Screening, Prevention and Treatment of Overweight/Obesity in Adult Populations

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Purpose and Background

This report will be used by the Canadian Task Force on Preventive Health Care to provide guidelines on the screening, prevention, and treatment of obesity in adults. The last Task Force guideline on the prevention of obesity was conducted in 2006 and published in 2007,¹ while obesity screening was last examined in 1994.² Since this time, other Canadian and international groups have provided guidance on obesity screening, management, and prevention, including the Obesity Canada Clinical Guidelines Expert Panel,(2006),³ the Scottish Intercollegiate Guidelines Network (2010),⁴ and the United States Preventive Services Task Force (2011).⁵ The lack of updated Canadian guidelines on this topic and the growing burden of obesity were key reasons for which this topic was chosen.

Definition

Obesity is characterized by an increase in total body fat and is defined by a body mass index $(BMI) \ge 30 \text{kg/m}^2$, based on the definition used by the World Health Organization and adopted by the Canadian Guidelines for Body Weight Classification in Adults.⁶ Adults (≥ 18 years) with BMI of 25kg/m^2 to 29.9kg/m^2 are considered overweight and at risk of becoming obese, whereas those with BMI of 18.5kg/m^2 to 24.9kg/m^2 are considered at low risk for morbidity.

Prevalence and Burden of Disease

Obesity has become a worldwide issue. According to the WHO report on global epidemic, an estimated one billion adults are overweight and at least 300 million are clinically obese⁷. Obesity occurs in all age and ethnic groups, and is associated with socioeconomic status (SES). According to a review by McLaren, the effect of SES differs by Human Development Index; Negative associations (i.e. lower SES associated with larger body size) for women in highly developed countries were most common with education and occupation, while positive associations for women in medium- and low-development countries were most common with income and material possessions⁸.

In 1980, the prevalence of obesity in Canadian adults was approximately 8 percent. Since then, the number of obese adults in Canada has drastically increased.⁹ The Canadian Health Measures Survey (CHMS) was conducted between 2007 and 2009 and estimated the prevalence of obesity in adults to be 24.1 percent.¹⁰ From 1978/1979 to 2004, the proportion of adults falling into obese class I (30.0 - 39kg/m²) increased from 10.5% to15.2%, Class II (35.0-34.9kg/m²) doubled, increasing from 2.3% to 5.1% and Class III (BMI≥40kg/m2) increased three-folds from 0.9% to 2.7% ^{8,11} respectively. Obesity is more prevalent among men than women; the average BMI was estimated to be 27.5(27-28.0) for men and 26.7(26-27.4) for women¹², however, females are more likely to fall into obese Class II and Class III than males ¹².In Canada, obesity does not appear to be associated with lower SES status, but is more prevalent in rural-dwelling adults and among people in Eastern and Northern Canada.¹³ Based on the 2008/2009 CCHS, regional, provincial and territorial variation were observed. Obesity varied across provinces and territories, from a low of 12.8% in British Columbia to a high of 25.4% in Labrador. The prevalence of obesity tends to be lower in urban regions and higher in rural areas. Obesity ranged from 5.3% in urban suburban (Richmond and British Columbia to a high of 35.9% in the Northern Region of Saskatchewan.¹¹

Etiology, Risk Factors, and the Natural History of Obesity

The etiology of weight gain and obesity is multi-factorial (Table 1), encompassing hereditary, metabolic, and drug-related conditions. The principal cause of obesity is an imbalance between calories consumed and calories expended as a result of an increased intake of energy-dense foods that are high in fat, salt and sugars but low in vitamins, minerals and other micronutrients as well as a decrease in physical activity due to the increasing sedentary life styles, ^{12,14-16}. Obesity can develop at any age but prevalence is highest in middle age and typically declines in the elderly, partly due to increased mortality and due to a multi-factorial age-related decline in BMI. There are several risk factors, metabolic, environmental and lifestyle-related, which contribute to obesity.^{14,16,17} Metabolic factors include a low baseline metabolic rate, increased carbohydrate oxidation, insulin resistance, and sympathetic activity. However, these factors are not easily measured and are less strongly linked to obesity than are lifestyle factors. Sedentary behaviours, such as prolonged screen time appears to contribute to weight gain.¹⁸ Similarly, among many lifestyle behaviours that predispose to obesity, sleep deprivation and smoking cessation may also be associated with weight gain.^{19,20} Among dietary factors, certain patterns of eating increase the risk for weight gain; these include consuming foods rich in saturated fats, fast-food consumption, and frequent snacking, especially during the evening hours.²¹ In recent years there has been increasing interest in determining the role of genetic factors in the pathogenesis of obesity. In general, genetic factors are considered to have a role in determining inter-individual variability in body weight. However, in adults with more severe obesity, less than 5% will harbour obesity-associated mutations such as those that cause leptin deficiency or leptin receptor dysfunction.¹⁵

Category	Condition/Disease
Neuroendocrine	 Cushing's syndrome²² hypothalamic obesity²³ hypothyroidism²⁴ polycystic ovary syndrome²⁵ growth hormone deficiency²⁶ weight cycling²⁷
Congenital	 Prader-Willi syndrome²⁸ Lawrence-Moon-Biedle syndrome²⁹
Dietary	 overeating relative to energy expenditure³⁰ increased dietary fat intake³¹ frequent fast-food consumption³² night-eating syndrome^{33,34}
Lifestyle	 sedentary lifestyle³⁵ decreased physical activity³⁶ sleep deprivation²⁰ smoking cessation³⁷ pregnancy/ post-pregnancy³⁸ Poor diet³⁹ Skipping meals³⁹ Snacking⁴⁰ Sugary soft drinks⁴¹
Psychiatric/Psychological/ Psychosocial	 binge eating and other eating disorders³⁰ seasonal affective disorder⁴² Depression/anxiety^{43,44} Boredom⁴⁵

	- Stress ⁴⁶
Drugs	- antipsychotics ⁴⁷
	- antidepressants
	- anticonvulsants ⁴⁹
	- corticosteroids ⁵⁰
	-
Biochemical	- Genetics ⁵¹
	- Metabolism ⁵¹
	- Injury ⁵²
	- Mobility issues ⁵³
	- Intrauterine growth ⁵⁴
Socio-Economic Determinants	- Education ⁵⁵
	- Low income ⁵⁵

Health Consequences of Obesity if Untreated

Obese adults are at increased risk for developing major diseases that include type 2 diabetes, coronary artery disease, stroke, depression, and certain cancers (Table 2).^{21,56,57} It is also estimated that approximately one in 10 premature deaths among adults, aged 20 to 64 years, are directly attributable to overweight and obesity.^{58,59}

Table 2: Health	Consequences	of Obesity
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Organ System	Condition/Disease
Cardiovascular	 coronary artery disease
	- hypertension
	 venous thromboembolism
	 varicose veins and venous hypertension
Respiratory	- obstructive sleep apnea
	 hypoventilation syndrome
	- cor pulmonale
Neurologic	- stroke
	 intracranial hypertension
	- meralgia paresthetica
Gastrointestinal	- cholelithiasis
	 gastroesophageal reflux disease
	- hepatic steatosis
	 non-alcoholic steatohepatitis
	 abdominal and inguinal hernias
	- colon cancer
Genitourinary	 urinary stress incontinence
	- hypogonadism
	- amenorrhea
	- prostate cancer
	- breast cancer
	- uterine cancer
Endocrine/Metabolic	- dyslipidemia
	 impaired glucose tolerance
	- type 2 diabetes
	- metabolic syndrome
	- infertility
	 polycystic ovarian syndrome
	- hypothyroidism
	- renal
Musculoskeletal	- degenerative osteoarthritis
	- low back strain
Skin	- cellulitis
	- intertrigo
Psychological	- depression
	 social and work-related discrimination

Rationale for Screening for Overweight and Obesity

- Screening directly for overweight and obesity may help guide clinical practice to improve patients' health.

Potential benefits of screening:

Screening for overweight and obesity can improve patients' health in three ways:

- In adults found to be obese and who have obesity-related diseases, modest weight loss (5% to 10% of total body weight) has been shown to improve control of such diseases and related symptoms and can reduce drug therapy requirements.^{3,60}
- In adults found to be obese but who do not have obesity-related diseases, lifestyle interventions such as starting a regular exercise program can reduce the risk of developing such diseases or can curtail their progression, (for example, prevention of diabetes in adults with impaired glucose tolerance).^{3,60}

• In adults found to be overweight but who are otherwise healthy, promoting healthy lifestyle practices may prevent the development of obesity.^{3,60}

Screening to guide clinical practice:

In clinical practice, an intervention relating to obesity could have two main goals:³

- <u>*Prevention of obesity*</u>. This can be considered in individual adults who are overweight and at risk for developing obesity, through interventions aimed at attaining a healthy weight or preventing weight gain.
- <u>*Treatment of obesity*</u>. Such interventions can be aimed to achieve weight loss in people who are already obese, thus reducing associated symptoms or burden of comorbidity. An example of this is weight loss in an obese adult with diabetes that aims to reduce hyperglycemia-related symptoms and reduce the need for glucose-lowering drugs.

Detection of Obesity and Overweight

There are several screening methods for assessing obesity and overweight. While methods include waist to hip and waist to height ratios, the two main methods used in everyday practice are BMI and waist circumference (WC).

- BMI is strongly correlated with direct measures of body fat, such as magnetic resonance imaging, and is a reliable determinant of adiposity-related health risks in adult men and women.⁶¹
- WC measures abdominal (or central) body fat, which is strongly correlated with an increased risk for type 2 diabetes, hypertension, dyslipidemia, and the metabolic syndrome, the latter of which combines all three of these conditions. Among those with BMI of 30-34.9kg/m², an increased WC results in a greater than 14- fold increased risk for type 2-diabetes, 28-fold increase for metabolic diseases and 15-fold increase for hypertension among women but a much lesser increase in risk among men .⁶¹

Practical considerations when using BMI and WC in clinical practice:

Combining BMI and WC to assess health risk. Although BMI and WC are related, WC provides an independent estimate of health risk beyond that provided by BMI.^{62,63} Considering both BMI and WC may be especially useful in adults with normal BMI, it can identify adults with an abdominal fat distribution who are at increased health risk despite normal BMI.⁶¹ The use of WC to assess health risk in adults at the extremes of BMI (\geq 35kg/m² or <18.5kg/m²) is not done because data are lacking as to associated health risks in this population.

BMI and WC as part of an overall health risk assessment. The classification schemes for BMI and WC were derived based on health risk assessments from large, heterogeneous populations: Consequently, the application of BMI and WC to assess health risk in individual adults will vary from person to person. The following should be considered when using BMI and WC as part of an overall risk assessment:

• BMI and WC should be combined with other determinants of individual health risk, which include smoking, concomitant disease, diet, physical activity, and personal and family weight history. However, what may be under-appreciated is the importance of BMI and

WC on health risk compared to the other, more traditional, risk factors. For example, obesity was, until recently, considered to increase the risk of coronary artery disease through its association with hypertension, dyslipidemia, and diabetes. However, BMI $\geq 30 \text{kg/m}^2$ appear to independently confer an increased risk for coronary artery disease which is comparable to the effect of hypertension.⁵⁷ A similar effect also occurs with WC, as adults with increased WC were more likely to develop hypertension, type 2 diabetes, and dyslipidemia.

- The Edmonton Staging System⁶⁴ contributes to our ability to assess obesity-related comorbidity. Applied to those with a BMI of ≥25kg/m², data from interview, exam or laboratory testing are used to assign a rating of 0 (no apparent comorbidity) to 4 (severe obesity related comorbidities or functional disability).⁶⁴ Using data from the NHANES 1999-2004, the scale independently predicted increased mortality.⁶⁵
- Because BMI and WC reflect an individual's risk at a single time point, longitudinal changes in BMI and WC may provide additional information on health risk. For example, an upward trend in the BMI and WC in adults with impaired glucose tolerance may place such individuals at increased risk for clinically overt type 2 diabetes. Conversely, a downward trend in BMI and WC with unintentional weight loss may indicate increased health risk due to the development of underlying disease.

Adult groups in which the BMI and WC classification may not be applicable:

The BMI and WC are not appropriate for the following populations and these will be considered when undertaking the review and making recommendations. Research questions have been developed to help to provide the evidence required to contextualize the recommendations for these populations.

Elderly >65 years. Interpreting the BMI and WC in the elderly requires caution. Health risk may not be increased in elderly people who are overweight, whereas an increased health risk may occur with a low BMI, between 18.5kg/m² and 21.9kg/m². A normal or low risk BMI range for mortality in the elderly may have different BMI boundaries, between 22kg/m² and 29.9kg/m². Involuntary weight loss in the elderly, especially loss of fat free mass, irrespective of BMI is associated with increased mortality.⁶⁶

Non-Caucasians. Although the body weight classification is intended for application to all ethnic and racial groups in Canada, health care providers should be aware of limitations in applying this classification to non-Caucasians.⁶⁷ In Asians, lower BMI cut-points for overweight (>23kg/m²) and obesity (>27kg/m²) may be warranted.⁶⁸ In African-Americans, health risks appear to be lower for a given BMI range and WC level than in Caucasians, thereby suggesting the need for higher BMI and WC cut-point levels to identify increased health risk.⁶⁷ Whether or not the BMI and WC cut-points used for the general population are appropriate for Inuit and First Nations adults requires additional research.

Adults with a healthy lifestyle. The BMI and WC should also be applied cautiously in adults who are physically active or have other healthy lifestyle habits as these factors appear to mitigate the health risks associated with an increased BMI.⁶⁹ Although physical activity and fitness seem to attenuate the negative effects of obesity, excess adiposity and physical inactivity appear to remain as independent contributors to both all-cause and cardiovascular mortality.⁶⁹

In the athletic population, BMI may inaccurately classify normal athletes as overweight because of their large muscle mass (42-43).

Current Clinical Practice: Prevention and Treatment of Obesity

Prevention of obesity:

Preventive interventions for individuals focus on the same treatment approaches as those used for the treatment of obesity. More aggressive methods (pharmacologic, surgical) are not typically considered and the focus is more on dietary habits, increased physical activity, and other lifestyle changes. Grade A level of recommendation according to 2006 Canadian clinical practice guideline.³

Treatment of obesity:

Therapeutic interventions aimed at weight loss to treat obesity and obesity-related complications are typically multi-component and include: dietary; physical exercise; behaviour modification; pharmacologic therapy; and bariatric surgery.

Most patients commence treatment with a non-pharmacologic, non-surgical approach which, over a 1 to 2 year period, can provide modest 3 to 5kg weight loss ⁷⁰This alone may be sufficient to meet weight loss goals. The addition of pharmacologic agents adds modestly to such weight loss (i.e., approx. 2.8-4.5kg).⁷¹

In instances where weight loss attempts do not respond to these interventions, bariatric surgery, typically with Roux-en-Y gastric bypass, can provide considerable weight loss of 50 to 70kg but is reserved for adults with severe obesity (BMI >40kg/m²) or those with less severe obesity (BMI >35kg/m²) that is associated with significant obesity-related comorbidities.⁷² Though bariatric surgery has been shown to be effective in severely obese patients, it is excluded from this review because the Working Group considers those with extreme BMIs for whom surgery would be indicated to be out of the scope of this review; they were also excluded in the USPTF review. Pharmacological therapy on the other hand could be used for all different levels of overweight and obese (e.g., not limited to those who are very obese) and as such remains within our scope.

Previous Review and Recommendations

The CTFPHC Guidelines from 2006 gave the following recommendations:¹

- There is insufficient evidence to recommend for or against community-wide cardiovascular disease preventive programs to prevent obesity (**I recommendation**).
- There is fair evidence to recommend intensive individual and small group counselling for a reduced calorie or low fat diet to prevent obesity (**B recommendation**).
- There is fair evidence to recommend an intensive individual or structured group program of endurance exercise to prevent obesity (**B recommendation**).
- There is insufficient evidence to recommend a program of strength training exercise to prevent obesity. (**I recommendation**).
- There is fair evidence to recommend an intensive individual or small group program of a combined low fat/reduced calorie diet and endurance exercise intervention to prevent obesity (**B recommendation**).

• There is fair evidence to recommend against low- intensity interventions employing telephone or mail support, or financial incentives to promote a low-fat/reduced calorie diet and endurance exercise as a means to prevent obesity. (**D recommendation**).

The 2011 CTFPHC Adult Obesity Working Group reviewed other guidelines. The Australian⁷³ and the New Zealand⁷⁴ guidelines only considered treatment of overweight and obesity. Neither the Obesity Canada Clinical Guidelines Expert Panel³ or NICE⁷⁵ considered mortality or morbidity outcomes of screening, but both made recommendations about treatment. The review for the SIGN⁴ guidelines searched for studies on the effectiveness of screening but found none. They also made recommendations for management. The USPSTF recently released a review⁵ and draft guidelines for comment. Draft recommendations state that clinicians should screen adults for obesity and that they should offer or refer patients with a body mass index (BMI) greater than 30kg/m² to intensive, multicomponent behavioural interventions (B recommendation).⁷⁶ A detailed summary of these guidelines recommendations is included in Appendix 2.

Review Approach

The Working Group first worked through an "ideal approach", considering the analytic framework and key questions for both screening and prevention of obesity in adults that they considered to be the most important for clinicians. An evidence based analysis on screening and prevention of obesity was planned to address key questions about the effectiveness of screening and preventive efforts for normal weight, overweight or obese adults in primary care on mortality, morbidity, various anthropometric measures of weight reduction or stabilization, costs, and harms. However, our preliminary search revealed recent reviews by the United States Preventive Services Task Force⁵ and SIGN⁴ that asked similar questions and identified no evidence that screening improved patient important outcomes. In order to avoid engaging in a full review when no evidence had previously been identified, we have removed these as key questions and have instead added a series of supplemental questions on screening. These will be examined in a condensed review process that will search for any studies published after the USPSTF review. This will allow us to capture any new evidence that may become available on screening for obesity since the last review.

The USPSTF⁵ also examined interventions for preventing obesity or further weight gain in overweight and obese populations. As a result, the Working Group decided to adopt a more pragmatic approach to selecting the questions that it wanted to have answered, based on those for which preliminary review had indicated that there would be sufficient evidence upon which to formulate a recommendation. In addition, in order to avoid duplication of the work that had already been completed by the USPSTF, the Working Group decided to:

- update the USPSTF review that examined interventions for those who were already overweight and obese (key question 2 below); and,
- conduct a new review to address the effectiveness of prevention interventions for those who are currently of normal weight (key question 1).

The key and contextual questions have been redefined to reflect these changes.

Figure 1: Analytic framework: prevention and treatment interventions for normal weight, overweight and obese adults



Key Questions

Normal Weight Adults (See Figure 1)

Key Question 1 (KQ1). Do primary care-relevant prevention interventions (behaviourally-based) in normal weight adults lead to improved health outcomes or sustained/short-term weight gain prevention, with or without improved physiological measures?

- a. How well is weight gain prevention or health outcomes maintained after an intervention is completed?
- b. What are common elements of efficacious weight gain prevention interventions?
- c. Are there differences in efficacy between adults subgroups (e.g., age 65 years or older; sex; race-ethnicity; baseline cardiovascular risk status)?
- d. What are the adverse effects of primary care-relevant prevention in normal weight adults (i.e.., labelling, disordered eating, psychological distress such as anxiety, depression and stigma, nutritional deficits and cost burden)?
- e. Are there differences in adverse effects between adults subgroups (e.g., age 65 years or older; sex; race-ethnicity; baseline cardiovascular risk status)?

Obese / Overweight Adult Population (See Figure 1)

Key Question 2 (KQ2). Do primary care-relevant prevention or treatment interventions (behaviourally-based and/or pharmacotherapy) in obese/overweight adults lead to short-term or sustained weight loss, or weight gain prevention, with or without improved physiological measures?

- a. How well is weight loss or health outcomes maintained after an intervention is completed?
- b. What are common elements of efficacious interventions (behaviourally-based and/or pharmacotherapy)?
- c. Are there differences in efficacy between patient subgroups (e.g., age 65 years or older; sex; race-ethnicity; degree of obesity/overweight; baseline cardiovascular risk status)?
- d. What are the adverse effects of primary care-relevant prevention or treatment interventions in obese/overweight adults (e.g., nutritional deficits, cardiovascular disease, bone mass loss, injuries, death, mental illness/psychological disorders)?
- e. Are there differences in adverse effects between patient subgroups (e.g., age 65 years or older; sex; race-ethnicity; degree of obesity/overweight; baseline cardiovascular risk status)?

Contextual questions for KQ1 and KQ2

1. Is there evidence that the burden of disease, the risk/benefit ratio of prevention or treatment, the optimal prevention or treatment method/access, and implementation differ in any ethnic subgroups or by age,rural and remote populations, or lower SES populations?

- 2. What are the resource implications and cost effectiveness of overweight and obesity prevention/treatment in Canada?
- 3. What are patients and practitioners' values and screening preferences regarding overweight and obesity prevention/treatment?
- 4. What process and outcome performance measures (indicators) have been identified in the literature to measure and monitor the impact of prevention/treatment for overweight and obesity?
- 5. What are the most effective (accurate and reliable) risk assessment tools* identified in the literature to assess future health risk as a result of obesity?
- 6. What are the most effective (accurate and reliable) risk assessment tools* identified in the literature to identify those at higher risk of obesity?

*Risk assessment tools are defined as those tools that combine known risk factors to identify risk of future obesity or of future health risk (e.g., diseases) associated with being obese now. Expedited searches are conducted to answer contextual questions. In these expedited searches, the ERSC searches selected databases to identify evidence (from any study type) published in the past five years. This search is supplemented by a search of key journals and websites for additional primary studies disseminated in the past two years (i.e., potentially too recent to have been included in published reviews). For these expedited reviews, the ERSC uses Canadian data sources wherever possible. The list of journals and databases to be searched is determined by the working group, with input from the ERSC and clinical and content experts.

Evidence used to address contextual questions does not require quality assessment and may be examined by only one reviewer. Qualitative analyses for all contextual questions will be performed. Study results addressing the above questions will be analysed descriptively.

Supplemental Questions

Is there direct evidence that primary care screening programs for adult obesity or overweight improve health outcomes or result in short-term (12 month) or sustained (>12 month) weight loss or improved physiological measures (i.e., glucose tolerance, blood pressure, and dyslipidemia)?

- a. How well is weight loss maintained after a screening intervention is completed?
- b. What is the most effective method of screening for overweight and obesity in adults in primary care?
- c. What is the optimal interval/frequency for screening for overweight and obesity in adults in primary care?
- d. What is the most effective type of screening (opportunistic vs. organized/systematic) for overweight and obesity in adults in primary care?
- e. What are the harms associated with screening for overweight and obesity in adults in primary care (psychological distress, disordered eating, nutritional deficits, labeling, and cost burden)?

Supplemental questions will be addressed by updating the literature from September 2010, which was the date of the last USPSTF search for articles that examined screening that yielded no results. All study designs will be eligible for inclusion.

For all key questions, the CTFPHC Adult Obesity Working Group will identify literature gaps that would not permit them to answer some of the key questions.

Inclusion/Exclusion Criteria

Language

• For key question 1, where we are conducting a new review, we will include studies published in English and French. Key question 2 is restricted to the language, inclusion, and exclusion criteria of the USPSTF review, as we are updating their work. As such, studies are restricted to English language publications only.

Study design

• For r KQ 1a-c and 2a-c we will include only RCTs with a no intervention control group. For KQ1 d-e 2d-e we will include cohort and case-control studies in addition to RCT's . More specifically, an acceptable control group couldn't receive a personalized intervention, athome workbook materials, and advice more frequently than annually, or participate in frequent weigh-ins (< 3 months). We will exclude case reports, case series and chart reviews for all key questions.

Population and Setting

- For key question 1 studies will be limited to:
 - human studies
 - adults (\geq 18 years) who are normal weight or studies with mixed weight populations who have as part of their sample a normal weight group
 - populations must either be unselected, selected for low cardiovascular disease risk, or selected for increased risk for specified conditions or people who had a diagnosis of the following conditions: cardiovascular disease, hypertension, dyslipidemia, or type II diabetes
 - <u>exclude</u> studies conducted in in-patient hospital settings, institutionalized settings, school-based programs, occupational settings, faith-based programs, and other settings deemed not generalizable to primary care, such as those with existing social networks among participants or the ability to offer intervention elements that could not be replicated in a health care setting, unless the intervention is primary care feasible
 - <u>exclude</u> studies that are focused only on pregnant women (post-partum women will be included) or underweight populations.
- Key question 2 is restricted to the study populations, inclusion, and exclusion criteria as in the USPSTF review, as we are updating their work. These include:
 - human studies
 - adults (\geq 18 years) who are obese or overweight
 - populations must either be unselected, selected for low cardiovascular disease risk, or selected for increased risk for specified conditions (cardiovascular disease, hypertension, dyslipidemia, or type 2 diabetes)
 - trials limited to participants with cardiovascular disease will not be included, though trials could include some participants with cardiovascular disease
 - Commercial programs are eligible for inclusion

- <u>exclude</u> trials conducted in inpatient hospital units, emergency departments, nursing homes or other institutionalized settings, school-based programs, occupational settings, churches and faith-based and other community based settings unless intervention is primary care feasible.
- <u>exclude</u> trials that include pregnant women, and medication induced obese patients
- <u>exclude</u> studies that only include patients with $BMI \ge 40$

Intervention

- For key question 1, interventions focusing on weight gain prevention such as behaviorallybased interventions will be included.
 - <u>exclude</u> pharmacological interventions for normal weight populations and surgical interventions
 - Include complementary/alternative therapies.
- Key question 2 is restricted to intervention inclusion and exclusion criteria as in the USPSTF review as we are updating their work. This includes restricting study interventions to:
 - interventions focusing on weight loss, including behaviourally-based interventions, pharmacological (orlistat and metformin), or a combination of behavioral and pharmacological interventions
 - <u>exclude</u> behavioral interventions that did not focus primarily on weight or that did not report weight-related outcomes, surgical interventions, primary prevention programs that did not involve a weight loss goal for all participants, and trials focusing on pharmacological agents other than orlistat or metformin
- For all KQs subgroup analyses by type of intervention would be performed (e.g. psychologically managed/supervised behavioural intervention and those that are not).

Outcome

- For key question 1a-c, health outcomes will include
- multiple health outcomes: decreased morbidity from diabetes mellitus, cardiovascular disease, cancer, arthritis, asthma, and sleep apnea; improved depression; improved emotional function (scores on emotional subscales of quality of life instruments); physical fitness capacity or performance (not behavioral), physical functioning (scores on physical subscales of quality of life measures), disability (global measures of disability, such as activities of daily living); and mortality
- intermediate outcomes: include a reduction of weight or adiposity (a required outcome). Acceptable measures included weight, relative weight, total adiposity measures, or change in any of these measures
- other intermediate outcomes: include weight maintenance after an intervention has ended; and metabolic consequences (glucose tolerance, blood pressure, dyslipidemia)
- adverse outcomes: include serious treatment-related harms at any time point after an intervention began (i.e., death, medical issue requiring hospitalization or urgent medical treatment) or other treatment-related harms reported in trials
- outcomes reported ≥ 12 months after the start of the intervention were included. Trials of treatment-related harms had no minimum follow-up requirement

- For key question 1d-e adverse effects will include: labeling, disordered eating, psychological distress, nutritional deficits and cost burden.
- Key question 2 is restricted to outcomes as in the USPSTF review as we are updating their work. This includes restricting study interventions to:
 - multiple health outcomes: decreased morbidity from diabetes mellitus, cardiovascular disease, cancer, arthritis, asthma, and sleep apnea; improved depression; improved emotional function (scores on emotional subscales of quality of life instruments); physical fitness capacity or performance (not behavioral), physical functioning (scores on physical subscales of quality of life measures), disability (global measures of disability, such as activities of daily living); and mortality
 - intermediate outcomes: include a reduction of weight or adiposity (a required outcome). Acceptable measures included weight, relative weight, total adiposity measures, or change in any of these measures
 - other intermediate outcomes: include weight maintenance after an intervention has ended; and metabolic consequences (glucose tolerance, blood pressure, dyslipidemia)
 - adverse outcomes: include serious treatment-related harms at any time point after an intervention began (i.e., death, medical issue requiring hospitalization or urgent medical treatment) or other treatment-related harms reported in trials
 - outcomes reported ≥ 12 months after the start of the intervention were included. Trials of treatment-related harms had no minimum follow-up requirement

<u>Time</u>

- For KQ1, there must be a minimum total time of 12 months or any combination of intervention and follow-up that is a minimum of 12 months with the exception of KQ1d-e, which had no time restriction.
- Key question 2 is restricted to time of outcome assessment as in the USPSTF review. This includes restricting outcomes reported at 12 months or longer with the exception of KQ1d-e, which had no time restriction.

Search Strategy

- 1. We will update the 2011 USPSTF reviews for screening and treatment for those who are overweight and obese. We will include all studies that they included except for pharmacological studies that examined drugs not approved by Health Canada, and will add any additional studies that have been published since their last search.
- 2. We will conduct a new search for prevention for those who are normal weight. Search terms will include Embase, Medline, Cochrane, CINAHL, PsychINFO, from 1980 to the present.

The search strategy to be taken from USPSTF 2011,⁵ with an additional search strategy for prevention in normal weight.

We will assess the overall strength of the evidence for key questions 1, 2, according to the GRADE framework.⁷⁷ The strength of evidence will be classified into four grades: high, moderate, low, and very low.

Definitions of Terms

Primary Care: Primary care is the provision of integrated, accessible health care services by clinicians who are accountable for addressing a large majority of personal health care needs, developing a sustained partnership with patients, and practicing in the context of family and community. (Primary Care: America's Health in a New Era. Institute of Medicine (IOM): National Academy Press, 1996.)

Primary Care Interventions Addressed by the CTFPHC: The CTFPHC considers primary care interventions to be those that are delivered in primary care settings or are judged to be feasible for delivery in primary care. To be feasible in primary care, an intervention could be applicable for patients seeking care in primary care settings, and the skills to deliver the intervention are typically present in clinicians and/or related staff or interdisciplinary primary care teams in the primary care setting, or the intervention can generally be ordered/initiated by a primary care clinician.

Reference List

- 1. Reeder BA and Katzmarzyk PT. Prevention of weight gain and obesity in adults: A systematic review. Canadian Task Force on Preventive Health Care; 2006.
- 2. Canadian Task Force on the Periodic Health Examination. Periodic health examination, 1994 update: 1. Obesity in childhood. Canadian Medical Association Journal. 1994;150:871-9.
- Lau DCW, Douketis JD, Morrison KM, et al. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children. Canadian Medical Association Journal. 2006;176(8 Suppl):1-117. www.cmaj.ca/cgi/content/full/176/8/S1/DC1
- 4. Scottish Intercollegiate Guidelines Network. Management of obesity: A national clinical guideline. 115. Edinburgh, Scotland: Scottish Intercollegiate Guidelines Network (SIGN); 2010.
- LeBlanc ES, O'Connor E, Whitlock EP et al. Effectiveness of primary care-Relevant treatments for obesity in adults. 2011. <u>http://www.uspreventiveservicestaskforce.org/uspstf11/obeseadult/obeseart.htm</u>
- 6. Health Canada. Canadian guidelines for body weight classification in adults. 2003. <u>http://www.hc-sc.gc.ca/fn-an/nutrition/weights-poids/guide-ld-adult/weight_book_tc-livres_des_poids_tm-eng.php</u>
- 7. Obesity and overweight. World Health Organization. 2011;
- 8. McLaren L. Socioeconomic status and obesity. Epidemiol Rev. 2007;29:29-48.
- 9. Shields M, Tjepkema M. Trends in adult obesity. Health Rep. 2006;17(3):9-25.
- Statistics Canada. Canadian Health Measures Survey: Adult obesity prevalence in Canada and the United States 2007-2009. Health Canada: The Daily. <u>http://www.statcan.gc.ca/daily-</u> <u>quotidien/110302/dq110302c-eng.htm</u>
- 11. Obesity in Canada: A joint report from the Public Health Agency of Canada and the Canadian Institute for Health Information. Canadian Institute for Health Information; Public Health Agency of Canada; 2011.
- 12. Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: Systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet. 2011;377:557-67.
- 13. Shields M, Tjepkema M. Regional differences in obesity. Health Rep. 2006;17(3):61-7.
- 14. Berthoud HR, Morrison C. The brain, appetite, and obesity. Annu Rev Psychol. 2008;59:55-92. PM:18154499
- 15. Katzmarzyk PT. Obesity and physical activity among Aboriginal Canadians. Obesity. 2008;16:184-90.
- Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: Shaped by global drivers and local environments. Lancet. 2011;378(9793):804-14. PM:21872749
- 17. Ramachandrappa S, Farooqi IS. Genetic approaches to understanding human obesity. J Clin Investig. 2011;121:2080-6.
- 18. Thorp AA, Owen N, Neuhaus M, et al. Sedentary behaviors and subsequent health outcomes in adults a systematic review of longitudinal studies, 1996-2011. Am J Prev Med. 2011;41(2):207-15. PM:21767729

- 19. Kamaura M, Fujii H, Mizushima S, et al. Weight gain and risk of impaired fasting glucose after smoking cessation. J Epidemiol. 2011;21(6):431-9. PM:22001544
- 20. Watanabe M, Kikuchi H, Tanaka K, et al. Association of short sleep duration with weight gain and obesity at 1-year follow-up: a large-scale prospective study. Sleep. 2010;33(2):161-7. PM:20175399
- 21. Eckersley RM. Losing the battle of the bulge: Causes and consequences of increasing obesity. Med J Aust. 2001;174:590-2.
- 22. Burt MG, Gibney J, Ho KK. Characterization of the metabolic phenotypes of Cushing's syndrome and growth hormone deficiency: a study of body composition and energy metabolism. Clin Endocrinol (Oxf). 2006;64(4):436-43. PM:16584517
- Blechman J, Amir-Zilberstein L, Gutnick A, et al. The metabolic regulator PGC-1alpha directly controls the expression of the hypothalamic neuropeptide oxytocin. J Neurosci. 2011;31(42):14835-40. PM:22016516
- 24. de Moura SA, Sichieri R. Association between serum TSH concentration within the normal range and adiposity. Eur J Endocrinol. 2011;165(1):11-5. PM:21543376
- 25. Al-Nuaim LA. The impact of obesity on reproduction in women. Saudi Med J. 2011;32(10):993-1002. PM:22008918
- 26. Scacchi M, Orsini F, Cattaneo A, et al. The diagnosis of GH deficiency in obese patients: a reappraisal with GHRH plus arginine testing after pharmacological blockade of lipolysis. Eur J Endocrinol. 2010;163(2):201-6. PM:20460421
- Barnes RD, Tantleff-Dunn S. A preliminary investigation of sex differences and the mediational role of food thought suppression in the relationship between stress and weight cycling. Eat Weight Disord. 2010;15(4):e265-e269 PM:21406950
- Butler MG. Prader-Willi Syndrome: Obesity due to Genomic Imprinting. Curr Genomics. 2011;12(3):204-15. PM:22043168
- 29. Croft JB, Morrell D, Chase CL, et al. Obesity in heterozygous carriers of the gene for the Bardet-Biedl syndrome. Am J Med Genet. 1995;55(1):12-5. PM:7702084
- 30. Davis C, Zai C, Levitan RD, et al. Opiates, overeating and obesity: a psychogenetic analysis. Int J Obes (Lond). 2011;35(10):1347-54. PM:21266954
- Austin GL, Ogden LG, Hill JO. Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals: 1971-2006. Am J Clin Nutr. 2011;93(4):836-43. PM:21310830
- 32. Bowman SA, Vinyard BT. Fast food consumption of U.S. adults: impact on energy and nutrient intakes and overweight status. J Am Coll Nutr. 2004;23(2):163-8. PM:15047683
- Lundgren JD, Rempfer MV, Brown CE, et al. The prevalence of night eating syndrome and binge eating disorder among overweight and obese individuals with serious mental illness. Psychiatry Res. 2010;175(3):233-6. PM:20031234
- 34. Striegel-Moore RH, Rosselli F, Wilson GT, et al. Nocturnal eating: association with binge eating, obesity, and psychological distress. Int J Eat Disord. 2010;43(6):520-6. PM:19708071

- 35. Thorp AA, Owen N, Neuhaus M, et al. Sedentary behaviors and subsequent health outcomes in adults a systematic review of longitudinal studies, 1996-2011. Am J Prev Med. 2011;41(2):207-15. PM:21767729
- 36. Kyrolainen H, Santtila M, Nindl BC, et al. Physical fitness profiles of young men: associations between physical fitness, obesity and health. Sports Med. 2010;40(11):907-20. PM:20942508
- Matsushita Y, Nakagawa T, Yamamoto S, et al. Associations of smoking cessation with visceral fat area and prevalence of metabolic syndrome in men: the Hitachi health study. Obesity (Silver Spring). 2011;19(3):647-51. PM:20966912
- 38. Van EP. Obesity in pregnancy. S D Med. 2011;Spec No:46-50. PM:21721188
- 39. Schlundt DG, Hill JO, Sbrocco T, et al. Obesity: a biogenetic or biobehavioral problem. Int J Obes. 1990;14(9):815-28. PM:2228413
- 40. Kong A, Beresford SA, Alfano CM, et al. Associations between snacking and weight loss and nutrient intake among postmenopausal overweight to obese women in a dietary weight-loss intervention. J Am Diet Assoc. 2011;111(12):1898-903. PM:22117666
- Brown CM, Dulloo AG, Montani JP. Sugary drinks in the pathogenesis of obesity and cardiovascular diseases. Int J Obes (Lond). 2008;32 Suppl 6:S28-S34 PM:19079277
- 42. Cizza G, Requena M, Galli G, et al. Chronic sleep deprivation and seasonality: implications for the obesity epidemic. J Endocrinol Invest. 2011;34(10):793-800. PM:21720205
- 43. Lester D, Iliceto P, Pompili M, et al. Depression and suicidality in obese patients. Psychol Rep. 2011;108(2):367-8. PM:21675551
- 44. Bodenlos JS, Lemon SC, Schneider KL, et al. Associations of mood and anxiety disorders with obesity: comparisons by ethnicity. J Psychosom Res. 2011;71(5):319-24. PM:21999975
- 45. Abramson EE, Stinson SG. Boredom and eating in obese and non-obese individuals. Addict Behav. 1977;2(4):181-5. PM:607789
- 46. Iversen LB, Strandberg-Larsen K, Prescott E, et al. Psychosocial risk factors, weight changes and risk of obesity: the Copenhagen City Heart Study. Eur J Epidemiol. 2012; PM:22350224
- 47. Choong E, Bondolfi G, Etter M, et al. Psychotropic drug-induced weight gain and other metabolic complications in a Swiss psychiatric population. J Psychiatr Res. 2012; PM:22316639
- 48. Serretti A, Mandelli L. Antidepressants and body weight: a comprehensive review and meta-analysis. J Clin Psychiatry. 2010;71(10):1259-72. PM:21062615
- 49. Verrotti A, D'Egidio C, Mohn A, et al. Weight gain following treatment with valproic acid: pathogenetic mechanisms and clinical implications. Obes Rev. 2011;12(5):e32-e43 PM:20880119
- 50. Fitzpatrick S, Joks R, Silverberg JI. Obesity is associated with increased asthma severity and exacerbations, and increased serum immunoglobulin E in inner-city adults. Clin Exp Allergy. 2011; PM:22092883
- 51. Ramachandrappa S, Farooqi IS. Genetic approaches to understanding human obesity. J Clin Invest. 2011;121(6):2080-6. PM:21633175
- 52. Rosenblatt NJ, Grabiner MD. Relationship Between Obesity and Falls by Middle-Aged and Older Women. Arch Phys Med Rehabil. 2012; PM:22218136

- Nicklas BJ, Gaukstern JE, Legault C, et al. Intervening on spontaneous physical activity to prevent weight regain in older adults: Design of a randomized, clinical trial. Contemp Clin Trials. 2012;33(2):450-5. PM:22155531
- 54. Desai M, Ross MG. Fetal programming of adipose tissue: effects of intrauterine growth restriction and maternal obesity/high-fat diet. Semin Reprod Med. 2011;29(3):237-45. PM:21710399
- 55. Wang Y, Chen X. How much of racial/ethnic disparities in dietary intakes, exercise, and weight status can be explained by nutrition- and health-related psychosocial factors and socioeconomic status among US adults? J Am Diet Assoc. 2011;111(12):1904-11. PM:22117667
- 56. Mokdad AH, Ford ES, Bowman BA, et al. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. JAMA. 2003;289(1):76-9. PM:12503980
- 57. Wilson PW, D'Agostino RB, Sullivan L, et al. Overweight and obesity as determinants of cardiovascular risk: The Framingham experience. Arch Intern Med. 2002;162(16):1867-72. PM:12196085
- 58. Flegal KM, Graubard BI, Williamson DF, et al. Excess deaths associated with underweight, overweight, and obesity. JAMA. 2005;293(15):1861-7. PM:15840860
- 59. Allison DB, Fontaine KR, Manson JE, et al. Annual deaths attributable to obesity in the United States. JAMA. 1999;282(16):1530-8. PM:10546692
- 60. Douketis JD, Macie C, Thabane L, et al. Systematic review of long-term weight loss studies in obese adults: Clinical significance and applicability to clinical practice. Int J Obes. 2005;29:1153-67.
- 61. Douketis JD, Paradis G, Keller H, et al. Canadian guidelines for body weight classification in adults: Application in clinical practice to screen for overweight and obesity and to assess disease risk. Canadian Medical Association Journal. 2005;172(8):995-8. PM:15824401
- 62. Pischon T, Boeing H, Hoffmann K, et al. General and abdominal adiposity and risk of death in Europe. New Engl J Med. 2008;359:2105-20.
- 63. Yusuf S, Hawken S, Ounpuu S, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: A case-control study. Lancet. 2005;366(9497):1640-9. PM:16271645
- 64. Sharma AM, Kushner R. A proposed clinical staging system for obesity. Int J Obes. 2009;33:289-95.
- 65. Padwal RS, Pajewski NM, Allison DB, et al. Using the Edmonton obesity staging system to predict mortality in a population-representative cohort of people with overweight and obesity. Canadian Medical Association Journal. 2011;183(14):E1059-E1066 PM:21844111
- Shea MK, Nicklas BJ, Houston DK, et al. The effect of intentional weight loss on all-cause mortality in older adults: Results of a randomized controlled weight-loss trial. Am J Clin Nutr. 2011;94(3):839-46. PM:21775558
- 67. Katzmarzyk PT, Bray GA, Greenway FL, et al. Ethnic-specific BMI and waist circumference thresholds. Obesity. 2011;19(6):1272-8. PM:21212770
- 68. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004;363:157-63.
- 69. Katzmarzyk PT, Janssen I, Ardern CI. Physical inactivity, excess adiposity and premature mortality. Obes Rev. 2003;4:257-90.

- 70. Norris SL, Zhang X, Avenell A, et al. Long-term non-pharmacological weight loss interventions for adults with prediabetes. Cochrane Database Syst Rev. 2005;(2):CD005270 PM:15846748
- 71. Rucker D, Padwal R, Li SK, et al. Long term pharmacotherapy for obesity and overweight: Updated metaanalysis. BMJ. 2007;335(7631):1194-9. PM:18006966
- 72. Padwal R, Klarenbach S, Wiebe N, et al. Bariatric surgery: A systematic review and network metaanalysis of randomized trials. Obes Rev. 2011;12:602-21.
- 73. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in adults. Australia: 2003.
- 74. Ministry of Health cTRU. Clinical guidelines for weight management in New Zealand adults. Wellington, New Zealand: Ministry of Health; 2009.
- 75. National Institute of Health and Clinical Excellence. Obesity: Guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children. Clinical guideline 43. National Institute of Health and Clinical Excellence; 2006.
- 76. Screening for and management of obesity in adults: U.S. Preventive Services Task Force recommendation statement. Draft 2011. USPSTF. 2011;
- 77. Owens DK, Lohr KN, Atkins D, et al. AHRQ series paper 5: grading the strength of a body of evidence when comparing medical interventions--agency for healthcare research and quality and the effective healthcare program. J Clin Epidemiol. 2010;63(5):513-23. PM:19595577