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Anthropometrically-Predicted Visceral Adipose Tissue and Blood-Based Biomarkers: A Cross-Sectional Analysis

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Abstract

Purpose—We hypothesized that anthropometrically-predicted visceral adipose tissue (apVAT) accounts for more variance in blood-based biomarkers of glucose homeostasis, inflammation, and lipid metabolism than body mass index (BMI), waist circumference (WC), and the combination of BMI and WC (BMI+WC).

Methods—This was a cross-sectional analysis of 10,624 males and females who participated in the Third National Health and Nutrition Survey (NHANES III; 1988–1994). apVAT was predicted from a validated regression equation that included age, height, weight, waist and thigh circumferences. Bootstrapped linear regression models were used to compare the proportion of variance (R^2) in biomarkers explained by apVAT, BMI, WC, and BMI+WC.

Results—apVAT accounted for more variance in biomarkers of glucose homeostasis than BMI ($R^2=8.4-11.8\%$; $P<0.001$), WC ($R^2=5.5-8.4\%$; $P<0.001$), and BMI+WC ($R^2=5.1-7.7\%$; $P<0.001$). apVAT accounted for more variance in biomarkers of inflammation than BMI ($R^2=3.8\%$; $P<0.001$), WC ($R^2=3.1\%$; $P<0.001$), and BMI+WC ($R^2=2.9\%$; $P<0.001$). apVAT accounted for more variance in biomarkers of lipid metabolism than BMI ($R^2=2.9-9.2\%$; $P<0.001$), WC ($R^2=2.9-5.2\%$; $P<0.001$), and BMI+WC ($R^2=2.4-4.1\%$; $P=0.01$).

Conclusions—apVAT, estimated with simple and widely used anthropometric measures, accounts for more variance in blood-based biomarkers than BMI, WC, and BMI+WC. Clinicians and researchers may consider utilizing apVAT to characterize cardio-metabolic health, particularly in settings with limited availability of imaging and laboratory data.

Keywords

adiposity; body composition; waist-hip ratio; population-based

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INTRODUCTION

Visceral adipose tissue (VAT) is one of the most deleterious fat deposits in the body [1]. Excess VAT is associated with an increased risk of developing cardiovascular disease [2, 3], and certain types of cancer [3]. Computed tomography (CT) and magnetic resonance (MR) imaging are gold-standard techniques to quantify VAT, yet their utilization is often hampered by complexities of access and high costs [4]. Waist circumference (WC) is utilized as an anthropometric proxy for VAT [5], however WC is more closely correlated with body mass index (BMI) and total fat mass than with VAT [6]. The ability to accurately quantify VAT is important, as visceral adiposity has been hypothesized to physiologically underpin the health risks associated with overweight and obesity [3].

Anthropometrically-predicted VAT (apVAT), which is calculated through the use of validated equations that simultaneously integrate multiple anthropometric dimensions, including BMI and circumferences of the waist and thigh, is strongly correlated with CT measured VAT ($r=0.90$) [7]. apVAT prognosticates all-cause, cardiovascular-specific, and cancer-specific mortality 10–25% more accurately than BMI, WC, or the combination of BMI+WC [8]. Blood-based laboratory biomarkers of glucose homeostasis [9, 10], inflammation [11, 12], and lipid metabolism [13] are also associated with premature mortality. Although CT and MR measures of VAT account for more variance in these blood-based biomarkers than BMI and WC [14–16], it is unknown if apVAT is superior in explaining variation in blood-based laboratory biomarkers of cardio-metabolic health compared to other more commonly used anthropometric measures.

We hypothesized that apVAT more accurately prognosticates mortality by accounting for a larger proportion of variance in blood-based laboratory biomarkers than BMI, WC, or BMI +WC. We explored this hypothesis in a population-based sample of Caucasian males and females. If apVAT explains patterns of cardio-metabolic health, this anthropometric measure may be particularly useful in clinical and research settings where CT or MR measures of VAT and laboratory testing are not feasible or widely available.

METHODS

Study Design and Participants

The Third National Health and Nutrition Examination Survey, 1988–1994 (NHANES III), was a stratified multistage study designed to provide health information on a nationally-representative sample of 33,994 males and females aged two-months and older. Our sample included males and females aged ≥20 years ($n=17,016$), with self-reported Caucasian race ($n=11,603$), who also completed the requisite measures necessary to calculate apVAT ($n=10,624$). All study participants provided informed consent.

Anthropometric Measures

Body mass (kg), height (m), WC (cm), and thigh circumference (cm) were measured following standardized techniques by trained study technicians [17, 18]. The reproducibility of anthropometric circumferences measures by trained technicians is excellent ($r=0.98$) [19].

BMI was calculated as body mass divided by the square of height (kg/m^2). apVAT (cm^2) was calculated using sex-specific validated equations that included age, BMI, WC, and circumferences of the waist and thigh [7]. The equation for males is:

$$6 \times \text{waist circumference} - 4.41 \times \text{proximal thigh circumference} + 1.19 \times \text{age} - 213.65,$$

and the equation for females is:

$$2.15 \times \text{waist circumference} - 3.63 \times \text{proximal thigh circumference} + 1.46 \times \text{age} + 6.22 \times \text{BMI} - 92.713$$

Blood-Based Laboratory Biomarkers

Study participants underwent a venipuncture in the fasting state using a sterile technique. Blood samples were stored following standardized laboratory procedures that have been described in detail elsewhere [20]. Biomarkers of glucose homeostasis included fasting plasma glucose and hemoglobin A1c (HbA1c). Biomarkers of inflammation included C-reactive protein (CRP) and fibrinogen. Biomarkers of lipid metabolism included apolipoprotein B, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, total cholesterol, and triglycerides. Laboratory procedures used to quantify each of the biomarkers has been described in detail elsewhere [21]. The coefficients of variation for all biomarker assays were 12% [22].

Covariates

Demographic information including date of birth and gender were self-reported. Clinical characteristics assessed using standardized questionnaires included smoking history, alcohol consumption, and use of medications for cholesterol, hypertension, and diabetes. The healthy eating index was calculated from a single 24-hour food recall to form a score that ranges from 0 to 100 to quantify aspects of a healthy diet [23]. Bouts of walking in the past week were self-reported and included any bout that was estimated to be 1 mile in duration, and of moderate or vigorous intensity. Impaired glucose tolerance was defined as a plasma glucose concentration of ≥ 5.6 mmol/L [24]. The metabolic syndrome was defined as meeting 3 of the following criteria: 1) waist circumference ≥ 102 cm (males) or ≥ 88 cm (females); 2) fasting plasma glucose ≥ 5.6 mmol/L; 3) blood pressure of $\geq 130/85$ mm Hg; 4) triglycerides ≥ 1.7 mmol/L; and 5) HDL cholesterol < 1.03 mmol/L (males) or < 1.29 mmol/L (females) [25].

Statistical Analysis

All biomarkers were log-transformed to reduce skewness and the influence of outliers. Biomarkers were then stratified by gender (male vs female) and standardized (mean of zero, standard deviation of one). Linear regression analyses were used to quantify the relationship between biomarkers and each of the four anthropometric measures (BMI, WC, BMI+WC, and apVAT). Multicollinearity was monitored using the variance inflation factor and tolerance statistics derived from the linear regression analyses [26]. All regression models integrated sampling weights to account for nonresponse bias and multistage sampling probabilities. Beta values from the linear regression models represent a one standard

deviation increase in the log-transformed biomarker. The coefficient of determination (R^2), calculated from each of the linear regression models, were used because its quantity represents the value of interest:

$$\text{Anthropometric measure } R^2 = \frac{\text{Variation explained by anthropometric measure}}{\text{Total variation of laboratory biomarker}}$$

The R^2 value ranges from 0 to 1 (or 0 to 100%), where a value of zero indicates that the anthropometric measure explains none of the variability of the distribution of the biomarker around its mean and a value of one indicates that the anthropometric measure explains all the variability of the distribution of the biomarker around its mean. Since all of the continuous variables are standardized this statistic is not susceptible to variation due to nonlinearity or non-normal distributions. The change () in R^2 was used to quantify the incremental increase in the proportion of variance explained between apVAT and BMI, WC, and BMI+WC, respectively, using 100 bootstrap replications to estimate normal-based standard errors. The area under the receiver operating characteristic (ROC) curve, known as the C-statistic, was used to compare the capacity of each anthropometric measure to predict the presence of impaired fasting glucose and the metabolic syndrome [27]. The C-statistic ranges from 0.5 to 1.0, with values of 0.6, 0.7, 0.8, and 0.9 representing poor, fair, good, and excellent discrimination, respectively. Stata/SE v.14.1 statistical software was used for all analyses.

RESULTS

Population Sample

The average age of study participants was 44.6 ± 0.30 years [range: 20–90], and 48.2% were male (Table 1). Males had a higher BMI (0.4 ± 0.15 kg/m²; $P=0.003$), WC (7.8 ± 0.39 cm; $P<0.001$), and apVAT (44.8 ± 1.97 cm²; $P<0.001$) than females. Males had higher fasting plasma glucose (0.23 ± 0.43 mmol/L; $P<0.001$), HbA1c ($0.1 \pm 0.02\%$; $P<0.001$), apolipoprotein B (0.04 ± 0.01 g/L; $P<0.001$), LDL cholesterol (0.13 ± 0.04 mmol/L; $P=0.002$), and triglyceride (0.28 ± 0.04 mmol/L; $P<0.001$) concentrations than females. Conversely, males were younger (-1.6 ± 0.42 yr; $P<0.001$), had lower CRP (-0.08 ± 0.02 mg/L; $P<0.001$), fibrinogen (-0.12 ± 0.03 umol/L; $P<0.001$), and total cholesterol (-0.11 ± 0.04 mmol/L; $P=0.001$) concentrations than females.

Laboratory Biomarkers of Glucose Homeostasis

All anthropometric measures were positively correlated with fasting plasma glucose ($P<0.001$; Table E1). apVAT accounted for 12.4% of the variance in fasting plasma glucose. apVAT accounted for more variance in fasting plasma glucose than BMI ($R^2=8.4\%$), WC ($R^2=5.5\%$), and BMI+WC ($R^2=5.1\%$) (all $P<0.001$; Figure 1). All anthropometric measures were positively correlated with HbA1c ($P<0.001$). apVAT accounted for 16.1% of the variance in HbA1c. apVAT accounted for more variance in HbA1c than BMI ($R^2=11.8\%$), WC ($R^2=8.4\%$), and BMI+WC ($R^2=7.7\%$) (all $P<0.001$).

Laboratory Biomarkers of Inflammation

All anthropometric measures were positively correlated with CRP (Table E2), except in models of BMI+WC modeled simultaneously, in which BMI no longer retained statistical significance ($P=0.554$). apVAT accounted for 8.2% of the variance in CRP. apVAT explained more variance in CRP than BMI ($R^2=2.2\%$; $P=0.006$), but not more than WC ($R^2=0.6\%$; $P=0.426$) or BMI+WC ($R^2=0.6\%$; $P=0.429$). All anthropometric measures were positively correlated with fibrinogen ($P<0.001$; Figure 1), except in models of BMI+WC modeled simultaneously, in which BMI no longer retained statistical significance ($P=0.115$). apVAT accounted for 4.6% of the variance in fibrinogen. apVAT accounted for more variance in fibrinogen than BMI ($R^2=3.8\%$), WC ($R^2=3.1\%$), and BMI+WC ($R^2=2.9\%$) (all $P<0.001$).

Laboratory Biomarkers of Lipid Metabolism

All anthropometric measures were positively correlated with apolipoprotein B, except in models of BMI+WC modeled simultaneously, in which BMI was negatively correlated with apolipoprotein B ($P<0.001$; Table E3). apVAT accounted for 15.2% of the variance in apolipoprotein B. apVAT explained more variance in apolipoprotein B than BMI ($R^2=9.2\%$), WC ($R^2=4.8\%$), and BMI+WC ($R^2=4.1\%$) (all $P<0.004$; Figure 1). All anthropometric measures were negatively correlated with HDL cholesterol ($P<0.001$). apVAT accounted for 4.5% of the variance in HDL cholesterol. apVAT explained less variance in HDL cholesterol than BMI ($R^2=-2.9\%$), WC ($R^2=-2.9\%$), and BMI+WC ($R^2=-3.4\%$) (all $P<0.001$). All anthropometric measures were positively correlated with LDL cholesterol, except in models of BMI+WC modeled simultaneously, in which BMI was negatively correlated with LDL cholesterol ($P<0.001$). apVAT accounted for 7.4% of the variance in LDL cholesterol. apVAT explained more variance in LDL cholesterol than BMI ($R^2=5.5\%$), WC ($R^2=3.3\%$), and BMI+WC ($R^2=2.4\%$) (all $P<0.011$). All anthropometric measures were positively correlated with total cholesterol ($P<0.001$). apVAT accounted for 10.8% of the variance in total cholesterol. apVAT accounted for more variance in total cholesterol than BMI ($R^2=6.5\%$), WC ($R^2=5.2\%$), and BMI+WC ($R^2=3.7\%$) (all $P<0.001$). All anthropometric measures were also positively correlated with triglycerides ($P<0.005$). apVAT accounted for 17.3% of the variance in triglycerides. apVAT explained more variance in triglycerides than BMI ($R^2=4.8\%$; $P<0.001$), but not significantly more than WC ($R^2=0.7\%$; $P=0.433$) or BMI+WC ($R^2=0.6\%$; $P=0.448$).

Impaired Fasting Glucose & Metabolic Syndrome

apVAT more accurately predicted the presence of impaired fasting glucose and the metabolic syndrome compared with BMI, WC, or BMI+WC (all $P<0.001$; Figure 2).

DISCUSSION

The major finding from this study is that apVAT, estimated with simple and widely used non-invasive anthropometric measures, accounts for more variance in laboratory biomarkers related to glucose homeostasis, inflammation, and lipid metabolism than BMI, WC, and BMI+WC. The proportion of variance in laboratory biomarkers explained by apVAT ranged from 4.5% (HDL cholesterol) to 17.3% (triglycerides), which is similar prior studies using

VAT quantified with CT or MR imaging [14–16]. Our finding that apVAT accounts for more variance in blood-based laboratory biomarkers than BMI, WC, and BMI+WC, may explain, in part, the superior prognostic capacity of apVAT to predict mortality [8].

The adipocytes of VAT are physiologically and pathologically distinct from subcutaneous adipose tissue [3]. VAT is an endocrine organ that secretes and up-regulates various adipokines, cytokines, hormone-like factors, and other metabolites that have been associated with poor health outcomes [28]. Despite the known deleterious effects of excess VAT on health outcomes, the measurement of VAT until recently has required the use of CT or MR imaging, which is not portable, costly, and in the case of CT, involves radiation exposure [3]. Dual-energy x-ray absorptiometry (DXA) has recently been validated to quantify VAT [29, 30]. DXA is more cost-effective than CT and MR imaging, and associated with lower radiation exposure than CT; however, DXA suffers from limited portability. As a result of limitations in feasibility, high cost, and difficulty of use, simple anthropometric measures such as waist-hip ratio (WHR) and WC have gained popularity over imaging techniques as surrogate measures of VAT [31]. However, WHR and WC are not reliable individual indicators of VAT as they cannot distinguish VAT from subcutaneous adipose tissue, and the interpretation of these measures are often obscured by total body adiposity or BMI [6]. Another metric, the hyper-triglyceridemic waist, defined as the co-occurrence of a large WC and elevated fasting triglyceride concentration, is predictive of excess VAT [32]. Fasting triglyceride concentrations may be easily obtained in clinical practice, however the quantification triglyceride concentrations in large-scale epidemiologic studies may not be feasible due to complexity and cost of serum or plasma sample ascertainment and quantification. apVAT circumvents many of these common barriers, is practical for use in large clinical or epidemiologic studies, and its uptake and application will promote our continued understanding of the deleterious role of excess VAT on health outcomes.

Excess VAT is a key contributor to metabolic dysregulation [33], making it a strategic target for lifestyle and pharmacotherapies designed to improve metabolic health [1]. The cornerstone for the management of excess VAT includes lifestyle modification consisting of physical activity, weight loss through caloric restriction, and consumption of a healthful diet [34]. More recently, various pharmacotherapies have been developed as adjuncts to lifestyle modification to promote weight loss [35]. Our findings suggest that therapies that reduce apVAT may also improve glucose, inflammatory, and lipid profiles, as has been observed with exercise and diet-induced reductions in VAT quantified with CT [36]. Future research clarifying the importance of apVAT relative to BMI and WC are warranted given the potential health implications of reducing excess adiposity in the population.

There are limitations to this study. Our sample was limited to Caucasian males and females because the anthropometric equation for apVAT was only validated in a white population. However, the population-based sampling framework of NHANES permitted our analyses to represent the national population of Caucasian males and females living in the United States. Although the predictive equation used to estimate VAT in our study has been validated [7], its predictive accuracy has not been replicated in a validation cohort. Other anthropometric equations have been developed to predict CT or MR imaging derived VAT with reasonable accuracy (reviewed here: [37]), however few have been validated in external populations.

Our study did not measure VAT with CT or MR imaging, thus we were unable to empirically validate the apVAT equation. Consequently, the magnitude of variance in laboratory biomarkers explained by apVAT in our sample may be underestimated. The sample size of our study was relative large, providing us with adequate statistical power to examine the relationship between multiple anthropometric measures and various blood-based laboratory biomarker concentrations.

In conclusion, apVAT accounts for more variance in blood-based laboratory biomarkers related to glucose homeostasis, inflammation, and lipid metabolism than BMI, WC, and BMI+WC. This may explain, in part, why apVAT more accurately prognosticates all-cause and cause-specific mortality than BMI, WC, and BMI+WC. This recently-established anthropometric measure represents a non-invasive tool to assess the health effects of body composition in the population and may quantify the efficacy of interventions designed to improve body composition and cardio-metabolic health. Clinicians and researchers with knowledge of apVAT may consider utilizing this tool to characterize cardio-metabolic health, particularly in settings where there is limited availability of imaging and laboratory data.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Figure 1.
Proportion of variance explained by each anthropometric measure.

Figure 2. Discriminative characteristics for impaired fasting glucose (Panel A) and the metabolic syndrome (Panel B) by each anthropometric measure.

Table 1

Characteristics of study participants

Characteristic	Full Sample (N=10,624)	Male (N=5,076; 48.2%)	Female (N=5,548; 51.8%)	P
Demographic Variables				
Age, yr	44.6±0.21	43.8±0.30	45.4±0.30	<0.001
Behavioral Variables				
Smoking Status, %				<0.001
Never	44.2	35.2	52.6	
Former	27.4	33.3	21.9	
Current	28.4	31.5	25.5	
Health Eating Index, 0–100	64.2±0.19	62.8±0.27	65.3±0.25	<0.001
Alcoholic Consumption, drinks/wk	1.1±0.02	1.6±0.04	0.8±0.30	<0.001
Bouts of Weekly Walking, %				0.774
0	64.8	65.2	64.4	
1–3	16.2	15.8	16.6	
3	19.0	19.0	19.0	
Clinical Variables				
Medication Use, %				
Diabetes	3.3	3.5	3.1	0.443
Cholesterol	3.1	2.8	3.4	0.177
Hypertension	11.7	10.2	13.7	<0.001
Impaired Fasting Glucose, %	23.9	28.2	19.8	<0.001
Metabolic Syndrome, %	12.0	12.3	11.7	0.428
Anthropometric Variables				
Predicted Visceral Adipose Tissue, cm ²	163.5±1.03	186.7±1.55	141.9±1.21	<0.001
Body Mass Index, kg/m ²	26.4±0.08	26.6±0.10	26.2±0.12	0.003
Waist Circumference, cm	91.9±0.21	96.0±0.27	88.2±0.29	<0.001
Serum & Plasma Biomarkers				
<i>Glucose Homeostasis</i>				
Glucose, mmol/L	5.44±0.02	5.56±0.03	5.33±0.03	<0.001
HbA1c, %	5.3±0.01	5.4±0.02	5.3±0.02	<0.001
<i>Inflammation</i>				
C-Reactive Protein, mg/L	0.40±0.008	0.35±0.011	0.44±0.010	<0.001
Fibrinogen, umol/L	3.02±0.01	2.96±0.02	3.08±0.02	<0.001
<i>Lipid Metabolism</i>				
Apolipoprotein B, g/L	1.05±0.005	1.07±0.007	1.03±0.008	<0.001
HDL Cholesterol, mmol/L	1.30±0.006	1.17±0.008	1.43±0.008	<0.001
LDL Cholesterol, mmol/L	3.30±0.02	3.37±0.03	3.24±0.03	0.002
Total Cholesterol, mmol/L	5.29±0.01	5.24±0.02	5.35±0.02	0.001
Triglycerides, mmol/L	1.65±0.02	1.80±0.03	1.51±0.02	<0.001

Values are means (standard error) or column percentages (%).