrated that DXA measurement of total abdominal adiposity correlated with insulin resistance in healthy men and women (11–13). Studies linking DXA-determined VAT (rather than total abdominal adipose tissue) to metabolic risk are limited. Bosch et al showed that VAT estimated by DXA correlated with insulin sensitivity as measured by euglycemic-hyperinsulinemic clamp and the homeostatic model assessment of insulin resistance (HOMA-IR) in children and adults (9,14). Bi et al demonstrated that DXA measurement of VAT was predictive of impaired glucose tolerance and metabolic syndrome in middle aged women with obesity (15). Miazgowski et al showed that DXA-determined VAT was associated with cardiometabolic risk factors, including HOMA-IR, in healthy young adults (16). Further validation in various populations would reinforce the utility of DXA for measurement of VAT and correlation with metabolic risk.

The use of DXA for this purpose may be especially relevant in a young adult population. The prevalence of overweight and obesity is increasing, including among young adults. Early intervention in this group may have a substantial beneficial impact (17,18). This makes it critical to identify which individuals are at highest metabolic risk, and might benefit most from targeted intervention.

The Training Effects of Fuel Metabolism (TrainMeUpMN) study is a randomized controlled clinical trial investigating the effects of supervised aerobic exercise in young (aged 18–40 yr), sedentary participants with over-weight and obesity. At baseline, all participants had a DXA scan to measure body composition and an oral glucose tolerance test (OGTT) to measure insulin sensitivity. We hypothesized that the amount of VAT measured by DXA would correlate with insulin sensitivity in this population of participants, supporting the use

of DXA-determined VAT as a tool for prediction of metabolic risk in sedentary young adults.

Methods

Cross-sectional data from the baseline visit of the TrainMeUpMN study were used for this analysis. Inclusion criteria were body mass index (BMI) 25–40 kg/m², stable weight for at least 3 mo prior to enrollment (defined as no change of >5 pounds), and sedentary status (self-report of <30 min of exercise per week). Participants were excluded if they had significant medical issues, were taking medications that could affect blood glucose levels, or were currently pregnant or breastfeeding. All study procedures were approved by the Institutional Review Board at the University of Minnesota, and all participants provided written informed consent prior to study participation.

The baseline study visit for each participant included an OGTT and measurement of body composition by DXA. Participants were included in this analysis if they attended a baseline visit between July 1, 2014 and August 31, 2017, and had data available from both the OGTT and DXA. Participants were included, if data were available even if incomplete, as follows: (1) 1 participant had a fasting glucose and insulin measured but was missing all other OGTT data because of loss of intravenous access, and (2) 3 participants were missing VAT measurement but had all other DXA data available. In these cases, all available data were used, and these participants were withheld from the specific analyses for which data were missing.

Assessment of Insulin Sensitivity

OGTT: Participants arrived for the baseline visit after an 8-h fast (hydration with water was allowed). Intravenous access was established and a fasting blood sample was taken for measurement of plasma glucose and serum insulin. An oral load of 75 g glucose was given, and blood was sampled every 30 min over the subsequent 2 h for measurement of plasma glucose and serum insulin. Samples were analyzed by the Clinical Chemistry Laboratory of the University of Minnesota Hospital.

Calculation of Indices of Insulin Resistance: Two indices of insulin resistance were calculated from OGTT data. HOMA-IR is an index of fasting glucose and fasting insulin concentrations. The Matsuda insulin sensitivity index (Matsuda ISI) is calculated from glucose and insulin concentrations fasting and for 2 h following the oral glucose load given during the OGTT. Both indices were calculated as previously described (19,20).

Assessment of Body Composition

Body composition was measured by DXA (GE Healthcare Lunar iDXA; General Electric Medical Systems, Madison, WI) and analyzed using its General Electric Medical Systems, Madison, WI, USA software (platform version 16.2). The Lunar iDXA has been shown to have improved precision for measurement of body composition when compared to older generation densitometers, with coefficient of variation values of 0.4%–0.5% for lean mass, 0.7%–1.0% for fat mass, and 0.6%–0.9% for percent body fat (21).

Standard positioning and imaging protocols were used. Participants had been fasting for 8 h prior to scanning (hydration with water was allowed). VAT was estimated using a method described previously, in which subcutaneous adipose mass is subtracted from total abdominal adipose mass in the android region (7). The android region is the area between the ribs and the pelvis. The gynoid region is located mid-pelvis to mid-thigh. Specifically, the android region was defined by a caudal limit placed at the top of the iliac crest, with height set to 20% of the distance from the top of the iliac crest to the base of the skull (22). The gynoid region was defined by an upper limit set a distance 1.5 times the height of the android region below the iliac crest, and a lower limit set a distance of 2 times the height of the android region below the iliac crest (22). All scans were reviewed for accurate placement of the android box.

Statistical Analyses

Characteristics of study participants were summarized using descriptive statistics and were compared between males and females using 2-sample *t* tests. Data distributions were examined for normality. Linear correlation between DXA measures of body composition and indices of insulin resistance (HOMA-IR or Matsuda ISI) were tested using Pearson's correlation. Tests were done for the overall cohort and for sex-specific cohorts. Correlations were adjusted for BMI. The effect sizes of correlations were interpreted as previously described (23). Statistical analyses were performed using statistical analysis software (version 9.3, SAS Institute Inc., Cary, NC). A $p < 0.05$ was considered to be statistically significant.

Results

A total of 123 participants were included in this analysis. Characteristics of the participants are shown in Table 1. The population was 70% female, with mean (standard error) age 30.1 (0.6) yr, mean weight 97.4 (1.6) kg, and mean BMI 34.0 (0.6) kg/m². The mean HOMA-IR value was 2.1 (range 0.1–13.1) and mean Matsuda ISI was 5.8 (range 0.7–24.4). A higher HOMA-IR value indicates insulin resistance, with insulin resistance suggested when HOMA-IR is > 2.5 (24). A lower Matsuda ISI indicates insulin resistance, with insulin resistance suggested when Matsuda ISI is < 2.5 (20).

Several DXA-based measures of regional adiposity were positively correlated with HOMA-IR (Table 2). The strongest correlations were observed between HOMA-IR and percent total body fat ($r = 0.34$, $p < 0.01$), total fat mass ($r = 0.35$, $p < 0.01$), and android fat mass ($r = 0.43$, $p < 0.01$). These correlations were similar or modestly stronger than the correlation between HOMA-IR and BMI ($r = 0.34$, $p < 0.01$). There was also a low positive correlation between HOMA-IR and VAT ($r = 0.30$, $p < 0.01$). The Matsuda ISI was negatively correlated with several DXA measures of adiposity. The strongest correlations were between Matsuda ISI and VAT ($r = 0.33$, $p < 0.01$), percent total body fat ($r = 0.35$, $p < 0.01$), and android fat mass ($r = 0.35$, $p < 0.01$). These correlations were somewhat stronger than that seen between Matsuda ISI and BMI ($r = 0.26$, $p < 0.01$).

There were sex-specific differences in the relationships between DXA measurements and HOMA-IR or Matsuda ISI (Table 3). In women, the strongest correlations were observed with the android-to-gynoid ratio ($r = 0.52$, $p < 0.001$ for HOMA-IR; $r = -0.46$, $p < 0.001$ for Matsuda ISI). In men, there was not a significant correlation between the android-to-gynoid ratio and either HOMA-IR or Matsuda ISI. For men, the strongest correlations were with android fat mass ($r = 0.40$, $p = 0.01$ for HOMA-IR; $r = -0.37$, $p = 0.02$ for Matsuda ISI). VAT was correlated with HOMA-IR and Matsuda ISI in women ($r = 0.42$, $p < 0.001$ for HOMA-IR; $r = -0.34$, $p < 0.01$ for Matsuda ISI), and only with Matsuda ISI in men ($r = -0.37$, $p = 0.02$). BMI was correlated with both indices in women ($r = 0.30$, $p = 0.005$ for HOMA-IR; $r = 0.25$, $p = 0.02$ for Matsuda ISI) and in men ($r = 0.37$, $p = 0.02$ for HOMA-IR; $r = -0.34$, $p = 0.04$ for Matsuda ISI).

Sex-specific correlations were adjusted for BMI (Table 4). In women, after adjustment for BMI, significant correlations remained between HOMA-IR or Matsuda ISI and VAT, VAT percent of total fat mass, and the android-to-gynoid ratio. No correlations remained significant in men; the sample size for men was small.

Discussion

We used cross-sectional data from the baseline visit of the TrainMeUpMN clinical trial to investigate the relationship between body composition and insulin resistance. Our analysis confirms that DXA-determined measures of regional adiposity correlate with 2 indices of insulin resistance, HOMA-IR and Matsuda ISI, in a sedentary, young adult population with overweight and obesity.

Previous work has shown that DXA measurements of abdominal adiposity correlate with markers of insulin resistance. This includes both DXA measures of total abdominal adipose tissue (11–13) and the newer technique of estimating VAT (9,14–16). Our analysis adds to these findings, and extends them to a sedentary, young adult population with overweight or obesity. For both women and men in our cohort, there were correlations between VAT and indices of insulin resistance, although these correlations were not as strong as initially hypothesized. For women, the strongest correlations were seen with the android-to-gynoid ratio, suggesting that this measure may be particularly useful for prediction of insulin resistance in women.

We found that BMI performed almost as well as the DXA-determined measures in both the overall and sex-specific cohorts, supporting the continued use of BMI for prediction of metabolic risk. However, DXA does offer some advantages over BMI, particularly in women. Specifically, when correlations were adjusted for BMI, several relationships remained significant in the cohort of women, including the correlations between HOMA-IR or Matsuda ISI and VAT, VAT percent of total fat mass, and the android-to-gynoid ratio. This suggests that, in women, an increase in adipose tissue in these regions has adverse metabolic consequences that are independent of BMI. This finding is in agreement with previous literature (25,26), and highlights the utility of DXA for providing information that cannot be obtained by anthropometric measures alone. We did not find significant correlations in men after adjustment for BMI; however, this may have been due to small sample size.

The US population is becoming increasingly overweight and obese. Young adulthood, especially for women, represents a period of particular risk for weight gain (27–29). Furthermore, weight gain as a young adult is associated with poor health outcomes, including type 2 diabetes, later in life (30). Thus, it is critical to find ways to predict which individuals are at most risk, and to design appropriate interventions. DXA is an easy-to-use tool that is widely available in both clinical and research settings. Our data provide evidence that DXA measures of adiposity correlate with insulin resistance in sedentary young adults with overweight and obesity, and can appropriately be used for this purpose. Our data also suggest that BMI is a useful marker, and although not as optimal, it could be substituted if resources are limited.

The strengths of this study include the novel subject population and the number of participants with available DXA and OGTT data. However, the moderate sample size was a limitation. In particular, our sample included only a small number of men, limiting any conclusions that can be drawn from this group. The cross-sectional design is another limitation, as the data are correlations only and do not allow any conclusions about cause and effect. The full TrainMeUpMN study, when completed, will provide longitudinal body composition and insulin sensitivity data both before and after an exercise intervention.

In conclusion, various DXA measures of abdominal adiposity correlate with insulin resistance in a cohort of sedentary young adults with overweight and obesity. In young women, these correlations are independent of BMI. We conclude that DXA is useful for prediction of insulin resistance in young adults with overweight and obesity, and could be used as a valuable tool to identify metabolically at-risk individuals for targeted interventions.

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Table 1

Characteristics of Study Participants

Characteristic	All participants (n = 123)	Female (n = 86)	Male (n = 37)	<i>p</i> value for male vs female
Age (yr)	30.1 (0.6)	29.5 (0.7)	31.3 (1.0)	0.15
Weight (kg)	97.4 (1.6)	96.3 (2.2)	99.8 (1.9)	0.24
BMI (kg/m ²)	34.0 (0.6)	35.1 (0.8)	31.3 (0.6)	<0.001
HOMA-IR	2.1 (0.2)	2.4 (0.2)	1.5 (0.1)	<0.01
Matsuda ISI	5.8 (0.4)	5.7 (0.5)	6.1 (0.7)	0.60
Fat-free mass (kg)	55.0 (0.8)	51.0 (0.7)	64.3 (1.1)	<0.0001
Muscle mass (kg)	52.3 (0.8)	48.5 (0.7)	61.0 (1.0)	<0.0001
Total fat mass (kg)	41.5 (1.2)	44.4 (1.6)	34.9 (1.4)	<0.0001
Percent total body fat (%)	43.6 (0.7)	46.9 (0.7)	36.1 (0.9)	<0.0001
VAT (kg)	1.2 (0.1)	1.1 (0.1)	1.5 (0.1)	<0.01
VAT percent of total fat mass (%)	3.0 (0.1)	2.4 (0.1)	4.3 (0.3)	<0.0001
Android fat mass (kg)	4.0 (0.1)	4.1 (0.2)	3.8 (0.2)	0.25
Gynoid fat mass (kg)	7.1 (0.2)	7.7 (0.3)	5.7 (0.3)	<0.0001
Android-to-gynoid ratio	1.1 (0.02)	1.1 (0.01)	1.3 (0.03)	<0.0001

Data are mean (standard error). The *p* values refer to the comparison for male vs female participants.

BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; Matsuda ISI, Matsuda insulin sensitivity index; VAT, visceral adipose tissue.

Table 2
Correlation of DXA Measures of Body Composition With Indices of Insulin Resistance All Participants (n = 123)

Body composition measure	HOMA-IR	Matsuda ISI
BMI	0.34 [*] (0.17, 0.49)	-0.26 [*] (-0.42, -0.08)
Fat-free mass	-0.02 (-0.19, 0.16)	0.08 (-0.11, 0.25)
Muscle mass	-0.01 (-0.18, 0.17)	0.06 (-0.12, 0.24)
Total fat mass	0.35 [*] (0.18, 0.49)	-0.28 [*] (-0.43, -0.10)
Percent total body fat	0.34 [*] (0.17, 0.48)	-0.35 [*] (-0.49, -0.18)
VAT	0.30 [*] (0.13, 0.46)	-0.33 [*] (-0.48, -0.16)
VAT percent of total fat mass	0.13 (-0.05, 0.31)	-0.28 [*] (-0.44, -0.11)
Android fat mass	0.43 [*] (0.27, 0.56)	-0.35 [*] (-0.49, -0.18)
Gynoid fat mass	0.19 [*] (0.01, 0.35)	-0.18 [*] (-0.34, 0.00)
Android-to-gynoid ratio	0.21 [*] (0.03, 0.37)	-0.27 [*] (-0.42, -0.90)

Data are correlation coefficients (95% confidence intervals).

BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; Matsuda ISI, Matsuda insulin sensitivity index; VAT, visceral adipose tissue.

* $p < 0.05$.

Table 3
Gender-Specific Correlation of DXA Measures of Body Composition With Indices of Insulin

Body composition measure	Female participants (n = 86)		Male participants (n = 37)	
	HOMA-IR	Matsuda ISI	HOMA-IR	Matsuda ISI
BMI	0.30 [*] (0.09, 0.48)	-0.25 [*] (-0.44, -0.03)	0.37 [*] (0.06, 0.62)	-0.34 [*] (-0.60, -0.02)
Fat-free mass	0.19 (-0.02, 0.39)	0.04 (-0.17, 0.25)	0.02 (-0.31, 0.34)	0.11 (-0.22, 0.42)
Muscle mass	0.20 (-0.02, 0.39)	0.02 (-0.20, 0.23)	0.02 (-0.31, 0.34)	0.12 (-0.21, 0.43)
Total fat mass	0.30 [*] (0.09, 0.48)	-0.27 [*] (-0.46, -0.06)	0.38 [*] (0.07, 0.63)	-0.32 (-0.58, 0.01)
Percent total body fat	0.28 [*] (0.07, 0.46)	-0.44 [*] (-0.59, -0.25)	0.36 [*] (0.04, 0.61)	-0.35 [*] (-0.60, -0.02)
VAT	0.42 [*] (0.22, 0.58)	-0.34 [*] (-0.52, -0.13)	0.25 (-0.08, 0.53)	-0.37 [*] (-0.62, -0.05)
VAT percent of total fat mass	0.41 [*] (0.21, 0.57)	-0.44 [*] (-0.60, -0.24)	0.09 (-0.24, 0.41)	-0.28 (-0.55, 0.05)
Android fat mass	0.44 [*] (0.24, 0.59)	-0.34 [*] (-0.51, -0.13)	0.40 [*] (0.09, 0.64)	-0.37 [*] (-0.62, -0.05)
Gynoid fat mass	0.11 (-0.11, 0.31)	-0.15 (-0.35, 0.07)	0.30 (-0.03, 0.57)	-0.25 (-0.53, 0.08)
Android-to-gynoid ratio	0.52 [*] (0.33, 0.65)	-0.46 [*] (-0.61, -0.27)	0.22 (-0.11, 0.51)	-0.17 (-0.47, 0.16)

Data are correlation coefficients (95% confidence intervals).

BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; Matsuda ISI, Matsuda insulin sensitivity index; VAT, visceral adipose tissue.

^{*} $p < 0.05$.

Table 4
Gender-Specific Correlation of DXA Measures of Body Composition With Indices of Insulin Resistance

Body composition measure	Female participants (n = 86)		Male participants (n = 37)	
	HOMA-IR	Matsuda ISI	HOMA-IR	Matsuda ISI
Fat-free mass	-0.02 (-0.23, 0.19)	0.30* (0.09, 0.48)	-0.14 (-0.44, 0.20)	0.27(-0.06, 0.55)
Muscle mass	-0.02 (-0.23, 0.20)	0.27* (0.06, 0.46)	-0.14 (-0.45, 0.20)	0.28(-0.05, 0.56)
Total fatmass	0.05 (-0.17, 0.26)	-0.12 (-0.33, 0.10)	0.15(-0.19, 0.45)	-0.07(-0.39, 0.26)
Percent body fat	0.07 (-0.15, 0.27)	-0.41* (-0.58, -0.22)	0.17(-0.17, 0.47)	-0.18(-0.48, 0.16)
VAT	0.30* (0.09, 0.49)	-0.26* (-0.45, -0.05)	-0.01 (-0.34, 0.32)	-0.20(-0.50, 0.14)
VAT percent of total fat mass	0.31* (0.10, 0.49)	-0.38* (-0.55, -0.17)	-0.03 (-0.35, 0.30)	-0.19(-0.49, 0.15)
Android fat mass	0.41* (0.22, 0.57)	-0.28* (-0.46, -0.07)	0.21(-0.12, 0.51)	-0.19(-0.49, 0.14)
Gynoid fat mass	-0.29* (-0.47, -0.08)	0.12 (-0.10, 0.32)	0.14(-0.20, 0.45)	-0.10(-0.41, 0.24)
Android-to-gynoid ratio	0.46* (0.27, 0.61)	-0.41* (-0.57, -0.21)	0.17(-0.17, 0.47)	-0.12(-0.43, 0.21)

Data are correlation coefficients (95% confidence intervals).

BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; Matsuda ISI, Matsuda insulin sensitivity index; VAT, visceral adipose tissue.

* $P < 0.05$.