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Diagnosis and Treatment of Obesity In The Elderly

Health Technology Assessment:

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Executive Summary

Background

Obesity, a disorder of body composition defined by a relative or absolute excess of body fat, [2] is extremely common among older US adults. In 1999-2000, 33% men and 39% of women aged 65-74 were obese, as were 20% and 25%, respectively, above the age of 74. [1] With steadily increasing prevalence in youth and younger adults, and links with adverse health outcomes, obesity has received increasing clinical and public health attention.

Diagnostic tests for obesity detect either generalized or central fat distribution. Body mass index (BMI = kg. of weight divided by the square of height in meters) is the standard clinical measure of generalized obesity. Skin fold thickness also correlates closely with overall body fat percentage. Waist circumference (WC) and waist-to-hip ratio (WHR) aim to detect central fat accumulation, as it carries increased cardiovascular risk. Accuracy and clinical relevance of these tools varies with gender and ethnicity, and possibly age.

In the general adult population, most treatment options (diet, exercise and behavioral, or pharmacologic) can lead to modest, sustainable improvements in weight, sufficient for improving intermediate health measures such as glycemic control, lipids, and blood pressure. [3-5] [6] Surgical intervention among the very obese can produce substantial weight loss and may markedly improve a number of health outcomes. However, high-quality surgical data are lacking, and while surgical adverse events are rare, they can be serious. [6] As there are age-related differences in fat distribution, physiology, and underlying health status, the generalizability of these findings to older populations is unclear.

Aim of the Review

To examine the data for the effectiveness of obesity diagnosis and treatment in the elderly, we conducted a systematic review of policy-relevant obesity diagnosis and treatment options in this group.

Methods

We developed a series of key questions for approaching six policy-pertinent tasks, posed by the Centers for Medicare and Medicaid Services (CMS) and the Agency for Healthcare Research and Quality (AHRQ), regarding obesity diagnosis and treatment in the elderly. We established eligibility criteria for literature inclusion, and rated evidence strength and study internal validity using standard criteria. [7] [8] To capture all data relevant to the aging population in the setting of limited studies, we considered data based on adults aged 60 years or above.

We searched MEDLINE and the Cochrane Library and reviewed bibliographies of published reviews for articles published in English between January 1980 and February 2003 (Appendix 2). At least two authors independently reviewed abstracts and articles, excluded those not meeting eligibility criteria, and abstracted the eligible articles. For topics on which data specific to the elderly were insufficient, we drew from findings of prior systematic reviews in the general population.

Results

Findings are organized according to the six policy-relevant questions.

1. Are there limitations in diagnosing obesity in the elderly with BMI? Should another measurement be used with BMI or in place of BMI for diagnosing obesity in the elderly?

Primary limitations to use of BMI in diagnosing obesity in the elderly include (1) A lower correlation with percentage body fat in the old than in the young, and (2) A weaker association with cardiovascular mortality, as well as several intermediaries of cardiovascular morbidity than measures of central adiposity (WC or WHR).

While the correlation between BMI and body fat percentage drops with age, most data show a reasonable correlation persists. In addition, body fat percentage is generally more closely correlated with BMI or WC than other common obesity diagnostic tests in the elderly. Likewise, BMI is the diagnostic measure linked with the broadest range of subsequent health states. Some of these outcomes (e.g., incident functional disability) [9] have not been evaluated by WHR or WC; others (e.g., hip fracture incidence in women) [10] are linked with BMI, but not with WHR or WC (likely reflecting that generalized, not central, obesity is important in their etiology). In addition, among the elderly, weight loss intervention efficacy trials have established that BMI changes with successful intervention; the ability to alter central measures of adiposity is unknown.

To best capture health risk in the aged, BMI should be combined with a measure of central adiposity. Data are inconsistent regarding the relative value of choosing WC versus WHR in the older population, however, WC is easier to measure.

2. Can intentional weight loss be used as a surrogate for improved net health outcomes? If so, how much weight loss and over what time period?

In the elderly, intentional weight loss is a reasonable surrogate for certain improvements in health outcomes. Evidence comes from consideration of (1) the health states for which older obese people are at increased risk, and (2) RCT evidence that such risk can be altered with intervention. Based on long term cohort evidence, the obese elderly have increased risk of several cardiovascular-related adverse outcomes (mortality, morbidity, and incidence of intermediary outcomes). In addition, multifaceted counseling-based weight loss interventions (combining dietary, exercise, and behavioral

components) can improve cardiovascular intermediaries, including fasting glucose tolerance, diabetes incidence, and a combined cardiovascular intermediary endpoint in generally healthy adults, compared with control participants. Modest weight loss (2 to 3 kg.), sustained over one to three years, was sufficient to produce these clinically meaningful results.

Intentional weight loss in the elderly has an unclear role in altering other obesity-related health risks. Observational data show increased cancer incidence, but not mortality, in older women (data are lacking for men) with increasing body size. However, the ability of intervention to alter this risk is uncertain: weight loss RCTs have limited duration of follow-up, and generally do not assess cancer or its intermediary outcomes. Similarly, while intentional weight loss may be a marker for improved function (cohort data indicate functional limitation increases with obesity), current RCTs in the elderly have not assessed this endpoint.

Longitudinal data linking higher BMI with lower incidence of and mortality from hip fracture in the elderly, coupled with RCT evidence of declining bone mineral density with weight loss interventions, indicate that intentional weight loss has adverse bone effects in older individuals. Obese individuals, though, are at a lower risk of osteoporosis than those with lower body weight. [11]

3. Which elderly patients with obesity would experience an improved health outcome with weight loss treatment?

We approached this question by reviewing the characteristics of people with evidence of obesity-associated health problems (cardiovascular disorders, cancer, or functional decline), and those with RCT evidence for health benefit (improved cardiovascular intermediaries) or harms (bone loss) from intentional weight loss.

Those at risk for obesity-associated health problems stand to benefit most from intervention, if such intervention alters their weight-related risk. The strongest evidence

for obesity intervention is for those with cardiovascular risk. Cardiovascular risk factors – including family history, diabetes, tobacco use, or dyslipidemia – can help identify this group. Risk for cancer or functional limitation is more complicated and often requires individualized patient assessment. However, several cancers with obesity-linked incidence (e.g., breast, colon) do have clear, identifiable risk factors. Finally, osteopenia, osteoporosis, or its risk factors may help identify those with risk of fracture – e.g., those most likely to experience harm from purposeful weight loss.

Among the elderly, the all-cause mortality risk associated with obesity (and therefore, potential benefit) diminishes with age. While risk of all-cause mortality with obesity is much diminished or absent by age 75 (age 85 in one large group of men), alterations in disease-specific mortality or morbidity risk are largely unknown. In addition, RCT evidence for clinical benefits with weight loss is based on a select sample, including reasonable gender and racial diversity, but lacking ethnic diversity. Trials focused on patients who were moderately overweight to mildly (stage I) obese and generally lacked substantial co-morbidities. It is unclear how these findings are generalized beyond this group.

4. Are there dietary or behavioral therapies that improve net health outcomes in obese elderly?

Obesity therapies with good evidence for improving health outcomes in the elderly incorporate both dietary and behavioral components; therefore, we consider these modes jointly here. All successful studies included exercise and used intensive counseling protocols. Effective interventions typically used diets based on reduced caloric intake, [12] often in the setting of low-saturated-fat and low-cholesterol intake goals. [13] Average weight loss was slightly less than found in younger samples (2-3 kg. versus 3-5 kg.); this may reflect the limited number of studies. Weight loss showed clinical utility, particularly for cardiovascular-related benefits such as oral glucose

tolerance testing or diabetes incidence. [13, 14] One intervention promoted a 71% reduction in diabetes incidence over placebo among glucose-intolerant elders (versus a corresponding reduction of 59% among those aged 45-59 years). In another trial, intervention participants showed a 30% reduction in a combined cardiovascular endpoint (persistently elevated blood pressure, re-initiation of antihypertensive medication, or a major blood pressure related clinical complication such as a cardiovascular event or surgery). [12] [15] While weight loss intervention efficacy data are very limited in the elderly, findings are consistent with the much larger body of literature focusing on younger adults. [6] The addition of long-term weight maintenance strategies helps to sustain weight loss in younger groups, but has not been evaluated among the aged. Although we did not specifically evaluate physical activity, it is important to note that both successful weight loss programs included a physical activity component. [13] [12] Also, incorporating exercise into weight dietary interventions may reduce the risk of bone loss. [16]

5. Are there surgical therapies that improve net health outcomes in obese elderly?

Current data are insufficient to assess the efficacy or safety of bariatric surgery in the elderly. We identified no RCTs evaluating surgical obesity treatment in the aged population. In the younger population, surgery can promote large degrees of weight loss among those with extreme obesity. [6] Its' complications are infrequent, but can be severe, including death. As chronic illness increases with age, and both age and co-morbidity have been linked with peri-operative risk, surgical adverse outcome rates based on younger populations may not generalize to the elderly.

Limitations

The primary limitation in these analyses is the lack of data specific to the elderly. Diagnostic testing studies were primarily limited by few calculated testing parameters

(e.g., sensitivity, specificity, and predictive values). Longitudinal studies often did not stratify findings by age or report attrition rates; also, statistical approaches frequently were inappropriate for our question. Dietary intervention studies showed high attrition rates, and sometimes, insufficient adjustment for potential confounders. Longitudinal studies had minimal racial diversity, and overall, the literature examined samples with limited age, ethnicity, and baseline weight status, potentially limiting generalizability of findings.

Conclusions

BMI is an adequate measure of body fat percentage in the elderly. WC and WHR provide additional information, particularly for identifying people at high cardiovascular risk. Skinfold thickness is a less desirable measure.

While, currently, there are no data directly measuring the effect that weight loss intervention has on mortality, intentional weight loss can serve as a valid marker for certain improved cardiovascular-related endpoints. Diabetes and blood pressure control are also important health problems, independent of their link to cardiovascular disease. [1] Possibly, additional benefits may be realized regarding cancer and functional disability. However, weight loss is also linked with adverse bone-related consequences.

The obese elderly most likely to benefit from weight loss are those with cardiovascular-related disease, or at high risk for these disorders. Those with high risk of cancer or functional decline may benefit as well. However, slowly developing disease processes and lack of reliable disease intermediaries makes evidence for cancer benefit difficult to establish. Intentional weight loss should be pursued with caution in the elderly with high osteoporosis risk. Among those with mixed risk patterns (e.g., cardiovascular and bone-related risk), relative benefits versus harms can only be individually assessed.

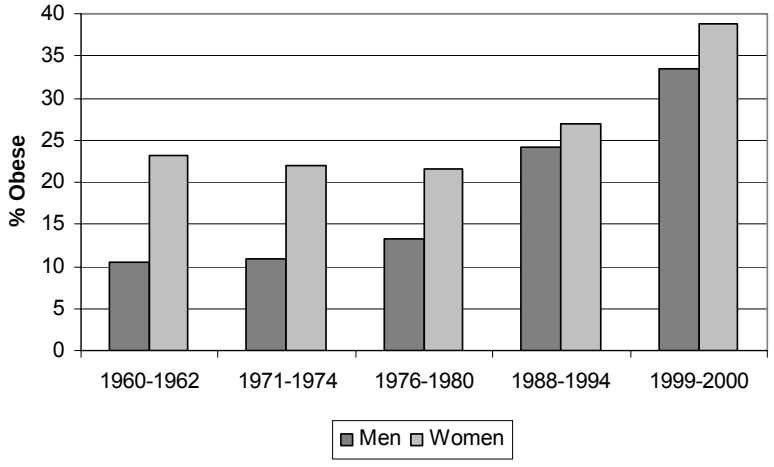
Overall, these findings reflect data from relatively healthy men and women; while race-specific data are limited, black participants' cardiovascular-related benefit from intentional weight loss appears similar to that of white participants. Modulation by ethnicity or underlying health status is unclear. As the relationship between all-cause mortality risk and obesity recedes with age and is absent after about age 74, any potential all-cause mortality benefit of weight loss programs likely diminishes as well. The relationship between morbidity or disease-specific mortality and obesity has not been assessed for age-related change in older adults.

Dietary interventions can promote clinically significant weight loss (2-3 kg.) in the aged over 1-3 years. Data reflect low calorie, often relatively low-fat diets, delivered with behavioral theory approaches. While we did not evaluate physical activity as an independent approach to obesity in the elderly, all successful dietary interventions included physical activity components, and physical activity may help offset the risk of bone loss found with intentional weight loss programs. These conclusions are based on efficacy (rather than effectiveness) data. Minimal data assessed harms of such counseling-based treatment. Data are insufficient to assess the safety or efficacy of bariatric surgery in the obese elderly.

I. BACKGROUND

Prevalence of obesity, a disorder of body composition defined by a relative or absolute excess of body fat, [2] is increasing among older US adults, paralleling the rise seen in the general adult and youth populations. [1] [17] In the setting of epidemic prevalence trends, the 2002 Surgeon General's Report establishes obesity as a health priority. [18] While older women are more likely to be obese then men, prevalence is increasing more rapidly among males: in 1960-1962, among US adults age 65-74, 10% of men and 23% of women were obese; 1999-2000 estimates are 33% and 39% respectively (Figure 1). [1] Over the age of 74, prevalence is lower, but still substantial: 20% of men and 25% of women in 1999-2000. These data reflect standard body weight definitions based on the Body Mass Index (BMI = kg./m^2). A BMI of 18.5-24.9 is considered healthy, 25-29.9 is overweight, and $\geq$ 30 is considered obese. [3]

Figure 1. Obesity prevalence trends among US adults age 65-74. [1]



In the general adult population, obesity has been associated with a diverse array of adverse health outcomes, including major causes of death such as cancer, diabetes, cardiovascular disease, as well as functional impairment from problems such as

osteoarthritis and sleep apnea. [3] A BMI of 30 kg./m^2 reflects the point at which obesity confers about 2-fold mortality risk in the general adult population. [3] The distribution of fat also mediates its' health implications. Central adiposity carries increased cardiovascular risk – even among the non-obese. [10, 19-22] In addition, visceral (versus subcutaneous) fat is particularly linked with adverse cardiovascular risk profiles in diverse ethnic/racial groups. [23] [24-29] In general, body composition varies with race/ethnicity; (e.g., Asians may be more likely [30] and African Americans less likely to accumulate visceral fat than Caucasians [24, 31, 32]) health implications may also vary. [29] [23-28]

Unique features of older Americans must be incorporated into consideration of obesity diagnosis, treatment benefits, and harms in this population. With aging, fat distribution shifts, potentially influencing both diagnostic accuracy of obesity tests and health risks. [33, 34] Central, versus peripheral fat accumulation, and the accumulation of deep fat relative to subcutaneous fat has been noted with aging. [35, 36] These changes may influence the accuracy of obesity diagnosis. Likewise, obesity treatment efficacy may be age dependent. As most obesity intervention trials focus on the middle-aged, efficacy and harms may not generalize to older populations.

Patterns of mortality and morbidity also vary with age, potentially influencing the health effects of intentional weight loss. Death rates rise with each decade of adult life and primary causes of mortality shift, with those associated with obesity becoming increasingly common. [1] [3] For example, among people 65 years of age or older, cardiovascular diseases surpass cancer as the top cause of death, and cerebrovascular disease displaces unintentional injuries as the third most common cause; [1] all three (though only certain cancers) are more common among the obese. [3] Hypertension prevalence increases with age (in 68% of men and 73% of women aged 65-74 years, versus 51% and 58% respectively, aged 55-64). Likewise, at age 60 or above, 19% of

people have diabetes and another 15% impaired fasting glucose; prevalence drops to 9% and 6% respectively in those aged 40-59. [37]

Functional limitation becomes increasingly important with age: 61% of US adults aged 85 or above report limitations in functional activities, up from 20% at age 55-64. Limitation in activities of daily living increase markedly (27% versus 3%) over the same age range [38] as does arthritis (the leading cause of US disability); [39] about 60% of those over the age of 65 report chronic joint disease or arthritis [40]. Likewise, falls and fractures become prevalent. [41] With escalating obesity prevalence in the old, functional concerns may be particularly relevant as short-term and cross-sectional data suggest a link with obesity. [42] [43, 44]

Obesity carries considerable societal cost, with estimated direct obesity costs of 5.7% of total US health expenditures. [45] Expected lifetime costs for cardiovascular disease and its risk factors increase by 20% with mild obesity, 50% with moderate obesity, and nearly 200% with severe obesity. [46]

Understanding the potential health effects of weight loss intervention is complicated by observational studies examining all-cause weight loss. A number of such studies have noted associations between weight loss and increased all-cause mortality. [47] [48] [49] [50] [51] Such findings, however, usually reflect lack of discrimination between intentional and unintentional weight loss; even when recognized, volition is difficult to measure, and transient weight loss may be misclassified. [52] [34] As clinically significant weight loss in the elderly frequently occurs in the setting of disease or psychological distress (depression, gastrointestinal complaints, and cancer are the most common causes), [52] unintentional weight loss is an important confounder. [53] [47] [34] Adjusting or excluding those with unintentional loss may eliminate [53] or diminish [50] this risk – the latter potentially reflects measurement problems as noted above. In the setting of such conflicting data and confounding, the evidence-based

medicine approach, focusing on randomized controlled trial (RCT) data over observational data, [7] is of particular importance for assessing the effects of intentional weight loss on health.

Options for intentional weight loss intervention include: counseling strategies for diet and/or exercise that often incorporate behavioral theory, as well as pharmacotherapy or surgery. Weight regain is a common problem which can be mitigated by long-term weight maintenance strategies. [3, 54] [4] [6] In the general adult population, counseling-based or pharmacologic treatment options are generally safe, and can lead to modest, sustainable improvements in weight sufficient for improving health measures such as glycemic control, lipids, and blood pressure. [3] [4] [5] [6] Improving these intermediaries may signal long-term cardiovascular benefit. [55, 56] Limited length of follow-up of weight-loss RCTs makes rigorous evaluation of changes in other health outcomes more difficult to assess. Surgical intervention, among the very obese, can produce substantial weight loss and marked improvement in blood pressure, lipid profile, glycemic control (including reversal of diabetes), and quality of life measures. [6] [3] [4, 57] Surgical data, though, are of lesser quality than counseling studies, and adverse events, while uncommon, can be serious, including death.

Thus, consideration of obesity treatment in the aged demands balancing multiple complex issues. The proportion of elderly with risk for diseases associated with obesity is considerable; if these risks improve with obesity treatment, intervention could have a large population effect. Among the frail, however, expected life span may be too limited to realize long-term health benefits of improving weight or cardiovascular intermediaries. Alternatively, any short-term improvement in functional status may substantially improve quality of life. Finally, accuracy of diagnosis and efficacy and harms of treatment are not well defined for older adults, and strongly influence the effects of intervention.

To determine how these factors interact among the aged, we conducted a systematic review of policy-relevant obesity diagnosis and treatment options.

II. METHODS

In this review we address five policy-relevant tasks posed by CMS and AHRQ: (1) Is BMI alone an adequate diagnostic test for obesity in the elderly or should another measurement be combined with or used instead of BMI?; (2) Can weight loss be used to predict improved health outcomes?; (3) Which obese elderly patients would experience improved health outcomes with weight reduction?; (4) Can dietary or behavioral therapies improve health outcomes in obese elderly?; (5) Can surgical therapies improve health outcomes in the obese elderly?

Key questions were adapted from an analytic framework for obesity treatment in the general adult population, [6] and address each step in the process from population at risk, diagnosis, treatment, and changes in intermediary and ultimate health outcomes. In answering these specific questions, we address a discrete subset of knowledge regarding body weight in the old. In order to capture all information pertinent to these questions for the aging population in the setting of limited data, we considered information on adults of at least 60 years of age (mean baseline age).

We defined obesity-related health outcomes to include: (1) mortality; (2) morbidity; including quality of life and function; and, (3) well-established intermediate outcomes of cardiovascular disease, such as glycemic control, blood pressure, and lipids. [55, 56] We were primarily interested in whether interventions could lead to improvements in the former, however, data on a number of pertinent health outcomes (e.g., cardiovascular events, cancer incidence, and death) are lacking because they require long-term follow-up or large sample sizes. Therefore, we followed the model of

other analyses assessing effectiveness of counseling interventions, and also examined the ability of intervention to affect intermediate health outcomes. [58]

We considered randomized controlled trials (RCTs) or systematic reviews of RCTs preferred evidence; when lacking, we evaluated other evidence such as cohort studies. [7] Evidence strength and study internal validity (Appendix 2) were rated using standard criteria. [7] [8] Because weight loss peaks at 6-months and tends to be transient, [3, 4] we excluded RCTs with less than 1-year of follow-up.

To determine long-term health implications of body weight, we considered cohort data for groups with at least 10 years of follow-up. Quality ratings of these observational studies included considerations of potential confounders identified in the medical literature: (1) we felt that adjusting for tobacco use was important, as smoking can both directly (by influencing appetite and eating patterns) and indirectly (by a causal role in chronic disease) alter weight dynamics; (2) because diet and exercise are primary mediators of obesity development, and we were interested in the net effect of obesity on health, we preferred relative risk estimates unadjusted for these factors; and, (3) we preferred exclusion of early deaths in studies examining mortality, to minimize the possibility that baseline weight, especially low weight (underweight), reflects abnormal weight dynamics due to concurrent disease.

We searched MEDLINE and the Cochrane Library for articles published in English between January 1980 and February 2003. We also reviewed bibliographies of published reviews to identify additional articles. Searches were limited to human population and English language. At least two authors independently reviewed abstracts and articles, excluded those not meeting eligibility criteria, and then retrieved eligible articles. If either reviewer felt an abstract met eligibility criteria, the article was obtained for review and data were abstracted into a standard abstraction form.

We examined evidence tables from well-done, prior systematic reviews of clinical approaches to obesity in the general population [3] [4] [54, 57] to identify any additional articles meeting our inclusion criteria. When these tables provided incomplete data to assess relevance, we obtained the primary literature for review. For topics on which insufficient data were available to assess key questions in the elderly, we drew from conclusions of these prior reviews in the general population. All data were synthesized into evidence tables and conclusions drawn from the aggregate findings.

III. RESULTS

Findings are organized according the five (A-E) policy relevant questions.

III.A. Are there limitations in diagnosing obesity in the elderly with BMI? Should another measurement be used with BMI or in place of BMI for diagnosing obesity in the elderly?

We approached the question of obesity diagnosis in the elderly by examining three facets of diagnostic tests: (1) evidence for diagnostic test validity and precision compared to referent adiposity measures, (2) evidence for associations of adiposity (as measured by different diagnostic tests) with long-term health risk, and (3) evidence that an alteration of body size with intervention is detected by the different adiposity measures.

III.A.1. Overview of clinical diagnostic tests

In clinical practice, obesity diagnosis is typically made through anthropometric measurements. Body mass index (BMI) is the most commonly used measure, expressed as kilograms of weight divided by the square of height in meters. In the general population, it is highly correlated (0.7 to 0.8) with adult body fat. [5] Chosen for its link with an approximately 2-fold increase in mortality risk, a BMI of 30 kg./m^2 or

higher is the standard clinical measure of obesity.; [3] Other health endpoints, particularly cardiovascular events, become increasingly prevalent throughout the BMI range of 25-30. [6]

Waist circumference (WC) and waist-to-hip ratio (WHR) are measures designed to better assess central adiposity. In the general population, they are especially useful for noting increasing cardiovascular risk within a non-obese BMI range. [10, 19-22] Measuring skin fold thickness can also identify adipose individuals, but requires significant training for accuracy. [5]

III A.2. Evidence for validity and precision of diagnostic tests for obesity in the elderly

Comparison between diagnostic tests and referent measures of body fat

Our searches identified 8 studies meeting our criteria for assessment of obesity diagnostic tests in elderly samples (Table 3). [10] [35, 59-64] They considered BMI, WHR, WC, and several skin-fold thickness measures. All were of fair internal validity, with problems including absent or atypical referent measures or lack of sensitivity, specificity, or predictive value calculations. None systematically evaluated adverse effects of testing. However, as these measures are entirely noninvasive the risk of harm is likely low.

Three studies compared BMI to a referent measure: body fat percentage as measured by bioelectrical impedance, [60, 61] or by dual-energy X-Ray absorbtiometry (DEXA). [59] [60, 61] Correlation with body fat percentage was somewhat lower than in the general population, with most data falling within the range of 0.59 to 0.73 in men, and 0.50 to 0.80 in women. In a small sample of older (> 66 years) individuals, a low correlation (0.37) was noted in men. [59] This finding is difficult to interpret, as it is discordant with that of a much larger (n=385) study with mean age of 80 years. [60, 61] An Asian (Hong Kong) study found that relatively low BMI corresponded with very high body fat (>40% in women, and >30% in men); this finding, which became more

pronounced with increasing age, likely reflects ethnic differences in fat accumulation. [61] In that study, from age 60-69, a BMI of 26 (mildly overweight) had a sensitivity of 91% in women, and 81% in men, with specificity of 86% and 83% respectively.

One study compared multiple adiposity measures to a referent measure of body fat percentage. [60] In men, among the common clinical diagnostic measures, body fat percentage (measured by DEXA) correlated highest with WC (0.78), followed by BMI (0.73), triceps (0.57), subscapularis (0.55) skin folds, and WHR (0.22). These correlations were minimally influenced by examining ratios between different skin fold measures, and generally slightly lower using bioelectrical impedance estimates of body fat percent. In women, BMI correlated most closely with body fat percentage (0.80), while triceps skin folds (0.67) and WC (0.64) showed similar, somewhat lower, correlations. Again, WHR did not correspond closely (0.16) to body fat percentage.

Of note, this study also measured the correlation between its two referent measures of body fat percentage (bioelectrical impedance versus DEXA – other data suggest DEXA is preferred in the elderly [65]). Correlation coefficients were 0.76 in men and 0.78 in women.

Comparison of diagnostic tests with each other

In five studies comparing BMI, WHR, and WC, [10, 35] [62] [63] [64]) pairwise comparisons typically showed the highest correlations between BMI and WC (0.73 to 0.83) [10, 62, 64], while the WC-WHR correlation was generally only slightly lower (0.72-0.76), [10] [62] [64] (although identical in one female sample). [62] WHR and BMI were weakly associated (0.26-0.55). In one study, age (55-69 versus $\geq$ 70 years) did not have an important effect on the relationship between these adiposity measures in either sex. [62]. However, the relative frequency of central adiposity (by WHR) increased with aging in one elderly cohort, while general adiposity (by BMI) dropped. [35]

One Swedish study evaluated the utility of WC to identify both generalized and central obesity in the aged. They found that an elevated waist circumference ($\geq$ 102 cm. in men, $\geq$ 88 cm. in women) was very specific (specificity 96.9 – 99.6, depending on gender and age) for identifying people who were either obese by BMI (BMI $\geq$ 30) or had an elevated WHR (men: $\geq$ 0.95; women: $\geq$ 0.80) [62] although, particularly among men, this measure showed poor sensitivity (sensitivity 33.4 to 63.7).

Another nationwide US study examined data on men and women age 60-90 years using ROC curves to determine gender- and ethnic-specific WC cut-points corresponding the a BMI of 30 kg./m^2. [63] Their derived WC values(103-105 cm. in men, 100-101 cm. in women) did not differ appreciably by race or ethnicity.

III.A.3. Consistency of health outcomes associated with adiposity diagnosed using different measures (BMI, WC, or WHR)

We reviewed 23 longitudinal studies with at least 10 years of follow-up, which examined long-term morbidity or mortality in relationship to baseline adiposity (by BMI, WC, or WHR) (Table 4). Studies addressed both mortality and morbidity risk. Internal validity was fair to good. In general, patterns of absolute (generally unadjusted) and relative (typically adjusted for smoking and sometimes other potential confounders) risk were very similar in these studies. Most studies (18 out of 21) adjusted relative risk estimates for tobacco or included only nonsmokers. [10] [66, 67] [68-70] [47, 71-76] [9, 64, 77, 78] [79] [80] Some studies adjusted for direct mediators of obesity (diet and/or exercise), [80] [10, 64, 69, 76] thus have the potential to underestimate obesity's net health effect. Studies typically did not exclude participants with early adverse health outcomes (e.g., ones in which baseline body weight may reflect an ongoing disease that subsequently leads to an event), however, consideration of studies with at least 10 years of follow-up should minimize this concern.

Two different methods typically assessed statistical significance of the relationship between long-term health risk and baseline adiposity: (1) comparison of risk in different body size categories with risk in a "referent" range, and (2) assessment for a trend in risk across the range of baseline body size. Notably, when comparing risk across body size categories, many studies used the sample's lowest values as referent. In a U-shaped relationship, with elevated risk for both the obese and the underweight, this technique limits ability to assess obesity risk, as it reflects risk of obesity versus underweight, rather than obesity versus "normal" weight.

III.A.3.i. Mortality risk and different measures of obesity

All Cause Mortality in Men: The relationship between anthropometric adiposity measures and long-term all-cause mortality was best described for BMI. Point estimates of mortality followed a U-shaped pattern with increasing BMI (higher mortality at both extremes of body weight) (Table 5). Longitudinal data consistently showed minimal long-term all-cause mortality among those in the normal-to-overweight BMI range. Overall, the strength of association between BMI and mortality lessened with age; the same has been seen in a review incorporating studies of shorter duration. [81] Five of eight studies finding a U-shaped relationship between relative risk for all-cause mortality and BMI [67, 68, 70] [72] [73] [74] [76] [80] compared risk between the heaviest participants and those with a middle-range BMI. [68] [80] [76] [70] [72] In one, relative risk was 2.75 (CI 2.17 to 3.49) for a BMI of $\geq$ 35 kg./m^2 versus 23.5 to 24.9 kg./m^2 among men age 65 to 74, but only 1.53 (CI 1.15 to 2.04) for the same body size comparison among men aged 75 or above. [68] In another, adjusted for multiple confounders, the risk associated with higher BMI dampened with age, and was not statistically significant by the age of 85 years (75 years in women). [80] The study with the youngest sample (mean age of 60 at baseline) found significantly increased mortality

risk with obesity: relative risk 1.5 (CI 1.09-1.86) for a BMI of > 30.2 kg./m^2 versus 23.8 to 25.4 kg./m^2. [76] Two with older baseline groups, (one with a range of 65-74 years [70] and the other with an average age of 70 years [72]) did not. Three studies comparing risk between the "tails" of the U-curve (e.g., heaviest versus thinnest participants), found no difference. [67] [73] [74] Only one [72] of three studies [67] [72] [74] assessing for trend across the baseline BMI range noted a significant effect.

Less data examined the relationship between central obesity (measured by WC or WHR) and long-term all-cause mortality risk. One study (baseline age $\geq$ 65; no mean noted) assessed BMI, WC, WHR, and total mortality (adjusted for multiple factors including age, family history, and smoking); all three showed U-shaped relationships of relative risk, no overall trend, and no difference in risk between the largest and smallest weight categories. [67] However, measures of central (versus general) adiposity showed higher estimates of relative risk: compared to participants in the lowest adiposity quintiles (BMI: < 23 kg./m^2; WC < 34.5 inches; or WHR <0.9), those in the largest quintiles (BMI $\geq$ 30 kg./m^2; WC $\geq$ 40.3 inches; WHR $\geq$ 0.98) showed relative risks of 0.85, 1.17, and 1.08 respectively. [67]

All Cause Mortality in Women: In women, seven studies found U-shaped relationships between BMI and point estimates of subsequent long-term mortality (Table 5). [10] [70, 72] [74] [68, 79] [80] In two age-specific studies, a similar age effect was seen as for men, with obesity generally associated with increased risk of mortality, but with smaller relative risk among older participants. [68] [80] Relative risks were overall slightly lower than men's. Three other studies examined all-cause mortality using a mid-BMI reference range. [79] [70] [72] Of these, one (baseline age range 55-84) found the largest participants had significantly increased mortality risk; [79] one (baseline age 65-74) found increased risk only among white participants; [70] and one (mean baseline age 70) found no statistical increase in risk. [72] Two studies compared the risk of

largest versus smallest participants; one showed no significant difference [10] and the other found the obese had lower risk of all-cause mortality. [74] Four studies assessing for trend across baseline BMI showed borderline [10] or significant [72] [74] [79] effects.

WC showed U-shaped relationships with all-cause mortality in two studies with broad baseline age ranges. [10, 67] One study illustrated a borderline but significant effect between largest and smallest quintiles, and a significant trend across baseline WC. [79] The second, also using the smallest participants as referent, was not significant. [67] Notably, WHR showed a different risk pattern in one of these studies – a fairly linear, positive (versus U-shaped) statistically significant increase in mortality risk with increasing WHR. [10] In this study (adjusted for multiple factors including age, education, physical activity, tobacco, hypertension, and diet), relative risk was consistently higher for central measures of adiposity (WC, WHR) than for BMI. Compared to participants in the lowest adiposity quintiles (BMI: < 22.8 kg./m^2; WC < 74.3 cm; WHR <0.76), those in the largest quintiles (BMI $\geq$ 30.2 kg./m^2; WC $\geq$ 96.3 cm; WHR $\geq$ 0.90) showed relative risks of 0.91, 1.3, and 1.5 respectively. [10]

Cardiovascular-Related Mortality in Men: Disease-specific mortality also varied across studies and between adiposity measures (Table 5). Five studies showed generally U-shaped relationship between BMI and cardiovascular-related mortality in the old, [67, 73-75, 82] and one showed a generally linear, positive relationship. [80] All relative risk estimates from these studies used the lowest quartile or quintile as referent and did not find any difference in risk between thinnest and most overweight participants. Trend in risk across baseline BMI was generally non-significant. [67, 74, 75] WC was a better predictor of cardiovascular-related mortality than BMI in one study (baseline age $\geq$ 65 years): coronary heart disease mortality increased directly (versus in a U-shaped pattern) with WC, with the largest participants (WC $\geq$ 40.3 inches) being 3.5 times (CI 1.6-7.7) more likely to die from cardiovascular disease than those with the

smallest waist (<34.5 inches). [67] Disease-specific mortality relationships with WHR were reported in only one study, and were non-significant after age 84. [80]

Cardiovascular-Related Mortality in Women: Similar patterns were found for disease-specific mortality as in men, though in the female studies, BMI at times showed U-shaped [74] [10] (cardiovascular disease), [10]at times negative (coronary heart disease), [74] and at times positive (ischemic heart disease, cardiovascular mortality) [78] relationships with cardiovascular-related risk (Table 5). These different relationships do not parallel age differences between the studies. As with men, some data suggest central adiposity has a more linear relationship with cardiovascular mortality than does generalized obesity. In one study, coronary heart disease mortality (but not "other cardiovascular disease" mortality) showed a statistically significant, generally positive, relationship with both WC and WHR (versus the U-shaped relationship seen in the same participants according to baseline BMI). [10] In this study, WC and WHR showed consistently higher relative risk estimates for CHD mortality than BMI. For example, among the largest quintiles, relative risk of coronary heart disease mortality (compared to the smallest quintiles) was 2.6 assessed by WC (CI 1.9-3.6), 2.5 assessed by WHR (CI 1.6-3.2), and 1.6 assessed by BMI (CI 1.2-2.2).

Cancer Mortality: We found little evidence for a link between BMI, WC, or WHR and cancer death in the elderly (Table 5). Three studies found no association between BMI and all cancer death, [78] [77] [67] or death from liver cancer. [77] One study in women showed a U-shaped relationship with a borderline significant trend between cancer mortality and BMI, no clear relationship between cancer mortality and WC, and a statistically significant positive relationship with WC. [10]

Other Disease-Specific Mortality: One study assessed hip fracture mortality by baseline BMI (Table 5). In women between the ages of 60-69, a U-shaped relationship was seen, with obese women having an 18% lower rate of fatal fracture than those with

a BMI less than 24.3 kg./m^2. Women aged 70-89 years and men aged 60-89 years showed a negative, generally linear relationship between baseline BMI and fatal hip fracture, with the quartile of largest participants (BMI >29-30 kg./m^2 in women; >27-28 in men) showing approximately one half the risk of the quartile of smallest participants (< 23-24 kg./m^2 in women; < 22-23 kg./m^2in men). [83]

Finally, one study found that death from respiratory disease showed a negative, statistically significant relationship with WHR. [67]

III.A.3.ii. Morbidity risk and different measures of obesity

Excess body weight (overweight to obese), as measured by BMI or WC, was significantly linked with incident stroke [66] [71] in men, but not women (Table 6). In one study, people with BMI above 27 were approximately 70% more likely to experience incident coronary heat disease. [47] Likewise, in a large group of US women, diabetes incidence was strongly linked with baseline adiposity, with a U-shaped pattern between baseline BMI and relative risk (RR for BMI > 30.2 kg./m^2 versus <22.8 kg./m^2: 13.8, CI 10.6 to 17.8) and a positive linear pattern for WC (RR for WC > 96.3 cm. versus <74.3 cm: 16.5, CI 9.0 to14.6) or WHR (RR for WHR $\geq$ 0.9 versus <0.76: 11.5, CI 9.0 to 14.6). Hypertension increased linearly with any of the diagnostic measures (relative risk for the same categories: 2.2 (CI 2.0-2.4), 2.3 (CI: 2.1 to 2.5), and 2.0 (CI 1.8 to 2.2) respectively).

Incident cancer (all causes) was 20% more common among largest (versus smallest) quintiles of women in the same study (Table 6). A similar increas in risk was seen for breast and colon cancer, regardless of adiposity measure, and higher risk noted for uterine cancer (RR in largest versus smallest quintiles for BMI: 3.5, CI 2.5 to 5.1; WC: 3.3, CI 2.3 to 4.8; WHR: 2.0, CI 1.4 to 2.8). No relationship was seen between adiposity and ovarian cancer. Obesity, measured by BMI, appeared protective of lung cancer;

[10, 67] risk disappeared after adjusting for multiple factors including smoking and physical activity. [64] In two other studies, non-significant U-shaped patterns were found between baseline BMI and both prostate cancer, [69] and non-Hodgkin's lymphoma [84] incidence.

Finally, weight in a moderately-overweight-to-obese range was predictive of incident limitations in functional mobility, showing approximately 2-fold risk. [9] However, increasing BMI (but not WC or WHR) was protective for hip fracture incidence. This effect was not limited to the obese range: risk was reduced by about 40% for anyone with a BMI over approximately 23 kg./m^2. [10]

Most longitudinal studies assessing body size and morbidity risk evaluated samples that were less than 70 years of age at baseline. Therefore, we were unable to assess for change in risk across old age.

III.A.4. Efficacy of weight loss intervention by different measures of obesity

All the weight loss RCTs we identified used kilograms or pounds as the primary outcome measure (Table 7). Two also assessed BMI; [85] [86] none evaluated for change in central adiposity. In addition, among younger adults, available data suggest that weight loss (kg.) corresponds with BMI loss. [86] [85]

III.A.5. Summary of Findings

Overall, among office-based diagnostic tests for obesity, BMI and WC showed very similar correlation with body fat percentage in men and women. Correlation with body fat percentage was slightly lower for skin fold thickness measurements, and markedly diminished for WHR. Correlation between anthropometric measures and body fat percentage was somewhat lower than found in younger samples. Limited, but mixed data suggest this correlation may decrease throughout old age. [59] [60, 61] Further evaluation of validity and precision of these measures is limited by a lack of sensitivity, specificity, or predictive value calculations. While WC correlates closely with body fat

percentage and aims to measure central adiposity, it showed low sensitivity when used as a single tool to identify older patients with either generalized (by BMI) or central (by WHR) obesity. [62] Gender did not appear to strongly affect these analyses' diagnostic accuracy, but the utility of diagnostic measures may differ across ethnic/racial groups. For example, in one Hong Kong sample, quite low (non-obese) BMI values were linked with high body fat percentage; [61] this finding is consistent with data for younger Asian populations. [87] Alternatively, a large US study including substantial proportions of white, black, and Hispanic participants found no ethnic difference in WC cutoffs corresponding to a BMI of at least 30 kg./m^2.

Data are mixed regarding the relative usefulness of WC or WHR in predicting long-term risk. BMI has been associated with the most health outcomes (supporting its clinical utility in the old); inconsistencies in BMI-mortality risk appear to be in part age-related. However, available data suggest that measures of central adiposity (WC or WHR) may be better predictors of total mortality and cardiovascular mortality and morbidity, as they exhibit more linear (versus U-shaped) relationships, and may identify people with higher degrees of relative risk than BMI across their respective ranges.

III.B. Can weight loss be used as a surrogate for improved net health outcomes? If so, how much weight loss and over what time period of time?

III.B.1 Health risk associated with obesity in the elderly

As discussed in detail in section III.A.3, obesity has been associated with increasing risk of a number of adverse health states in the elderly, including both mortality and morbidity (Tables 5 and 6). Conversely it may be protective of hip fracture and lung cancer.

Mortality Risk: Among men and women, point-estimates of long-term all-cause mortality typically showed a U-shaped pattern with increasing BMI. [10] [70, 72] [67, 68] [74] [79] [80] [73] [76] This relationship's inconsistent statistical significance appears largely a result of confounding by age or comparison of risk between the largest and smallest individuals, rather than between the largest individuals and ones with "healthy" baseline weight. All-cause mortality risk dampened with age and was typically much reduced or absent after approximately age 75 (age 85 among men in one study [61]). Limited data examining relationships between WC or WHR and all-cause mortality also typically showed U-shaped risk patterns [67, 10] (only one study found a linear pattern between WHR and all-cause mortality in women [10]), and showed higher estimates of relative risk, compared with BMI, across the examined range of baseline body size.

Point estimates of cardiovascular-related mortality risk typically was increased among men or women with general obesity (by BMI) when compared to those with "healthy" or "overweight" baseline status. [67, 73, 74, 75, 10, 82, 78] Some variance in findings and statistical significance was noted by outcome, across studies, and by sex. As with all-cause mortality, statistical or (in men) age-related related factors appear to explain much of this variance. In a single study, the relationship between BMI and cardiovascular-related death risk was evaluated according to age: it declined, and was absent after age 84 in men, and after age 74 in women.

In contrast to BMI, limited data indicate that baseline WC (and WHR in women; data are lacking for men) shows a positive, generally linear, relationship with long-term cardiovascular-related mortality risk. [67, 10] In addition, within samples relative risk for

cardiovascular-related death was consistently greater across the range of baseline central obesity measures than across the baseline range of BMI.

We found little evidence for a link between BMI, WC, or WHR and cancer death in the elderly. Hip fracture mortality increased with decreasing BMI; in women, this pattern was stronger among the oldest participants. [64] Likewise, death from respiratory disease was lowest among those with elevated WC. [47]

Morbidity Risk: A number of measures of cardiovascular-related disease were linked with general or central baseline adiposity: stroke (only in men), [46] [52] coronary heart disease, [47] diabetes, and hypertension. [10] In one large female study, incident breast cancer, colon cancer, and uterine cancer were more common among the obese. [10] Obesity, however, may be protective against lung cancer. [64] No significant relationship was found between obesity and ovarian cancer, [10] prostate cancer, [49] or non-Hodgkins lymphoma. [65] Moderately-overweight-to-obese body weight was linked with about 2-fold risk of incident limitation in functional mobility. [8] Finally, a BMI over approximately 23 kg./m^2 (in the middle of the "healthy" range) was protective of hip fracture.

III.B.2. RCT evidence for change in health status with weight loss in the elderly

Randomized controlled trials of weight loss in the elderly report mixed health effects of weight loss (Table 7). Overall, intervention participants in trials with modest weight loss success showed improved glycemic control as well as lower incidence of an outcome combining hypertension and cardiovascular events. However, they showed diminished bone mineral density (see section III.D.2). Although additional health effects of weight loss in the elderly are likely, others have not been evaluated in an RCT setting.

III.C. Which elderly patients with obesity would experience an improved health outcome with weight loss treatment?

We used two approaches to identify people likely to benefit from weight loss therapy: (1) consideration of who is at risk for weight-related adverse outcomes, and (2) consideration of the features of those with demonstrated health improvement following weight reduction intervention.

III.C.1. Characteristics of people with evidence for health risk associated with body weight

Data for long-term health effects of body weight in the aged were drawn from cohort studies of fairly diverse patient populations (Table 4). Average baseline age range was 60 to at least 96 years; as discussed above, relative risk of all-cause mortality was generally significant in a wide age range over 60 years, but the magnitude of the risk tended to be reduced or absent by age 75 (age 85 in one male sample). Both genders were reasonably represented with five female studies, [10] [9, 84] [64, 78] six male studies [66, 67, 69, 73, 75, 82] and ten studies where 43% to 79% of participants were female. [68, 70-72, 74] [76] [77] [79, 80, 83, 88] Fourteen analyses were US-based. [9, 10, 47, 64, 66-70, 73, 78-80, 84] Others were carried out in Scandinavian countries, [71, 72] [74, 76, 83] [88] England, [75] [82] or Italy. [77] Five studies noted entirely white populations [73, 78-80, 84]; one included 17% Black participants, [70] and one followed Japanese-Americans; [66] the rest did not report racial/ethnic composition. Most used community samples; about a quarter restricted their sample to participants who were generally healthy at baseline.

Overall, health risk associated with excess weight was established for men and women over the age of 60 years. Race- and ethnic-specific data were limited; conclusions largely apply to populations of European origin. While all-cause mortality

risk was considerably elevated among 60-74 year olds, it was largely non-significant after age 74. Data were insufficient to establish whether morbidity risk changes across old age.

III.C.2. Review of the characteristics of people for whom there is RCT evidence of benefit from weight loss

The best evidence for health status change accompanying intentional weight loss in the elderly comes from US-based studies with good gender and racial diversity (Table 7). [89] [15] [86] [13] [85] These patients were relatively healthy. In one trial all participants were glucose intolerant and therefore at high risk of diabetes. [13] In another, participants had hypertension controlled by a single antihypertensive medication as enrollment and no other significant co-morbidities. [12] Additional data reflect Swedish outpatients, [14] and US female inpatients following bypass grafting. [85]

III.C.3. Summary of Findings

Older people with evidence of obesity-associated health problems (cardiovascular disorders, cancer, or functional decline) stand to benefit most from intervention, if such intervention will alter their weight-related risk. Evidence for benefit from intentional weight loss in the elderly from well conducted RCTs is currently limited to improved cardiovascular-related measures, while harms focus on bone loss.

Cardiovascular risk factors – including family history, diabetes, tobacco use, or dyslipidemia – can identify those at high risk for cardiovascular disease. Risk for cancer or functional limitation is more complicated and often requires individualized patient assessment. However, several cancers with obesity-linked incidence (e.g., breast and colon) do have clear, identifiable risk factors. Finally, osteopenia, osteoporosis, or its risk factors may help identify those with risk of fracture – e.g., those most likely to experience harm from purposeful weight loss.

Among the elderly, the all-cause mortality risk associated with obesity (and therefore any potential all-cause mortality related benefit) diminishes with age. While risk of all-cause mortality with obesity is much reduced or absent by the mid-70's to early-80's (possibly persisting longer in men), age-related alteration in disease-specific mortality or morbidity risk among the elderly is largely unknown. In addition, RCT evidence for clinical benefits of weight loss is based on a select sample, including reasonable gender and racial diversity, but lacking ethnic diversity. These trials focused on patients who were moderately overweight to mildly (Stage I) obese, and often with at least one cardiac risk factor, but without substantial co-morbidities; the generalizability of findings beyond this group is unclear.

III.D. Are there dietary or behavioral therapies that improve net health outcomes in obese elderly?

III.D.1. Evaluation of evidence for dietary or behavioral therapy leading to weight loss in the elderly

We identified seven RCTs meeting eligibility criteria (Table 7). Because all dietary studies except one incorporated behavioral components, the two treatment modes are discussed jointly here. Four of the included studies [86] [15] [89] [90] analyzed data from the Trial of Non-Pharmacologic Interventions in the Elderly (TONE) study. To better compare across studies, we assessed treatment mode (diet, exercise, and behavioral) and intensity (based on frequency of encounter in the first 3 months). [6] Interventions which met monthly were considered of moderate intensity; more frequent were considered high intensity and less frequent of low intensity. One study did not record information to assess treatment intensity; [14] one was considered moderate-to-high [85]; five (four based on TONE study participants) were of high intensity. [86] [15]

[89] [13] [90] All except one [14] incorporated behavioral components. Effective interventions typically used diets based on reduced caloric intake, [12] often in the setting of low-saturated-fat and low-cholesterol intake goals. [13]

Internal validity of the included studies was fair [14] [85] [86] to good. [13] [15] [89] [90] Problems included high attrition (7.5 to 37%) and insufficient adjustment for potential confounders. In addition, studies generally did not report adverse effects of treatment. Most studies were based on US participants. [15, 89] [13, 86] [85] One focused on men, [14] two on women, [85] [86] and three included both. [13] [15] [89] Participants were generally fairly healthy and, when recorded, baseline age was typically in the 60's. Duration of follow-up was 1 to 3.3 years.

All high-intensity interventions showed statistically significant weight loss. [86] [15] [89] [13] Over 1-2.5 years, the TONE study, incorporating behavioral and exercise components, showed significant ($p<0.05$) average weight loss over placebo. One analysis showed an average 2.6 kg. loss after one year; one an average 3 kg. loss after 2.5 years; the third, stratified by race, a 3-year loss of 1.9 kg. among black participants and 3.3 kg. among white participants. One TONE analysis reported frequency of clinically significant weight loss ($\geq$ 4.5 kg.): 44% of those treated with the weight loss intervention versus 13% not counseled for weight loss (significance not reported). [15] The other (non-TONE) high-intensity intervention study's participants were aged 25 or above, with 20% of participants over the age of 60. While this study did not report age stratified weight results, it did show a significant weight loss (5.6 vs. 0.1 kg., $p<0.001$) in the entire sample over a mean of 2.8 years. [13]

The single study that evaluated a moderate-to-high-intensity behavioral approach did not show a statistically significant outcome. [85] The study examining diet without exercise or behavioral interventions was likewise not successful. [14] This study did not provide adequate information to assess intervention intensity. Results of both non-

significant trials are difficult to interpret, as tobacco cessation is a potential confounder: both showed higher (but not statistically significant) quit rates in the intervention groups than the control: 16% versus 5% in one, [85] and 32% versus12% in the other. [14]

No studies evaluated long-term maintenance interventions. However, one analysis of TONE data showed that at four years of follow-up (approximately two years after discontinuing intervention), the difference in weight change between weight loss and control groups was no longer statistically significant. [90]

III.D.2. Health status change with intentional weight loss or dietary therapies

The RCTs of weight loss intervention that reported health outcomes in addition to weight change focused on cardiovascular-related endpoints. Two dietary RCTs considered glycemic outcomes. In one, 20% of the overall group was over the age of 60; age-specific weight loss was not presented. Overall mean weight loss in the lifestyle intervention was 5.5 kg. beyond control after 2.8 years; elders in the lifestyle group showed a 71% reduction in diabetes incidence (CI 51 to 83%) compared with the placebo group. [13] Among those aged 45-59 years, corresponding diabetes reduction was only 59% (CI 44 to 70%). In another diet-only study of Swedish men, although there was no net weight change in intervention versus control participants, the sum of pre-and post-serum blood glucose from an oral glucose tolerance test was significantly lower in the intervention versus control group (sum of pre- and post- blood glucose concentrations: 24.6 +/- 3.9 mmol/L vs. 28.2 +/- 10.2 mmol/L). [14]

Two analyses of the TONE data considered a combined endpoint of either poorly controlled blood pressure, re-initiation of antihypertensive medications, or a cardiovascular surgery or event. [15] [89] They found an average 3.0 kg. weight loss corresponding to an approximately 30% reduction (RR 0.70, CI 0.57-0.87) in combined outcome after 2.5 years. [15] [89] When these authors stratified by race, trends were

similar, but non-significant, likely due to sample size limitations; they found no significant ethnic difference in incidence of the combined outcome (white: HR 0.72, CI 0.57 to 0.93; black: HR 0.74, CI 0.49-1.11). [89] A four year follow-up from the TONE study showed that although weight change was no longer significant, some benefit persisted in terms of hypertension: 17% of those in the weight loss group versus 7% of those in the usual care group had not needed re-initiation of antihypertensive medication. The two groups did not differ in cardiac event rate, but were not powered for this outcome. [90]

Another TONE sub study considered bone implications of weight loss in a subset of women: a 2.6 kg. loss corresponded to a decrease in bone mineral density (BMD) (0.00063 +/- 0.00021 g/cm^2 for each pound of weight loss). As the risk of hip fracture increases directly with age, and inversely with BMD, this finding may be clinically significant. [91] While the degree of BMD change is unlikely to have a large impact over the typical range of diet-induced weight loss (sustained 8 kg. of weight loss corresponds to 0.01 g/cm^2 decrement in BMD), for those at high risk of osteoporosis, it could be important.

III.D.3. Limitations and conclusions

Diet and behavioral treatment results are drawn from relatively few RCTs examining older adults. The findings, however, are very consistent with efficacy trials enrolling younger participants. [6] Intensity of intervention and a multifaceted approach (diet, behavioral, exercise) appear linked with intervention success. High-intensity dietary intervention, delivered with behavioral approaches and incorporating physical activity, led to about 3 kg. of weight loss. White participants generally lost slightly more weight than African American ones. The modest weight change seen with such diet and exercise interventions was associated with beneficial health outcomes, including glycemic improvement and diabetes prevention. [13, 14] Similarly, improved

cardiovascular risk, as assessed by incidence of a composite outcome (based on blood pressure control and cardiovascular events), was reported. [15] [89] Notably, there are also negative effects of intentional weight loss in the aged: bone mineral density loss showed a significant linear decline with weight loss in one study.

III.E. Are there surgical therapies that improve net health outcomes in obese elderly?

Bariatric surgery is restricted to patients with BMI > 40 or BMI $\geq$ 35 with associated health complications and past failure to respond to other treatment modalities. [92] Common procedures are either restrictive or malabsorptive, and include gastric bypass, adjustable gastric banding, vertical banded gastroplasty, and the duodenal switch procedure.

We identified no RCTs evaluating surgical obesity treatment in the elderly (Table 1). In the younger population, surgery can promote large degrees of weight loss (18-36 kg.), over a prolonged (18-36 months) period of time. [3, 4, 57, 93] [6] [54] Data must be interpreted with caution, however, as there are practical and ethical constraints to RCT study designs. These data reflect randomized trials comparing more than one procedure, and non-randomized controlled studies.

Surgical weight loss has been accompanied by marked improvements in a number of health outcomes including diabetes (improved blood glucose control or resolution of diabetes), hypertension (although there some data indicate that hypertension resolution is transient), dyslipidemia, and quality of life. [6, 93] [6] While bariatric surgery complications are infrequent, they can be severe, including death. As chronic illness increases with age, and both age and co-morbidity have been linked with peri-operative risk, [94-96] relatively low rates of adverse surgical outcomes based on younger populations may not generalize to the older group.

Overall, there are insufficient data to support the efficacy and safety of bariatric surgery among the elderly at this time. We draw this conclusion from the lack of bariatric efficacy and adverse effect data in the elderly, coupled with increased likelihood of adverse outcomes with age itself, and with the co-morbidities that tend to accompany obesity with age.

IV. DISCUSSION

IV.A. General Conclusions

Obesity is an increasingly common problem among US elderly, found in about a third of those aged 65 to 74 years, and 20-25% of those over the age of 74. Among the general adult population, there is good evidence to promote the diagnosis and treatment of obesity; respective findings are more complicated among the aged.

Diagnosis of obesity using anthropometric measures is easy and inexpensive. Although the correlation between BMI and fat mass is lower in older adults than in the general population, the difference is typically small. Data showing lower correlations among older men are limited and conflicting, while long-term health outcomes associated with BMI in the aged support its clinical utility. Two measures of central weight accumulation are also common: WC and WHR. Of the two, only WC correlates closely with body fat percent; limited data suggest both may be linked with higher relative risk of total or coronary heart disease mortality, as well as cardiovascular morbidity, than is BMI. At this time, data are insufficient to distinguish the relative utility of WC and WHR for identifying elderly persons with cardiovascular risk, but the ease of use of WC may make it more desirable. BMI, however, has been associated with a much wider range of health outcomes, including some (such as protection against fatal hip fractures) that are not linked to central adiposity. Likewise, RCTs evaluating

sustained efficacy of lifestyle intervention in the elderly have measured success using either BMI or weight change (which is directly correlated with BMI). Therefore, use of a central adiposity measure should not replace BMI as a diagnostic tool, but rather be used as an adjunct particularly for the identification of those with cardiovascular risk.

To date, there are no RCT data evaluating the effect of weight loss interventions on long term mortality. However, intentional weight loss is a reasonable surrogate for certain improvements in health outcomes in the elderly. A number of measures of cardiovascular disease (mortality, morbidity, and incidence of intermediary outcomes) are not only prospectively linked with obesity in the elderly, but RCT evidence shows that counseling-based interventions (combining dietary, exercise, and behavioral components) can improve cardiovascular risk profile. For example, improved fasting glucose tolerance, decreased diabetes incidence, and a decreased combined cardiovascular-related endpoint (either high blood pressure, re-initiation of antihypertensive medication, or a cardiovascular event or surgery) are associated with intervention. Only modest weight loss (2 to 3 kg.), sustained over one to three years, was sufficient to produce these clinically meaningful results.

As long-term longitudinal data support an increased risk of cancer incidence (but not mortality) in obese women (data are lacking for men), intentional weight loss potentially may influence cancer morbidity among the obese elderly. Because weight loss RCTs have limited duration of follow-up, and most cancers do not have reliable intermediary outcomes, it is difficult to assess the effect of intentional weight loss on cancer incidence; current data do not address the question. Similarly, there is strong evidence for incident functional limitation with increasing BMI; RCTs in the elderly have not assessed functional improvement to date.

Both longitudinal data showing that obese BMI is linked with lower hip fracture incidence and hip fracture mortality rate in the elderly, and RCT evidence of declining

bone mineral density with weight loss intervention suggest that intentional weight loss may be a marker for adverse bone consequences.

The obese elderly most likely to benefit from weight loss are those with cardiovascular-related disease, or those at high risk of such disorders. Those with high risk of cancer or functional decline would possibly benefit as well. Markers of cardiovascular risk – including age, family history, personal history of diabetes or hypertension, tobacco use, or dyslipidemia – have been well-defined, and can be used to identify a high risk group. Risk for any cancer is much more complicated. However several of the cancers with demonstrated obesity-linked incidence (e.g., breast, colon) have clear risk factors that could be identified clinically and are biologically plausible. Predicting functional limitation, however, is likely to require individualized physician assessment. Finally, osteopenia, osteoporosis, or its' risk factors are likely to best identify those with risk of fracture – e.g., those most likely to experience harm from purposeful weight loss.

Observational data suggest that any potential mortality risk from obesity diminishes with age and is much reduced or absent by the mid-70's to early 80's. Change in disease-specific mortality risk or morbidity risk across old age, however, can not be accurately assessed from the current medical literature. Successful dietary weight loss intervention has focused on participants with a relatively young [12] or poorly defined [13] age range. In addition, while diet and behavioral RCTs included participants with reasonable gender and racial diversity, ethnic diversity was lacking. Trials focused on patients who were moderately overweight to mildly (stage I) obese, generally at risk for cardiovascular disease, but without substantial co-morbidities.

Obesity therapies shown in RCTs to improve health outcomes in the elderly incorporate both dietary and behavioral components. All successful studies also included exercise and were delivered with a high-intensity (based on frequency)

approach. Average weight loss appeared slightly less (2-3 kg. versus 3-5 kg.) than among younger samples; this may reflect the limited number of studies. Clinical utility of this loss is supported by evidence for associated improved glycemic parameters, including improved oral glucose tolerance test values, as well as marked reductions in diabetes incidence among glucose intolerant individuals. [13] [14] Similarly, participants treated with an intensive intervention with diet, exercise, and behavioral components showed reduction in an endpoint combining hypertension and cardiovascular events or surgery; [15] improvements in hypertension control persisted over a year after discontinuation of intervention, though weight gain recurred. [90] While data in the elderly were very limited, findings are consistent with the much larger body of literature in the younger adult population. For example, data from the younger adult population suggest that intensive counseling techniques improve treatment efficacy, as does the combination of multiple modes of therapy. [6] Likewise, in younger adults, addition of long-term weight maintenance strategies has been shown to help sustain weight loss. We did not identify any RCTs evaluating such maintenance studies in older adults.

In this review we did not specifically evaluate physical activity or pharmacotherapy treatment options for obesity, as they were not considered policy options by CMS. However, physical activity has extensive health benefits, including potentially lessening the adverse health effects of obesity. [97] [98] In addition, exercise is an integral part of successful obesity lifestyle interventions in the elderly. Also, as weight bearing physical activity is protective of bone loss, [16] incorporation of exercise into weight loss programs may help offset bone risk.

Current data are insufficient to assess the efficacy or safety of bariatric surgery in the elderly. We identified no RCTs evaluating surgical obesity treatment in the elderly. In the younger population, surgery can promote large degrees of weight loss among a select group of patients. Its' complications are infrequent, but can be severe, including

death. As chronic illness increases with age, and both age and co-morbidity have been linked with peri-operative risk, [94-96] rates of adverse outcomes of surgery based on younger populations may not generalize to the older group.

IV.B. Future Research Topics

Consideration of the effectiveness of obesity diagnosis and treatment in the elderly is primarily limited by the lack of age-specific data in this field. Observational studies assessing health sequellae of obesity show a clear age-related gradient in total mortality risk, but such age-stratified analyses are lacking for disease-specific mortality, or for morbidity. As quality-of-life, morbidity, and health care costs are strongly influenced by these factors, such data are essential. Although available studies include well-done trials set in racially diverse populations, treatment efficacy data in the elderly are quite limited. Quality issues in some studies include high attrition, minimal reporting of harms, and sometimes inappropriate adjustment for potential confounders. No studies evaluated weight maintenance strategies in this group. Incorporation of greater ethnic diversity, and participants with higher stage obesity, as well as consideration of alternate measures of adiposity (e.g., WC, WHR), could add to understanding of the role of purposeful weight loss in elders' health.

A better understanding is needed of both how obesity risk for morbidity and how benefit from intervention changes across old age. Notably, our searches included studies with mean baseline age of at least 60 years – findings based on samples at the lower end of this range may not generalize to the very old. Likewise, assessment of a wider array of health consequences with intentional weight loss is needed – including characterizing the mechanisms of functional improvement with loss (especially important given the high prevalence and costs of disability in the aged), long-term assessment of cardiovascular or cancer outcomes, and impact on health care costs. Finally all the

dietary and behavioral interventions were efficacy trials; translation into clinical effectiveness is uncertain from these data.

The absolute lack of surgical RCT data makes this mode of treatment impossible to assess in the aged. Because safety concerns could be considerable, a first step may be analysis of existing data to determine adverse event rates in older bariatric surgery patients. If safe, it is potentially an important treatment mode for patients with extreme obesity.

As noted above, better characterization of the role of physical activity in weight loss and health in the elderly is needed. Similarly, the role of pharmacotherapy was not addressed here – as adverse effects of drugs are particularly problematic in the old, data from the younger population may not generalize to the old.

Finally, consideration of the net costs and benefits of obesity treatment in the elderly is essential in understanding the long-term policy implications of diagnosis and treatment strategies.

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Table 1. Policy-relevant tasks and Key Question approach for literature evaluation.

Tasks	Key Question Approach
1. Are there limitations in diagnosing obesity in the elderly with BMI? Should another measurement be used with BMI or in place of BMI for diagnosing obesity in the elderly?	• Evaluate evidence for the validity and precision of the different diagnostic studies in the elderly? • Assess how the different clinical measures of obesity are associated with adverse health outcome incidence • Assess use of different adiposity measures in randomized, controlled trials for weight loss, and compare findings (efficacy, related health effects) across these measures.
2. Can weight loss be used as a surrogate for improved net health outcomes? If so, how much weight loss and over what time period of time?	• Evaluate evidence for health risk associated with obesity in the elderly • Evaluate evidence that intentional weight loss leads to improved health outcomes. • RCT evidence • If RCT evidence lacking, draw inferences from general population evidence
3. Which elderly patients with obesity would experience an improved health outcome with weight loss treatment?	• Review of the characteristics of people for whom there is evidence of health risk associated with body weight • Review of the characteristics of people for whom there is RCT evidence of benefit from weight loss.
4. Are there dietary therapies that improve net health outcomes in obese elderly? (including discussion of duration, circumstances, co-interventions, harms)	• Evaluate evidence for dietary therapy leading to weight loss in the elderly • Evaluate evidence that intentional weight loss leads to improved health outcomes. • Evaluate evidence that dietary obesity therapy leads to improved health outcomes
5. Are there behavioral therapies that improve net health outcomes in obese elderly? (including discussion of duration, circumstances, co-interventions, harms)	• Evaluate evidence for behavioral therapy leading to weight loss in the elderly • Evaluate evidence that intentional weight loss leads to improved health outcomes. • Evaluate evidence that behavioral obesity therapy leads to improved health outcomes
6. Are there surgical therapies that improve net health outcomes in obese elderly? (including discussion of duration, circumstances, co-interventions, harms)	• Evaluate evidence for surgical therapy leading to weight loss in the elderly • Evaluate evidence that intentional weight loss leads to improved health outcomes. • Evaluate evidence that surgical obesity therapy leads to improved health outcomes

Table 2. Search and Abstraction Summary

Search	# Abstracts from Search	References for Article Review	References Meeting Full Criteria
Diagnostic tools	198	49	7
Longitudinal Studies	2042	200	23
Diet: Diagnostic	14	3	7
Diet: RCTs	288	79	
Diet: Reviews	145	24	
BT: Diagnostic	55	9	
BT: RCTs	263	20	
BT: Reviews	151	14	
Surgery: Diagnostic	0	0	0
Surgery: RCTs	9	1	
Surgery: Reviews	9	1	

BT = Behavioral Therapy
No studies evaluated behavioral therapy alone in this age group, so diet and behavioral interventions were grouped.

Table 3. Diagnostic Tests for Obesity.

Citation	% Female Mean Age Race	Sample	Tests	Gold Standard	Internal Validity
Deurenberg (59)	58% Female in the larger group (sex NR by age) Race NR	A subset of 1129 healthy participants age 7-83	BMI	BF% by bone density	Fair
Folsom (10)	100 % Female 55-69 years White	Iowa Women's Health Study Subset (n=31702): post-menopausal women had no history of CA, CHD or diabetes, and full BMI, WHR data (31,702=n)	WHR* WC† BMI	None	Fair
Goodman-Gruen (60)	64% Female Women: 79.4 years, Men: 80.3 years White	385 community dwelling ambulatory participants of the Rancho Bernardo Heard and Chronic Disease Study	BMI BF%§ (DEXA) Subscapular (SS) Skinfold Triceps Skinfold WHR Trunk Fat: Leg Fat (DEXA) SS: Triceps Ratio	BF % by Bioelectrical Impedence	Fair
Ko (61)	73% Female 51.5 years Race NR	5153 community volunteers for a primary care health check in Hong Kong in 1996-7	BMI	Very High BF > 40% (female) > 30% (male) by BI†	Fair
Logue (35)	67% Female ≥ 60 years In the larger (age ≥ 45) sample: 22% Black 75% White	Consecutive age-eligible outpatients (n=225 over the age of 60) seen in 1993 at 2 community-hospital-based, medical school-affiliated family practice centers in Ohio.	Elevated BMI >27.8 kg. (men) >27.3 kg. (women) Elevated WHR >0.95 (men) >0.80 (women)	None	Fair
Molarius (62)	% Female NR ≥ 55 years Race NR	Population sample (73%) of all people at least 55 years of age in Ommoord Sweden: 6423 participants	Elevated WC ≥ 102 cm (men) ≥ 88 cm (women)	BMI ≥ 30 OR elevated WHR Definition of Elevated WHR ≥ 0.95 (men) ≥ 0.80 (women)	Fair
Okosun (63)	Older Subset: 60-90 years (correlations reflect this group) General Sample: 53% Female; 42% White, 29% Black, 29% Hispanic	US NHANES III participants (sample size NR by age)	WC	BMI ≥ 30	Fair

Olson (64)	100% Female 55-69 Years (mean not reported) Race NR	Iowa Women's health study participants (n=38006) initially age 55-69 years in 1986, who were cancer-free (except skin cancer) at baseline	WHR WC BMI	None	Fair

Citation	Subset	Sens.	Spec	Correlations or Other Analyses
Deurenberg (59)		NR	NR	Correlation: BMI & BF% (BF% measured by densitometry) Age 56-65 MEN 0.72 p<0.01 WOMEN 0.50 p<0.01 Age > 66 MEN 0.37 NS WOMEN 0.51 NS
Folsom (10)		NR	NR	Pearson product moment correlation WC: BMI 0.82; WHR 0.72 WHR: BMI 0.4
Goodman-Gruen (60)	NR	NR	NR	Correlation with BF% (DEXA) (Age Adjusted) MEN: BMI 0.73; BF % (BI) 0.76; WC 0.78; WHR 0.22; SS Skinfold 0.55; Triceps Skinfold 0.57; Trunk : Leg 0.57; SS : Triceps -0.3 WOMEN: BMI 0.80; BF % (BI) 0.78; WC 0.64; WHR 0.16; SS Skinfold 0.59; Triceps Skinfold 0.67; Trunk : Leg 0.65; SS : Triceps 0.13
Ko (61)	WOMEN			NR
Age 60-69	BMI 26.2	0.91	0.86	
Age ≥ 70	BMI 25.5	0.97	0.86	
	MEN			
Age 60-69	BMI 25.9	0.81	0.83	
Age ≥ 70	BMI 24.1	1	0.67	
Logue (35)	NR	NR	NR	NR Linear decrease in elevated BMI frequency seen with age (p<0.001 for men, p=0.05 in women). In women: Linear increase in central obesity (by WHR) with age (p=0.003), but not men. In men: elevated BMI dropped from just over 60% at age 60-69 to approximately 12% at age > 80, while elevated WHR fell from about 55% to 30%. In women: Elevated BMI dropped from approximately 60% at age 60-69 to about 25% at age ≥ 80. Elevated WHR rose over the same timeframe: from about 80% to about 90%.
Molarius (62)	MEN			MEN (Pearson correlation coefficients) Age 55-69: BMI 0.77; WC 0.45 WC 0.76 Age ≥ 70: BMI 0.8; WC 0.55 WC 0.75
	Age 55-67	33.4	97.6	
	Age ≥ 70	37.9	96.9	
	WOMEN			
	Age 55-67	55.3	98	
	Age ≥ 70	63.7	99.6	WOMEN Age 55-69: BMI 0.75; WC 0.32 WC 0.76 Age ≥ 70: BMI 0.73; WC 0.26 WC 0.73
Okosun (63)	NR	NR	NR	NR ROC curves were use to determine the gender- and ethnic-specific WC cut-point corresponding to a BMI of ≥ 30. Among men and women, the derived WC value did not differ appreciably by race/ethnicity (103-105 in men, 100-101 in women). Larger WC correlated with lower BMI in older people
Olson (64)				(Pearson correlation coefficients) WHR: BMI 0.4 WC: BMI 0.83; WHR 0.72

* WHR = Waist: Hip Ratio
† WC = Waist Circumference
‡ BI = Bioelectrical Impedence
§ BF = Body Fat

Table 4. Description of longitudinal studies examining the relationships between different measures of body weight and health risk. Included studies have at least 10 years of follow-up, with a mean baseline age of at least 60 years. *NR: Not reported; † PA: Physical Activity; ‡RR: Relative Risk

Author	Sample	Duration	Race	% Female	Baseline Age	Baseline BMI	% Never smoked	% Loss to f/u	Potential Confounders, Diet, Activity	Outcome
Abbot (66)	1163 Non-smoking men with baseline age 55-68, of the Honolulu Heart Program, without high baseline CVA risk (hypertension, DM, LVH, CAD, CVA)	22 years	Japanese Americans	0	59.8	NR	100	>99% mortality, very high morbidity	Nonsmokers RR‡ not adjusted for diet or PA†	1st thromboembolic stroke
Baik (67)	Subset of the Health Professionals Follow-up Study: male health professionals followed since 1986, without recent significant weight loss at baseline	10 yr.	NR	0	≥ 65 (no mean given)	NR	44-54% in the larger group (varied with BMI)	NR	RR adjusted for smoking, age, FH MI or colon CA, profession, marital status, height, alcohol, vitamin and fiber intake	Mortality (Total and Specific)
Calle (68)	Generally healthy adults with no history of smoking, a subset (n not reported) of 1046154 participants with a broader age range, in the Cancer Prevention Study II.	14 years	NR	56% (larger cohort, not age specific)	≥ 65 subset	NR by age	NR by age	0.20%	Nonsmokers RR adjusted for age, education, physical activity, alcohol, marriage, aspirin or estrogen use, fat or vegetable consumption	Total Mortality
Cerhan (69)	Iowa 65+ Rural Health Study: 80% of the non-institutionalized residents at least 65 y/o in 2 Iowa counties: 81% completed baseline data and had no history of prior prostate cancer	10 yr.	NR	0	73.5	26	42	NR	RR adjusted for age, smoking, physical activity	Prostate Cancer Incidence
Cerhan (84)	37932 Iowa women, randomly selected from those with drivers' licenses, and no cancer at baseline. Self-reported height, weight, and waist circumference.	13 years	"Caucasian population"	100	55-69	NR	NR	NR	RR not adjusted for smoking, diet, activity	Non-Hodgkin's Lymphoma Incidence

Author	Sample	Duration	Race	% Female	Baseline Age	Baseline BMI	% Never smoked	% Loss to f/u	Potential Confounders, Diet, Activity	Outcome
Cornoni-Huntley (70)	NHEFS (f/u of HNANES I) - non-institutionalized civilian US Population	10 years	3339 total, 17% Black	52%	65-74 (mean NR)	NR	NR	NR	RR adjusted for Age and Smoking	Mortality
Dey (71)	2287 men and women measured between 1971 & 1981 in Goteborg, Sweden, with no prior stroke	15 years	NR	54	70	26	NR	19%	RR adjusted for adjusted for cohort, height, smoking, lipids, diabetes, heart disease, blood pressure.	1st stroke
Dey et al (72)	2628 participants with BMI <40 and no cancer at baseline, recruited via 3 random community samples, examined in at age 70 between 1971-1981 in Gothenburg Sweden	15 years	NR	53%	70	25.7 (Men) 26.0 (Women)	22% Male 79%Female	NR	Spline analysis for trend adjusted for smoking.	Mortality
Dorn (73)	190 Residents of Buffalo NY, a subset of the Buffalo Blood Pressure Study, randomly selected from the general population	29 year	100% White	0	65-96: Mean 71	Men: 25.3 Women: 26.3	NR age-specific	NR	RR adjusted for age, education, smoking	All Cause and Cause-specific Mortality
Ellekjaer (74)	6392 Norwegian men and women initially free from stroke, heart disease, diabetes, and without BP medication use.	10 years	NR	51	77	Men: 24-25 (varied by age) Women: 25-27	current smokers: men: 15-33%, women 2-12% (decreased with age)	Very low - used national death registry	RR adjusted for age, blood pressure, smoking	Total and Cause-Specific Mortality
Fitzgerald (75)	Subset of 18403 male civil servants in London (the Whitehall Study): 2387 men age 60-64 years between 1968-1970	15 years	NR	0%	60-64	NR	15.70%	NR	RR not adjusted for smoking, diet, or exercise	CHD Mortality

Author	Sample	Duration	Race	% Female	Baseline Age	Baseline BMI	% Never smoked	% Loss to f/u	Potential Confounders, Diet, Activity	Outcome
Folsom (10)	Iowa Women's Health Study Subset: post-menopausal women with no history of CA, CHD or diabetes, and full BMI, WHR data (31,702=n)	11-12 years	99% White	100%	55-69	NR	55-72 (differs by BMI)	NR	Overall RR adjusted for: Age, education, physical activity, alcohol, smoking, first birth,, estrogen use, vitamin use, diet, hypertension	All Cause & Specific Mortality, multiple measures of morbidity incidence
Harris (47)	1581 participants of the Epidemiologic Follow-up Study of NHANES I, who provided a baseline BMI in 1971-1975, and were free of heart disease in 1982-1984.	From 1971-1975 to mid 1980's.	White	61	60-76 in 1972-1974	NR	60%	NR	RR adjusted for age and smoking	CHD Event
Heitmann (76)	787 men from a community sample in Gothenburg, Sweden	22 years	NR	0	60	25.5	21	NR	RR adjusted for smoking and physical activity	Mortality
Launer (9)	NHEFS subset: 426 white women age 60-74 at the NHANES I sample, who did not report any disability in 1982-1984: looked for development of disability by 1986-1987	Approximately 10 years	White	100	66.1	26.4	79	NR	RR adjusted for age, smoking, education, time to follow-up	Mobility disability
Mazza (77)	1028 elderly subjects from a rural, low-income Italian populations in the "Cardiovascular Study in the Elderly"	12 years	NR	59%	74.3	27.3	NR	NR	Relative Risk adjusted for age, gender, smoking, resp. symptoms, ALT, ALP, town	Cancer mortality
Meyer (83)	Men and women age 50-89 at in a national tuberculosis screening exam in Norway (1963-1975). Data was available for 673,848 people: population attendance at initial screening was 80% age 50-69, 70% age 70-79, 42% for age 80 or over	mean 16.4 years	NR	53%	50-89 (mean NR)	NR	NR	NR	RR not adjusted for smoking	Fatal Hip Fracture

Author	Sample	Duration	Race	% Female	Baseline Age	Baseline BMI	% Never smoked	% Loss to f/u	Potential Confounders, Diet, Activity	Outcome
Olson (64)	Iowa Women's health study participants initially age 55-69 years in 1986, who were cancer-free (except skin cancer) at baseline (n=38,006)	12 years	NR	100%	55-69 years (mean not noted)	NR	NR	NR	Relative Risk adjusted for age, smoking, physical activity, education, beer consumption	Lung Cancer
Shinton (82)	Subset of the Whitehall Study of 18403 male civil servants examined between 1967-1969 until 1985	15 years	NR	0	55-64 (no mean reported)	NR	19% (larger group)	NR	RR not adjusted for smoking, though sub-analyses showed smokers risk was $\geq$ 2-fold that of lean non-smokers for.	Stroke Mortality
Singh and Lindsted (78)	12576 non-Hispanic white California Seventh-Day Adventist women who had never smoked	26 years	White	100%	55-74 (no mean noted)	NR	100	6% in a subset	Nonsmokers. RR is adjusted for age.	Cause-Specific Mortality
Singh (79)	Subset of the Adventist Health Study, non-Hispanic white adults in California	12 years	White	65%	55-84 (no mean reported)	13.4-65.6 Range	100%	3-4% per subsample	Never smokers	All Cause Mortality
Stevens (80)	A subset (n=46954) of participants in the Cancer Prevention Study I who had not history of smoking, were generally healthy, and without recent unintentional weight loss at enrollment.	12 years	100% White	79%	$\geq$ 65	Men: 23.2-25.1 Women: 23.4-25.0	100%	NR	Nonsmokers RR adjusted for age, education, physical activity, alcohol.	Total and Coronary Heart Disease Mortality
Takala (88)	721 (80% of the total aged population) participants over the age of 65, initially sampled in coordination with tuberculosis screening in rural Hankasalmi Finland in 1971	10 years	NR	57%	71.6	27.3	NR	NR	RR not adjusted for smoking, diet, exercise	Mortality

Table 5. Summary of studies examining relationships between a) BMI b) Waist Circumference and c) Waist to Hip Ratio and prospective mortality risk.

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	p (trend: RR)
A. Studies Examining BMI and Mortality							
Total Mortality							
Baik (67)	<23	NR		NR	1		.59 (trend)
	23-24.9	NR			0.69	.48-0.99	
	25-26.9	NR			0.75	.53-1.08	
	27-29.9	NR			0.83	.56-1.23	
	≥ 30	NR			0.85	.49-1.46	
Calle (68)	*MEN*						
	Age 65-74	Deaths / 100,000 py			Multivar RR	95% CI	NR
	<20.5	1089	NR	NR	1.21	0.95-1.53	
	20.5-21.9	816	NR		0.9	0.74-1.08	
	22.0-23.4	854	NR		0.96	0.84-1.09	
	23.5-24.9	898	NR		1		
	25.0-26.4	941	NR		1.04	0.93-1.16	
	26.5-27.9	1038	NR		1.12	1.00-1.26	
	28.0-29.9	1270	NR		1.34	1.19-1.52	
	30.0-31.9	1370	NR		1.42	1.22-1.66	
	32.0-34.9	1798	NR		1.85	1.54-2.22	
	≥ 35	2767	NR		2.75	2.17-3.49	
	AGE ≥ 75						
	<20.5	4564	NR	NR	1.16	1.02-1.32	NR
	20.5-21.9	4344	NR		1.12	1.01-1.24	
	22.0-23.4	3956	NR		1.01	0.93-1.09	
	23.5-24.9	3924	NR		1		
	25.0-26.4	4024	NR		1.01	0.94-1.09	
	26.5-27.9	4206	NR		1.06	0.97-1.15	
	28.0-29.9	4840	NR		1.21	1.10-1.33	
	30.0-31.9	4687	NR		1.16	1.01-1.32	
	32.0-34.9	5393	NR		1.31	1.10-1.55	
	≥ 35	6154	NR		1.53	1.15-2.04	
	WOMEN						
	Age 65-74						
	<18.5	594	NR	NR	0.99	0.78-1.25	NR
	18.5-20.4	531	NR		0.91	0.80-1.03	
	20.5-21.9	503	NR		0.86	0.77-0.96	
	22.0-23.4	498	NR		0.84	0.76-0.93	
	23.5-24.9	595	NR		1		
	25.0-26.4	598	NR		0.99	0.89-1.10	
	26.5-27.9	636	NR		1.04	0.94-1.16	
	28.0-29.9	796	NR		1.28	1.15-1.43	
	30.0-31.9	836	NR		1.32	1.17-1.50	
	32.0-34.9	1111	NR		1.71	1.51-1.95	
	35.0-39.9	1299	NR		1.99	1.69-2.34	
	≥ 40	1434	NR		2.09	1.57-2.78	
	AGE ≥ 75						
	<18.5	4051	NR	NR	1.44	1.31-1.58	NR
	18.5-20.4	3124	NR		1.16	1.09-1.25	
	20.5-21.9	2848	NR		1.06	1.00-1.13	
	22.0-23.4	2732	NR		1.01	0.96-1.08	
	23.5-24.9	2711	NR		1		
	25.0-26.4	2837	NR		1.04	1.97-1.10	
	26.5-27.9	2960	NR		1.07	1.00-1.15	
	28.0-29.9	3209	NR		1.15	1.07-1.23	
	30.0-31.9	3514	NR		1.25	1.15-1.35	
	32.0-34.9	3939	NR		1.36	1.23-1.49	
	35.0-39.9	4348	NR		1.53	1.33-1.75	
	≥ 40	4363	NR		1.41	1.10-1.81	
Cornoni-Huntley (70)	*BLACK MEN*						
	<20.5	U-shaped graph		NR	2.3	1.4-3.9	NR
	20.5-22.95				1.8	1.1-2.8	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	p (trend: RR)
Cornoni-Huntley (70)	22.95-25.58				1		
	25.58-29.55				1.2	0.8-1.9	
	≥ 29.55				1.4	0.8-2.4	
	BLACK WOMEN						
	< 22.18	U-shaped graph		NR	1.9	1.1-3.3	NR
	22.18-26.22				1.3	0.8-2.2	
	26.22-28.82				1		
	28.82-34.37				0.9	0.5-1.6	
	≥34.37				1.3	0.7-2.3	
	WHITE MEN						
	<21.68	U-shaped graph		NR	1.3	1-1.6	NR
	21.68-24.42				1.2	0.9-1.4	
	24.42-26.39				1		
	26.39-29.3				1	0.8-1.2	
	≥ 29.30				1.1	0.9-1.4	
	WHITE WOMEN						
	<21.43	U-shaped graph		NR	1.7	1.3-2.2	NR
	21.43-24.37				1.2	0.9-1.6	
	24.37-26.95				1		
	26.95-31.26				1.4	1.1-1.8	
	≥ 31.26				1.6	1.2-2.1	
Dey (72)	*MEN*						
	14.0-22.6	NR		NR	1.2	0.96-1.51	0.000
	22.7-24.6	NR			1.07	0.85-1.34	(nonlinear)
	24.7-26.4	NR			1	ref	
	26.5-28.5	NR			1.01	0.81-1.26	
	28.6-39.2	NR			1.19	0.95-1.49	
	WOMEN						
	14.1-22.5	NR		NR	1.49	1.14-1.96	0.004
	22.6-24.5	NR			1.16	0.88-1.53	(nonlinear)
	24.6-26.5	NR			1		
	26.6-29.2	NR			1.16	0.88-1.52	
	29.3-39.8	NR			1.25	0.95-1.64	
Dorn (73)	17.9-22.9	NR		NR	1		
	23.0-25.1	NR			0.67		NS (Graph)
	25.2-27.4	NR			0.63		NS (Graph)
	27.5-47.0	NR			0.77		NS (Graph)
Ellekjaer (74)	MEN						
	≤ 22.95	9.09	NR	NR	1		0.11
	22.96-25.10	6.68	NR		0.8	.69-.93	
	25.11-27.35	6.69	NR		0.8	.69-.93	
	27.36	6.61	NR		0.88	.75-1.02	
	WOMEN						
	≤ 23.23	6.46	NR		1		<0.01
	23.24-25.97	4.54	NR		0.74	.62-.88	
	25.98-29.00	3.97	NR		0.62	.52-.75	
	≥ 29.01	4.13	NR		0.68	.57-.81	
Folsom (10)		Cases/100py					
	< 22.8	0.88	NR	NR	1		0.05
	22.80- 24.87	0.70	NR		0.76	0.7-0.9	Multivariable
	24.87-27.06	0.66	NR		0.74	0.7-0.8	RR for Quintile
	27.06-30.21	0.64	NR		0.71	0.6-0.8	5 vs. 1:
	>30.21	0.82	NR		0.91	0.8-1.0	0.91(0.8-1.9)
Heitmann (76)	21.3	NR		NR	1.3	0.94-1.68	NR
	23.8	NR			1.1	NR	
	25.4	NR			1		
	27.1	NR			1.2	NR	
	30.2	NR			1.5	1.09-1.96	
Singh (79)	WOMEN						
	13.4-20.6	NR		NR	1.3	1.1-1.6	<0.00001
	20.7-22.4	NR			0.9	0.7-1.2	(nonlinear)
	22.5-24.2	NR			1		
	24.3-27.4	NR			1.1	0.9-1.3	
	27.5-65.6	NR			1.5	1.2-1.8	
	MEN						
	13.5-22.3	NR		NR	1.3	1.0-1.9	0.009
	22.4-23.7	NR			1.5	1.1-2.0	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	p (trend: RR)
Singh (79)	23.7-25.3	NR			1		
	25.4-27.3	NR			1.2	0.9-1.7	
	27.4-40.5	NR			1.8	1.3-2.5	
Stevens (80)	*MEN*	Deaths per 100,000 person-years					
	Men age 65-74	U-Shaped Curves		NR	U-Curves		NR
	Men age 75-84	U-Shaped Curves		NR	U-Curves		NR
	Men age ≥ 85	U-Shaped Curves		NR	U-Curves		NR
	WOMEN						
	Women age 65-74	U-Shaped Curves		NR	U-Curves		NR
	Women age 75-84	U-Shaped Curves		NR	U-Curves		NR
	Women age > 85	U-Shaped Curves		NR	U-Curves		NR
Takala (88)	All	% Survival					
	<23	39.90	NR	NR	NR		NR
	23-25	42.30	NR		NR		
	26-29	51.00	NR		NR		
	≥ 30	54.20	NR		NR		
	MEN						
	<23	39.00	NR	NR	NR		NR
	23-25	38.60	NR		NR		
	26-29	40.90	NR		NR		
	≥ 30	48.70	NR		NR		
	WOMEN						
	<23	41.10	NR	NR	NR		NR
	23-25	45.70	NR		NR		
	26-29	58.20	NR		NR		
	≥ 30	55.90	NR		NR		
Disease-Specific Mortality							
Baik (67)	*Cancer Mortality*						
	23	NR		NR	1		0.21
	23-24.9	NR			0.87	.48-1.6	
	25-26.9	NR			0.77	.53-1.08	
	27-29.9	NR			0.64	.31-1.34	
	≥ 30	NR			0.67	.24-1.87	
	CVD Mortality						
	23	11.00		NR	1		0.98
	23-24.9	11.00			0.58	.25-1.38	
	25-26.9	NR			0.95	.43-2.11	
	27-29.9	NR			0.94	.39-2.24	
	≥ 30	NR			0.7	.19-2.66	
Dorn (73)	*Cardiovascular Mortality*	NR					
	17.9-22.9	NR		NR	1	NR	
	23.0-25.1	NR			0.77	NR	NS (Graph)
	25.2-27.4	NR			0.66	NR	NS (Graph)
	27.5-47.0	NR			0.73	NR	NS (Graph)
	CHD Mortality						
	17.9-22.9	NR		NR	1	NR	
	23.0-25.1	NR			0.73	NR	NS (Graph)
	25.2-27.4	NR			0.7	NR	NS (Graph)
	27.5-47.0	NR			0.78	NR	NS (Graph)
Ellekjaer (74)		Cases/100 py					
	Death *from CVD: MEN*						
	≤ 22.95	0.93	NR	NR	1		0.71
	22.96-25.10	0.96	NR		1.1	0.72-1.67	
	25.11-27.35	0.71	NR		0.74	0.46-1.18	
	27.36	1.02	NR		1.21	0.79-1.84	
	Death from CVD: Women						
	≤ 23.23	1.08	NR	NR	1		0.16
	23.24-25.97	0.72	NR		0.85	.55-1.32	
	25.98-29.00	0.66	NR		0.69	.43-1.10	
	≥ 29.01	0.63	NR		0.76	.48-1.19	
	Death from CHD: Men						
	≤ 22.95	1.91	NR	NR	1		0.76
	22.96-25.10	1.39	NR		0.8	.58-1.11	
	25.11-27.35	1.62	NR		0.98	.71-1.34	
	27.36	1.56	NR		0.99	.71-1.36	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	p (trend: RR)
Ellekjaer (74)	*Death from CHD: Women*						
	≤ 23.23	1.04	NR	NR	1		0.35
	23.24-25.97	0.84	NR		0.97	.65-1.45	
	25.98-29.00	1.07	NR		0.99	.67-1.48	
	≥ 29.01	0.80	NR		0.81	.86-2.52	
Fitzgerald (75)	*CHD Mortality*	Cases/100 py					
	<22.4	1.18	0.90-1.49	0.28	NR		NR
	22.4-24.0	0.93	0.70-1.25	(linear)	NR		
	24.1-25.4	1.23	0.97-1.55		NR		
	25.5-27.0	1.21	0.96-1.52		NR		
	>27.0	1.27	1.03-1.56		NR		
Folsom (10)	*CHD Mortality*						
	< 22.8	0.11	NR	NR	1		<0.001
	22.80- 24.87	0.11	NR		0.94	0.7-1.3	Multivar RR
	24.87-27.06	0.13	NR		1.1	0.8-1.6	1.7 (1.2-2.3)
	27.06-30.21	0.12	NR		1.1	0.8-1.5	
	>30.21	0.18	NR		1.6	1.2-2.2	
	Other CVD Mortality						
	< 22.8	0.11	NR	NR	1		0.7
	22.80- 24.87	0.09	NR		0.77	0.6-1.1	Multivar RR
	24.87-27.06	0.07	NR		0.60	0.4-0.9	0.72 (0.5-1.1)
	27.06-30.21	0.10	NR		0.86	0.6-1.2	
	>30.21	0.10	NR		0.89	0.6-1.2	
	Cancer Mortality						
	< 22.8	0.40	NR	NR	1		
	22.80- 24.87	0.35	NR		0.84	0.7-1.0	0.06
	24.87-27.06	0.31	NR		0.77	0.6-0.9	Multivar RR
	27.06-30.21	0.30	NR		0.72	0.6-0.9	1.0 (0.8-1.2)
	>30.21	0.36	NR		0.89	0.7-1.1	
Mazza (77)	*Cancer Mortality*						
	≤ 22.7	0.14	NR	0.04	1.63	.96-2.56	NR
	22.8-25	0.08	NR		1 (middle 3 quintiles)		
	25.1-26.7	0.12	NR				
	26.8-29.5	0.11	NR				
	≥29.6	0.10	NR		0.91	0.53-1.56	
	Lung Cancer Mortality						
	≤ 22.7	0.05	NR	<0.004	2	1.13-4.52	NR
	22.8-25	0.05	NR		1.24	0.98-1.46	
	25.1-26.7	0.03	NR		1 (largest 3 quintiles)		
	26.8-29.5	0.03	NR				
	≥29.6	0.02	NR				
	Liver Cancer Mortality						
	≤ 22.7	0.03	NR	<0.02	2.69	1.04-5.6	NR
	22.8-25	0.03	NR		1 (middle 3 quintiles)		
	25.1-26.7	0.03	NR				
	26.8-29.5	0.02	NR				
	≥29.6	0.01	NR		0.88	0.32-1.21	
Meyer (83)	Fatal Hip Fracture: Women						
	AGE 60-69						
	<24.3	8.34	NR	NR	1		NR
	24.3 – 27.0	7.09	NR		0.82	0.72-0.95	
	27.1-30	6.33	NR		0.73	0.62-0.84	
	>30	6.81	NR		0.82	0.72-0.95	
	AGE 70-79						
	< 24.1	34.03	NR	NR	1		NR
	24.1-26.9	22.61	NR		0.65	0.57-0.73	
	27-29.9	20.36	NR		0.59	0.52-0.67	
	>29.9	18.64	NR		0.57	0.50-0.65	
	AGE 80-89						
	< 23.1	70.84	NR	NR	1		NR
	23.1 – 25.9	52.76	NR		0.73	0.56-0.94	
	2.6-28.9	44.64	NR		0.59	0.45-0.78	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	p (trend: RR)
	>28.9	38.87	NR		0.55	0.41-0.73	
Meyer (83)	Fatal Hip Fracture: Men						
	AGE 60-69						
	<23.1	7.47	NR	NR	1		NR
	23.1-25.2	5.10	NR		0.66	0.56-0.80	
	25.3-27.5	4.36	NR		0.57	0.48-0.69	
	>27.5	3.73	NR		0.52	0.42-0.64	
	AGE 70-79						
	<22.9	23.80	NR	NR	1		NR
	22.9-25.2	16.09	NR		0.68	0.57-0.81	
	25.3-27.5	11.59	NR		0.48	0.39-0.58	
	>27.5	10.65	NR		0.48	0.39-0.59	
	AGE 80-89						
	<22.4	49.12	NR	NR	1		NR
	22.4-24.6	29.41	NR		0.58	0.40-0.85	
	24.7-26.9	31.41	NR		0.62	0.43-0.90	
	>26.9	23.50	NR		0.52	0.340-.78	
Shinton (82)		*# Stroke Deaths/100 Py*					
	≤22.4	0.163485	NR	NR	1		NR
	22.5-24.0	0.153561			0.97	NR	
	24.1-25.4	0.136304			0.83	NR	
	25.5-27.0	1.490826			0.91	NR	
	≥ 27.1	0.194901			1.19	0.7-2.0	
Singh (78)	*Ischemic Heart Disease Death*	487 deaths					
	<21.3	NR			0.9	0.7-1.2	NR
	21.4-27.4	NR			1		
	>27.4	NR			1.2	1.0-1.5	
	CVD Mortality	232 deaths					
	<21.3	NR			0.8	0.5-1.2	NR
	21.4-27.4	NR			1.00		
	>27.4	NR			1.10	0.8-1.5	
	Other Cardiovascular Mortality	200 deaths					
	<21.3	NR			0.9	0.6-1.4	NR
	21.4-27.4	NR			1		
	>27.4	NR			1.5	1.1-2.0	
	Cancer Mortality	188 deaths					
	<21.3	NR			0.9	0.6-1.3	NR
	21.4-27.4	NR			1.00		
	>27.4	NR			1.00	0.7-1.4	
	Other Mortality	238 deaths					
	<21.3	NR			0.9	0.6-1.3	NR
	21.4-27.4	NR			1		
	>27.4	NR			1	0.8-1.4	
Stevens (80)	*MEN*	Cardiovascular Deaths per 100,000 p-y					
	Men age 65-74	U-Shaped Curves		NR	U-Curves		NR
	Men age 75-84	U-Shaped Curves		NR	U-Curves		NR
	Men age ≥ 85	U-Shaped Curves		NR	U-Curves		NR
	WOMEN						
	Women age 65-74	U-Shaped Curves		NR	U-Curves		NR
	Women age 75-84	U-Shaped Curves		NR	U-Curves		NR
	Women age > 85	U-Shaped Curves		NR	U-Curves		NR

B. Studies Examining Waist Circumference and Mortality

Total Mortality

Reference	BaselineWC	Incidence	95% CI	p (incidence)	Relative Risk	95% CI	p (RR)
Baik (67)	WC (inches)	*Total Mortality*					
	<34.5	NR		NR	1		0.11
	34.5-36.2	NR		NR	0.97	.70-1.33	
	36.3-37.9	NR		NR	0.82	.59-1.16	
	38.0-40.2	NR		NR	0.97	.72-1.32	
	≥ 40.3	NR		NR	1.17	.87-1.59	
Folsom (10)	WC (cm)	*Total Mortality*					
	< 74.3	0.67	NR	NR	1		<0.001
	74.3-80	0.71	NR	NR	0.99	0.9-1.1	Multivar RR 1.1
	80-87.3	0.66	NR	NR	0.90	0.8-1.0	(1.0-1.3)
	87.3-96.3	0.71	NR	NR	0.93	0.8-1.1	
	>96.3	0.96	NR	NR	1.3	1.1-1.5	
Disease-Specific Mortality							
Baik (67)	WC (inches)	*CVD Death*					
	<34.5	NR		NR	1		<0.001
	34.5-36.2	NR			0.72	0.27-1.96	
	36.3-37.9	NR			2.37	1.03-5.45	
	38.0-40.2	NR			1.98	0.88-4.48	
	≥ 40.3	NR			3.5	1.6-7.66	
	WC (inches)	*Cancer Death*					
	<34.5	NR		NR	1		NR
	34.5-36.2	NR			1.34	.82-2.19	
	36.3-37.9	NR			0.75	.43-1.32	
	38.0-40.2	NR			0.96	0.59-1.59	
	≥ 40.3	NR			0.93	0.56-1.55	
		Respiratory Disease Death					
	Lowest tertile	NR		NR	1		0.01
	2nd tertile	NR			0.59	0.27-1.29	
	Largest Tertile	NR			0.28	0.10-0.75	
Folsom (10)	WC (cm)	*CHD Mortality*					
	< 74.3	0.07		NR	1		<0.001
	74.3-80	0.11			1.5	1.0-2.1	Multivar RR 2.2
	80-87.3	0.12			1.6	1.1-2.2	(1.5-3.2)
	87.3-96.3	0.14			1.7	1.2-2.5	
	>96.3	0.21			2.6	1.9-3.6	
	WC (cm)	*Other CVD Mortaltiy*					
	< 74.3	0.07		NR	1		0.1
	74.3-80	0.11			1.4	.97-2	Multivar RR 1.2
	80-87.3	0.08			1	.7-1.5	(0.8-1.2)
	87.3-96.3	0.10			1.2	.8-1.7	
	>96.3	0.13			1.5	1.1-2.2	
	WC (cm)	*Cancer Mortality*					
	< 74.3	0.33		NR	1		0.31
	74.3-80	0.35			1	0.8-1.2	Multivar RR 1.2
	80-87.3	0.30			0.84	0.7-1.0	(0.96-1.4)
	87.3-96.3	0.33			0.88	0.7-1.1	
	>96.3	0.42			1.2	0.97-1.4	

C. Studies Examining Waist to Hip Ratio and Mortality

Total Mortality

Reference	Baseline WHR	Incidence	95% CI	p (incidence)	Relative Risk	95% CI	p (RR)
Baik (67)	WHR						
	<0.9	NR		NR	1		0.1
	0.9-0.91	NR			0.78	.52-1.17	
	0.92-0.94	NR			0.85	0.62-1.18	
	0.95-0.97	NR			1.06	0.78-1.43	
	≥ 0.98	NR			1.08	0.81-1.43	
Folsom (10)	WHR						
	< 0.76	0.55	NR	NR	1		<0.001
	0.76- 0.81	0.61	NR		1.1	0.9-1.2	Multivar RR 1.2
	0.81-0.85	6.70	NR		1.2	1.1-1.4	(1.1-1.4)
	0.85-0.9	0.83	NR		1.3	1.2-1.5	
	>0.90	0.97	NR		1.5	1.4-1.8	

Disease-Specific Mortality							
Reference	**Baseline WHR**	**Incidence**	**95% CI**	**p (incidence)**	**Relative Risk**	**95% CI**	**p (RR)**
Folsom (10)	WHR	*CHD Mortality*					
	< 0.76	0.07	NR	NR	1		<0.001
	0.76- 0.81	0.09	NR		1.3		Multivar RR 1.9
	0.81-0.85	0.09	NR		1.6	0.9-1.9	(1.3-2.9)
	0.85-0.9	0.10	NR		2.3	1.1-2.3	
	>0.90	0.14	NR		2.5	1.6-3.2	
		Other CVD Mortaltiy					
	< 0.76	0.07	NR	NR	1		0.003
	0.76- 0.81	0.09	NR		1.2	0.8-1.8	Multivar RR 1.4
	0.81-0.85	0.09	NR		1.2	0.8-1.8	(0.9-2.1)
	0.85-0.9	0.10	NR		1.2	0.8-1.8	
	>0.90	0.14	NR		1.7	1.2-2.4	
		Cancer Mortality					
	< 0.76	0.28	NR	NR	1		0.003
	0.76- 0.81	0.31	NR		1.1	0.9-13	Multivar RR 1.2
	0.81-0.85	0.37	NR		1.2	1.0-1.5	(0.9-1.4)
	0.85-0.9	0.36	NR		1.2	1.0-1.4	
	>0.90	0.40	NR		1.3	1.1-1.6	
Study Examining % Body Fat and (Total) Mortality							
Reference	**Baseline %BF**	**Incidence**	**95% CI**	**p (incidence)**	**Relative Risk**	**95% CI**	**p (RR)**
Heitmann (76)	% Body Fat (quintile mean)						
	25.7	NR		NR	1.0		NR
	28.2	NR			1.2		
	30	NR			1.0		
	32	NR			1.3		
	35.3	NR			1.5	1.1-2.0	

NR = Not reported
WC = Waist Circumferance
WHR = Waist:Hip Ratio
BF = Body Fat
PY = Person-Years

Table 6. Summary of studies examining relationships between a) BMI b) Waist Circumference, and c) Waist to Hip Ratio and prospective morbidity risk.

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	P (RR)
Studies Examining BMI and Morbidity							
Abbot (66)	1st Thrombo-embolic CVA (#/1000)						
	15.33-22.31	28.70	NR	<0.01	27.2	NR	NR
	22.32-24.71	40.70	NR		42.2	NR	
	24.75-35.96	54.26	NR		55.4	NR	
Cerhan (69)	Prostate Cancer Incidence (Cases/100 PY)						
	<23.6	0.80	NR	NR	1		0.1
	23.6-25.8	0.67	NR		0.9	0.5-1.9	
	25.9-27.8	0.80	NR		1.2	0.6-2.5	
	>27.8	1.07	NR		1.7	0.8-3.3	
Cerhan (84)	Non-Hodgkins Lymphoma: Cases/100 PY						
	<23.5	0.06	NR	NR	1		NR
	23.5-26.1	0.05	NR		0.7	0.5-1.0	
	26.2-29.7	0.05	NR		0.9	0.6-1.2	
	≥ 29.8	0.07	NR		1	0.7-1.4	
Dey (71)	1st CVA: WOMEN						
	≤ 23.2	NR		NR	1		NS (linear &
	23.3-25.5	NR			0.96	.66-1.38	nonlinear)
	25.6-28.5	NR			0.86	.59-1.26	
	≥28.6	NR			1	.69-1.45	
	1st CVA: MEN						
	≤ 23.3	NR		NR	1		0.006 linear
	23.4-25.6	NR			1.27	.83-1.95	(Nonlinear NS)
	25.7-28	NR			1.35	.89-2.05	
	≥ 28.1	NR			1.68	1.12-2.53	
Folsom (10)	Diabetes Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			1.9	1.4-2.5	
	24.87-27.06	NR			2.9	2.2-3.6	
	27.06-30.21	NR			6.6	5.0-8.5	
	>30.21	NR			13.8	10.6-17.8	
	Hypertension Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			1.1	1.0-1.3	
	24.87-27.06	NR			1.4	1.2-1.5	
	27.06-30.21	NR			1.7	1.6-1.9	
	>30.21	NR			2.2	2.0-2.4	
	Hip Fracture						
	< 22.8	NR		NR	1		0.002
	22.80- 24.87	NR			0.61	0.5-0.8	
	24.87-27.06	NR			0.63	0.5-0.8	
	27.06-30.21	NR			0.62	0.5-0.8	
	>30.21	NR			0.64	0.5-0.8	
	Any Cancer Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			0.95	0.9-1.1	
	24.87-27.06	NR			0.97	0.9-1.1	
	27.06-30.21	NR			1.0	0.9-1.1	
	>30.21	NR			1.2	1.1-1.3	
	Breast Cancer Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			1.2	0.97-1.4	
	24.87-27.06	NR			1.2	0.99-1.4	
	27.06-30.21	NR			1.3	1.1-1.5	
	>30.21	NR			1.4	1.2-1.7	
	Colon Cancer Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			0.93	0.7-1.3	
	24.87-27.06	NR			1.2	0.9-1.6	
	27.06-30.21	NR			1.3	0.99-1.8	
	>30.21	NR			1.5	1.1-2.0	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	P (RR)
Folsom (10)	Lung Cancer Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			0.59	0.4-0.8	
	24.87-27.06	NR			0.44	0.3-0.6	
	27.06-30.21	NR			0.45	0.3-0.6	
	>30.21	NR			0.40	0.3-0.5	
	Uterine Cancer Incidence						
	< 22.8	NR		NR	1.0		<0.001
	22.80- 24.87	NR			0.94	0.6-1.5	
	24.87-27.06	NR			1.0	0.7-1.6	
	27.06-30.21	NR			1.4	0.95-2.2	
	>30.21	NR			3.5	2.5-5.1	
	Ovarian Cancer Incidence						
	< 22.8	NR		NR	1.0		0.27
	22.80- 24.87	NR			0.95	0.6-1.6	
	24.87-27.06	NR			0.68	0.4-1.2	
	27.06-30.21	NR			1.2	0.7-1.9	
	>30.21	NR			1.2	0.7-2.0	
Harris (47)	CHD Incidence						
	<23.47	NR		NR	1		NR
	23.47-27.28	NR			1.1	0.7-1.8	
	>27.28	NR			1.7	1.1-2.5	
	<23.97	NR		NR	1		NR
	23.97-26.96	NR			1.4	0.7-2.2	
	>26.96	NR			1.7	1.1-2.7	
Launer(9)	Incident Mobility Disability (cases/100,000 pop)						
	≤ 23.8	3476		NR	1		NR
	23.81-28.10	4169			1.31	0.82-2.08	
	≥ 28.10	5031			2.04	1.20-3.48	
Olson (64)	Lung Cancer Cases/100 PY						
	<22.89	0.00	NR	<0.001	1		<0.001
	22.90-25.04	0.19		(trend)	0.92	0.72-1.16	
	25.05 - 27.43	0.14			0.76	0.58-0.98	
	27.44-30.69	0.10			0.69	0.52-0.90	
	≥ 30.70	0.09			0.66	0.50-0.89	
Studies Examining Waist Circumference and Morbidity							
Dey (71)	1st CVA: WOMEN						
	≤ 78 cm	NR		NR	1		NS (linear and
	79-85	NR			1.16	0.81-1.67	nonlinear)
	86-93	NR			1.18	0.80-1.71	
	≥ 94 cm	NR			1.31	0.88-1.92	
	1st CVA: MEN						
	≤ 84 CM	NR		NR	1		0.004 linear
	85-91	NR			1.4	0.91-2.14	(Nonlinear NS)
	92-98	NR			1.5	0.98-2.29	
	≥ 99	NR			1.65	1.08-2.51	
Folsom (10)	Diabetes Incidence						
	< 74.3	NR		NR	1.0		<0.001
	74.3-80	NR			1.7	1.2-2.3	
	80-87.3	NR			3.4	2.5-4.6	
	87.3-96.3	NR			7.2	5.5-9.6	
	>96.3	NR			16.5	12.6-21.7	
	Hypertension Incidence						
	< 74.3	NR		NR	1.0		<0.001
	74.3-80	NR			1.1	1.0-1.3	
	80-87.3	NR			1.5	1.3-1.6	
	87.3-96.3	NR			1.8	1.6-2.0	
	>96.3	NR			2.3	2.1-2.5	
	Hip Fracture						
	< 74.3	NR		NR	1.0		0.56
	74.3-80	NR			0.85	0.6-1.1	
	80-87.3	NR			0.91	0.7-1.2	
	87.3-96.3	NR			0.83	0.6-1.1	
	>96.3	NR			0.91	0.7-1.2	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	P (RR)
Folsom (10)	Any Cancer Incidence						
	< 74.3	NR		NR	1,0		<0.001
	74.3-80	NR			1.1	0.96-1.2	
	80-87.3	NR			1.0	0.9-1.1	
	87.3-96.3	NR			1.1	1.0-1.2	
	>96.3	NR			1.4	1.2-1.5	
	Breast Cancer Incidence						
	< 74.3	NR		NR	1.0		<0.001
	74.3-80	NR			1.4	1.1-1.7	
	80-87.3	NR			1.2	1.0-1.5	
	87.3-96.3	NR			1.4	1.2-1.7	
	>96.3	NR			1.7	1.4-2.0	
	Colon Cancer Incidence						
	< 74.3	NR		NR	1.0		<0.001
	74.3-80	NR			0.82	0.6-1.1	
	80-87.3	NR			1.1	0.8-1.5	
	87.3-96.3	NR			1.1	0.8-1.5	
	>96.3	NR			1.5	1.1-2.0	
	Lung Cancer Incidence						
	< 74.3	NR		NR	1.0		0.003
	74.3-80	NR			0.93	0.7-1.2	
	80-87.3	NR			0.56	0.4-0.8	
	87.3-96.3	NR			0.55	0.4-0.8	
	>96.3	NR			0.78	0.6-1.0	
	Uterine Cancer Incidence						
	< 74.3	NR		NR	1.0		<0.001
	74.3-80	NR			1.2	0.8-1.8	
	80-87.3	NR			1.2	0.8-1.9	
	87.3-96.3	NR			1.4	0.9-2.1	
	>96.3	NR			3.3	2.3-4.8	
	Ovarian Cancer Incidence						
	< 74.3	NR		NR	1.0		0.14
	74.3-80	NR			1.1	0.6-1.9	
	80-87.3	NR			0.65	0.3-1.2	
	87.3-96.3	NR			1.5	0.9-2.5	
	>96.3	NR			1.3	0.8-2.2	
Olson (64)	Lung Cancer Incidence: Cases/100 person years						
	≤ 76.56 cm	0.14	NR	0.004	1		0.15
	75.57-81.92	0.14	NR	(trend)	1.04	0.81-1.34	
	81.93-89.54	0.10	NR		0.8	0.62-1.05	
	89.55-99.0	0.10	NR		0.82	0.63-1.09	
	≥ 99	0.11	NR		0.91	0.69-1.19	
Studies Examining Waist to Hip Ratio and Morbidity							
Cerhan (84)	Non-Hodgkins Lymphoma: Cases/100 PY						
	<0.777	0.05	NR	NR	1		
	0.777-0.831	0.05	NR		0.9	0.6-1.4	NR
	0.832-0.892	0.07	NR		1.4	1.0-2.0	
	≥ 0.893	0.06	NR		1.1	0.8-1.6	
Folsom (10)	Diabetes Incidence						
	< 0.76	NR		NR	1.0		<0.001
	0.76- 0.81	NR			1.9	1.4-2.5	
	0.81-0.85	NR			3.0	2.3-3.9	
	0.85-0.9	NR			6.0	4.7-7.7	
	>0.90	NR			11.5	9.0-14.6	
	Hypertension Incidence						
	< 0.76	NR		NR	1		<0.001
	0.76- 0.81	NR			1.2	1.1-1.4	
	0.81-0.85	NR			1.4	1.3-1.6	
	0.85-0.9	NR			1.6	1.5-1.8	
	>0.90	NR			2.0	1.8-2.2	
	Hip Fracture						
	< 0.76	NR		NR	1		0.14
	0.76- 0.81	NR			1.1	0.8-1.5	
	0.81-0.85	NR			1.3	0.9-1.7	
	0.85-0.9	NR			1.2	0.9-1.6	
	>0.90	NR			1.2	0.9-1.6	

Reference	Baseline BMI	Incidence	95% CI	p (Incid)	Relative Risk	95% CI	P (RR)
Folsom (10)	Any Cancer Incidence						
	< 0.76	NR		NR	1		<0.001
	0.76- 0.81	NR			1.0	0.9-1.1	
	0.81-0.85	NR			1.1	0.98-1.2	
	0.85-0.9	NR			1.1	1.0-1.3	
	>0.90	NR			1.3	1.2-1.4	
	Breast Cancer Incidence						0.002
	< 0.76	NR		NR	1		
	0.76- 0.81	NR			1.0	0.9-1.2	
	0.81-0.85	NR			1.0	0.8-1.2	
	0.85-0.9	NR			1.2	1.0-1.4	
	>0.90	NR			1.3	1.1-1.5	
	Colon Cancer Incidence						0.007
	< 0.76	NR		NR	1.0		
	0.76- 0.81	NR			0.89	0.7-1.2	
	0.81-0.85	NR			0.95	0.7-1.3	
	0.85-0.9	NR			1.2	0.9-1.6	
	>0.90	NR			1.3	0.97-1.7	
	Lung Cancer Incidence						0.26
	< 0.76	NR		NR	1		
	0.76- 0.81	NR			1.0	0.8-1.5	
	0.81-0.85	NR			1.2	0.9-1.6	
	0.85-0.9	NR			1.0	0.7-1.4	
	>0.90	NR			1.2	0.9-1.7	
	Uterine Cancer Incidence						<0.001
	< 0.76	NR		NR	1		
	0.76- 0.81	NR			1.0	0.7-1.5	
	0.81-0.85	NR			1.4	0.9-2.0	
	0.85-0.9	NR			1.2	0.8-1.8	
	>0.90	NR			2.0	1.4-2.8	
	Ovarian Cancer Incidence						0.13
	< 0.76	NR		NR	1		
	0.76- 0.81	NR			1.3	0.6-2.4	
	0.81-0.85	NR			1.7	1.0-3.0	
	0.85-0.9	NR			1.2	0.6-2.1	
	>0.90	NR			1.7	0.96-3.0	
Olson (64)	Lung Cancer Incidence: Cases/100 PY						
	≤ 0.76	0.10	NR	<0.001	1		0.92
	0.77-0.80	0.12	NR		1.01	0.76-1.34	
	0.81-0.85	0.12	NR		0.95	0.73-1.24	
	0.86-0.90	0.12	NR		0.97	0.73-1.28	
	>0.90	0.14	NR		1	0.76-1.32	

NR = Not reported
WC = Waist Circumferance
WHR = Waist:Hip Ratio
BF = Body Fat
PY = Person-Years

Table 7. Summary of Diet and Behavioral Therapy Randomized Controlled Trials

Citation	Goal* Intensity Mode[§]	Follow Up	Sample	Baseline Weight †	Groups	Weight Change	Delta [¶]	p-value	Secondary Outcomes	Drop Out	Quality
Allen (85)	L Mod/High DEB	12 mo	116 women s/p initial CABG without concurrent surgical procedures at a large E Coast teaching hospital	29-30	 BT Standard advice BT Standard advice BT Standard advice	Kg. Change -2 -1 BMI Change -0.9 -0.3 Freq. BMI Loss 55.4% 53.8%	 -1 -0.6 1.6%	 NS NS NR	Quit smoking: 16% 5% NS	16%	Fair
Chao (86)	L/M High DEB	52 wk	67 Women (TONE subset)	31	 Intervention Control Intervention Control	Kg. Change -3.5 -0.9 BMI Change -1.33 -0.34	-2.6 -0.99	<0.001 <0.0001	BMD decreased by 6.3 +/- 2.1 g/cm2x10^{-4} for each pound of weight loss	20%	Fair
Fagerberg (14)	L NR[‖] D	3.3 yr	112 Hypertensive men in a CVD study in Gothenberg Sweden	27	 Diet/Lipids/Tob Control	Kg. Change -0.56 -0.31	-0.26	NS**	Oral GTT 24.6 mmol/L 28.2 mmol/L p=0.008 (sum of pre & post serum BG) Lipids reported, but not noted here as the intervention included lipid-lowering agents for those with unmet treatment goals	37%	Fair
Knowler (13)	L/M High DEB	2.8 yr (mean)	Minorities over-represented. Included 648 older adults (≥ 60 y/o); DM (but not weight) outcomes specific to this older group	34	 Lifestyle Placebo	Kg. Change (entire group, not the older subset) -5.6 -0.1	 -5.5	 <0.001	71% (CI 51 to 83%) reduction in diabetes incidence in lifestyle vs. placebo	7.5%	Good

Table 7. Summary of Diet and Behavioral Therapy Randomized Controlled Trials

Citation	Goal* Intensity Mode§	Follow Up	Sample	Baseline Weight †	Groups	Weight Change	Delta ¶	p-value	Secondary Outcomes	Drop Out	Quality
Kostis (90)	L/M High DEB	48 mo	141 obese TONE participants at University of Medicine and Dentistry of New Jersey-Robert Wood Johnson Medical School center		WT Loss Non-Wt Loss	-14.8 -5.4	-9.4	NS	Off antihypertensive medication: 23% wt loss and low Na, 17% wt loss, 15% low Na, 7% UC (p=0.012) Cardiovascular events: 10% wt loss and low Na, 15% wt loss, 11% low Na, 16% UC (NS)	10%	Good
Kumanyika (89)	L/M High DEB	30 mo (mean)	TONE participants at least moderately obese		Wt Loss‡ Non-Wt loss Wt Loss‡ Non-Wt loss	-3.3 -1.4 -4.2 -0.9	-1.9 -3.3	<0.05 (Black) <0.001 (White) Ethnic Diff p=0.12	HRs (Combined TONE endpoint) Total: 0.71 (CI: 0.58-0.88) Black: 0.74 (CI: 0.49-1.11) White: 0.72 (CI: 0.57-0.93) p for ethnic difference 0.92	Black: 5% White: 10%	Good
Whelton (15)	L/M High DEB	30 mo	585 Obese participants (TONE)	Women: 32 Men: 30-31	Wt Loss‡ Non-Wt loss Wt Loss‡ Non-Wt loss	Kg. Change -3.9 -0.9 Freq. 4.5 kg. Loss 44% 13%	-3.0 31%	<0.05 NR	HR for weight loss vs. usual care: 0.70 (0.57-0.87) Combined TONE endpoint: New hypertension, antihypertensive or cardiovascular event	9-11%	Good

* Trial Types: L=Weight Loss; M=Maintenance of Weight Loss

† Weight: presented as baseline mean (or range) BMI unless otherwise noted.

‡ Components: D=Diet; E=Exercise; B=Behavioral Therapy

§ Mode of Intervention: G=Group-based; I=Individual-based

|| NR = Not Recorded

¶ Delta = Weight Change in Intervention – Weight Change in Control group.

** NS = Not Significant ($p \geq 0.05$)

‡ Here wt loss group includes those with and without sodium reduction

Appendix 1. Topic Area, Key Questions and Eligibility Criteria

Topic and Key Questions	Eligibility Criteria*
A. Validity and Precision of Diagnostic Tests for Obesity • What is the evidence for validity and precision of the different diagnostic studies in the elderly?	Obesity diagnostic test carried out in an out-patient setting Comparison with gold standard or alternative measures of adiposity Sample mean age (median or mid-point if mean not presented) ≥ 60 years
B. Health Risks of Obesity in the elderly • Does obesity entail health risk in the elderly? • How are the different measures of obesity associated with adverse health outcome incidence? • What are the characteristics of people for whom there is evidence of health risk associated with body weight?	Prospective cohort studies with absolute or relative rates of health risk reported over at least 10 years of follow-up No systematic body weight intervention Outcome measures (morbidity or mortality) presented according to baseline weight status measures. Sample mean age ≥ 60 years
C. Efficacy and harms of policy-relevant treatment for weight reduction or intermediate outcomes • Are there dietary, behavioral, or surgical therapies that can lead to weight loss? • Does intentional weight loss in the elderly lead to improved health outcomes? • Do dietary, behavioral, or surgical therapies lead to improved health outcomes? • Do trials assessing weight loss interventions (leading to weight loss or improved health outcomes) address the	**Dietary Interventions** - RCT (of fair or good quality) - Outcome: weight loss or BMI, WC or WHR reduction - BMI ≥ 25 - Minimum 12 month follow-up - Population: generalizable to typical US primary care population - Sample mean age ≥ 60 years **Behavioral Treatment** - RCT (of fair or good quality) - Outcome: weight loss or BMI, WC or WHR reduction - BMI ≥ 25 - Minimum 12 month follow-up - Population: generalizable to typical US primary care population - Sample mean age ≥ 60 years **Surgical Treatment** - RCT (of fair or good quality), or Cohort study with a control group - Surgical procedure currently in clinical use - Outcome: weight loss or BMI reduction - BMI ≥ 35 - Minimum 12 month follow-up - Initial BMI ≥35 - Sample mean age ≥ 60 years

	different measures of adiposity, and if so, show similar outcomes?

*RCT, randomized controlled trial; BMI, body mass index; WC, Waist Circumference; WHR, Waist to Hip ratio.

Appendix 2. Criteria for grading the internal validity of individual studies [5]

Study design	Criteria
Systematic reviews	Comprehensiveness of sources/search strategy used Standard appraisal of included studies Validity of conclusions Recency and relevance
Case-control studies	Accurate ascertainment of cases Nonbiased selection of cases/controls with exclusion criteria applied equally to both Response rate Diagnostic testing procedures applied equally to each group Appropriate attention to potential confounding variables
Randomized controlled trials (RCTs) and cohort studies	Initial assembly of comparable groups: For RCTs: adequate randomization, including concealment and whether potential confounders were distributed equally among groups For cohort studies: consideration of potential confounders with either restriction or measurement for adjustment in the analysis; consideration of inception cohorts Maintenance of comparable groups (includes attrition, crossovers, adherence, contamination) Important differential loss to follow-up or overall high loss to follow-up Measurements: equal, reliable, and valid (includes masking of outcome assessment) Clear definition of interventions All important outcomes considered Analysis: adjustment for potential confounders for cohort studies, or intention-to-treat analysis for RCTs
Diagnostic accuracy studies	Diagnostic test relevant, available for primary care, adequately described Study uses a credible reference standard, performed regardless of test results Reference standard interpreted independently of diagnostic test Handles indeterminate results in a reasonable manner Spectrum of patients included in study Sample size Administration of reliable diagnostic test

Appendix 3. Search Strategies

Task 1: Diagnosis of obesity in the elderly

a) Validity and reliability of diagnostic tests

b) How are the different measures of obesity associated with the incidence of adverse health outcomes in the elderly: longitudinal studies by baseline weight in studies from question 2.1

c) Review of which adiposity measures are linked with health risk in the trials reviewed in questions 2b, 4-6

Validity and Reliability of Diagnostic Tests

Search History
exp body mass index/ or exp anthropometry/ or exp body composition/ or exp skinfold thickness/
exp "Sensitivity and Specificity"/ or exp Predictive Value of Tests/ or positive predictive value.mp. or negative predictive value.mp.
exp reproducibility of results/ or validity.mp. or precision.mp.
2 or 3
1 and 4
limit 5 to (human and english language and yr=1980-2003)
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp.
limit 6 to ("all aged <65 and over>" or "aged <80 and over>")
6 and 7
9 or 8
exp obesity/
exp DIAGNOSIS/
11 and 12
13 and 4
limit 14 to (human and english language and yr=1980-2003)
limit 15 to ("all aged <65 and over>" or "aged <80 and over>")
15 and 7
16 or 17

Task 2: Does weight loss lead to improved health in the elderly? (how much weight and over how long?)
a**) Does obesity entail health risk in the elderly? (Longitudinal studies search)**
b) Does intentional weight loss in the elderly lead to improved health outcomes? – review of health outcome change in trials reviewed for questions 4-6; if no information from the elderly, draw inferences from the USPSTF searches.

Longitudinal Studies Search

Search History
exp body mass index/ or exp body composition/ or exp skinfold thickness/
exp Longitudinal Studies/
exp cohort studies/
2 or 3
1 and 4
limit 5 to (human and english language and ("all aged <65 and over>" or "aged <80 and over>"))
aged/ or postmenopause/ or geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp.
5 and 7
limit 8 to (human and english language)
9 or 6
limit 10 to yr=1980-2003

Task 3: Which elderly patients with obesity would experience improved health outcomes with weight loss

a) review of the characteristics of those at highest risk from Longitudinal Studies above (2a)
b) review characteristics of the participants who show benefit in the RCTs reviewed for tasks 4-6.

Task 4: Are there dietary therapies that improve net health outcomes in the obese elderly? (Include duration, circumstances, harms)
a) Are there dietary therapies that can lead to weight loss?

Dietary Searches: Diagnostic Studies

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp diet, reducing/ or exp diet/ or exp diet therapy/ or low calorie diet.mp. or exp vegetarianism/ or vegetarian diet.mp. or aha diet.mp. or aha guidelines.mp. or ncep.mp. or caloric restriction.mp.
6 and 7
exp mass screening/
8 and 9

Diet RCTs

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp diet, reducing/ or exp diet/ or exp diet therapy/ or low calorie diet.mp. or exp vegetarianism/ or vegetarian diet.mp. or aha diet.mp. or aha guidelines.mp. or ncep.mp. or caloric restriction.mp.
6 and 7
exp "randomized controlled trials"/ or exp single-blind method/ or exp double-blind method/ or exp random allocation/
8 and 9
limit 8 to randomized controlled trial
10 or 11

Dietary Searches: Review Articles

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp diet, reducing/ or exp diet/ or exp diet therapy/ or low calorie diet.mp. or exp vegetarianism/ or vegetarian diet.mp. or aha diet.mp. or aha guidelines.mp. or ncep.mp. or caloric restriction.mp.
6 and 7
limit 8 to review articles
limit 8 to meta analysis
9 or 10

b) **Does intentional weight loss lead to improved health?** (from 2b; same searches as for Task 4a but looking at health outcomes as available; if needed, refer to USPSTF general population findings)

c) **Do dietary therapies lead to improved health?** (evaluate health outcomes from the RCTs in dietary searches above)

Task 5: Are there behavioral therapies that lead to weight loss?
Behavioral Therapy Diagnostic Studies

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp behavior/ or exp behavior therapy/ or exp goals/ or goal setting.mp. or self monitoring.mp. or exp self concept/ or behavior modification.mp. or exp cognitive therapy/ or cognitive behavior.mp.
6 and 7
exp mass screening/
8 and 9

Behavioral Therapy RCTs

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp behavior/ or exp behavior therapy/ or exp goals/ or goal setting.mp. or self monitoring.mp. or exp self concept/ or behavior modification.mp. or exp cognitive therapy/ or cognitive behavior.mp.
6 and 7
exp "randomized controlled trials"/ or exp single-blind method/ or exp double-blind method/ or exp random allocation/
8 and 9
limit 8 to randomized controlled trial
10 or 11

Behavioral Therapy Reviews

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
exp behavior/ or exp behavior therapy/ or exp goals/ or goal setting.mp. or self monitoring.mp. or exp self concept/ or behavior modification.mp. or exp cognitive therapy/ or cognitive behavior.mp.
6 and 7
limit 8 to review articles
limit 8 to meta analysis
9 or 10

d) **Does intentional weight loss lead to improved health?** (from 2b; same searches as for Task 5a but looking at health outcomes as available; if needed, refer to USPSTF general population findings)

e) **Do behavioral therapies lead to improved health?** (evaluate health outcomes from the RCTs in behavioral searches above)

Task 6: Are there weight loss surgeries that improve net health outcomes in the elderly?

a) Are there surgical therapies that can lead to weight loss?

Surgical Searches: RCTs

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
obesity/su or exp GASTROPLASTY/ or exp Gastric Bypass/ or exp ANASTOMOSIS, ROUX-EN-Y/ or exp ANASTOMOSIS, SURGICAL/ or exp jejunoileal bypass/ or duodenal switch.mp. or exp Biliopancreatic Diversion/ or gastric resection.mp.
6 and 7
exp "randomized controlled trials"/ or exp single-blind method/ or exp double-blind method/ or exp random allocation/
8 and 9
limit 8 to randomized controlled trial
10 or 11

Surgical Searches: Review Articles

Search History
exp obesity/ or exp body mass index/ or exp body composition/ or exp skinfold thickness/ or exp weight loss/
exp aged/ or exp postmenopause/ or exp geriatrics/ or aged.mp. or elderly.mp. or gerontologic.mp. or post-menopausal.mp. or geriatric.mp.
1 and 2
limit 1 to ("all aged <65 and over>" or "aged <80 and over>")
3 or 4
limit 5 to (human and english language and yr=1980-2003)
obesity/su or exp GASTROPLASTY/ or exp Gastric Bypass/ or exp ANASTOMOSIS, ROUX-EN-Y/ or exp ANASTOMOSIS, SURGICAL/ or exp jejunoileal bypass/ or duodenal switch.mp. or exp Biliopancreatic Diversion/ or gastric resection.mp.
6 and 7
limit 8 to review articles
limit 8 to meta analysis
9 or 10

b) Does intentional weight loss lead to improved health outcomes? (as in 2b)

c) Does surgical therapy lead to improved health outcomes (we will review health outcome benefits reported in the trials reviewed for 6a).