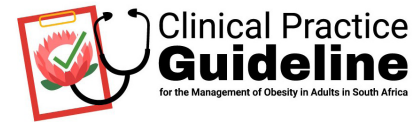






Prevention and harm reduction of obesity (clinical prevention)



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SOUTH AFRICAN METABOLIC MEDICINE AND SURGERY SOCIETY

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KEY MESSAGES FOR HEALTHCARE PROVIDERS

- Obesity is a complex chronic disease in which abnormal or excess body fat (adiposity) impairs health, increases the risk of long-term medical complications, and reduces lifespan.
- Obesity arises from a complex interplay of genetic, biological, behavioural, psychosocial and environmental factors, and can develop via slow and steady weight gain over an extended period, or from rapid bursts of weight gain.
- Obesity prevention should take place in a range of settings that access whole populations or high-risk groups. The individual-based approach to prevention is primarily used by healthcare providers and targets those with the highest level of risk of obesity. The population-based approach addresses the behavioural, sociocultural and environmental factors that contribute to non-communicable diseases in populations, including obesity.
- Primary care clinicians have an important role in early identification of people living with obesity. Regular assessments of body weight are needed to catch early weight gain. (See the chapters '[Assessment of people living with obesity](#)' and '[Primary care and primary healthcare in obesity management](#).')
- Primary care clinicians should initiate discussion around weight gain early and contemplate interventions that consider its complex causes, providing guidance beyond 'eat less and move more'.
- Many medications are associated with weight gain side-effects that can contribute to long-term weight gain. The risks and benefits of such medications should be weighed up for each specific person before prescribing.
- Excess pregnancy weight gain and post-pregnancy weight retention are significantly reduced with behavioural interventions. Primary care clinicians should counsel women attending prenatal care not to exceed pregnancy weight gain guidelines, in the course of dietary, physical activity and psychological interventions during prenatal visits.
- Health benefits of smoking cessation outweigh the cardiovascular consequences associated with smoking cessation-related weight gain.
- Short-term behavioural interventions (generally 6 months or less) aimed at preventing weight gain during young adulthood, menopause, smoking cessation and breast cancer treatment have not yet been shown to be effective.
- Longer-term interventions are likely to be needed to properly examine strategies for preventing weight gain for many of these high-risk groups and in the general population.

KEY MESSAGES FOR PEOPLE LIVING WITH OBESITY

- Preventing or delaying obesity is likely to be easier than long-term weight reduction.
- Causes of and contributors to weight gain are wide ranging, extending beyond personal lifestyle choices such as food intake and exercise, and include factors that you may or may not be able to control.
- Obesity rates increase with age. Reducing weight gain and preventing obesity as you get older is preferable to trying to lose significant weight, considering the difficulty in sustaining weight loss, especially as we age.
- Obesity can develop with small gains in weight over a long period of time, or from rapid bursts of weight gain.
- People are prone to greater weight gain during certain life stages, including adolescence, young adulthood and pregnancy.
- Raise your weight gain concerns with your primary care physician, even if you have experienced modest weight gain.
- Regular weighing by healthcare providers can help to identify patterns and factors contributing to weight gain early on.

Introduction

In our modern environment, there are numerous factors that put adults at risk for weight gain and the development of obesity. From a public health standpoint, prevention of obesity and related health consequences should be a focus of healthcare systems. Preventing obesity can be targeted at the primary, secondary and tertiary care levels, and aimed anywhere from the individual to the population level. These factors are wide ranging, and our understanding of them is growing at a rapid pace. Most research interventions and public health initiatives have focused on nutrition and physical activity. However, there are other modifiable factors, such as sleep, stress, use of medications that cause weight gain, gut dysbiosis secondary to antibiotic use,^[1] other chronic conditions and smoking, that may also influence weight regulation. Factors that influence weight gain but are much less modifiable include age, genetics, epigenetics, income, physical environment, sociopolitical environment and adverse childhood events, including abuse and neglect.^[2] These factors are not modifiable or are less subject to individual control, but may also be potentially important influencers of weight management effectiveness.

This chapter specifically discusses the evidence supporting obesity prevention interventions at the primary, secondary and tertiary levels. In addition, it highlights important opportunities for healthcare providers (HCPs) to engage in preventive, early diagnosis and early intervention activities for overweight and obesity.

Primary prevention

The aim of primary prevention is to minimise weight gain and prevent overweight and obesity from developing in the first place. It has been suggested that primary prevention is the most cost-effective option for addressing obesity.^[3] Owing to the high prevalence of people living with obesity (PLWO), it can be argued that population-level interventions aimed at primary prevention may be more appropriate than interventions aimed at individual-level factors. Commonly proposed targets include unhealthy food/beverage taxation,^[4] calories on menus,^[5] healthy food programmes, limiting food and beverage advertisement, affordable physical activity options, increasing mixed land use, and improving the walkability of the built environment.^[6] It is also important to address social determinants of health that negatively affect an individual's ability to dedicate time or resources to healthy living fundamentals.

Although it is clear that poor nutrition and a lack of adequate physical activity are important risk factors for the development of obesity, there may be other factors that play a significant role. This means that HCPs must consider the underlying root causes of and contributors to weight gain, as opposed to only targeting the symptom of weight gain. For example, stress, shift work or insufficient sleep could be the underlying reason for increased dietary intake; depression and fatigue can lead to decreased physical activity. Further, depending on the situation, attenuating weight gain as opposed to preventing weight gain or achieving weight loss may be a more reasonable goal.

Currently, there are very few randomised controlled trials that examine primary prevention for obesity. Of those published, most examine short time periods that are associated with high risk for rapid weight gain. Very few published studies demonstrate the effectiveness of interventions at the population level. Most of the studies demonstrate effects on eating or physical activity behaviour, but it is unclear whether these results translate into clinically relevant differences in obesity.^[7]

Most of the evidence suggesting that diet and physical activity play a role in preventing unhealthy weight gain and obesity comes

from observational trials.^[8-11] Observational trials do not provide strong evidence on which to base recommendations. Nevertheless, the lack of strong evidence supporting recommendations for obesity prevention should not necessarily preclude HCPs from incorporating these recommendations into practice. It is important also to consider that there are several challenges and barriers that are inherent to conducting prevention research in general.

Primary prevention randomised controlled trials that examine risk factors for weight gain are limited. Within the general population, there have been short studies of dietary interventions examining specific foods, such as yoghurt,^[12] whey protein supplementation^[13] or polyunsaturated fatty acids,^[14] but with limited success. Short-term studies report that behavioural changes, such as self-weighing, frequent reminders or self-reflection, may be associated with significantly less weight gain or even weight loss over the holidays.^[15,16] There are also several published reports on workplace interventions that have been successful in improving knowledge and behaviour but are less likely to report improvements in obesity or weight gain,^[17,18] unless they target weight loss in workers with obesity.^[19,20] Primary prevention studies tend to be short in duration (less than 6 months) and are predominantly nutrition and physical activity interventions. This is problematic, as primary prevention efforts will probably need to be far longer in order to demonstrate weight gains less than the typical 0.5 - 1.0 kg per year. There are a few longer-term, randomised controlled trials, with most showing no differences in weight gain over time, unless a study was able to induce weight loss.^[21-24]

Most prevention research examines weight gain in high-risk populations or during short periods associated with high risk for weight gain, such as pregnancy or postpartum weight retention,^[25-27] smoking cessation,^[28,29] certain cancer treatments,^[30] people using medications associated with weight gain,^[31,32] menopause^[33] and young adults.^[34] Of these, limiting pregnancy and postnatal weight gain has received the most attention; behavioural intervention has been demonstrated to be effective. The limited pharmacological options for weight gain prevention have mainly been examined in populations that are more likely to include PLWO, such as people living with diabetes or those taking antipsychotic medications.

Pregnancy: Gestational weight gain and postpartum weight retention

The causes of maternal obesity are often multifaceted, including environmental, societal and other factors. This necessitates a multi-system, life-course approach to the prevention and management of PLWO. Pregnancy is a period of rapid weight gain. International recommendations suggest that weight gained through pregnancy should be between 5 and 18 kg,^[35] depending on the woman's pre-pregnancy body mass index (BMI) category. The International Federation of Gynaecology and Obstetrics recommends that pregnant women with a BMI ≥ 30 kg/m² should be advised to avoid high gestational weight gain.^[36] Studies demonstrate that many women retain 2 - 5 kg per pregnancy.^[37] Pregnancy and the postnatal period may therefore be particularly important periods for targeted primary weight gain prevention.

Behavioural interventions to prevent excessive gestational weight gain have ranged in their intensiveness and delivery methods. Most use medical nutrition therapy and/or exercise interventions.^[37] Some interventions also incorporate behavioural change strategies to supplement the programme. To date, it is unclear which aspects of the intervention, or which combination, are the most effective.

A Cochrane review of high-quality evidence published in 2015^[38] reported that nutrition and/or exercise randomised controlled trials are associated with a 20% reduction in risk for excessive gestational

weight gain. In this review, the effectiveness of the interventions was not clearly demonstrated in women with overweight or obesity, which is concerning given the already increased risk for negative pregnancy outcomes for both mother and baby in these populations.^[37] The authors hypothesised that there may be differences in physiology and/or other barriers that may require a more intensive intervention to prevent excessive gestational weight gain in women already affected by overweight or obesity. In a study by Yeo *et al.*,^[27] the authors suggest that interventions delivered by prenatal care providers may be more successful than those delivered outside prenatal care, resulting in 3 kg less gestational weight gain. Although behavioural interventions are effective in reducing gestational weight gain, it is less clear whether these interventions are sufficient to improve maternal and fetal complications.^[39] Nevertheless, prenatal behavioural interventions may present a unique opportunity for prevention of obesity at a life stage when women are regularly engaged with their HCP.^[37]

Smoking cessation

Smoking cessation is associated with substantial cardiovascular benefits, but also with substantial weight gain. For example, Tian *et al.*^[40] reported that individuals who quit smoking gained 2.6 kg more than those who continued to smoke over 6 years. However, it is important to note that a recent meta-analysis suggests that the mortality risk associated with the weight gain associated with smoking cessation is far less than the mortality rate^[41] associated with continuing to smoke.

Nevertheless, post-cessation weight gain is a significant concern,^[28] and may have a negative impact on smoking cessation efforts, particularly in individuals of white ethnicity and those with existing weight concerns.^[28,29] Interventions that address post-cessation weight gain may therefore be important for improving smoking cessation success.

Weight gain associated with smoking cessation is largely attributed to increased energy intake and reduced energy expenditure.^[41] Several studies that tested a combination of smoking cessation and traditional calorie restriction interventions (using meal replacement or low-calorie diets) reported mixed results on cessation and weight gain.^[42] Further, there is a concern that strict caloric restriction may impede smoking cessation attempts.^[41,43,44] Not all agree on this point, as some suggest that combined weight and smoking cessation programmes may in fact improve abstinence in the short term (less than 3 months). Unfortunately, there do not appear to be long-term benefits of behavioural interventions for weight gain prevention.^[42] Similarly, it does not appear that exercise alone is associated with improved weight gain prevention.^[45] That said, evidence from observational trials suggests that individuals who quit smoking are better able to manage their weight if they are physically active.^[41,45] A recent study reports that post-cessation weight gain in young adults was not related to dietary and physical activity patterns,^[29] suggesting that post-smoking weight management may be far more complicated than can be explained by behavioural habits alone. Nevertheless, physical activity and improved dietary habits are likely to have beneficial health effects independent of changes in body weight.

A recent Cochrane review^[46] suggests that there is short-term evidence to support the effectiveness of pharmacotherapies to attenuate post-cessation weight gain, but as with short-term behavioural interventions, it is unclear whether these benefits extend past 1 year, or which, if any, pharmacotherapy is superior.^[46,47] It therefore appears that pharmacotherapy delays but does not prevent post-cessation weight gain.

In summary, individuals who attempt to quit smoking should be aware of the risk of weight gain. Nevertheless, the health benefits of smoking cessation generally exceed the consequences of some weight gain. However, there is insufficient evidence to strongly recommend any single type of intervention to prevent post-cessation weight gain. Adoption of healthy behavioural habits is recommended as an adjunct for smoking cessation programmes.

Cancer treatment

Although weight loss is more common with cancer treatment, some people gain weight, and weight gain is particularly common with breast, colorectal, prostate and ovarian cancers.^[30,48,49] Weight gain may be related to certain medications, chemotherapy and hormonal changes.^[50] The vast majority of literature has focused on the efficacy of behavioural interventions for the prevention of weight gain for breast cancer patients, as most women experience weight gain during breast cancer treatment.^[51] In fact, fewer than 10% of women who gain weight after receiving a breast cancer diagnosis return to their pre-diagnosis weight, even after 6 years.^[30]

According to a recent review by Thomson and Reeves,^[30] most studies do not demonstrate significant weight gain prevention, and none demonstrate longer-lasting weight effects after the cessation of the intervention. Although the evidence is limited, some studies suggest that outcomes may be better in postmenopausal women, and that initiating interventions while the patient is still undergoing chemotherapy may be key in preventing weight gain.^[30] That said, the short-term negative outcomes of weight gain are less consistent than those seen with weight loss,^[49,52-54] with increased mortality risk reported only when the weight gains exceed 10%.^[49] Care must therefore be given to ensuring that weight management efforts do not mask negative health outcomes that require intervention.

Medication use

Use of several classes of medications, such as antipsychotics, antidepressants, antihyperglycaemic agents, corticosteroids and antiretroviral medication, is associated with weight gain.^[31,32] The amount of weight gain they are associated with varies; some can be a potentially large contributor to PLWO. From a primary prevention standpoint, HCPs may therefore wish to consider the weight-gaining side-effects, if possible, when initiating medications. In general, there is insufficient evidence to suggest the routine prescription of adjunct medications for preventing weight gain, and it is probably inappropriate from a primary prevention perspective. However, for many of these medications, the magnitude of associated weight gain and the potential for cardiometabolic consequences may warrant consideration.

Antipsychotics

Several antipsychotic medications are well documented to be associated with weight gain and are associated with the highest levels of weight gain.^[55] In the short term, antipsychotics are associated with weight gains of approximately 3.2 kg, and long term with gains of 5.3 kg, compared with placebo control.^[31] Of these medications, olanzapine and clozapine are associated with the largest amounts of weight gain,^[32] with as much as 10 kg reported.^[55] Antipsychotics are thought to relate to weight gain through changes in appetite and altered metabolism.^[56] (See the chapter '[The role of mental health in obesity management](#)'.)

Initiating medications with less weight gain tendency^[57,58] may therefore be a preferable option, if clinically appropriate. It may also be important to consider whether medications are needed for long-

term management, and whether acutely switching to a medication with a better weight gain profile for maintenance therapy may be appropriate.^[59] If the decision is made to switch medications, symptoms should be closely monitored to address side-effects, such as rebound insomnia, and to ensure that relapse does not occur. It is also likely that switching to an agent with a lower propensity to cause weight gain may prevent ongoing weight gain, but may not significantly reverse the weight already gained; early switching, where appropriate, is therefore worth considering.^[60]

To prevent weight gain, pharmacological and behavioural interventions have been examined and have been found to have variable success. Medical nutrition therapy, physical activity and cognitive behavioural strategies are associated with medium effect sizes for weight loss trials and large effect sizes for weight gain prevention trials.^[61] However, even with intervention, many people are still likely to gain weight. Weight gain is also associated with untreated mental illness, and not treating is not an option. Consequently, it may be more beneficial to initiate behavioural interventions early after initiating antipsychotic use. Of the pharmacological choices, support exists for metformin as an adjunct therapy,^[60] but this is likely only to be applicable to populations with already prevalent obesity. In general, there is no strong evidence to suggest the routine prescription of adjunct medications for preventing antipsychotic-associated weight gain or for achieving weight reduction after weight gain.^[61]

Antidepressants

Antidepressants are associated with a more moderate amount of weight gain than antipsychotics, with a recent review citing a 2 - 5 kg weight gain associated with tricyclic antidepressants, monoamine oxidase inhibitors, mirtazapine and selective serotonin reuptake inhibitors (SSRIs).^[55] SSRIs were noted to have the lowest propensity to cause weight gain in this study, especially when prescribed for shorter periods of time. Furthermore, paroxetine was most strongly associated with weight gain, while fluoxetine caused the least weight gain in this class of drugs.^[55] However, antidepressants may have a higher global weight gain burden, as there are more individuals with depression than schizophrenia.^[32] Upon initiation of an antidepressant, close monitoring of weight changes is needed, as early changes in body weight are highly predictive of long-term changes.^[62]

Clinicians should therefore consider early intervention in preventing excessive weight gain if possible. Weight gain with antidepressant use may be associated with increased appetite, but could also indicate changes in the underlying mood disorder.^[55] In observational trials, dietary choices are associated with differences in weight gain,^[63] but depression is often an exclusion criterion for weight management trials. It is therefore unclear whether medical nutrition therapy or physical activity therapy are effective in preventing weight gain associated with antidepressant use, particularly in populations without obesity. Again, weight gain is associated with untreated illness as well, so whenever possible, engagement in healthy nutrition and eating behaviour, as well as weight monitoring, are important when initiating and continuing treatment of depression. (See the chapter [‘The role of mental health in obesity management’](#).)

Diabetes medications

Most individuals with type 2 diabetes have obesity, and PLWO are recommended weight loss to improve risk factors. Some diabetes medications are paradoxically associated with improved insulin sensitivity and increased weight.^[55] Thiazolidinediones, sulphonylureas and meglitinides may be associated with weight

gains of between 1 and 4 kg, while insulin is associated with higher weight gains of 5 - 6 kg.^[55] The mechanisms responsible for weight gain vary between the medications, but include increases in appetite, increased lipid storage and fluid retention.^[55] People who are prescribed sulphonylureas as a first-treatment strategy typically have greater weight gains^[32] than with other medications. However, there is variation between sulphonylureas with regard to the degree of weight gain.^[64] Metformin is the most commonly prescribed first-line treatment option and is associated with modest weight losses of 1.0 - 2.9 kg,^[32] and may help prevent some of the weight gain associated with other type 2 diabetes medications such as insulin therapy.^[65]

Menopause

The transition to menopause is associated with greater than normal fat gain, but with only normal rates of age-related weight gain.^[33] Owing to the hormonal changes in menopause, there are losses in muscle mass that mask the accelerated gains in fat mass. Despite the beneficial effects of hormone replacement on body fat distribution, there is insufficient evidence to support its recommendation as a treatment for obesity.^[33] Menopause is associated with increases in sedentary time and physical inactivity.^[66] Several large studies have examined the impact of behavioural interventions on weight management, although most examined middle-aged women,^[67] and not necessarily the period of transition into menopause. Simkin-Silverman *et al.*^[24] undertook one of the few studies to demonstrate that behavioural intervention is successful in preventing weight gain over 5 years in women transitioning to menopause. Similarly, Kuller *et al.*^[68] reported success preventing weight gain over 54 months. In short, more research is needed to determine which components are most important in preventing menopausal-related increases in adiposity.

Young adults

Early adulthood has been a life phase associated with increased risk for weight gain, with one study reporting an average weight gain of 14 kg over 15 years of follow-up in young adulthood.^[69] Onset of weight gain is common in this age group, and it may therefore represent an important life stage to target with weight gain prevention incentives. In particular, young adults attending post-secondary education are reported to have significant weight gain. However, a meta-analysis suggests the weight gain in the first year of college to be less than 2 kg,^[70] comparable to the average weight gain for the general population. Most of the literature in young adults that examines obesity prevention in fact involves weight loss or weight loss maintenance trials^[71] or observational studies. These interventions use nutrition and physical activity approaches, behavioural change strategies, technology-based programmes and educational programmes. Interventions in this age group tend to be disappointing, with several reporting no effect of intervention^[72-74] on weight gain prevention. Interventions that do demonstrate significant effects are typically weight loss studies,^[34,75] and overall show modest results of less than 2 kg,^[71] with very limited evidence that this is maintained long term.^[71] This finding is in accordance with other clinical weight loss research suggesting that younger age is associated with worse weight outcomes.^[76] Younger adults may therefore be a particularly high-risk group for weight gain and poor primary prevention intervention success.

Secondary prevention

Secondary prevention aims to reduce the impact of the disease that has already developed. This is accomplished by early detection and treating the disease as soon as possible in order to slow or stop its

progression. Ultimately, the aim of secondary prevention is to return the person to their original health and functional status to prevent long-term problems.

In terms of obesity, this can be thought of as regular screening and preventing further weight gain in individuals with uncomplicated obesity (i.e. Edmonton Obesity Staging System (EOSS) stage 0 or 1). Although obesity is strongly associated with morbidity and mortality, there is substantial variation in the health profiles observed between individuals with the same BMI. Further, at the lower border of obesity, there are individuals who have not yet developed obesity-related comorbidities, such as hypertension, dyslipidaemia, orthopaedic problems or diabetes. It has been reported that up to 40% of the population may present with an elevated BMI, yet can be described as having EOSS stage 0 or 1 (depending on the definition used to define healthy).^[77,78] In PLWO with EOSS stage 0 or 1, there are reports that up to 20 - 25% have not yet developed a comorbidity.^[79]

Unlike tertiary prevention, where weight loss is clearly associated with health benefits for PLWO with prevalent obesity-related morbidity, it is unclear what, if any, benefits there may be for people who present with EOSS stage 0 or 1.^[80,81] There is debate in the published literature as to whether people with EOSS stage 0 or 1 have better long-term health outcomes and a lower mortality risk compared with PLWO and obesity-related complications.^[77,82] Individuals with EOSS stage 0 or 1 tend to be more physically active, with less consistent evidence reported for dietary differences.^[83] This may suggest that behavioural strategies may also play an important role in secondary prevention in this patient population.

Importance of self-weighing

One of the key considerations for primary and secondary prevention is the concept of regular monitoring and early diagnosis. Obesity is surprisingly hard to recognise without objective assessments,^[84] and may be harder to recognise as the average BMI in the population is now within the overweight range.^[85] Consequently, despite the attention given to PLWO, HCPs and the general population may be less likely to recognise the need for or pursue obesity prevention interventions.^[85] This would suggest that regular assessments need to be done, preferably as a primary prevention method at primary care level. (See the chapter '[Primary care and primary healthcare in obesity management](#)').

However, to our knowledge there are no randomised controlled trials that examine regular self-weighing in a primary or secondary prevention context. In observational trials such as the Pound of Prevention study^[86] and the STOP Regain trial,^[87] individuals who engaged in self-weighing had less weight gain over time. In the context of weight loss or weight loss maintenance, self-weighing is also associated with better weight outcomes.^[88,89] However, in populations with severe obesity, regular weighing may be a source of stress and frustration that needs to be considered on an individual basis with the patient. HCPs should therefore initiate respectful conversations around weight and weight gain before the development of obesity.

Tertiary prevention

Tertiary prevention aims to soften the impact of an ongoing illness or injury that has lasting effects. This is done by helping people manage long-term, often complex health problems and injuries (e.g. chronic diseases, permanent impairments) in order to improve as much as possible their ability to function, their quality of life and their life expectancy. For PLWO, this would be synonymous with weight loss and long-term obesity management. This is where the majority of research lies and is the topic of the other guideline chapters.

Conclusion

Obesity prevention represents a critical public health priority for all healthcare systems. Primary prevention requires a comprehensive, multi-level approach addressing the complex array of factors contributing to weight gain in modern society. In South Africa, population-based interventions targeting both nutrition and physical activity by way of addressing the food and built environments are essential. Effective obesity prevention must, however, also consider emerging modifiable factors at an individual level, such as sleep quality, stress management, medication effects and gut health. Although many contributors such as socioeconomic status and environmental factors present significant challenges, recognising their influence is essential for developing realistic and effective weight management strategies. Success in obesity prevention will ultimately depend on co-ordinated efforts spanning primary, secondary and tertiary levels, involving multiple governmental departments, and implemented across individual and population-based interventions that acknowledge both the causes of and contributors to weight regulation in modern society.

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- Vallianou N, Stratigou T, Christodoulatos GS, Dalamaga M. Understanding the role of the gut microbiome and microbial metabolites in obesity and obesity-associated metabolic disorders: Current evidence and perspectives. *Curr Obes Rep* 2019;8(3):317-332. <https://doi.org/10.1007/s13679-019-00352-2>
- Williamson DF, Thompson TJ, Anda RF, Dietz WH, Felitti V. Body weight and obesity in adults and self-reported abuse in childhood. *Int J Obes Relat Metab Disord* 2002;26(8):1075-1082. <https://doi.org/10.1038/sj.ijo.0802038>
- Gill TP. Key issues in the prevention of obesity. *Br Med Bull* 1997;53(2):359-388. <https://doi.org/10.1093/oxfordjournals.bmb.a011618>
- Redondo M, Hernandez-Aguado I, Lumbreras B. The impact of the tax on sweetened beverages: A systematic review. *Am J Clin Nutr* 2018;108(3):548-563. <https://doi.org/10.1093/ajcn/nqy135>
- Sarink D, Peeters A, Freak-Poli R, et al. The impact of menu energy labelling across socioeconomic groups: A systematic review. *Appetite* 2016;99:59-75. <https://doi.org/10.1016/j.appet.2015.12.022>
- Kumanyika SK, Obarzanek E, Stettler N, et al. Population-based prevention of obesity: The need for comprehensive promotion of healthful eating, physical activity, and energy balance: A scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the Expert Panel on Population and Prevention Science). *Circulation* 2008;118(4):428-464. <https://doi.org/10.1161/CIRCULATIONAHA.108.189702>
- Walls HL, Peeters A, Proietto J, McNeil JJ. Public health campaigns and obesity – a critique. *BMC Public Health* 2011;11:136. <https://doi.org/10.1186/1471-2458-11-136>
- Fung MD, Canning KL, Mirdamadi P, Ardern CI, Kuk JL. Lifestyle and weight predictors of a healthy overweight profile over a 20-year follow-up. *Obesity (Silver Spring)* 2015;23(6):1320-1325. <https://doi.org/10.1002/oby.21087>
- Fung TT, Pan A, Hou T, et al. Long-term change in diet quality is associated with body weight change in men and women. *J Nutr* 2015;145(8):1850-1856. <https://doi.org/10.3945/jn.114.208785>
- Golubic R, Wijndaele K, Sharp SJ, et al. Physical activity, sedentary time and gain in overall and central body fat: 7-year follow-up of the ProActive trial cohort. *Int J Obes (Lond)* 2015;39(1):142-148. <https://doi.org/10.1038/ijo.2014.66>
- Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med* 2011;364(25):2392-2404. <https://doi.org/10.1056/NEJMoa1014296>
- Sayon-Orea C, Martinez-Gonzalez MA, Ruiz-Canela M, Bes-Rastrollo M. Associations between yogurt consumption and weight gain and risk of obesity and metabolic syndrome: A systematic review. *Adv Nutr* 2017;8(1):146S-154S. <https://doi.org/10.3945/an.115.011536>
- Ooi EM, Adams L, Zhu K, et al. Consumption of a whey protein-enriched diet may prevent hepatic steatosis associated with weight gain in elderly women. *Nutr Metab Cardiovasc Dis* 2015;25(4):388-395. <https://doi.org/10.1016/j.numecd.2014.11.005>

81. Liu RH, Wharton S, Sharma AM, Ardern CI, Kuk JL. Influence of a clinical lifestyle-based weight loss program on the metabolic risk profile of metabolically normal and abnormal obese adults. *Obesity (Silver Spring)* 2013;21(8):1533-1539. <https://doi.org/10.1002/oby.20219>
82. Kuk J, Rotondi M, Sui X, Blair SN, Ardern CI. Individuals with obesity but no other metabolic risk factors are not at significantly elevated all-cause mortality risk in men and women. *Clin Obes* 2018;8(5):305-312. <https://doi.org/10.1111/cob.12263>
83. Phillips CM. Metabolically healthy obesity across the life course: Epidemiology, determinants, and implications. *Ann N Y Acad Sci* 2017;1391(1):85-100. <https://doi.org/10.1111/nyas.13230>
84. Harris CV, Bradlyn AS, Coffman J, Gunel E, Cottrell L. BMI-based body size guides for women and men: Development and validation of a novel pictorial method to assess weight-related concepts. *Int J Obes (Lond)* 2008;32(2):336-342. <https://doi.org/10.1038/sj.ijo.0803704>
85. Yates EA, Macpherson AK, Kuk JL. Secular trends in the diagnosis and treatment of obesity among US adults in the primary care setting. *Obesity (Silver Spring)* 2012;20(9):1909-1914. <https://doi.org/10.1038/oby.2011.271>
86. Jeffery RW, French SA. Preventing weight gain in adults: The Pound of Prevention study. *Am J Public Health* 1999;89(5):747-751. <https://doi.org/10.2105/ajph.89.5.747>
87. Wing RR, Tate DF, Gorin AA, Raynor HA, Fava JL, Machan J. STOP Regain: Are there negative effects of daily weighing? *J Consult Clin Psychol* 2007;75(4):652-656. <https://doi.org/10.1037/0022-006X.75.4.652>
88. Madigan CD, Aveyard P, Jolly K, Denley J, Lewis A, Daley AJ. Regular self-weighing to promote weight maintenance after intentional weight loss: A quasi-randomised controlled trial. *J Public Health (Oxf)* 2014;36(2):259-267. <https://doi.org/10.1093/pubmed/ftd061>
89. Shieh C, Knisely MR, Clark D, Carpenter JS. Self-weighing in weight management interventions: A systematic review of literature. *Obes Res Clin Pract* 2016;10(5):493-519. <https://doi.org/10.1016/j.orcp.2016.01.004>