Normal-weight central obesity

Unique hazard of the toxic waist

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Abstract

Objective To examine the mortality risk presented by normal-weight central obesity, to identify a clinical measure to aid in the identification of this phenotype, and to explore the means for mitigation of this risk.

Quality of evidence Only prospective cohort studies (level II) comparing participants with central obesity at normal weight with those at higher levels of body mass index (BMI) were found. Good level I studies were available to demonstrate the effect of diet and exercise interventions on central obesity and mortality.

Main message Participants with atherogenic dyslipidemia who are centrally obese at normal BMI are at similar, and possibly higher, mortality risk compared with those who are centrally obese and overweight or obese according to their BMI. Waist-to-height ratio might be the most pragmatic clinical measure of central obesity. The Mediterranean diet is an effective intervention to prevent ongoing weight gain while reducing abdominal girth. Low levels of exercise can also reduce waist circumference. Weight loss need not be an objective.

Conclusion A waist-to-height ratio exceeding 0.5 at normal BMI identifies elevated mortality risk for cardiometabolic disease. This risk might equal or exceed that of centrally obese patients who are overweight or obese. Modest dietary and exercise interventions can be effective in mitigation of this risk.

Editor's key points

- Visceral obesity is increasing faster in the North American population than generalized obesity is and it has a more profound effect on morbidity and mortality. Individuals with a normal-weight body mass index and central obesity are at equivalent, and possibly higher, risk than people with central obesity who are overweight or obese by body mass index.
- The simplest and most valid measure of central obesity is waist-to-height ratio. The cutoff value is 0.5 and is the same regardless of sex and ethnic origin. It can be easily measured with minimal equipment. A piece of string representing patient height can be retained, folded in half, and periodically pulled around the waist.
- Reduction in central obesity and weight stabilization can be achieved with a Mediterranean-style eating pattern. Exercise with minimal weight loss can reduce visceral fat. Small changes in exercise and eating patterns can produce cardiometabolic and mortality benefit.

The World Health Organization defines *obesity* as "abnormal or excessive fat accumulation that may impair health."[1] This document concedes that body mass index (BMI) can only be a rough guide to the degree of adiposity. People with normal BMI can have a proportion of body fat exceeding 30%.[2] If this fat is distributed primarily as central or visceral fat, it is strongly associated with cardiometabolic risk.[3] Such people have abnormal adipose tissue distribution and function, with increased risk of diabetes and cardiovascular disease. These abnormally functioning fat deposits can produce atherosclerotic, dysmetabolic, and mechanical challenges leading to deteriorating health,[4] thus fulfilling the World Health Organization criteria for obesity.

Rates of obesity in North America have continued to rise since the 1980s. As of 2015, 38.2% of the adult population in the United States (US) and 25.8% of the adult population in Canada is obese.[5] Between 1990 and 2015, rates of ischemic heart disease fell 55% in the US and 60% in Canada[5]; however, while Canadian rates continue to fall, decline in the US has leveled off since 2011.[6,7] Uniquely among Organisation for Economic Co-operation and Development countries, US life expectancy has begun to fall in the past few years,[7] driven in part by rising obesity and social inequality.[8]

Prevalence of the normal-weight metabolically obese phenotype by direct measurement of fat distribution or metabolic characteristics might vary between 13% and 38%.[9,10] Truncal fat, as estimated by waist circumference (WC), waist-to-hip ratio, or waist-to-height ratio (WHtR) (**Table 1**),[11-15] is positively correlated with metabolic abnormalities, while fat in the lower body has

Table 1. Anthropometric measures of body fat distribution: *A) Mass-based and B) distribution-based measures.*

A)		
MASS-BASED MEASURE	**DEFINITION**	**COMMENTS**
Body mass index[11]	Weight in kg divided by the square of the height in m	Does not distinguish between lean and fat tissue mass
• Underweight	< 18.5 kg/m^2	Associated with higher mortality
• Normal weight	18.5-24.9 kg/m^2	Lowest mortality associated with these categories
• Overweight	25.0-29.9 kg/m^2	
• Obesity class 1	30.0-34.9 kg/m^2	No consistent association with increased mortality
• Obesity class 2	35.0-39.9 kg/m^2	Direct association with increased mortality
• Obesity class 3	≥ 40.0 kg/m^2	
B)		
DISTRIBUTION-BASED MEASURES	**VALUES REPRESENTING INCREASED RISK**	**SURROGATE MEASURES OF CENTRAL OR VISCERAL ADIPOSITY**
Waist circumference	Females ≥ 80 cm Males ≥ 95 cm	Cut points vary according to ethnicity, sex, and age[12]
Waist-to-hip ratio	Females ≥ 0.85 Males ≥ 0.95	Cut points not well established for ethnicity[13]
Waist-to-height ratio	Increased risk 0.50-0.60 Substantial risk > 0.60	Cut points the same for ethnicity, sex, and age[12] Best predicts visceral fat mass[14,15]

a negative correlation. Subcutaneous fat, particularly in the femorogluteal area, might provide a depot helping to prevent lipid deposition at intra-abdominal and visceral sites, where it might be more damaging.[16-18] Prevalence of abdominal obesity as measured by WC is currently rising faster than general obesity as measured by BMI.[19,20]

Objectives for this review are as follows:

- to quantify the mortality risk posed by normal-weight central obesity;
- to identify a pragmatic clinical measure to aid in identifying those at risk; and
- to explore mitigation of this risk.

Quality of evidence

The initial PubMed search included the following MeSH headings and key words: *normal weight* and *central obesity* (title and abstract) or *visceral obesity* (title and abstract) or *visceral fat* (title and abstract) or *ectopic fat* (title and abstract) or *hypertriglyceridemic waist* (title and abstract) or *metabolically obese* (title and abstract) or *abdominal adiposity* (title and abstract) or *waist* (title and abstract) and *mortality* not *cancer*. References from appropriate retrieved papers were also scanned. Key word searches were also done in Google Scholar and the Cochrane database. No level I studies were found. All included studies were observational, and pertinent prospective cohort studies or systematic reviews were selected.

Main message

Visceral obesity is uniquely atherogenic. Central or abdominal obesity is contained in discrete compartments. Subcutaneous fat can be considerable over the abdominal area. Visceral intra-abdominal fat collects in the omentum, mesentery, liver, and pancreas. This visceral fat can be found at extra-abdominal sites such as the pericardium, myocardium, and skeletal muscle as well.[21] Retroperitoneal fat can also contribute to abdominal girth. The intra-abdominal compartment, together with ectopic fat in other organs of the body and in skeletal muscle, constitutes metabolically active visceral fat, which behaves quite differently from subcutaneous fat.[22] Their differing characteristics are summarized in **Table 2**.[15-18,22-28]

It is still not clear whether a population trend to increasing hyperinsulinemia is instrumental as a cause of obesity or whether established obesity is a cause of hyperinsulinemia.[29] A central factor is the rising sugar content of the food supply. Apart from sugar added in home preparation, 66% of Canadian packaged foods and beverages now contain added sugars.[30] This triggers insulin release, which drives circulating lipid into adipocytes to be stored as fat. Increasing fat in turn induces insulin resistance, and even higher levels of insulin are required to drive glucose into cells. Some individuals can store more fat peripherally than others, and as these deposits become replete, fat begins to be deposited in visceral sites as well, appearing in the liver, omentum, skeletal muscle, and peripheral organs. The insulin-resistant state tends to accelerate lipolysis, releasing free fatty acids (FFAs) into circulation. Visceral adipocytes are also very sensitive to catecholamine-induced lipolysis, releasing FFAs into the portal circulation and presenting the liver with increased lipid for processing.[28] Levels of these FFAs in circulation are high in the abdominally obese. Although they can be used as a substrate for energy

Table 2. Characteristics of subcutaneous and visceral fat

VARIABLES	CHARACTERISTICS	
	SUBCUTANEOUS FAT	VISCERAL FAT
Clinical measurement	Body mass index	Waist circumference, waist-to-height ratio, waist-to-hip ratio
Association with cardiometabolic disease[16]	Association with mortality is inconsistent	Direct linear association with mortality
Function[15,18]	Metabolic sink and longer-term energy storage	Short-term energy source
Cardiac risk[23]	Moderate	High
Metabolic risk[24]	Moderate	High
Inflammation[22]	Moderate	High
Catecholamine response[22]	Moderate	Rapid
Insulin sensitivity[17]	Moderate	Low
Metabolic flux[18]	Low	High
Trend with age[25]	Increased to age 65 y, then reduced	Gradual increase
Storage duration[26]	Long	Short
Effect of exercise[27]	High levels needed for weight change	Low levels effective for cardiometabolic benefit
Adverse effects of refined carbohydrate[28]	Moderate	High

production, they also contribute to insulin resistance, inhibiting glucose uptake by muscle and other organs, further contributing to hyperglycemia.[18]

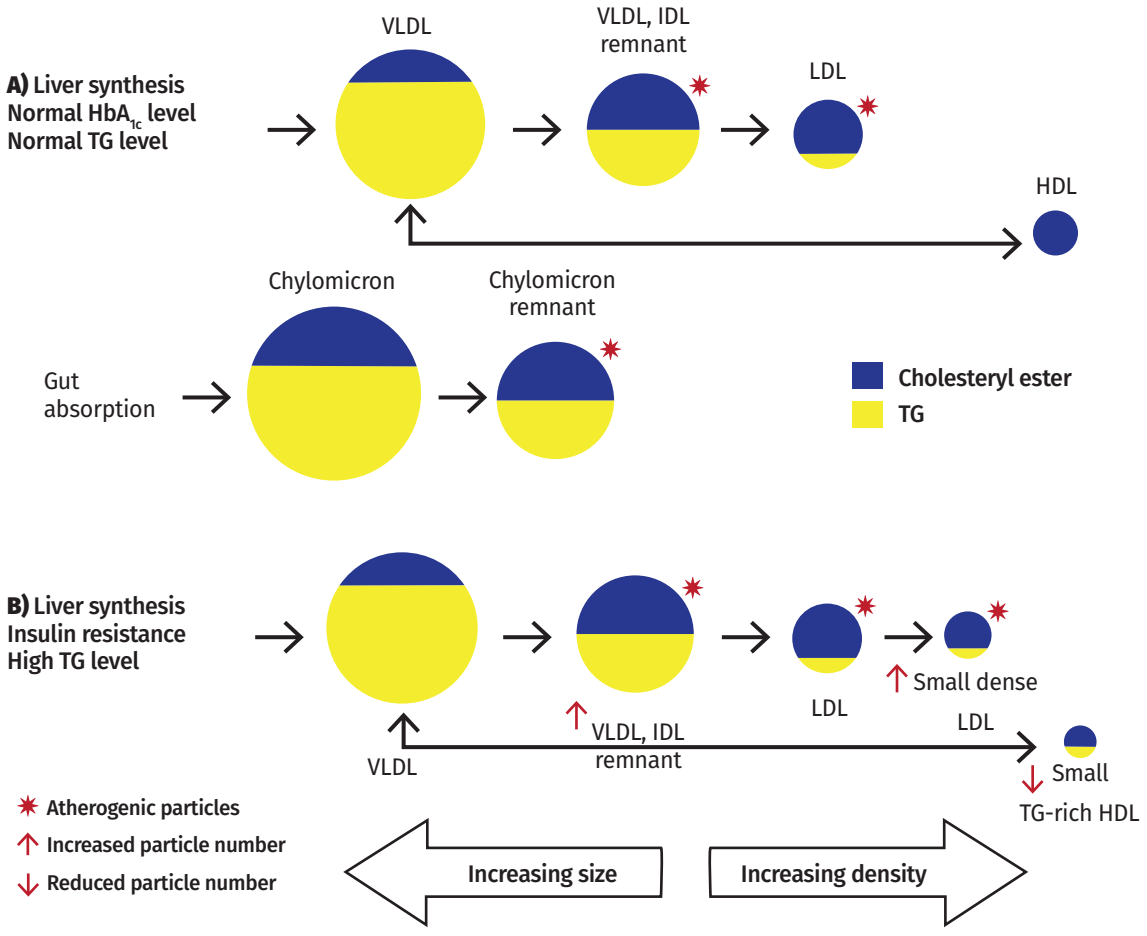
Free fatty acid lipid is combined with glycerol to form triglyceride (TG) and then packaged by the liver in water-soluble form as very low-density lipoprotein (VLDL) particles. These particles are large and contain predominantly TG, along with some cholesterol. Triglyceride, being an energy substrate, is hydrolyzed in various tissues, and these particles gradually become smaller and more dense, forming intermediate-density lipoprotein (IDL) and low-density lipoprotein (LDL) cholesterol particles.[31] As particles become smaller they contain an increasing proportion of cholesterol, which is not an energy substrate (**Figure 1A**).

In the presence of insulin resistance and hypertriglyceridemia the characteristics of these particles change. The VLDL and IDL remnant particle numbers increase, and LDL particles become even smaller, more dense, and much more numerous. All these particles easily penetrate the vascular endothelium, where they become intensely atherogenic. At the same time, high-density lipoprotein (HDL) particles, which are involved in reverse cholesterol transport, tend to become TG rich and smaller, to the extent that some are lost in the urine. The result is a smaller number of HDL particles, which cannot contain as much cholesterol for clearance from the vasculature (**Figure 1B**).[21,32]

A patient with this metabolic profile is likely to have high TG, low and dysfunctional HDL, and increased numbers of small LDL particles, characteristic of atherogenic dyslipidemia.[33,34] Low-density lipoprotein is not reliable for risk evaluation, as much of the cholesterol might be hiding in VLDL, IDL, and other remnant lipoproteins, including chylomicron remnants in the non-fasting state. These particles are at least as potent as LDL in predicting cardiovascular events.[35] The most common clinical characteristic is increased visceral fat as indicated by central obesity.[36]

Clinical evaluation of central obesity. There is consensus that an anthropometric measure of abdominal or central obesity is a better predictor of cardiometabolic risk, diabetes risk, and all-cause mortality than BMI is, and that combining the 2 indices might be even better. Usually, mortality risk plotted against BMI is *J* shaped, with increased mortality only in underweight and high class 1 to class 3 obesity (**Table 1**).[11-15] Being overweight carries consistently lower mortality risk than being normal weight does.[37] Removal of competing causes of mortality (smoking, comorbid disease, advanced age) tends to remove the steep increase in the underweight category and move the nadir for mortality into the normal BMI range.[38] The relation between BMI and mortality then becomes more progressive and linear. A similar plot of mortality against waist-to-hip ratio, WC, or WHtR typically shows a much more steeply progressive linear relation, allowing a more granular assessment of mortality risk (**Figure 2**).[39,40] There are mixed opinions in the literature as to which of these central obesity measurements

Figure 1. Plasma lipoproteins comparing hepatic synthesis and lipid trajectory of TG-rich particles: *A) With and B) without insulin resistance.*



HbA_{1c}—hemoglobin A_{1c}, HDL—high-density lipoprotein cholesterol, IDL—intermediate-density lipoprotein cholesterol, LDL—low-density lipoprotein cholesterol, TG—triglyceride, VLDL—very low-density lipoprotein cholesterol.

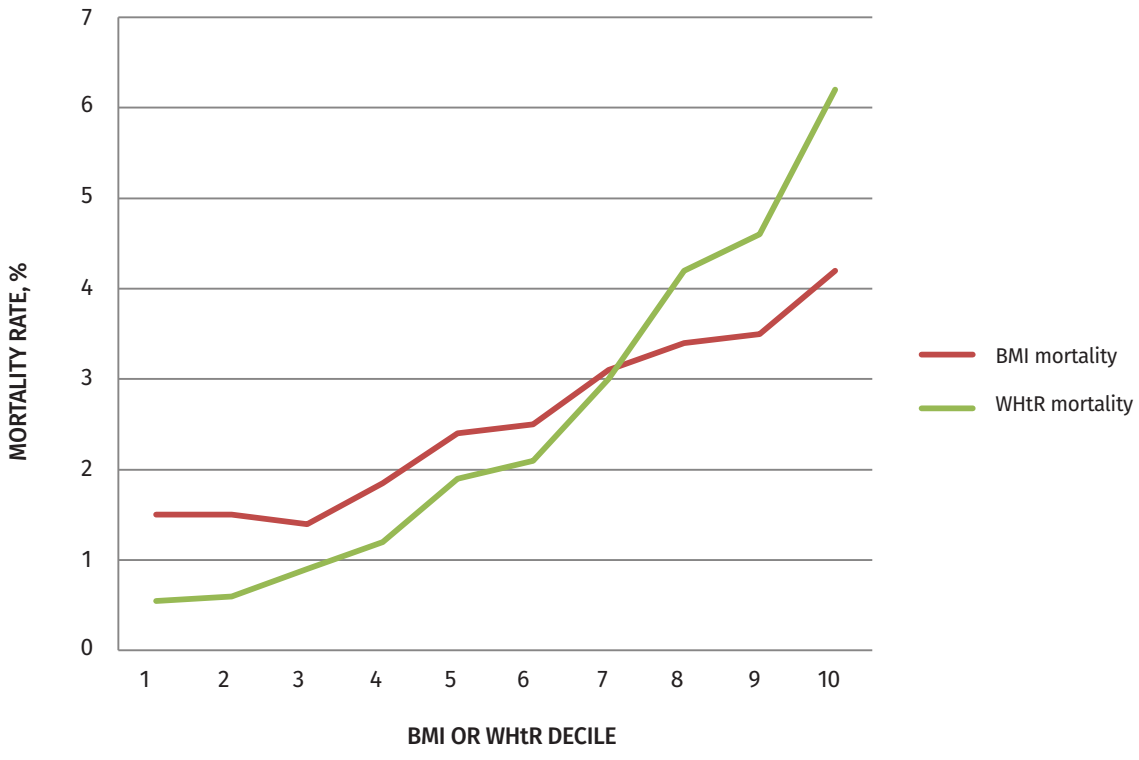
is best, but, of the systematic reviews and meta-analyses comparing the 3,[12,14,23,39] WHtR is favoured. There are pragmatic reasons for use of WHtR as well:

- It can be used without the aid of a scale or reference to BMI categories.
- The cutoff value for WHtR is 0.5 and is the same regardless of sex and ethnic origin, although children might require further study.[39]
- It allows simple home monitoring. Patients can be told, "Your waist should not exceed half your height."
- It is easily used in austere settings with minimal equipment.[41] A piece of string representing patient height can be retained, folded in half, and periodically pulled around the waist.[42]

Mortality risk presented by normal-weight central obesity. Available prospective studies documenting all-cause mortality associated with increasing central obesity at any given BMI are presented in **Table 3**.[39,43-57] The striking finding is that, with one exception,[51] the mortality risk of those with central obesity at normal BMI is similar to or greater than the risk of those with central obesity who are overweight or obese. In most cases these normal-weight participants have the highest mortality of any group combination of body mass and central fat distribution. In some studies the magnitude of this difference is statistically significant.[46,52,54] The implication of this is that normal-weight centrally obese people might be at uniquely high risk of all-cause mortality.

The other consistent finding from these studies is that, when central obesity is present, the existence of subcutaneous fat seems to offer protection. Centrally obese participants in the overweight and class 1 obesity BMI range actually have lower mortality risk than

Figure 2. Comparison of mortality rates in non-smokers by BMI or WHtR deciles over 20 years



BMI—body mass index, WHtR—waist-to-height ratio.
Data from Ashwell et al.[40]

their normal-weight centrally obese counterparts (**Figure 3**).[46-49,51-54,57] The limited capacity to store lipid subcutaneously might lead to a mortality disadvantage, particularly in those with the highest levels of visceral adipose tissue.[58]

Mitigating risk without weight loss. It seems reasonable that interventions targeting visceral fat should target causation—primarily the environment of processed, calorie-dense foods and sedentary lifestyle.[59] Weight loss need not be an objective. In a review of randomized and non-randomized trials primarily using exercise for weight control, 11 of 29 studies showed a significant reduction in visceral fat despite no clinically relevant weight loss.[60] Other studies[61] suggest that with exercise-induced weight management, visceral fat is preferentially lost in those with minimal weight loss, whereas higher weight-loss categories showed predominant loss of subcutaneous fat.

In addition to reduction of both visceral fat and some cardiometabolic risk factors, exercise is effective in preservation of muscle mass and facilitation of mobility.[60] This is particularly important in the elderly, who might have difficulty maintaining weight, but tend to accumulate visceral fat and lose muscle mass and subcutaneous fat mass.[62,63] Exercise is an essential component in minimizing central obesity and maintaining muscle mass. A meta-analysis of studies lasting 4 to 52 weeks[27] showed that, with weight held stable, visceral adipose tissue is reduced 6.1% by exercise and only 1.1% by diet.

Each 5-cm reduction in WC is associated with reduction in mortality by as much as 9% over 6.7 years at any level of BMI.[64] The risk of disability and mortality can be mitigated by exercise interventions well below targets currently recommended.[19] Large cohort studies now exist demonstrating mortality reduction associated with walking 15 minutes a day[65] or jogging 5 to 10 minutes a day.[66] The focus in this population should be on reduction in central obesity and prevention of weight gain. Emphasis should be on small and achievable changes in behaviour. More intensive physical activity can remain an option, as the mechanical disadvantage of obesity in these patients is not an issue.

Dietary constituents can affect visceral fat. The PREDIMED (Prevención con Dieta Mediterránea) study was a randomized controlled trial comparing the Mediterranean diet with a low-fat diet over 4.8 years[67];

Table 3. Prospective observational studies and related SRs and MAs of studies relating normal-weight central obesity to mortality

STUDY AND YEAR	STUDY TYPE (DURATION)	STUDY OBJECTIVES	NO. AND AGE OF PARTICIPANTS	RESULTS	COMMENTS
Coutinho et al, 2013[43]	SR of prospective observational studies and collaborative analysis (0.5-7.4 y)	Relation of measures of central obesity to mortality	15 923 patients with known CAD Mean (SD) age 65.7 (11.5) y	Highest mortality category combined lowest BMI with highest WHR or WC; HR = 1.7	Central obesity associated with equal increase in mortality risk in lean and obese patients. Increased BMI was associated with reduced risk in these patients
Kramer et al, 2013[44]	MA of prospective observational studies (3-30 y)	Relation of cardiometabolic risk and BMI category to CVD events and all-cause mortality	61 386 adults from the general population Mean age range 44-60 y	Highest mortality or CVD event rate was similar in metabolically unhealthy normal-weight and obese participants; HR = 2.65 compared with those of normal weight and metabolic health	Did not specifically consider central obesity. Metabolic health was based on absence of metabolic syndrome components, insulin resistance, or inflammatory markers
Carmienke et al, 2013[39]	SR and regression MA of prospective cohorts (5-24 y)	Relation of measures of abdominal obesity parameters to mortality	689 465 healthy adults Age ≥ 18 y	Highest mortality category combined lowest BMI with highest WHR or WC	WHR, WC, or WHtR combined with BMI gives best mortality prediction. In participants > 65 y there was a non-significant or negative association with increasing BMI, WC, and WHR
Folsom et al, 2000[45]	Prospective observational study (11-12 y)	Relation of BMI, WC, and WHR to mortality, cancer, diabetes, hypertension, and fracture	31 702 healthy US women Age 55-69 y	Highest mortality category combined lowest BMI with highest WHR	WHR had the best mortality prediction
Pischon et al, 2008[46]	Prospective observational study (9.7 y)	Association of distribution of adiposity with risk of death	359 387 participants from general European population Age 25-70 y	Highest mortality associated with lowest BMI percentile having highest WC or WHR	Association of BMI with mortality is *J* shaped. Measures of central obesity showed positive linear association with mortality when adjusted for BMI
Zhang et al, 2008[47]	Prospective observational study (16 y)	Relation of abdominal adiposity to premature death	44 636 US women from the Nurses' Health Study Age 30-55 y	Highest mortality category combined highest BMI and highest WC or WHR. Risk was only slightly lower for normal BMI with high central obesity	WC and WHR were both directly associated with mortality
Koster et al, 2008[48]	Prospective observational study (9 y)	Relation of WC to all-cause mortality	245 533 US adults Age 51-72 y	Normal weight with high WC increased mortality by 22%. Exceeded only by class 2 and 3 obesity with high WC	WC used as measure of central obesity in mortality prediction
Reis et al, 2009[49]	Prospective observational study (12 y)	Relation of BMI, WHR, and WTR to mortality	13 065 participants from the general US population Age 30-102 y	Highest mortality category combined lowest BMI with highest WHR or WTR	WHR or WTR had the best mortality prediction. No association at > 65 y of age
Romero-Corral et al, 2009[50]	Prospective observational study (8.8 y)	Relation of body fat percentage to cardiovascular mortality and metabolic dysregulation in participants with normal BMI	6171 US patients with normal BMI and CAD Age ≥ 20 y	High body fat percentage and WC were associated with increased risk of metabolic syndrome. CVD mortality increased in normal-weight obese women (HR = 2.20) compared with nonobese women	Body fat measured by bioimpedance. Body fat percentage did not correlate with all-cause mortality in women or men at any BMI

Table 3 *continued on page 405*

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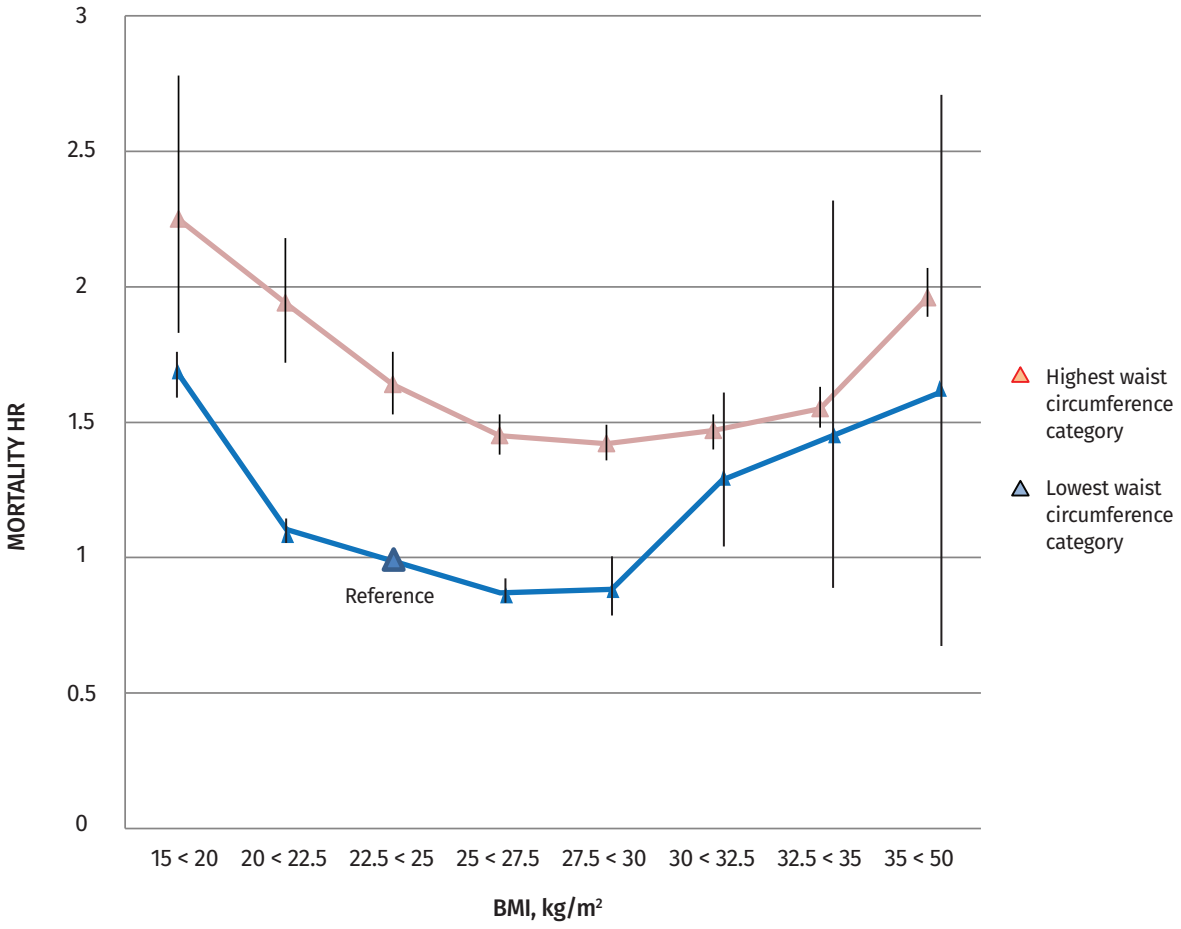
STUDY AND YEAR	STUDY TYPE (DURATION)	STUDY OBJECTIVES	NO. AND AGE OF PARTICIPANTS	RESULTS	COMMENTS
Staiano et al, 2012[51]	Prospective observational study (13 y)	Relation of BMI, WC, and WHR to CVD and all-cause mortality	8061 Canadian adults Age 18-74 y	No mortality increase with WC in normal-weight adults. Highest mortality in highest WC terciles of obese adults	WC had the best association with CVD and all-cause mortality
Thomas et al, 2013[52]	Prospective observational study (mean [SD] 5.6 [2.4] y)	Relation of obesity measured by BMI or WC on mortality at differing ages	119 010 French participants with BMI > 20 kg/m^2 Age 17-85 y	Mortality at BMI 20-25 kg/m^2 and WC ≥ 102 cm … • increased 2-fold at age < 55 y and • increased 5-fold at age 55-65 y No association at age > 65 y	Mortality risk higher for central obesity in normal BMI range than in class 2 and 3 obesity. WC gives best mortality prediction at < 65 y of age. Neither WC nor BMI are useful in the elderly
Cerhan et al, 2014[53]	Pooled data from 11 prospective observational studies (median 9 y, maximum 21 y)	Relation of WC to mortality across entire range of BMI categories	650 386 non-Hispanic white adults Age 20-83 y	Highest mortality associated with BMI ranges < 20 and ≥ 35 kg/m^2 in those with highest WC. Highest WC in those with BMI 20-22.5 kg/m^2 had higher mortality than those with class 1 obesity at highest WC	WC should be assessed in combination with BMI even in those in normal BMI range. WC is directly associated with mortality at all levels of BMI
Sahakyan et al, 2015[54]	Prospective observational study (14.3 y)	Relation of central obesity and survival in adults of normal body weight	16 124 US adults with BMI ≥ 18.5 kg/m^2 Age 18-90 y	Men with normal-weight central obesity had higher mortality risk than any other BMI and WHR combination. Similar women had 40% and 32% relative risk increase compared with overweight and obese women without central obesity	WHR used as measure of central obesity in mortality prediction
Klingberg et al, 2015[55]	Prospective observational study (average 6 y)	Relation of baseline WC and change in WC to morality and CVD	2492 healthy Danish and Swedish women Age 44-74 y at baseline	Association of mortality with both high baseline WC and large increase in WC over time; particularly high in those with BMI < 25 kg/m^2	WC used in mortality prediction. Hip circumference was unrelated to mortality
Sharma et al, 2016[56]	Prospective observational study (average 7.1 y)	Relation of WC or WHR and BMI to mortality in elderly patients with CVD	7057 elderly patients with CAD Mean age 73 y	Highest mortality in patients with normal BMI and central obesity	Highlights importance of including WC or WHR along with BMI when making adiposity-related mortality assessment
Hamer et al, 2017[57]	Prospective observational study (average 9 y)	Determine whether WHR is more predictive of mortality than BMI is	42 702 UK adults Mean age 57.7 y	Normal weight with central obesity showed highest mortality; HR = 1.22 for death	WHR used as measure of central obesity in mortality prediction

BMI—body mass index, CAD—coronary artery disease, CVD—cardiovascular disease, HR—hazard ratio, MA—meta-analysis, SR—systematic review, UK—United Kingdom, US—United States, WC—waist circumference, WHR—waist-to-hip ratio, WHtR—waist-to-height ratio, WTR—waist-to-thigh ratio.

Figure 3. Adjusted mortality HRs (with 95% CIs represented by error bars) for waist circumference comparing highest and lowest values by category of BMI



BMI—body mass index, HR—hazard ratio.
Data from Cerhan et al.[53]

it showed statistically significant mortality reduction with the Mediterranean diet. Despite unrestricted intake, no weight gain was seen with the intervention. A secondary analysis[68] revealed that those assigned to the Mediterranean diet were significantly more likely to no longer meet the criterion of central obesity compared with those in the control group ($P<.001$). As avoidance of weight gain is an objective along with reduction in visceral fat, the Mediterranean eating pattern could be a preferred option for those with normal-weight central obesity, particularly as it tends to be low in refined carbohydrate. It is important to recognize that the benefits of exercise might be completely negated by poor dietary choices.[69]

Increased WC can be an indication for statin therapy according to Canadian guidelines in men older than 50 years and women older than 60 years who remain at intermediate Framingham risk despite optimized individual uptake of lifestyle recommendations.[70]

Conclusion

Visceral obesity is increasing faster in the North American population than generalized obesity is and it has a more profound effect on morbidity and mortality. The simplest and most valid measure of central obesity is WHtR. This phenotype is closely linked to atherogenic dyslipidemia, which predisposes one to the deposition of cholesterol in the vascular endothelium and resultant atherosclerosis. Individuals with normal-weight central obesity are at equivalent, and possibly higher, risk than people with central obesity who are overweight or obese by BMI.

Reduction in central obesity and weight stabilization can be achieved with a Mediterranean-style eating

pattern. Exercise with minimal weight loss can reduce visceral fat. Small changes in exercise and eating patterns can produce cardiometabolic and mortality benefit.

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Competing interests
None declared

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