



## Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association<sup>1-4</sup>

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### INTRODUCTION

Obesity is an important risk factor for cardiometabolic diseases, including diabetes, hypertension, dyslipidemia, and coronary heart disease (CHD). Several leading national and international institutions, including the World Health Organization and the National Institutes of Health, have provided guidelines for classifying weight status based on body mass index (BMI; in kg/m<sup>2</sup>) (1, 2). Data from epidemiologic studies demonstrate a direct correlation between BMI and the risk of medical complications and mortality rate (eg, 3, 4). Men and women who have a BMI  $\geq$  30 are considered obese and are generally at higher risk for adverse health events than are those who are considered overweight (BMI between 25.0 and 29.9) or lean (BMI between 18.5 and 24.9). Therefore, BMI has become the gold standard for identifying patients at increased risk of adiposity-related adverse health outcomes.

Body fat distribution is also an important risk factor for obesity-related diseases. Excess abdominal fat (also known as central or upper-body fat) is associated with an increased risk of cardiometabolic disease. However, precise measurement of abdominal fat content requires the use of expensive radiological imaging techniques. Therefore, waist circumference (WC) is often used as a surrogate marker of abdominal fat mass, because WC correlates with abdominal fat mass (subcutaneous and intraabdominal) (5) and is associated with cardiometabolic disease risk (6). Men and women who have WCs  $>$  40 in (102 cm) and 35 in (88 cm), respectively, are considered to be at increased risk for cardiometabolic disease (7). These cutpoints were derived from a regression curve that identified the WC values associated with a BMI  $\geq$  30 in primarily Caucasian men and women living in north Glasgow, Scotland (8).

An expert panel, organized by the National Heart, Lung, and Blood Institute (NHLBI), has recommended that WC be measured as part of the initial assessment and be used to monitor the

efficacy of weight-loss therapy in overweight and obese patients who have a BMI  $<$  35 (7). However, measurement of WC has not been widely adopted in clinical practice, and the anatomical, metabolic, and clinical implications of WC data can be confusing. Therefore, Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; and the American Diabetes Association convened a panel, composed of members with expertise in obesity management, obesity-related epidemiology, adipose tissue metabolic pathophysiology, statistics, and nutrition science, to review the published scientific literature and hear presentations from other experts in these fields. The Consensus Panel met

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December 17–20, 2006, in Washington, DC, and was charged to provide answers to the following 4 questions:

1. What does waist circumference measure?
2. What are the biological mechanisms responsible for the association between waist circumference and cardiometabolic risk?
3. What is the power of waist circumference to predict adverse cardiometabolic outcomes? How does the predictive power of waist circumference compare with that of BMI? Does measuring waist circumference in addition to BMI improve predictability?
4. Should waist circumference be measured in clinical practice?

## QUESTION 1: WHAT DOES WAIST CIRCUMFERENCE MEASURE?

### Measurement technique

WC is actually a perimeter, which provides an estimate of body girth at the level of the abdomen. Different anatomic landmarks have been used to determine the exact location for measuring WC in different clinical studies, including 1) midpoint between the lowest rib and the iliac crest; 2) the umbilicus; 3) narrowest (minimum) or widest (maximum) WC; 4) just below the lowest rib; and 5) just above the iliac crest. The specific site used to measure WC influences the absolute WC value that is obtained (9). The most commonly used sites reported in studies that evaluated the relation between morbidity or mortality rate and WC were the midpoint between the lowest rib and the iliac crest (29%), the umbilicus (28%), and the narrowest WC (22%). Although sites that use an easily identifiable and reproducible landmark (eg, just above the bony landmark of the iliac crest) might be more precise and easier to use than other sites, we are not aware of data from any studies that demonstrate an advantage of one measurement site over others.

WC measurements should be made around a patient's bare midriff, after the patient exhales while standing without shoes and with both feet touching and arms hanging freely. The measuring tape should be made of a material that is not easily stretched, such as fiberglass. The tape should be placed perpendicular to the long axis of the body and horizontal to the floor and applied with sufficient tension to conform to the measurement surface. In a research setting, WC measurements are typically taken 3 times and recorded to the nearest 0.1 cm. Although specific techniques have been recommended for measuring WC in the clinical setting (2, 10), there is no uniformly accepted approach. Training technicians and even patients to use an appropriate technique for measuring WC is essential to obtaining reliable data; special tape measures, instructional manuals, and videotapes are available for this purpose (11).

The reproducibility of WC measurements at all sites is high for both men and women (eg, iliac crest site, intraclass correlation coefficient,  $r = 0.998$  and  $r = 0.999$ , respectively) (9, 12, 13). The correlation between technician- and self-measured WC after proper training can also be high for men ( $r = 0.95$ ) and women (0.89), respectively (14). However, self-reported measurements are prone to a systematic bias, and there is a nontrivial underestimate of self-measured WC at all anatomic sites (15).

**TABLE 1**  
Distribution of adipose tissue mass in lean and obese men<sup>1</sup>

	Lean men	Obese men
BMI (kg/m <sup>2</sup> )	23	37
Body weight (kg)	71	116
Body fat (%)	15	32
Total body fat (kg)	10	37
Total subcutaneous fat (kg)	9	32
Abdominal fat (kg)	4.3	12.3
Subcutaneous (kg)	2.4	7.2
Intraabdominal (kg)	1.9	5.1
Intraperitoneal (kg)	1.1	3.5
Retroperitoneal (kg)	0.8	1.6

<sup>1</sup> Adapted from Reference 16.

### Anatomic relations

Adipose tissue consists of adipocytes, inflammatory cells, and vascular, connective, and neural tissues. Adipose tissue is distributed throughout the body as large homogeneous discrete compartments and as small numbers of cells "marbling" or adjacent to other tissues. Most adipose tissue (~85% of total adipose tissue mass) is located under the skin (subcutaneous fat), and a smaller amount (~15%) is located within the abdomen (intra-abdominal fat) in lean and obese persons (Table 1) (16). The relative contribution of intra-abdominal fat mass to total body fat is influenced by sex, age, race-ethnicity, physical activity, and total adiposity. The term "visceral fat" is commonly used to describe intra-abdominal fat, and it includes both intraperitoneal fat (mesenteric and omental fat), which drains directly into the portal circulation, and retroperitoneal fat, which drains into the systemic circulation.

Magnetic resonance imaging (MRI) and computed tomography (CT) are considered the gold-standard methods for determining the quantity of subcutaneous abdominal adipose tissue (SAAT) and intra-abdominal adipose tissue (IAAT) (17). Most MRI and CT methods involve acquisition of cross-sectional abdominal images, which are then analyzed for fat content. A single slice is often acquired at the L<sub>4</sub>-L<sub>5</sub> inter-vertebral level to estimate SAAT and IAAT volume, expressed as cm<sup>3</sup>. However, L<sub>4</sub>-L<sub>5</sub> imaging does not provide the best estimate of total IAAT mass, which is more reliably estimated several centimeters cephalad of the L<sub>4</sub>-L<sub>5</sub> inter-vertebral space (17, 18). In addition, measurement site influences the relation between IAAT volume and cardiometabolic risk; the association between IAAT volume and the presence of the metabolic syndrome is greater when IAAT volume is determined at the L<sub>1</sub>-L<sub>2</sub> than at the L<sub>4</sub>-L<sub>5</sub> level (19). Currently, there is no universally accepted site for measuring IAAT and SAAT.

The relation between WC, weight, and BMI can be conceptualized by using simple geometric relations that consider the body as a cylinder; WC is the cylinder's circumference, height is its length, and weight is a measure of mass. Therefore, BMI provides information about body volume and mass, and WC provides information about body shape. In general, BMI and WC are highly correlated, typically with  $r$  values in the range of 0.80–0.95 (20), and WC reflects both SAAT and IAAT volumes (21). The relations among WC, BMI, and adipose tissue compartments in primarily Caucasian and African American men and women are shown in Table 2 (18). These data demonstrate that both BMI



**TABLE 2**

Relationships among waist circumference, BMI, and adipose tissue compartments in men and women<sup>1</sup>

	Men		Women	
	BMI	Waist circumference	BMI	Waist circumference
Total adipose tissue	0.82	0.87	0.91	0.87
Percentage body fat	0.70	0.79	0.86	0.82
Total subcutaneous adipose tissue	0.82	0.83	0.91	0.86
Total intraabdominal adipose tissue	0.59	0.79	0.69	0.77

<sup>1</sup> Values are correlation coefficients. Adapted from Reference 18.

and WC are strongly correlated with total-body adipose tissue mass, but that WC is a better predictor of IAAT than is BMI.

Assessment of WC provides a measure of fat distribution that cannot be obtained by measuring BMI. However, there is no standardized approach for measuring WC, and different anatomic landmarks have been used to measure WC in different studies. Moreover, the measurement site that provides the best correlation with disease risk and best reflects changes in abdominal adipose tissue mass has not been established. Nonetheless, the precision of WC measurement is high at any given landmark. Even self-measurement can be highly reproducible when performed by properly trained subjects, although self-measurement results in an underestimation of true WC. Measurement of WC cannot determine the individual contributions of SAAT and IAAT to abdominal girth, which require imaging by MRI or CT. The value of these scanning techniques in clinical practice has not been determined.

**QUESTION 2: WHAT ARE THE BIOLOGICAL MECHANISMS RESPONSIBLE FOR THE ASSOCIATION BETWEEN WAIST CIRCUMFERENCE AND METABOLIC AND CARDIOMETABOLIC RISK?**

It is not known whether the storage of an absolute or relative excess amount of triacylglycerols in abdominal fat depots is directly responsible for increased disease risk, whether such deposition is simply associated with other processes that cause risk, or both. In addition, WC values provide a measure of both SAAT and IAAT masses. Therefore, the relation between WC and cardiometabolic risk cannot determine whether risk is associated with SAAT, IAAT, or both.

The mechanism(s) responsible for the relation between excess abdominal fat distribution and cardiometabolic disease is not known, but several hypotheses have been proposed. One of the earliest hypotheses that implicated IAAT as a metabolic risk factor suggested that activation of the central nervous system–adrenal axis by environmental stressors caused both the preferential deposition of adipose tissue in the trunk and the cardiovascular and metabolic disorders associated with that deposition (22). More recently, it has been suggested that a limited ability of subcutaneous fat depots to store excess energy results in an “overflow” of chemical energy to IAAT and “ectopic” sites, such as liver and skeletal muscle. Excessive ectopic fat accumulation then causes metabolic dysfunction in those organs. In fact, increased intrahepatic fat is associated with dyslipidemia and hepatic insulin resistance (23), and increased intramyocellular fat is associated with skeletal muscle insulin resistance (24). In this

paradigm, IAAT is primarily a marker of the magnitude of overflow of fatty acids from subcutaneous depots. Therefore, increased WC could be a discernible marker of a systemwide impairment in energy storage regulation, in which an increase in IAAT reflects a reduced capacity for energy storage in other adipose tissues. A third hypothesis proposes a direct effect of omental and mesenteric adipose tissue depots on insulin resistance, lipoprotein metabolism, and blood pressure. Metabolic products of omental and mesenteric adipose tissue depots are released into the portal vein, which provides direct delivery to the liver. Lipolysis of omental and mesenteric adipose tissue triacylglycerols releases free fatty acids that can induce hepatic insulin resistance and provide substrate for lipoprotein synthesis and neutral lipid storage in hepatocytes. In addition, specific proteins and hormones produced by omental and mesenteric adipose tissue, such as inflammatory adipokines, angiotensinogen, and cortisol (generated by local activity of 11  $\beta$ -hydroxysteroid dehydrogenase), can also contribute to cardiometabolic disease. A fourth hypothesis is that genes that predispose to preferential deposition of fat in abdominal depots independently cause cardiometabolic disease.

These hypotheses are not mutually exclusive, and it is possible that all, and other unknown mechanisms, are involved in the association between abdominal fat mass and adverse metabolic consequences.

**QUESTION 3: WHAT IS THE POWER OF WAIST CIRCUMFERENCE TO PREDICT ADVERSE CARDIOMETABOLIC OUTCOMES? HOW DOES THE PREDICTIVE POWER OF WAIST CIRCUMFERENCE COMPARE WITH THAT OF BMI? DOES WAIST CIRCUMFERENCE MEASUREMENT IN ADDITION TO BMI IMPROVE PREDICTABILITY?**

The importance of WC in predicting cardiometabolic risk factors (eg, elevated blood pressure, dyslipidemia, and hyperglycemia) and adverse outcomes (eg, diabetes, CHD, and death rate) has been examined in many large epidemiologic studies (7, 24–33). Specific relative risks between WC and these outcomes vary, depending on the population sampled and the outcome measured. The relation between WC and clinical outcome is consistently strong for diabetes risk, and WC is a stronger predictor of diabetes than is BMI. The relative risk of developing diabetes between subjects in the highest and lowest categories of reported WC often exceeds 10, and it remains statistically significant after adjustment for BMI. These data demonstrate that WC can identify persons who are at greater cardiometabolic risk than are those



identified by BMI alone. Values for WC are also consistently related to the risk of developing CHD, and the relative risk of developing CHD between subjects in the highest and lowest categories of WC ranges from 1.5 to 2.5 and remains statistically significant after adjustment for BMI. Values for WC are usually strongly associated with all-cause and selected cause-specific mortality rates. Data from several studies support the notion that WC is an important predictor of diabetes, CHD, and mortality rate, independent of traditional clinical tests such as blood pressure, blood glucose, and lipoproteins (7, 26). However, there is not yet a compelling body of evidence demonstrating that WC provides clinically meaningful information that is independent of well-known cardiometabolic risk factors.

The relations between WC and health outcomes are affected by demographic variables, including sex, race-ethnicity, and age. WC is an important predictor of health outcomes in men and women; Caucasians, African Americans, Asians, and Hispanics; and adults of all age groups. In fact, the relation between WC and health outcome changes much less with increasing age than does the relation between BMI and health outcome (31). However, it is not known whether WC can provide a better assessment of health risk in one sex, racial-ethnic group, or age category than another.

The shape of the relation between WC and health outcomes (eg, linear, monotonic, step-function, or U-shaped) influences the WC value that can most efficiently distinguish between “normal” and “abnormal” and serve as a basis for considering clinical action. Data from most studies suggest that the shape of the relation between WC and health outcome lends itself to identifying clinically meaningful cutpoint values, because risk often accelerates monotonically above, and can be relatively flat below, a specific WC value. Optimum WC cutpoints will likely vary according to the population studied, the health outcome of interest, and demographic factors.

Data from most clinical weight-loss and exercise training trials have shown that reductions in WC occur concomitantly with reductions in obesity-related cardiometabolic risk factors and disease. However, these results do not prove that the reduction in WC was responsible for the beneficial effect on health outcome. Additional studies are needed to evaluate the effect of decreasing WC on cardiometabolic outcomes.

#### QUESTION 4: SHOULD WAIST CIRCUMFERENCE BE MEASURED IN CLINICAL PRACTICE?

The panel concluded that determining whether WC should be measured in clinical practice depends on the responses to the following 4 key questions:

1. Can waist circumference be reliably measured? Answer: **Yes.**

Health care personnel and even patients themselves, who are given appropriate training in technique, can provide highly reproducible measurements of WC in men and women. However, it is not known whether measurement at one anatomical site is a better indicator of cardiometabolic risk than is measurement at other sites.

2. Does waist circumference provide *a*) good prediction of diabetes, CHD, and mortality rate? Answer: **Yes;** *b*) incremental value in predicting diabetes, CHD, and mortality rate above and beyond that provided by BMI? Answer: **Yes;** and *c*) sufficient incremental value in these predictions above and beyond that

offered by BMI and commonly evaluated cardiometabolic risk factors, such as blood glucose concentration, lipid profile, and blood pressure? Answer: **Uncertain.**

Data from many large population studies have found WC to be a strong correlate of clinical outcome, particularly diabetes, and to be independent of BMI. In addition, data from a limited number of studies demonstrate that WC remains a predictor of diabetes, CHD, and mortality rate, even after adjustment for BMI and several other cardiometabolic risk factors. Additional studies are needed to confirm that WC remains an independent predictor of risk.

3. Do the current definitions used to determine a high WC identify a nontrivial number of patients who are at increased cardiometabolic risk, but who would not otherwise be identified by having a BMI  $\geq 25$  and an assessment of commonly evaluated cardiometabolic risk factors? Answer: **Yes.**

The recommended WC thresholds for increased cardiometabolic risk in men [ $>40$  in (102 cm)] and women [ $>35$  in (88 cm)] were derived from WC values that correlated with a BMI  $\geq 30$  (2). The National Health and Nutrition Examination Survey III (NHANES III) found that about 14% of women and about 1% of men had a “high” WC but a normal BMI (18.5–24.9) (36). In addition,  $\approx 70\%$  of women who were overweight (BMI 25.0–29.9) had a WC  $> 35$  in and  $\approx 25\%$  of men who were overweight had a WC  $> 40$  in. An estimate based on data available from the World Health Organization’s Monica Project, conducted in  $>32\,000$  men and women from Europe, Australia, and New Zealand, suggest that about 10% of participants who had a BMI  $< 30$  had a WC above the recommended cutpoints for increased risk (36). It is not known what portion of subjects who had a large WC would have been identified as having increased cardiometabolic risk based on findings from a standard medical evaluation. Therefore, the optimal WC criteria needed to identify patients at increased risk of metabolic disease, who would otherwise not be identified by evaluating BMI and/or other standard cardiometabolic risk factors, is not known and will likely require adjustments based on BMI, sex, age, and race-ethnicity.

4. Would assessment of WC in patients who have a BMI  $\geq 25$  affect clinical management if NHLBI obesity treatment guidelines are followed? Answer: **Probably not.**

Measurement of WC in clinical practice is not trivial, because providing this assessment competes for the limited time available in a busy office practice and requires specific training to ensure that reliable data are obtained. Therefore, WC should only be measured if it can provide additional information that influences patient management. Based on NHANES III data, 99.9% of men and 98.49% of women would have received the same treatment recommendations proposed by the NHLBI Expert Panel by evaluating BMI and other cardiovascular risk factors, without an assessment of WC (37). However, it is likely that different WC cutpoint values could provide more useful clinical information. For example, an analysis of data obtained from the NHANES III and the Canadian Heart Health Surveys found that BMI-specific WC cutpoints provided a better indicator of cardiometabolic risk than did the recommended WC thresholds (35). For normal-weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), class I obesity (BMI 30.0–34.9) and class II/class III obesity (BMI  $\geq 35.0$ ), the optimal WC cutpoints were 87, 98, 109, and 124 cm in men and 79, 92, 103, and 115 cm in women, respectively. Therefore, it is possible that WC measurement could be an effective clinical tool for identifying “metabolically obese, lean” patients,

who might benefit from lifestyle therapy but would not have been considered for treatment because of a normal BMI. WC could also identify “metabolically normal, obese” subjects, who do not require aggressive obesity therapy because they do not have a marked increase in cardiometabolic risk.

**CONCLUSIONS**

WC provides a unique indicator of body fat distribution, which can identify patients who are at increased risk of obesity-related cardiometabolic disease, above and beyond the measurement of BMI. However, the current WC cutpoints recommended to determine health risk (2) were derived by regression from an “obese” BMI and are unlikely to affect clinical management when BMI and other obesity-related cardiometabolic risk factors are already being determined. Therefore, the clinical usefulness of measuring WC, when risk is based on the currently accepted guidelines, is limited. However, WC measurement can sometimes provide additional information to help the clinician determine which patients should be evaluated for the presence of cardiometabolic risk factors, such as dyslipidemia, and hyperglycemia. In addition, measuring WC can be useful in monitoring a patient’s response to diet and exercise treatment, because regular aerobic exercise can cause a reduction in both WC and cardiometabolic risk, without a change in BMI (38). Further studies are needed to establish WC cutpoints that can assess cardiometabolic risk that is not adequately captured by BMI and routine clinical assessments. Selection of the most appropriate WC values will be complex, because they are likely influenced by sex, race-ethnicity, age, BMI, and other factors. Nonetheless, it should be possible to determine more useful WC cutpoints than are currently recommended, by carefully reviewing published data and re-evaluating datasets available from existing population studies. These additional analyses will define the future role of WC measurement in clinical practice.

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