



Published in final edited form as:

Am J Med. 2016 January ; 129(1): 74–81.e2. doi:10.1016/j.amjmed.2015.08.010.

Beyond Body Mass Index: Advantages of Abdominal Measurements for Recognizing Cardiometabolic Disorders

Henry S. Kahn, MD and Kai McKeever Bullard, PhD

Division of Diabetes Translation, Centers for Disease Control and Prevention, Atlanta, Ga

Abstract

BACKGROUND—The clinical recognition of cardiometabolic disorders might be enhanced by anthropometry based on the sagittal abdominal diameter (SAD; also called “abdominal height”) or waist circumference rather than on weight. Direct comparisons of body mass index (BMI, weight/height²) with SAD/height ratio (SADHtR) or waist circumference/height ratio (WHtR) have not previously been tested in nationally representative populations.

METHODS—Nonpregnant adults without diagnosed diabetes (ages 20–64 years; n = 3071) provided conventional anthropometry and supine SAD (by sliding-beam caliper) in the 2011–2012 US National Health and Nutrition Examination Survey. Population-weighted, logistic models estimated how strongly each anthropometric indicator was associated with 5 cardiometabolic disorders: *Dysglycemia* (glycated hemoglobin $\geq 5.7\%$), *HyperNonHDLc* (non-high-density-lipoprotein [HDL] cholesterol ≥ 4.14 mmol/L, or taking anticholesteremic medications), *Hypertension* (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg, or taking antihypertensive medications), *HyperALT* (alanine transaminase $\geq p75$ [75th percentile, sex-specific]), and *HyperGGT* (gamma-glutamyltransferase $\geq p75$ [sex-specific]).

RESULTS—After scaling each indicator, adjusted odds ratios (aORs) tended to be highest for SADHtR and lowest for BMI when identifying each disorder except dysglycemia. When SADHtR entered models simultaneously with BMI, the aORs for BMI no longer directly identified any condition, whereas SADHtR identified persons with HyperNonHDLc by aOR 2.78 (95% confidence interval [CI], 1.71–4.51), Hypertension by aOR 2.51 (95% CI, 1.22–5.15), HyperALT by aOR 2.89 (95% CI, 1.56–5.37), and HyperGGT by aOR 5.43 (95% CI, 3.01–9.79). WHtR competed successfully against BMI with regard to Dysglycemia, Hyper-NonHDLc, and HyperGGT. c-Statistics of SADHtR and WHtR were higher than those of BMI ($P < .001$) for identifying HyperNonHDLc and HyperGGT.

CONCLUSIONS—Among nonelderly adults, SADHtR or WHtR recognized cardiometabolic disorders better than did the BMI.

Requests for reprints should be addressed to Henry S. Kahn, MD, Division of Diabetes Translation, Centers for Disease Control and Prevention, CDC Mail Stop F-75, 4770 Buford Highway, NE, Atlanta, GA 30341. hkahn@cdc.gov.

Conflict of Interest: None.

Authorship: Both authors had access to the data and a role in writing the manuscript.

A portion of this report was presented as a moderated poster at the Epidemiology and Prevention/Lifestyle and Cardiometabolic Health, American Heart Association Scientific Session, Baltimore, MD, March 3–6, 2015.

SUPPLEMENTAL DATA

Supplemental tables accompanying this article can be found in the online version at <http://dx.doi.org/10.1016/j.amjmed.2015.08.010>.

Keywords

Body mass index; Epidemiologic measurements; Hypercholesterolemia; Hypertension; Sagittal abdominal diameter

Since 1980, epidemiologists and many clinicians have identified persons with excess adiposity by calculating their body mass index (BMI; kg/m²).¹ Nevertheless, commentators have pointed out that increased weight may represent lean tissues as well as fat mass,² or that adipose tissue accumulated in non-truncal regions (eg, gluteofemoral) may be relatively benign in comparison with adipose tissue in the abdominal depots.³ Some authorities recommend that the waist circumference be added to BMI as a primary tool for assessing adiposity.^{4,5}

CLINICAL SIGNIFICANCE

- For identifying 5 cardiometabolic disorders, body mass index was no better than sagittal abdominal diameter (SAD)/height or waist/height ratios.
- The SAD/height ratio may perform best for finding hypertension, elevated alanine transaminase, or gamma-glutamyltransferase.

During the past 2 decades, the prevalence of abdominal adiposity among US women and overweight men has increased, irrespective of BMI categories.⁶ This trend in body fat distribution suggests a need for alternative approaches to anthropometry in clinical and epidemiologic settings. Rather than concentrate on total body weight, examiners might focus on external measurements of the abdomen. The waist circumference has been widely employed as a proxy measure of abdominal adipose tissue,^{7,8} although the waist circumference/height ratio (WHtR) may represent an anthropometric improvement because it roughly controls for variation in adult stature.^{9–11} Neither waist circumference nor WHtR, however, distinguishes between adipose tissue accumulated within, as opposed to outside, the visceral compartment.

The supine sagittal abdominal diameter (SAD, also described as “abdominal height”) arguably estimates intra-abdominal adipose tissue better than the waist circumference.^{12–14} Recent studies reported that SAD was more strongly associated than waist circumference with men’s atherogenic lipoprotein subfractions¹⁵ or women’s insulin resistance.¹⁶ Studies in selected research samples have reported that the SAD/height ratio (SADHtR) also could serve to identify cardiometabolic risk.^{17,18}

Any discussion of whether to adopt WHtR or SADHtR more widely would first require confirmation that these indicators are indeed associated with known cardiometabolic risk factors in a representative population. Thereafter, it would help to demonstrate that these associations are independent of BMI, and that they might be of superior strength. In 2011–2012, the National Health and Examination Survey (NHANES) began measuring the supine SAD.¹⁹ Simultaneous measurements of weight, height, and waist circumference were obtained, along with clinical and laboratory information that established aspects of

cardiometabolic risk. These cross-sectional data drawn from a large, representative sample of nonelderly adults have allowed us to assess how well the adiposity indicators SADHtR, WHtR, and BMI are associated with 5 common cardiometabolic disorders.

METHODS

Survey Participants and Their Measurements

NHANES is an ongoing, nationally representative, cross-sectional survey of the resident civilian, noninstitutionalized, US population.²⁰ Participants chosen for NHANES undergo home interviews followed by standardized anthropometric and laboratory assessments in mobile examination centers. The NHANES protocol was approved by the Research Ethics Review Board of the National Center for Health Statistics; participants provided informed consent. Our report is restricted to participants from 2011–2012.

We excluded those who responded affirmatively to the question “Have you ever been told by a doctor or other health professional that you have diabetes or sugar diabetes?” (for women “...other than during pregnancy”). By restricting analysis to adults without diagnosed diabetes, we attempted to minimize confounding by behaviors or medication use that might influence adiposity indicators or the cardiometabolic disorders of interest. We defined adults as individuals ≥ 20 years old, but we excluded pregnant women and those ≥ 65 years out of concern primarily for survivorship bias. Also excluded were 787 eligible participants who lacked information on SAD, waist circumference, BMI, or the clinical information required to confirm the 5 cardiometabolic disorders described in the following paragraph. After these exclusions, our analytic sample included 3071 nonelderly, nonpregnant adults without diagnosed diabetes.

For analytic modeling, we chose as dependent variables 5 cardiometabolic disorders (Dysglycemia,²¹ HyperNonHDLc,^{22–24} Hypertension,^{25–27} HyperALT,^{28–30} and HyperGGT^{31–34}) that could be easily identified in the NHANES data (see Table 1 for the definition of each condition).

Online documents describe the pertinent laboratory and blood-pressure measurements acquired by NHANES.³⁵ Within our represented study population, the 75th percentile (p75) thresholds for HyperALT were 33 U/L for men, 22 U/L for women; for HyperGGT they were 33 U/L for men, 21 U/L for women. Blood pressure values were the means of second and third readings. Medication use was ascertained from responses to the questions “To lower your blood cholesterol, are you now following advice to take prescribed medicine?” or “Are you now taking prescribed medicine for your high blood pressure/hypertension?”

SAD was measured using a portable, sliding-beam caliper (Holtain, Ltd, Wales, UK).^{19,36} Supine participants rested on a lightly padded examination table with their hips in fixed position as the examiner marked the level of their iliac crests. The lower arm of the caliper was then positioned under the small of the back, and the upper arm was raised above the belly in alignment with their iliac-crest level. The examiner asked the participant to inhale gently, slowly let the air out, and then relax. The examiner then lowered the caliper’s upper arm, letting it lightly touch the abdomen but without compressing it. The SAD value was

read directly from a centimeter scale on the caliper shaft, recorded to the nearest 0.1 cm. For 94.4% of the participants we defined SAD as the mean of 2 initial measurements; for 5.6% we used the mean of up to 4 measurements.³⁷ Weight, height, and waist circumference (standing position, just above the iliac crest) were measured by established methods.³⁶

Statistical Analyses

NHANES selected participants through a complex, multistage-probability design requiring a sampling weight for each participant. We used SAS (release 9.3; SAS Institute Inc., Cary, NC) and SUDAAN (release 11.1; RTI International, Research Triangle Park, NC) to account for the complex design and sampling weights so that characteristics of the represented population could be correctly described.

We compared the strengths of the adiposity indicators using 3 approaches. Our initial approach prepared separate, sex-stratified, multivariable, logistic regression models (RLOGIST procedure of SUDAAN) to evaluate SADHtR, WHtR, or BMI as alternative independent variables. In order to compare the magnitude of adjusted odds ratios (aORs) from these alternative models, we scaled each adiposity indicator according to its sex-specific interquartile range (75th percentile [p75] minus 25th percentile [p25]) in the estimated population. Each model included adjustments for age (continuous), age,² and a quadratic term for the adiposity indicator. An additional term adjusted for self-identified race and ethnicity summarized in 5 mutually exclusive ancestral categories (non-Hispanic white, non-Hispanic black, non-Hispanic Asian, Hispanic, and “other, including multi-racial”). For outcome disorders HyperNonHDLc and Hypertension, we also repeated these sex-stratified modeling exercises after excluding persons who reported current use of the medication class prescribed to control that condition.

Our second approach tested whether an adiposity indicator might retain its independent association with the outcome while another indicator, despite recognized collinearity, was competing simultaneously in the model. For direct comparisons against the BMI, we prepared expanded logistic models in which the scaled BMI and the scaled SADHtR (along with their quadratic terms) were both participating. Similar logistic models tested the BMI in competition with the WHtR, or the SADHtR in competition with WHtR, for each cardiometabolic disorder. These models with 2 simultaneous indicators retained the covariates for age, age,² and ancestry. For the competing adiposity indicators in this report we present the simultaneous odds ratios based on the full sample of men and women. These models also included a covariate for sex, except for HyperALT and HyperGGT, in which outcome thresholds were defined in a sex-specific manner.

Our third approach calculated a c-statistic (area under the curve of the receiver operating characteristic [ROC]) and its jackknife variance. This approach covered a full range of discrimination thresholds for each continuous independent variable, and did not depend on scaling each indicator to its interquartile range. We used the “somersd” procedure in STATA (version 13; StataCorp LP, College Station, TX) to calculate each c-statistic for the sample of men plus women, with adjustment for 5 ancestral groups, using restricted cubic splines (3 knots) for each adiposity indicator and age. A sex adjustment was included when the

outcome condition was Dysglycemia, HyperNonHDLc, or Hypertension. We conducted pairwise comparisons of the c-statistics (goodness of fit) by the “lincom” function.

RESULTS

Population Distributions of Cardiometabolic Disorders and Adiposity Indicators

Prevalence estimates for the outcome disorders in our represented population ranged from 21.5% (Hypertension in women) to 42.4% (HyperNonHDLc in men) (Table 1). Table 2 shows the sex-stratified population distributions of age and adiposity indicators. Women tended to have a wider dispersion of the anthropometric variables, with the exception of height, as shown by their larger interquartile ranges.

Individual Adiposity Indicators Associated with Each Cardiometabolic Disorder (by Sex)

Each of the scaled adiposity indicators was associated with all 5 of the cardiometabolic disorders. Among men, the weakest associations were with Dysglycemia (aOR point estimates 1.59 to 1.60), and the strongest were with HyperALT (aORs 3.30 to 4.02) (Figure 1). Among women, the aORs fell in a narrower range (1.51 to 2.42) across all 5 of the cardiometabolic disorders.

For HyperNonHDLc, Hypertension, HyperALT, and HyperGGT, the aORs tended to be largest for the scaled SADHtR and lowest for scaled BMI. The aORs for scaled WHtR generally had intermediate values. (See Supplemental Table 1, available online for exact aORs and 95% confidence intervals [CIs] associated with each model). Among these 4 cardiometabolic disorders for men (Figure 1), the largest aOR was 4.02 (SADHtR to identify HyperALT), while the smallest aOR was 1.67 (BMI to identify HyperNonHDLc). For women, the largest aOR was 2.42 (SADHtR to identify HyperGGT), while the smallest was 1.51 (BMI to identify HyperNonHDLc).

With regard to outcome disorders other than Dysglycemia, some adiposity indicators had significant curvilinear relationships ($P < .05$ for quadratic terms). These instances are identified in Supplemental Table 1, available online).

When we excluded persons whose HyperNonHDLc was defined by currently taking anticholesteremic medication, the rankings of adiposity indicators were similar (ie, SADHtR > WHtR > BMI). In this analysis of a reduced population, the prevalence of HyperNonHDLc decreased to 36.4% for men, and their aORs were 2.24 for SADHtR, 2.14 for WHtR, and 1.70 for BMI. The prevalence of Hyper-NonHDLc decreased to 27.7% for women, and their aORs were 2.02 for SADHtR, 2.00 for WHtR, and 1.51 for BMI. When we excluded persons whose Hypertension was defined by currently taking antihypertensive medication, the prevalence of Hypertension in this reduced population decreased to 10.4% for men, and their aORs were 2.38 for SADHtR, 2.04 for WHtR, and 1.75 for BMI. The prevalence of Hypertension decreased to 6.1% for women, and their aORs were 1.73 for SADHtR, 1.64 for WHtR, and 1.59 for BMI.

Competing Adiposity Indicators Associated with Each Cardiometabolic Disorder (Sexes Combined)

In our multivariable logistic models with competing (simultaneous) terms for SADHtR and BMI, the BMI was unable to positively identify any of the cardiometabolic disorders. In this competing situation, SADHtR identified persons with Hyper-NonHDLc by aOR (95% CI) 2.78 (1.71–4.51), Hypertension by aOR 2.51 (1.22–5.15), HyperALT by aOR 2.89 (1.56–5.37), and HyperGGT by aOR 5.43 (3.01–9.79), but did not identify persons with Dysglycemia (aOR 1.44 [0.92–2.25]) (Figure 2 and Supplemental Table 2, available online). In these models based on the combined sexes, being male contributed to HyperNonHDLc (aOR 1.5; $P = .007$), but did not significantly influence the other 4 outcomes.

When WHtR competed simultaneously with BMI, once again BMI lost the ability to positively identify any of the cardiometabolic disorders (Figure 3 and Supplemental Table 2, available online). The WHtR identified persons with Dysglycemia by aOR 1.67 (95% CI, 1.06–2.62), HyperNonHDLc by aOR 3.87 (95% CI, 2.14–7.01), and HyperGGT by aOR 4.71 (95% CI, 1.68–13.2), but did not identify persons with Hypertension (aOR 1.93; 95% CI, 0.96–3.85) or with HyperALT (aOR 1.63; 95% CI, 0.77–3.44).

In models that directly compared SADHtR with WHtR, we found for all 5 cardiometabolic disorders that the aORs for WHtR were indistinguishable from 1.00 (Supplemental Table 2, available online). SADHtR likewise became a nonsignificant term for identifying Dysglycemia and HyperNonHDLc. However, in simultaneous competition with WHtR, SADHtR continued to identify Hypertension (aOR 2.42; 95% CI, 1.00–5.89), HyperALT (aOR 3.75; 95% CI, 1.81–7.74), and HyperGGT (aOR 3.06; 95% CI, 2.08–4.52).

c-Statistics for Individual Adiposity Indicators and Their Comparisons (Sexes Combined)

Calculated c-statistics (areas under the ROC curve [95% CI]) ranged from a high value of 0.812 [0.788–0.835] for Hypertension identified by SADHtR to a low value of 0.664 [0.636–0.691] for HyperGGT identified by BMI (Table 3). For identification of HyperNonHDLc and HyperGGT, the c-statistics for SADHtR and WHtR were higher ($P < .001$) than those for BMI. For all 5 of the outcomes, the c-statistics for SADHtR tended to be higher than those for WHtR, but these differences did not reach statistical significance.

DISCUSSION

In this representative, cross-sectional, population sample we found that the SADHtR and WHtR provided some advantages over BMI for identifying persons with 5 cardiometabolic disorders. SADHtR competed successfully against BMI for 4 of the 5 disorders (Figure 2, Supplemental Table 2, available online), and WHtR competed successfully against BMI for 3 of the 5 disorders (Figure 3, Supplemental Table 2, available online). We found no instances in which the BMI had advantages over SADHtR or WHtR.

In models that simultaneously compared SADHtR with WHtR, these 2 indicators were roughly equivalent for identifying Dysglycemia and HyperNonHDLc; however, SADHtR remained independently capable of identifying Hypertension, HyperALT, and HyperGGT (Supplemental Table 2, available online). When comparing their c-statistics (overall

goodness of fit), SADHtR and WHtR were each superior to BMI for identifying HyperNonHDLc or HyperGGT (Table 3).

SADHtR and WHtR are proxy indicators for enlargement of abdominal adipose tissue while roughly controlling for stature. However, it is expansion more specifically of the intra-abdominal adipose tissue that reflects the dyslipidemic, hormonal, and inflammatory features that constitute dysfunctional adiposity.^{5,7,38–40} SADHtR is presumably the better estimator of expanded intra-abdominal adipose tissue. By contrast, WHtR also incorporates an estimate of the abdominal subcutaneous adipose tissue, which generally performs relatively benign functions. Subcutaneous adipose tissue serves primarily as a metabolic sink during periods of caloric excess and as the source of necessary fuels when they are required.^{41,42} However, when subcutaneous adipose tissue does not adequately expand to sequester excessive circulating fatty acids, the intra-abdominal adipose tissue sites become further enlarged.^{27,43,44} Smaller depots of adipose tissue (eg, epicardial, perivascular, renal sinus) may likewise expand under these circumstances, and these expansions are associated with adverse consequences for, respectively, the heart, great vessels, or renal function.^{45,46} In addition, excessive caloric intake may be directed to organs (liver, skeletal muscle, pancreas, and myocardium) where “ectopic” lipid accumulation in these nonadipose tissues can alter usual cellular metabolism.^{47,48} The magnitude of lipid accumulation in these small depots or ectopic sites, however, cannot be estimated directly except by costly imaging or spectroscopic technologies. These considerations support the logic of quantifying dysfunctional adiposity primarily through an external, inexpensive estimate of intra-abdominal adipose tissue accumulation.

In recent years, external abdominal anthropometry has primarily depended on the waist circumference^{7,8} or the WHtR.^{9–11} Reports employing the SADHtR have been infrequent,^{17,18} but among Finnish adults (ages > 30 years) the population-based distribution of SADHtR has been described from a national survey conducted in 2000–2001.⁴⁹ The Finnish distributions of SADHtR resembled the US distributions in 2011–2012, reported here in Table 2.

While the SAD is an unfamiliar dimension to many clinicians, its measurement by a sliding-beam caliper can be no more complex or expensive than the accurate measurement of body weight. SAD requires an examination table and a portable caliper, whereas weight measurement requires the presence and regular calibration of a good-quality scale. For the SAD measurement, clothing must be loosened sufficiently to expose the mid-abdominal area, but there is no need to empty pockets or to remove outer garments, shoes, heavy jewelry, or most medical appliances. The amputation of a limb ordinarily limits the utility of a weight measurement, but this is rarely true for the SAD. The SAD protocol requires attention to the phases of respiration. Measurement of the standing waist circumference likewise requires attention to respiratory phases, along with methods to standardize the tension applied to the measuring tape and ensure its horizontal orientation at the specified position on the abdomen.

Our report is limited by the absence in NHANES 2011–2012 of circumferences measured at the hip or thigh. If these circumferences had been obtained, these data might have been used

to calculate the waist/hip ratio,⁵⁰ the waist-hip-height ratio,¹⁸ or the SAD/thigh circumference ratio.⁵¹ Each of these adiposity indicators has been reported to predict incident cardiovascular disease in prospective studies.

Anthropometry repeated over time could test the value of SADHtR or WHtR for monitoring changes in cardiometabolic risk, but NHANES does not include any longitudinal anthropometric data. A recent report from the MESA Study,⁴⁰ however, has demonstrated that changes of intra-abdominal adipose tissue (as measured by sequential computed tomography images) are superior to changes in weight for the prediction of future cardiometabolic outcomes.

We would like also to have considered outcome variables that are usually measured in the fasting state. However, fasting blood was obtained in NHANES only from participants attending the morning examination sessions. The reduced sample size for these fasting variables provided insufficient outcome information to yield reliable results.

The NHANES is an ongoing program providing public-access information collected in 2-year examination cycles from representative US population samples.²⁰ With the continued availability of SAD measurements in NHANES, researchers will have opportunities to validate our results with larger sample sizes and outcome biomarkers that require fasting blood samples. We anticipate increasing interest in the uses of abdominal anthropometry to estimate chronic-disease risks in the USA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding: None.

The authors acknowledge the participants in the 2011–2012 National Health and Nutrition Examination Survey (NHANES), and the efforts of the NHANES field staff and laboratory personnel.

All original data described in this article are available for public use through the Web site http://wwwn.cdc.gov/nchs/nhanes/search/nhanes11_12.aspx.

The findings and conclusions in this article are those of the authors and do not necessarily reflect the official position of the Centers for Disease Control and Prevention.

References

1. Kuczmarski RJ, Flegal KM. Criteria for definition of overweight in transition: background and recommendations for the United States. *Am J Clin Nutr.* 2000; 72(5):1074–1081. [PubMed: 11063431]
2. Garn SM, Leonard WR, Hawthorne VM. Three limitations of the body mass index. *Am J Clin Nutr.* 1986; 44(6):996–997. [PubMed: 3788846]
3. Landsberg L. Body fat distribution and cardiovascular risk: a tale of 2 sites. *Arch Intern Med.* 2008; 168(15):1607–1608. [PubMed: 18695073]
4. Cornier MA, Despres JP, Davis N, et al. Assessing adiposity: a scientific statement from the American Heart Association. *Circulation.* 2011; 124(18):1996–2019. [PubMed: 21947291]

5. Rao G, Powell-Wiley TM, Ancheta I, et al. Identification of obesity and cardiovascular risk in ethnically and racially diverse populations: a scientific statement from the American Heart Association. *Circulation*. 2015; 132(5):457–472. [PubMed: 26149446]
6. Ladabaum U, Mannalithara A, Myer PA, Singh G. Obesity, abdominal obesity, physical activity, and caloric intake in US adults: 1988 to 2010. *Am J Med*. 2014; 127(8):717–727. e12. [PubMed: 24631411]
7. Tchernof A, Despres JP. Pathophysiology of human visceral obesity: an update. *Physiol Rev*. 2013; 93(1):359–404. [PubMed: 23303913]
8. Katzmarzyk PT, Heymsfield SB, Bouchard C. Clinical utility of visceral adipose tissue for the identification of cardiometabolic risk in white and African American adults. *Am J Clin Nutr*. 2013; 97(3):480–486. [PubMed: 23364010]
9. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: Systematic review and meta-analysis. *Obes Rev*. 2012; 13(3):275–286. [PubMed: 22106927]
10. Roriz AKC, Passos LCS, de Oliveira CC, et al. Evaluation of the accuracy of anthropometric clinical indicators of visceral fat in adults and elderly. *PLoS One*. 2014; 9:e103499. [Accessed September 7, 2015] Available at: <http://dx.doi.org/10.1371/journal.pone.0103499>. [PubMed: 25078454]
11. Meseri R, Ucku R, Unal B. Waist:height ratio: a superior index in estimating cardiovascular risks in Turkish adults. *Public Health Nutr*. 2014; 17(10):2246–2252. [PubMed: 24103435]
12. Sjostrom, L., Lonn, L., Chowdhury, B., et al. The sagittal diameter is a valid marker of the visceral adipose tissue volume. In: Angel, A.Anderson, H.Bouchard, C.Lau, D.Leiter, L., Mendelson, R., editors. *Progress in Obesity Research: 7*. London: John Libbey; 1996. p. 309-319.
13. Jensen MD, Kanaley JA, Reed JE, Sheedy PF. Measurement of abdominal and visceral fat with computed tomography and dual-energy x-ray absorptiometry. *Am J Clin Nutr*. 1995; 61(2):274–278. [PubMed: 7840063]
14. Sampaio LR, Simoes EJ, Assis AM, Ramos LR. Validity and reliability of the sagittal abdominal diameter as a predictor of visceral abdominal fat. *Arq Bras Endocrinol Metabol*. 2007; 51:980–986. [Accessed September 7, 2015] Available at: <http://www.scielo.br/pdf/abem/v51n6/a13v51n6.pdf>. [PubMed: 17934666]
15. Nakata K, Choo J, Hopson MJ, et al. Stronger associations of sagittal abdominal diameter with atherogenic lipoprotein subfractions than waist circumference in middle-aged US white and Japanese men. *Metabolism*. 2010; 59(12):1742–1751. [PubMed: 20580038]
16. Vasques AC, Cassani RS, Forti AC, et al. Sagittal abdominal diameter as a surrogate marker of insulin resistance in an admixed population-Brazilian Metabolic Syndrome Study (BRAMS). *PLoS One*. 2015; 10:e0125365. [Accessed September 7, 2015] Available at: <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0125365>. [PubMed: 25951458]
17. Kumlin L, Dimberg L, Marin P. Ratio of abdominal sagittal diameter to height is strong indicator of coronary risk. *BMJ*. 1997; 314:830.
18. Carlsson AC, Riserus U, Engstrom G, et al. Novel and established anthropometric measures and the prediction of incident cardiovascular disease: a cohort study. *Int J Obes (Lond)*. 2013; 37(12):1579–1585. [PubMed: 23609935]
19. Kahn HS, Gu Q, Bullard KM, et al. Population distribution of the sagittal abdominal diameter (SAD) from a representative sample of US adults: Comparison of SAD, waist circumference and body mass index for identifying dysglycemia. *PLoS One*. 2014; 9:e108707. [Accessed September 7, 2015] Available at: <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0108707>. [PubMed: 25272003]
20. Centers for Disease Control and Prevention (CDC)/National Center for Health Statistics. [Accessed July 15, 2015] About the National Health and Nutrition Examination Survey. Available at: http://www.cdc.gov/nchs/nhanes/about_nhanes.htm
21. Ackermann RT, Cheng YJ, Williamson DF, Gregg EW. Identifying adults at high risk for diabetes and cardiovascular disease using hemoglobin A1c—National Health and Nutrition Examination Survey 2005–2006. *Am J Prev Med*. 2011; 40(1):11–17. [PubMed: 21146762]

22. Boekholdt SM, Arsenault BJ, Mora S, et al. Association of LDL cholesterol, non-HDL cholesterol, and apolipoprotein B levels with risk of cardiovascular events among patients treated with statins: a meta-analysis. *JAMA*. 2012; 307(12):1302–1309. [PubMed: 22453571]
23. Gaziano JM, Gaziano TA. What's new with measuring cholesterol? *JAMA*. 2013; 310(19):2043–2044. [PubMed: 24240929]
24. Expert Panel on Dyslipidemia. An International Atherosclerosis Society position paper: global recommendations for the management of dyslipidemia: Executive summary. *Atherosclerosis*. 2014; 232(2):410–413. [PubMed: 24468156]
25. Strazzullo P, Barba G, Cappuccio FP, et al. Altered renal sodium handling in men with abdominal adiposity: a link to hypertension. *J Hypertens*. 2001; 19(12):2157–2164. [PubMed: 11725158]
26. Rapsomaniki E, Timmis A, George J, et al. Blood pressure and incidence of twelve cardiovascular diseases: lifetime risks, healthy life-years lost, and age-specific associations in 1.25 million people. *Lancet*. 2014; 383(9932):1899–1911. [PubMed: 24881994]
27. Chandra A, Neeland IJ, Berry JD, et al. The relationship of body mass and fat distribution with incident hypertension: observations from the Dallas Heart Study. *J Am Coll Cardiol*. 2014; 64(10):997–1002. [PubMed: 25190234]
28. Lorenzo C, Hanley AJ, Rewers MJ, Haffner SM. The association of alanine aminotransferase within the normal and mildly elevated range with lipoproteins and apolipoproteins: the Insulin Resistance Atherosclerosis Study. *Diabetologia*. 2013; 56(4):746–757. [PubMed: 23344727]
29. Schindhelm RK, Dekker JM, Nijpels G, et al. Alanine aminotransferase predicts coronary heart disease events: a 10-year follow-up of the Hoorn Study. *Atherosclerosis*. 2007; 191(2):391–396. [PubMed: 16682043]
30. Schindhelm RK, Diamant M, Bakker SJ, et al. Liver alanine amino-transferase, insulin resistance and endothelial dysfunction in normotriglyceridaemic subjects with type 2 diabetes mellitus. *Eur J Clin Invest*. 2005; 35(6):369–374. [PubMed: 15948897]
31. Stranges S, Trevisan M, Dorn JM, et al. Body fat distribution, liver enzymes, and risk of hypertension: evidence from the Western New York Study. *Hypertension*. 2005; 46(5):1186–1193. [PubMed: 16203871]
32. Ghouri N, Preiss D, Sattar N. Liver enzymes, nonalcoholic fatty liver disease, and incident cardiovascular disease: a narrative review and clinical perspective of prospective data. *Hepatology*. 2010; 52(3):1156–1161. [PubMed: 20658466]
33. Elsurer R, Afsar B. Morning blood pressure surge is associated with serum gamma-glutamyltransferase activity in essential hypertensive patients. *J Hum Hypertens*. 2015; 29(5):331–336. [PubMed: 25355010]
34. Ruhl CE, Everhart JE. Fatty liver indices in the multiethnic United States National Health and Nutrition Examination Survey. *Aliment Pharmacol Ther*. 2015; 41(1):65–76. [PubMed: 25376360]
35. National Center for Health Statistics. [Accessed July 15, 2015] NHANES 2011–2012 Examination Data. Available at: <http://wwwn.cdc.gov/nchs/nhanes/search/datapage.aspx?Component=Examination&CycleBeginYear=2011>
36. National Center for Health Statistics. [Accessed July 15, 2015] Anthropometry Procedures Manual – National Health and Nutrition Examination Survey (NHANES). Available at: http://www.cdc.gov/nchs/data/nhanes/nhanes_13_14/2013_Anthropometry.pdf
37. National Center for Health Statistics. [Accessed July 15, 2015] NHANES 2011–2012 data documentation, codebook, and frequencies—body measures (BMX_G). Available at: http://www.cdc.gov/nchs/nhanes/nhanes2011-2012/BMX_G.htm#Analytic_Notes
38. Fox CS, Massaro JM, Hoffmann U, et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation*. 2007; 116(1):39–48. [PubMed: 17576866]
39. McArdle MA, Finucane OM, Connaughton RM, et al. Mechanisms of obesity-induced inflammation and insulin resistance: insights into the emerging role of nutritional strategies. *Front Endocrinol (Lausanne)*. 2013; 4:52. [Accessed September 7, 2015] Available at: <http://journal.frontiersin.org/Journal/10.3389/fendo.2013.00052/full>. [PubMed: 23675368]

40. Shah RV, Murthy VL, Abbasi SA, et al. Visceral adiposity and the risk of metabolic syndrome across body mass index: the MESA Study. *JACC Cardiovasc Imaging*. 2014; 7(12):1221–1235. [PubMed: 25440591]
41. Manolopoulos KN, Karpe F, Frayn KN. Gluteofemoral body fat as a determinant of metabolic health. *Int J Obes (Lond)*. 2010; 34(6):949–959. [PubMed: 20065965]
42. Karastergiou K, Fried SK. Multiple adipose depots increase cardiovascular risk via local and systemic effects. *Curr Atheroscler Rep*. 2013; 15(10):361. [PubMed: 23982264]
43. Hardy OT, Czech MP, Corvera S. What causes the insulin resistance underlying obesity? *Curr Opin Endocrinol Diabetes Obes*. 2012; 19(2):81–87. [PubMed: 22327367]
44. Schleinitz D, Bottcher Y, Bluher M, Kovacs P. The genetics of fat distribution. *Diabetologia*. 2014; 57(7):1276–1286. [PubMed: 24632736]
45. Fitzgibbons TP, Czech MP. Epicardial and perivascular adipose tissues and their influence on cardiovascular disease: basic mechanisms and clinical associations. *J Am Heart Assoc*. 2014; 3:e000582. [Accessed September 7, 2015] Available at: <http://jaha.ahajournals.org/content/3/2/e000582.full.pdf>. [PubMed: 24595191]
46. Foster MC, Hwang SJ, Porter SA, et al. Fatty kidney, hypertension, and chronic kidney disease: the Framingham Heart Study. *Hypertension*. 2011; 58(5):784–790. [PubMed: 21931075]
47. Shulman GI. Ectopic fat in insulin resistance, dyslipidemia, and cardiometabolic disease. *N Engl J Med*. 2014; 371(12):1131–1141. [PubMed: 25229917]
48. Borel AL, Nazare JA, Smith J, et al. Visceral, subcutaneous abdominal adiposity and liver fat content distribution in normal glucose tolerance, impaired fasting glucose and/or impaired glucose tolerance. *Int J Obes (Lond)*. 2015; 39(3):495–501. [PubMed: 25179244]
49. Kahn HS, Rissanen H, Bullard KM, Knekt P. The population distribution of the sagittal abdominal diameter (SAD) and SAD/height ratio among Finnish adults. *Clin Obes*. 2014; 4(6):333–341. [PubMed: 25826163]
50. Myint PK, Kwok CS, Luben RN, et al. Body fat percentage, body mass index and waist-to-hip ratio as predictors of mortality and cardiovascular disease. *Heart*. 2014; 100(20):1613–1619. [PubMed: 24966306]
51. Ehrlich AC, Smith DA. Abdominal diameter index and 12-year cardiovascular disease incidence in male bridge and tunnel workers. *Int J Obes (Lond)*. 2011; 35(3):409–415. [PubMed: 20714330]

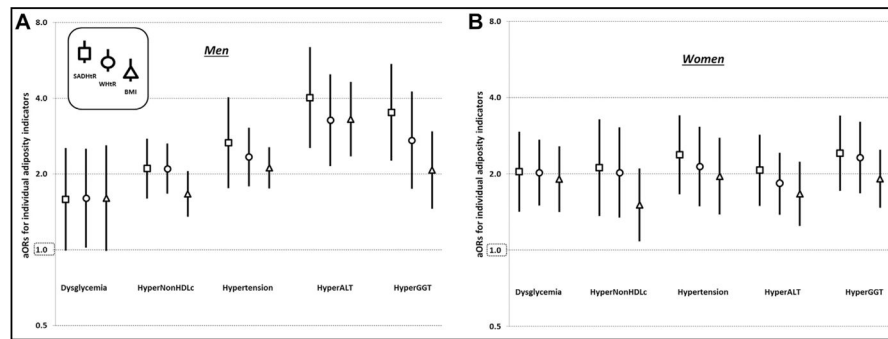


Figure 1. Comparisons in the population for men (panel A) and women (panel B) among SADHtR, WHtR, and BMI in association with 5 cardiometabolic disorders. Each adiposity indicator is scaled to the sex-specific interquartile range. Adjusted odds ratios (aOR; with 95% confidence intervals) are plotted logarithmically on the Y-axis. ALT = alanine transaminase; BMI = body mass index; GGT = gamma-glutamyltransferase; HDL = high-density lipoprotein; SADHtR = sagittal abdominal diameter/height ratio; WHtR = waist circumference/height ratio.

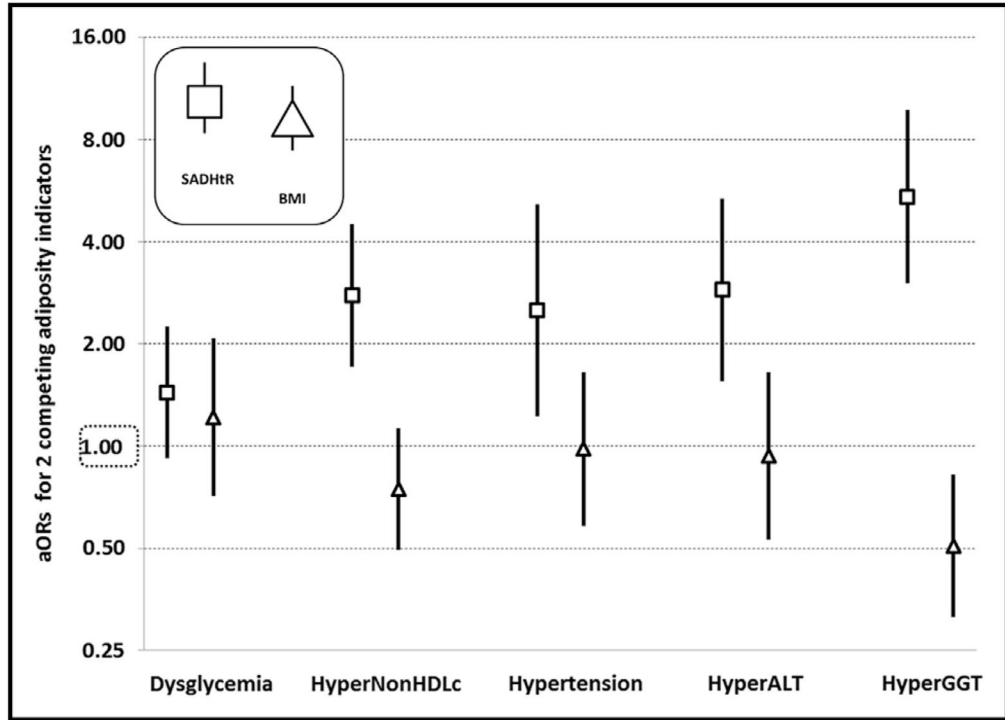


Figure 2. Comparisons of adjusted odds ratios (95% confidence intervals) for SADHtR and BMI as they compete simultaneously in models identifying 5 cardiometabolic disorders. The estimated population includes both sexes, and each adiposity indicator is scaled to its sex-specific interquartile range. Y-axis values are plotted logarithmically. aOR = adjusted odds ratio; BMI = body mass index; GGT = gamma-glutamyltransferase; HDL = high-density lipoprotein; SADHtR = sagittal abdominal diameter/height ratio.

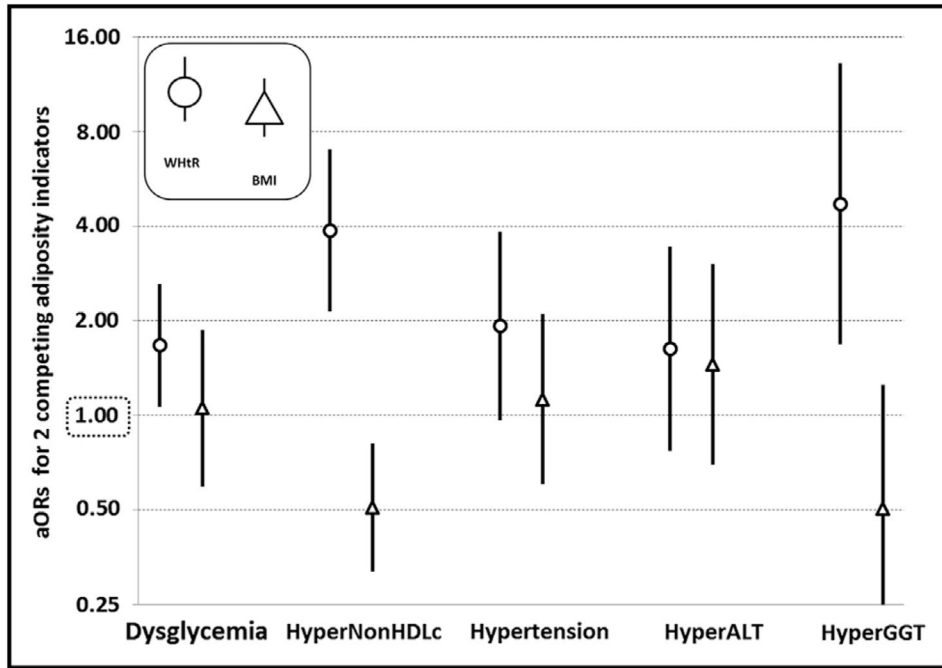


Figure 3. Comparisons of adjusted odds ratios (95% confidence intervals) for WHtR and BMI as they compete simultaneously in models identifying 5 cardiometabolic disorders. The estimated population includes both sexes, and each adiposity indicator is scaled to its sex-specific interquartile range. Y-axis values are plotted logarithmically. aOR = adjusted odds ratio; BMI = body mass index; GGT = gamma-glutamyltransferase; HDL = high-density lipoprotein; WHtR = waist circumference/height ratio.

Table 1
 Definitions and Population Prevalence of Five Cardiometabolic Disorders Among Nonelderly, Nonpregnant US Adults Without Diagnosed Diabetes, By Sex, in 2011–2012

	Prevalence in the Represented Population			
	Men		Women	
	n, Million	%	n, Million	%
Represented population	73.76	100.0	70.93	100.0
With Dysglycemia	16.24	22.0	15.55	21.9
With HyperNonHDLc	31.26	42.4	24.88	35.1
With Hypertension	16.40	22.2	15.25	21.5
With HyperALT	19.71	26.7	20.15	28.4
With HyperGGT	19.45	26.4	19.40	27.3

Criteria for Each Cardiometabolic Disorder

—				
Glycated hemoglobin (HbA1c)	5.7%			
Non-HDL-cholesterol	4.14 mmol/L or taking anticholesteremic medications			
SBP	140 mm Hg or DBP 90 mm Hg or taking antihypertensive medications			
Alanine transaminase (ALT)	p75, [sex-specific 75 th percentile]			
Gamma-glutamyltransferase (GGT)	p75, [sex-specific 75 th percentile]			

DBP = diastolic blood pressure; HDL = high-density lipoprotein; SBP = systolic blood pressure.

Population Distribution of Age and Adiposity Indicators Among Nonelderly, Nonpregnant US Adults, Without Diagnosed Diabetes, By Sex, 2011–2012

Table 2

	Men (n = 1575)					Women (n = 1496)				
	Quartile Outpoints					Quartile Outpoints				
	Mean	p25	p50	p75	IQR	Mean	p25	p50	p75	IQR
Age, y	41.0	29.3	40.3	51.1	21.8	42.1	30.4	42.3	52.2	21.8
SAD, cm	22.8	19.8	22.2	25.3	5.5	21.3	18.2	20.7	23.9	5.7
WC, cm	99.4	89.1	98.1	108.1	18.9	94.6	82.7	92.6	103.9	21.2
Height, cm	176.5	171.4	176.1	181.4	10.0	163.2	158.1	163.2	167.8	9.6
SADHtR	0.129	0.112	0.126	0.144	0.032	0.131	0.112	0.126	0.148	0.036
WHtR	0.564	0.505	0.556	0.613	0.108	0.580	0.510	0.569	0.636	0.126
BMI, kg/m ²	28.2	24.2	27.5	31.0	6.8	28.3	23.4	26.9	31.7	8.3

BMI = body mass index; IQR = interquartile range; SAD = sagittal abdominal diameter; SADHtR = sagittal abdominal diameter/height ratio; WC = waist circumference; WHtR = waist circumference/height ratio.

Table 3
 c-Statistics (Areas Under the Curve of the Receiver Operating Characteristic) for Identifying 5 Cardiometabolic Disorders by 3 Adiposity Indicators Adjusted for Age, Sex, and Ancestry

Cardiometabolic Disorder	c-Statistic (Area Under ROC Curve)					P-Value* for Difference in Areas		
	SADHR	WHGR	BMI	SADHR vs BMI	WHGR vs BMI	WHGR vs BMI	SADHR vs WHGR	SADHR vs WHGR
Dysglycemia	0.768	0.768	0.766	.56	.40		.90	
95% CI	.744-.792	.744-.792	.743-.790					
HyperNonHDLc	0.767	0.766	0.753	<.001	<.001		.73	
95% CI	.744-.791	.743-.790	.730-.777					
Hypertension	0.812	0.808	0.807	.13	.52		.19	
95% CI	.788-.835	.785-.832	.783-.831					
HyperALT [‡]	0.685	0.673	0.673	.054	.85		.034	
95% CI	.658-.712	.647-.701	.646-.699					
HyperGGT [‡]	0.695	0.687	0.664	<.001	<.001		.14	
95% CI	.668-.722	.660-.714	.636-.691					

BMI = body mass index; CI = confidence interval; ROC = receiver operating characteristic; SADHR = sagittal abdominal diameter/height ratio; WHGR = waist circumference/height ratio.

These population estimates were calculated for nonelderly, nonpregnant US adults without diagnosed diabetes in 2011–2012.

* P-values are presented without Bonferroni corrections; when testing 3 hypotheses, statistical significance may be established conservatively at $\alpha = 0.017$.

[‡] Models for HyperALT and HyperGGT do not include a term for sex adjustment.