

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

SAMUEL KLEIN, MD¹
DAVID B. ALLISON, PHD²
STEVEN B. HEYMSFIELD, MD³
DAVID E. KELLEY, MD⁴

RUDOLPH L. LEIBEL, MD⁵
CATHY NONAS, MS, RD, CDE⁶
RICHARD KAHN, PHD⁷

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 (WHO) and the National Institutes of Health, have provided guidelines for classifying weight status based on BMI (1,2). Data from epidemiological studies demonstrate a direct correlation between BMI and the risk of medical com-

plications and mortality rate (e.g., 3,4). Men and women who have a BMI ≥ 30 kg/m² 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 kg/m²) or lean (BMI between 18.5 and 24.9 kg/m²). Therefore, BMI has become the "gold standard" for identifying patients at increased risk for adiposity-related adverse health outcomes.

Body fat distribution is also an impor-

tant 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 intra-abdominal) (5) and is associated with cardiometabolic disease risk (6). Men and women who have waist circumferences greater than 40 inches (102 cm) and 35 inches (88 cm), respectively, are considered to be at increased risk for cardiometabolic disease (7). These cut points were derived from a regression curve that identified the waist circumference values associated with a BMI ≥ 30 kg/m² in primarily Caucasian men and women living in north Glasgow (8).

An expert panel, organized by the National Heart, Lung and Blood Institute, 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 kg/m² (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, comprised 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 from December 17 to 20, 2006, in Wash-

From the ¹Division of Geriatrics and Nutritional Science, Center for Human Nutrition, Washington University School of Medicine, St. Louis, Missouri; the ²Clinical Nutrition Research Unit, University of Alabama at Birmingham, Birmingham, Alabama; the ³Clinical Research Department, Metabolism, Merck Pharmaceutical Company, Rahway, New Jersey; the ⁴Obesity and Nutrition Research Center, University of Pittsburgh, Pittsburgh, Pennsylvania; the ⁵Naomi Berrie Diabetes Center, Columbia University, New York, New York; the ⁶Obesity and Diabetes Programs, North General Hospital, New York, New York; and the ⁷American Diabetes Association, Alexandria, Virginia.

Address correspondence and reprint requests to Samuel Klein, MD, Washington University School of Medicine, 660 South Euclid Ave., Campus Box 8031, St. Louis, MO 63110. E-mail: sklein@wustl.edu.

Approved for publication 7 March 2007.

D.B.A. has received research grants from Frito-Lay and OMP; has served as a consultant to Kraft Foods, Pfizer, Bristol-Myers Squibb, and Bio Era; and has received financial support from Lilly, Pfizer, Merck Pharmaceutical Company, Unilever, Coca-Cola, General Mills, International Life Sciences Institute, Glaxo-SmithKline, OMP, Jansen Pharmaceuticals, and Frito-Lay. S.K. has received research grants from Sanofi-Aventis, Merck, and Takeda for clinical trials; has served as a consultant to Sanofi-Aventis, Amylin Pharmaceuticals, EnteroMedics, Dannon-Yakult, and Merck Pharmaceutical Company. S.B.H. is an employee of Merck Pharmaceutical Company. D.E.K. has received research grants from Novartis Pharmaceuticals, Sanofi-Aventis, and Pfizer; has served as a consultant/advisor to Novartis Pharmaceuticals, Sanofi-Aventis, Pfizer, Merck Pharmaceutical Company, and GlaxoSmithKline; and has been on speaker's bureaus for Novartis Pharmaceuticals, Sanofi-Aventis, and Merck Pharmaceutical Company. R.L.L. has received research grants from GlaxoSmithKline and has been a consultant/advisor to Amylin Pharmaceuticals, Merck Pharmaceutical Company, Arisaph Pharmaceuticals, and Genaera Corporation. C.N. has been a consultant/advisor to Amylin Pharmaceuticals, GlaxoSmithKline, and Slim Fast.

Abbreviations: CHD, coronary heart disease; CT, computed tomography; IAAT, intra-abdominal adipose tissue; MRI, magnetic resonance imaging; NHANES III, National Health and Nutrition Examination Survey III; SAAT, subcutaneous abdominal adipose tissue; WC, waist circumference; WHO, World Health Organization.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

DOI: 10.2337/dc07-9921

© 2007 by NAASO and the American Diabetes Association.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

ington, DC, and was charged to provide answers to the following four 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. Waist circumference is actually a perimeter, which provides an estimate of body girth at the level of the abdomen. Different anatomical 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) waist circumference; 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 relationship between morbidity or mortality rate and WC were at the midpoint between the lowest rib and the iliac crest (29%), umbilicus (28%), and narrowest waist circumference (22%). Although sites that use an easily identifiable and reproducible landmark (e.g., 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.

Waist circumference measurements should be made around a patient's bare midriff, after the patient exhales while standing without shoes, 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 mea-

Table 1—Distribution of adipose tissue mass in lean and obese men

	Lean men	Obese men
BMI (kg/m ²)	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
Intra-abdominal (kg)	1.9	5.1
Intraperitoneal (kg)	1.1	3.5
Retroperitoneal (kg)	0.8	1.6

Adapted from reference 16.

surements are typically taken three 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 obtain 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 (e.g., iliac crest site, intra-class 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 both 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).

Anatomical relationships. 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 includes both intra-peritoneal 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 L₄-L₅ intervertebral level to estimate SAAT and IAAT volume, expressed as cm³. However, L₄-L₅ imaging does not provide the best estimate of total IAAT mass, which is more reliably estimated several centimeters cephalad of the L₄-L₅ intervertebral space (17,18). In addition, measurement site influences the relationship between IAAT volume and cardiometabolic risk; the association between IAAT volume and presence of the metabolic syndrome is greater when IAAT volume is determined at the L₁-L₂ than at the L₄-L₅ level (19). Currently, there is no universally accepted site for measuring IAAT and SAAT.

The relationship between WC, weight, and BMI can be conceptualized by using simple geometric relationships 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 relationships 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 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 anatomical 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

Table 2—Relationships among waist circumference, BMI, and adipose tissue compartments in men and women

	Men		Women	
	BMI	Waist circumference	BMI	Waist circumference
Total adipose tissue	0.82	0.87	0.91	0.87
Percent body fat	0.70	0.79	0.86	0.82
Total subcutaneous adipose tissue	0.82	0.83	0.91	0.86
Total intra-abdominal adipose tissue	0.59	0.79	0.69	0.77

Data are correlation coefficients. Adapted from reference 18.

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 triglycerides in abdominal fat depots is directly responsible for increased disease risk or 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 relationship between WC and cardiometabolic risk cannot determine whether risk is associated with SAAT, IAAT, or both.

The mechanism(s) responsible for the relationship 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 “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 system-wide 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 triglycerides release 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 β -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 in-

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 (e.g., elevated blood pressure, dyslipidemia, and hyperglycemia) and adverse outcomes (e.g., diabetes, CHD, and death rate) has been examined in many large epidemiological studies (7,24–33). Specific relative risks between WC and these outcomes vary, depending on the population sampled and the outcome measured. The relationship 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 remains statistically significant after adjusting for BMI. These data demonstrate that WC can identify persons who are at greater cardiometabolic risk than 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 adjusting 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 relationships 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 relationship be-

tween WC and health outcome changes much less with increasing age than does the relationship 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 relationship between WC and health outcomes (e.g., 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 relationship between WC and health outcome lends itself to identifying clinically meaningful cut point values because risk often accelerates monotonically above, and can be relatively flat below, a specific WC value. Optimum WC cut points 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 waist circumference should be measured in clinical practice depends on the responses to the following four 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 of one anatomical site is a better indicator of cardiometabolic risk than 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*; 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 waist circumference 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 demonstrates that WC remains a predictor of diabetes, CHD, and mortality rate, even after adjusting 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 ≥ 25 kg/m² and an assessment of commonly evaluated cardiometabolic risk factors? Answer: *Yes*.

The recommended WC thresholds for increased cardiometabolic risk in men (>40 inches [102 cm]) and women (>35 inches [88 cm]) were derived from WC values that correlated with a BMI ≥ 30 kg/m² (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 kg/m²) (36). In addition, ~70% of women who were overweight (BMI 25.0–29.9 kg/m²) had a WC >35 inches and ~25% of men who were overweight had a WC >40 inches. An estimate based on data available from the WHO Monica Project, conducted in more than 32,000 men and women from Europe, Australia, and New Zealand, suggest that about 10% of participants who had BMI values <30 kg/m² had a WC above the recommended cut points 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 ≥ 25 kg/m² 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, waist circumference should only be measured if it can provide additional information that influences patient management. Based on NHANES III data, 99.9% of men and 98.4% 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 cut point 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 cut points provided a better indicator of cardiometabolic risk than the recommended WC thresholds (35). For normal-weight (BMI 18.5–24.9 kg/m²), overweight (BMI 25.0–29.9 kg/m²), class I obesity (BMI 30.0–34.9 kg/m²), and class II/class III obesity (BMI ≥ 35.0 kg/m²), the optimal WC cut points 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. Waist circumference 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

Waist circumference provides a unique indicator of body fat distribution, which can identify patients who are at increased risk for obesity-related cardiometabolic disease, above and beyond the measurement of BMI. However, the current WC cut points 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 cur-

rently 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 cut points that can assess cardiometabolic risk, 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 cut points than are currently recommended, by carefully reviewing published data and reevaluating datasets available from existing population studies. These additional analyses will define the future role of WC measurement in clinical practice.

Acknowledgments—This conference was supported in part by an educational grant from the Campbell Soup Company.

References

- World Health Organization: *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity*. Geneva, World Health Organization, 1998
- National Institutes of Health, National Heart, Lung, and Blood Institute: Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obes Res* 6 (Suppl. 2):51S–209S, 1998
- Colditz GA, Willett WC, Rotnitzky A, Manson JE: Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med* 122:481–486, 1995
- Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr: Body-mass index and mortality in a prospective cohort of U.S. adults. *N Engl J Med* 341:1097–1105, 1999
- Pouliot MC, Despres JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Nadeau A, Lupien PJ: Waist circumference and abdominal sagittal diameter: best simple anthropometric indices of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *Am J Cardiol* 73:460–468, 1994
- Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, Adams PW: Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 54:254–260, 1982
- Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB: Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr* 81:555–563, 2005
- Lean ME, Han TS, Morrison CE: Waist circumference as a measure for indicating need for weight management. *BMJ* 311:158–161, 1995
- Wang J, Thornton JC, Bari S, Williamson B, Gallagher D, Heymsfield SB, Horlick M, Kolter D, Laferrere B, Mayer L, Pi-Sunyer FX, Pierson RN Jr: Comparison of waist circumferences measured at 4 sites. *Am J Clin Nutr* 77:379–384, 2003
- Douketis JD, Paradis G, Keller H, Martineau C: Canadian guidelines for body weight classification in adults: application in clinical practice to screen for overweight and obesity and to assess disease risk. *CMAJ* 172:995–998, 2005
- NHANES III Anthropometric Procedures Video. Available from <http://www.cdc.gov/nchs/about/major/nhanes/avideo.htm>.
- Nordhamm K, Sodergren E, Olsson E, Karlstrom B, Vessby B, Berglund L: Reliability of anthropometric measurements in overweight and lean subjects: consequences for correlations between anthropometric and other variables. *Int J Obes Relat Metab Disord* 24:652–657, 2000
- Chen MM, Lear SA, Gao M, Frohlich JJ, Birmingham CL: Intraobserver and interobserver reliability of waist circumference and the waist-to-hip ratio. *Obes Res* 9:651, 2001
- Rimm EB, Stampfer MJ, Colditz GA, Chute CG, Litin LB, Willett WC: Validity of self-reported waist and hip circumferences in men and women. *Epidemiology* 1:466–473, 1990
- Bigaard J, Spanggaard I, Thomsen BL, Overvad K, Tjonneland A: Self-reported and technician-measured waist circumferences differ in middle-aged men and women. *J Nutr* 135:2263–2270, 2005
- Abate N, Garg A, Peshock RM, Stray-Gundersen J, Grundy SM: Relationships of generalized and regional adiposity to insulin sensitivity in men. *J Clin Invest* 96:88–98, 1995
- Shen W, Wang Z, Punyanyita M, Lei J, Sinav A, Kral JG, Imielinska C, Ross R, Heymsfield SB: Adipose tissue quantification by imaging methods: a proposed classification. *Obes Res* 11:5–16, 2003
- Shen W, Punyanyita M, Wang Z, Gallagher D, St-Onge MP, Albu J, Heymsfield SB: Total body skeletal muscle and adipose tissue volumes: estimation from a single abdominal cross-sectional image. *J Appl Physiol* 97:2333–2338, 2004
- Kuk JL, Church TS, Blair SN, Ross R: Does measurement site for visceral and abdominal subcutaneous adipose tissue alter associations with the metabolic syndrome? *Diabetes Care* 29:679–684, 2006
- Ford ES, Mokdad AH, Giles WH: Trends in waist circumference among U.S. adults. *Obes Res* 11:1223–1231, 2003
- Chan DC, Watts GF, Barrett PH, Burke V: Waist circumference, waist-to-hip ratio and body mass index as predictors of adipose tissue compartments in men. *QJM* 96:441–447, 2003
- Bjorntorp P: Body fat distribution, insulin resistance, and metabolic diseases. *Nutrition* 13:795–803, 1997
- Seppala-Lindroos A, Vehkavaara S, Hakkinen AM, Goto T, Westerbacka J, Sovijarvi A, Halavaara J, Yki-Jarvinen H: Fat accumulation in the liver is associated with defects in insulin suppression of glucose production and serum free fatty acids independent of obesity in normal men. *J Clin Endocrinol Metab* 87:3023–3028, 2002
- Sinha R, Dufour S, Petersen KF, LeBon V, Enoksson S, Ma YZ, Savoye M, Rothman DL, Shulman GI, Caprio S: Assessment of skeletal muscle triglyceride content by (1)H nuclear magnetic resonance spectroscopy in lean and obese adolescents: relationships to insulin sensitivity, total body fat, and central adiposity. *Diabetes* 51:1022–1027, 2002
- Katzmarzyk PT, Craig CL: Independent effects of waist circumference and physical activity on all-cause mortality in Canadian women. *Appl Physiol Nut Metab* 31:271–276, 2006
- Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS; INTERHEART Study Investigators: Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 366:1640–1649, 2005
- Hu G, Tuomilehto J, Silventoinen K, Barengo N, Jousilahti P: Joint effects of physical activity, body mass index, waist circumference and waist-to-hip ratio with the risk of cardiovascular disease among middle-aged Finnish men and women. *Eur Heart J* 24:2212–2219, 2004
- Lofgren I, Herron K, Zern T, West K, Patalay M, Shachter NS, Koo SI, Fernandez ML: Waist circumference is a better predictor than body mass index of coronary heart disease risk in overweight premenopausal women. *J Nutr* 134:1071–1076, 2004
- Suk SH, Sacco RL, Boden-Albala B, Cheun JF, Pittman JG, Elkind MS, Paik MC; Northern Manhattan Stroke Study: Abdominal obesity and risk of ischemic stroke: the Northern Manhattan Stroke Study. *Stroke* 34:1586–1592, 2003
- Carey VJ, Walters EE, Colditz GA, Solomon CG, Willett WC, Rosner BA,

Consensus Statement

- Speizer FE, Manson JE: Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women: the Nurses' Health Study. *Am J Epidemiol* 145:614–619, 1997
31. Visscher TL, Seidell JC, Molarius A, van der Kuip D, Hofman A, Witteman JC: A comparison of body mass index, waist-hip ratio and waist circumference as predictors of all-cause mortality among the elderly: the Rotterdam study. *Int J Obes Relat Metab Disord* 25:1730–1735, 2001
32. Folsom AR, Kushi LH, Anderson KE, Mink PJ, Olson JE, Hong CP, Sellers TA, Lazovich D, Prineas RJ: Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's Health Study. *Arch Intern Med* 160:2117–2128, 2000
33. Hu FB, Willett WC, Li T, Stampfer MJ, Colditz GA, Manson JE: Adiposity as compared with physical activity in predicting mortality among women. *N Engl J Med* 351:2694–2703, 2004
34. Baik I, Ascherio A, Rimm EB, Giovannucci E, Spiegelman D, Stampfer MJ, Willett WC: Adiposity and mortality in men. *Am J Epidemiol* 152:264–271, 2000
35. Ardern CI, Janssen I, Ross R, Katzmarzyk PT: Development of health-related waist circumference thresholds within BMI categories. *Obes Res* 12:1094–1103, 2004
36. Meisinger C, Doring A, Thorand B, Heier M, Lowel H: Body fat distribution and risk of type 2 diabetes in the general population: are there differences between men and women? The MONICA/KORA Augsburg cohort study. *Am J Clin Nutr* 84:483–489, 2006
37. Kiernan M, Winkleby MA: Identifying patients for weight-loss treatment: an empirical evaluation of the NHLBI Obesity Education Initiative Expert Panel treatment recommendations. *Arch Intern Med* 160:2169–2176, 2000
38. Dekker MJ, Lee S, Hudson R, Kilpatrick K, Graham TE, Ross R, Robinson LE: An exercise intervention without weight loss decreases circulating interleukin-6 in lean and obese men with and without type 2 diabetes mellitus. *Metabolism* 56:332–338, 2007