CHAPTER 7.

What existing epidemiological data could serve to better understand the relationship of energy intake and expenditure to obesity and the obesity epidemic?

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Understanding the relationship between energy balance and obesity is a challenge, particularly in low- and middle-income countries (LMICs), where data are limited. Most of the studies have been conducted in high-income countries, and the results may not be generalizable to other settings because of differences in population characteristics.

This chapter provides an overview of studies focusing on determinants of obesity (adiposity) relating to dietary intake (energy intake) and physical activity (energy expenditure) and proposes study designs that could be implemented in LMICs to improve the understanding of the main drivers of obesity in these countries.

Determinants of obesity

Obesity is defined as a state of excess adiposity that presents a risk to health, for example increased risk of chronic diseases [1], and is the consequence of sustained positive energy balance over time.

Factors that influence energy balance can be considered as relating to the host (i.e. people), the environment (i.e. the set of external factors to which people are exposed), and the vector (i.e. foods and drinks). These factors interact in a complex way to influence eating and drinking patterns as well as activity behaviours. Although these factors are experienced at the individual level as the acceptability, availability, and affordability of foods, drinks, and activity behaviours, their roots lie in policies and actions that determine the environment, which may be local, national, or international [2].

Apart from non-modifiable factors, other characteristics may influence energy balance – in particular, the amount of energy expended in physical activity. Increasing energy expenditure might be expected not to influence energy balance, because of appetite control mechanisms that feed back and tend to maintain balance. However, there is evidence that at the low levels of activity characteristic of many high-income countries and increasingly of LMICs, this feedback operates imperfectly and does not suppress appetite to the low levels necessary to maintain energy balance [3]. Many factors relating to the foods and drinks consumed have been shown to influence the amount consumed or energy balance over the short to medium term, such as energy density and portion size [4, 5].

Measurement of adiposity

Several measures for overweight and obesity have been used in epidemiological studies. However, it is important to be aware that such measures are imperfect markers of the internal physiological processes that are the actual drivers of cancer development.

Body mass index (BMI) is defined as the quotient between weight in kilograms and height in metres squared (kg/m^2); overweight and obesity are conventionally defined in relation to cut-offs of 25 kg/m^2 and 30 kg/m^2, respectively [6]. BMI is the most commonly used marker of body composition in epidemiological studies, because of the simplicity of assessment and the high precision and accuracy. However, it does not differentiate between lean and adipose tissue or take into account fat distribution, which varies across individuals, among ethnicities, and throughout the lifespan. Nevertheless, BMI at a population level is a useful marker of or proxy for adiposity.

Waist circumference (WC) and waist–hip ratio (WHR) are useful tools to identify abdominal obesity, commonly defined as WHR ≥ 0.90 for men and ≥ 0.85 for women, with WC cut-offs varying according to sex and ethnicity [7]. However, these measures cannot clearly differentiate between visceral and subcutaneous fat compartments [8]. Skinfold thickness can be used to predict body fat and its distribution, but it is particularly prone to measurement error and is generally unfeasible for use in large studies. Bioelectrical impedance analysis estimates lean and fat mass based on the principle that resistance to an electrical current is greater in adipose tissue than in lean tissue. Bioelectrical impedance analysis is an accurate and reproducible measure of body composition [9], but the body composition estimates from this method do not appear to yield stronger correlations with biomarkers of chronic disease risk than BMI does [10].

More direct measures of body composition are available, such as air displacement plethysmography, underwater weighing (hydrodensitometry), dual-energy X-ray absorptiometry (DEXA), ultrasonography, computed tomography, and magnetic resonance imaging [11, 12]. These methods show excellent reproducibility and validity [13] and are increasingly being used to measure body composition at the tissue or organ level, particularly in small-scale studies that require a high level of accuracy. However, because of high costs and lack of portability, their use in large-scale epidemiological studies tends to be as reference methods [14].

Understanding nutritional determinants of obesity

Experimental data

Prevention of weight gain and/or maintenance of weight loss

Short- to medium-term experimental data in humans can illuminate the possible physiological and other mechanisms underpinning how foods, drinks, and nutrients might promote energy overconsumption and positive energy balance.

In such studies, factors relating to the host include genetic variants that are associated with higher risk of obesity; these variants tend to be related to appetite regulation rather than to energy metabolism at a whole-body or cellular level [15]. Such studies may also include the impact of early-life events, so that child growth trajectories can predict later risk of obesity. Thus, children tend to maintain their BMI ranking within their population throughout childhood, particularly after the age of 11 years [16]. Also, the timing of the rebound in BMI in childhood (the so-called adiposity rebound) predicts later obesity, with an earlier rebound indicating a higher risk of obesity [17]. However, such factors are not modifiable in later life, although the effects might be mitigated.

There is evidence that people who are more physically active, and those who spend less time sedentary, are less likely to gain excess weight in adulthood. Results from intervention trials have been inconsistent, but based on observational studies conducted among Caucasian adults, an increase in energy expenditure through physical activity of approximately 6300–8400 kJ/week (1500–2000 kcal/week) was associated with improved weight maintenance [18].

These host factors may also interact with other factors related to the vector (foods and drinks). For example, in studies of young men eating ad libitum diets of different energy density for up to 3 weeks, Stubbs et al. found that the higher the level of activity, the more likely people were to avoid positive energy balance [19]. At high levels of energy density (60% of energy as fat), only the most active people remained in energy balance, whereas the least active were able to maintain energy balance only at low levels of energy density (20% of energy as fat). However, this trial was conducted among a small number of participants and for a short duration (2 weeks).

Long-term (> 1 year) experimental data are also available. In the long-term Prevención con Dieta Mediterránea (PREDIMED) study, with a follow-up of 4 years, the impact of a non-calorie-restricted traditional Mediterranean diet enriched with nuts and olive oil (two high-energy foods) was compared with that of a control diet consisting of advice on a low-fat diet. No substantial weight gain was observed in the group eating the enriched Mediterranean diet [20]. In the European Prospective Investigation into Cancer and Nutrition (EPIC), among participants with baseline BMI < 25 kg/m^2, dietary energy density was weakly associated with weight change, whereas among participants with

BMI > 25 kg/m^2, greater energy density was inversely associated with weight change [21]. The long-term impact of different macronutrient proportions, of the food sources of those macronutrients, and of energy density on adiposity and the development of obesity remains to be more precisely clarified.

There is also evidence that specific foods, drinks, or food components can have an impact on energy homeostasis. A recent systematic review of randomized controlled trials (RCTs) and prospective cohort data on dietary sugar and body weight concluded that among free-living people eating ad libitum diets, intake of free sugars or sugar-sweetened beverages is a determinant of body weight. The change in body fatness that occurs when intake is modified appears to be mediated via changes in energy intakes, because isoenergetic exchange of sugars with other carbohydrates was not associated with weight change [22]. In addition, in a small experimental study that compared isoenergetic meals with low and high glycaemic index (GI), plasma glucose (2-hour area under the curve) after the high-GI meal was 2.4 times that after the low-GI meal. Thereafter, compared with the low-GI meal, the high-GI meal decreased plasma glucose, increased hunger, and selectively stimulated brain regions associated with reward and craving in the late postprandial period. This suggests that reduced consumption of high-GI carbohydrates (specifically, highly processed grain products, potatoes, and concentrated sugar) may ameliorate overeating and facilitate maintenance of a healthy weight in overweight and obese individuals [23].

Evidence from RCTs conducted in *children and adolescents* indicates that consumption of sugar-sweetened beverages, compared with non-calorically sweetened beverages, results in greater weight gain and larger increases in BMI; however, the evidence is limited to a small number of studies. The findings of these trials suggest that there is inadequate energy compensation (degree of reduction in intake of other foods and drinks) for energy delivered as sugar [24]. However, a recent RCT showed a similar effect of a low-fat diet and a low-glycaemic-load diet on BMI over 24 months among Hispanic children [25]. There is inconsistent evidence that dietary fibre has an impact on energy balance but some evidence that wholegrain foods may help maintain energy balance [24].

For prevention of weight gain, few reported interventions have continued for more than 12 months; therefore, extrapolation to lifelong effectiveness should be done with caution. A review of diet and nutrition factors in relation to prevention of weight gain found evidence that protective factors against obesity were regular physical activity (convincing), a high intake of dietary non-starch polysaccharides/fibre (convincing), home and school environments that support healthy food choices for children (probable), and breastfeeding (probable). Risk factors for obesity were sedentary lifestyles (convincing), a high intake of energy-dense, micronutrient-poor foods (convincing), heavy marketing of energy-dense foods and fast-food outlets (probable), sugar-sweetened soft drinks and fruit juices (probable), and adverse social and economic conditions (in developed countries, especially for women) (probable) [26].

Understanding weight loss

There is copious evidence relating to various strategies for promoting weight loss through negative energy balance among overweight and obese people, although there is less evidence relating specifically to maintaining energy balance (or preventing weight gain and obesity). In addition, long-term data from RCTs for obesity prevention are more difficult to obtain than those from trials for weight loss.

For weight loss, there is widespread but not universal agreement that limiting the proportion of dietary energy from fat (or energy density) helps to reduce ad libitum energy intake [27]. More recently, evidence has accrued for the effects of intermittent partial fasting for 2 days per week [28]. Avoiding calorific beverages, especially sugar-sweetened beverages, aids weight control [29], because consumption of these beverages is not followed by adequate compensation in subsequent intake. Simple calorie counting appears to be relatively ineffective. A recent meta-analysis of RCTs comparing the long-term effect (≥ 1 year) of dietary interventions on weight loss showed that low-carbohydrate interventions led to significantly greater weight loss than did low-fat interventions (18 studies), and that low-fat interventions did not lead to differences in weight change compared with other, higher-fat interventions (19 studies) [30]. In a 2-year trial, obese subjects were randomly assigned to one of three diets: low-fat, restricted-calorie; Mediterranean, restricted-calorie; or low-carbohydrate, restricted-calorie. For the Mediterranean-diet group and the low-carbohydrate group, weight loss was similar and significantly higher than that for the low-fat group. Among the subjects with diabetes, the Mediterranean diet led to larger changes in plasma glucose and insulin levels compared with the low-fat diet [31].

The addition of a physical activity component improves the efficacy of a dietary intervention, but physical activity alone has not been shown to lead to weight loss (possibly because in obese people it is difficult to achieve a sufficient volume of activity). Physical activity is helpful in weight maintenance after weight loss [18].

A review of workplace and community interventions found some evidence for the effectiveness of these interventions in changing diet and physical activity behaviours but limited evidence for their effectiveness in changing BMI [32]. However, many interventions are of relatively short duration, whereas changes in BMI are more distal outcomes.

Most of these studies were conducted in high-income countries. This emphasizes the importance of conducting studies in LMICs, in particular long-term dietary intervention trials focusing on alternative dietary patterns with foods readily available in these countries to propose viable changes in nutritional behaviours.

Epidemiological data

Population surveys, etiological and population intervention studies, and implementation research are all important to develop the evidence base to tackle the rise in the prevalence of obesity associated with changing dietary and physical activity patterns.

Population surveys in representative samples combining the evaluation of diet, physical activity, and anthropometry and chronic outcomes enable the capture of baseline information and provide an indicator of the health status of the population [33]. Repeated surveys would enable the evaluation of trends and changes over time at the population level. Although cross-sectional analysis of these data limits interpretations on causality, it can provide good indications of the major determinants of obesity in that population. For example, Aburto et al. compared overweight and obese children with non-overweight children and observed a strong association between dietary energy density and body mass status [34]. An analysis of data from the National Health and Nutrition Examination Survey (NHANES) showed that among children and adolescents, replacement of sugar-sweetened beverages with water was associated with reductions in total energy intake for all groups studied, a reduction not negated by compensatory increases in intake of other foods or beverages [35]. Other analyses using population surveys can also be implemented, for example to determine the adequacy of the diet with respect to national recommendations.

The scientific value of *prospective epidemiological cohorts* has been solidly established for evaluating exogenous and endogenous exposures in relation to change in weight and obesity. Their primary advantage is the ability to measure exposures before the onset of obesity. Cohorts are also instrumental in spurring mechanistic and translational research. One example is the investigation by Mozaffarian et al. of the relationship between changes in lifestyle factors and weight change at 4-year intervals over a 20-year period in a large cohort of men and women in the USA. The study identified food items and lifestyle factors that were highly associated with weight change within each 4-year period; 4-year weight gain was most strongly associated with the intake of potato chips, potatoes, sugar-sweetened beverages, unprocessed red meats, and processed meats, and was inversely associated with the intake of vegetables, whole grains, fruits, nuts, and yogurt. Physical activity was independently associated with weight loss, whereas alcohol consumption, sleep duration (> 8 hours), and television watching were associated with weight gain [36]. A further analysis of the relationship between change of diet quality indexes and concurrent weight change showed that improvement of diet quality was associated with less weight gain [37].

Several cohort studies in high-income countries have shown an impact of healthy dietary patterns on obesity [38] (see Chapter 14). In a 4-year follow-up to an RCT for weight loss, subjects assigned to the Mediterranean diet during the RCT were more likely to maintain weight loss (during the 2-year trial) compared with subjects assigned to the low-fat diet or the low-carbohydrate diet. This finding suggests that compliance with the Mediterranean diet may be easier in the long term [39]. A recent analysis of the EPIC study using biomarkers of highly processed foods, which are increasingly consumed worldwide, reported that increasing blood levels of industrial trans-fatty acids were associated with an increased risk of weight gain and a decreased likelihood of weight loss, particularly among women; this might be particularly relevant in the context of LMICs [40].

Cohort studies conducted in LMICs would be valuable resources for understanding the impact of the nutrition and lifestyle transition on obesity. Some longitudinal studies have already been initiated in LMICs, for instance those included in the Consortium of Health-Orientated Research in Transitioning Societies (COHORTS) [41] and the Mexican Teachers' Cohort (MTC) [42]. Building on these continuing initiatives may prove informative and cost-efficient. Data from the MTC showed that women with a dietary pattern characterized by high intakes of carbohydrates, sweet drinks, and refined foods had a higher risk of having a larger body shape silhouette and higher BMI, whereas women with a dietary pattern characterized by high intakes of fruits, vegetables, grains, and nuts had a lower risk [43]. This finding emphasizes the need for public health interventions that improve access to healthy diets, healthy food choices in the workplace, and ways of limiting consumption of beverages with a high sugar content and of highly processed foods, particularly those rich in refined starches.

Research suggests that obesity and chronic diseases in adulthood may be traced back to exposures that occurred during fetal development, childhood, and adolescence. Therefore, cohort *studies covering the whole life-course*, focusing on critical windows of exposure and the time course of exposure to disease (birth cohorts, adolescent cohorts, and young adult cohorts), should be considered. Of particular interest are multicentre cohorts and intergenerational cohorts, which would create resources to enable research on the interplay between genetics, lifestyle, and the environment. For example, in the Avon Longitudinal Study of Parents and Children (ALSPAC), increased intake of energy-dense, nutrient-poor foods during childhood (mainly due to a rise in free [added] sugars) was associated with obesity development, and diets with higher energy density were associated with increased fat mass [44]. Most relevant to LMICs is the observation that children who were stunted in infancy are more likely to be relatively fat at puberty at the same BMI, compared with children who were not stunted in infancy [45]. Poor maternal prenatal dietary intakes of energy, protein, and micronutrients have been shown to be associated with an increased risk of adult obesity in the offspring, and a high-protein diet during the first 2 years of life was also associated with increased obesity later in life, whereas exclusive breastfeeding was associated with a lower risk of later obesity [46]. Results from a cohort study conducted in Mexico showed that children exclusively or predominantly breastfed for 3 months or longer had lower adiposity at age 4 years [47].

Conclusions

Obesity is a state of excess adiposity and is a consequence of sustained positive energy balance over time. Measurements of energy intake, expenditure, and balance are difficult. Measurements of anthropometric indices such as BMI or WC, although imperfect, are useful markers of or proxies for adiposity or visceral adiposity. Establishing repeated population-based measurements of such anthropometric measures, together with estimates of the macronutrient composition of diets, food consumption patterns, and estimates of physical activity and time spent sedentary (preferably with objective measures, such as accelerometers, used in subsamples) will help track changes in the population levels of overweight and obesity, and point to the key nutritional drivers.

Most research has tended to focus on Caucasians, but these findings may not be generalizable to other populations because of differences in age structure, genotype, body composition, lifestyle, culture, religion, and socioeconomic status. Detailed analyses that consider differences in genetic and environmental factors and gene–environment interactions between populations, and take account of the whole life-course, are required to elucidate these complex relationships. Importantly, measures of obesity prevention and control in LMICs will benefit if they are evaluated in the context within which they will be implemented.

Key points

- Obesity is a state of excess adiposity and is a consequence of sustained positive energy balance over time.
- Short-term experimental studies show that among men eating ad libitum diets of different energy density, the higher the level of activity, the less likely was positive energy balance, and at any level of activity, higher energy density led to more positive energy balance.
- In long-term experiments, intake of free sugars or sugar-sweetened beverages was a determinant of body weight. The change in body fatness when intake is modified appears to be mediated via changes in energy intakes.
- Studies in children and adolescents show that consumption of sugar-sweetened beverages results in increases in BMI, mainly by inadequate energy compensation (degree of reduction in intake of other foods and drinks).
- In weight-loss trials, low-carbohydrate interventions led to significantly greater weight loss than did low-fat interventions. Little attention has been paid to the effects of different types of carbohydrate.
- Cohort studies with repeated measurements have identified unhealthy dietary patterns and lifestyle factors associated with weight gain over time, and healthy dietary patterns have been shown to decrease the risk of obesity.
- Exposures during fetal life and early childhood may affect the risk of obesity.

Research needs

- Data from LMICs are limited, and further research is needed in these countries.
- Population surveys, etiological and population intervention studies, and implementation research are all important to develop the evidence base to tackle the rise in the prevalence of obesity associated with changing dietary and physical activity patterns.
- Research needs to take account of differences in age structure, genotype, body composition, lifestyle, culture, religion, and socioeconomic status. In this context, measurements of anthropometric indices such as BMI or WC are useful markers of or proxies for adiposity.
- Measures of obesity prevention and control in LMICs will benefit if they are evaluated in the context within which they will be implemented.

References

1. Anderson AS, Key TJ, Norat T, Scoccianti C, Cecchini M, Berrino F, et al. (2015). European Code against Cancer 4th Edition: obesity, body fatness and cancer. Cancer Epidemiol. 39(Suppl 1):S34–45. http://dx.doi.org/10.1016/j.canep.2015.01.017 PMID:26205840

2. World Cancer Research Fund/American Institute for Cancer Research (2009). Policy and action for cancer prevention: food, nutrition, physical activity: a global perspective. Washington (DC), USA: American Institute for Cancer Research. Available from: www.wcrf-hk.org/sites/default/files/Policy_Report.pdf.

3. Blundell JE, Gibbons C, Caudwell P, Finlayson G, Hopkins M (2015). Appetite control and energy balance: impact of exercise. Obes Rev. 16(Suppl 1):67–76. http://dx.doi.org/10.1111/obr.12257 PMID:25614205

4. Ello-Martin JA, Ledikwe JH, Rolls BJ (2005). The influence of food portion size and energy density on energy intake: implications for weight management. Am J Clin Nutr. 82(1 Suppl):236S–41S. PMID:16002828

5. Prentice AM (1998). Manipulation of dietary fat and energy density and subsequent effects on substrate flux and food intake. Am J Clin Nutr. 67(3 Suppl):535S–41S. PMID:9497166

6. WHO (2000). Obesity: preventing and managing the global epidemic: report of a WHO consultation. Geneva, Switzerland: World Health Organization (WHO Technical Report Series, No. 894). Available from: http://www.who.int/nutrition/publications/obesity/WHO_TRS_894/en/.

7. WHO (2011). Waist circumference and waist–hip ratio: report of a WHO Expert Consultation, Geneva, 8–11 December 2008. Geneva, Switzerland: World Health Organization. Available from: http://www.who.int/nutrition/publications/obesity/WHO_report_waistcircumference_and_waisthip_ratio/en/.

8. van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Droop A, Bakker CJ (1993). Waist-hip ratio is a poor predictor of changes in visceral fat. Am J Clin Nutr. 57(3):327–33. PMID:8438766

9. Segal KR, Van Loan M, Fitzgerald PI, Hodgdon JA, Van Itallie TB (1988). Lean body mass estimation by bioelectrical impedance analysis: a four-site cross-validation study. Am J Clin Nutr. 47(1):7–14. PMID:3337041

10. Willett K, Jiang R, Lenart E, Spiegelman D, Willett W (2006). Comparison of bioelectrical impedance and BMI in predicting obesity-related medical conditions. Obesity (Silver Spring). 14(3):480–90. http://dx.doi.org/10.1038/oby.2006.63 PMID:16648620

11. Heymsfield SB, Lohman TG, Wang Z, Going SB, editors (2005). Human body composition. 2nd ed. Champaign (IL), USA: Human Kinetics.

12. McCrory MA, Gomez TD, Bernauer EM, Molé PA (1995). Evaluation of a new air displacement plethysmograph for measuring human body composition. Med Sci Sports Exerc. 27(12):1686–91. http://dx.doi.org/10.1249/00005768-199512000-00016 PMID:8614326

13. Glickman SG, Marn CS, Supiano MA, Dengel DR (2004). Validity and reliability of dual-energy X-ray absorptiometry for the assessment of abdominal adiposity. J Appl Physiol (1985). 97(2):509–14. http://dx.doi.org/10.1152/japplphysiol.01234.2003 PMID:15075304

14. Bandera EV, Maskarinec G, Romieu I, John EM (2015). Racial and ethnic disparities in the impact of obesity on breast cancer risk and survival: a global perspective. Adv Nutr. 6(6):803–19. http://dx.doi.org/10.3945/an.115.009647 PMID:26567202

15. Karra E, O'Daly OG, Choudhury AI, Yousseif A, Millership S, Neary MT, et al. (2013). A link between FTO, ghrelin, and impaired brain food-cue responsivity. J Clin Invest. 123(8):3539–51. http://dx.doi.org/10.1172/JCI44403 PMID:23867619

16. Guo SS, Chumlea WC (1999). Tracking of body mass index in children in relation to overweight in adulthood. Am J Clin Nutr. 70(1):145S–8S.

17. Brisbois TD, Farmer AP, McCargar LJ (2012). Early markers of adult obesity: a review. Obes Rev. 13(4):347–67. http://dx.doi.org/10.1111/j.1467-789X.2011.00965.x PMID:22171945

18. Fogelholm M, Kukkonen-Harjula K (2000). Does physical activity prevent weight gain – a systematic review. Obes Rev. 1(2):95–111. http://dx.doi.org/10.1046/j.1467-789x.2000.00016.x PMID:12119991

19. Stubbs RJ, Harbron CG, Murgatroyd PR, Prentice AM (1995). Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. Am J Clin Nutr. 62(2):316–29. PMID:7625338

20. Salas-Salvadó J, Bulló M, Babio N, Martínez-González MA, Ibarrola-Jurado N, Basora J, et al.; PREDIMED Study Investigators (2011). Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of the PREDIMED-Reus nutrition intervention randomized trial. Diabetes Care. 34(1):14–9. http://dx.doi.org/10.2337/dc10-1288 PMID:20929998

21. Du H, van der A DL, Ginder V, Jebb SA, Forouhi NG, Wareham NJ, et al. (2009). Dietary energy density in relation to subsequent changes of weight and waist circumference in European men and women. PLoS One. 4(4):e5339. http://dx.doi.org/10.1371/journal.pone.0005339 PMID:19396357

22. Te Morenga L, Mallard S, Mann J (2013). Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 346:e7492. http://dx.doi.org/10.1136/bmj.e7492 PMID:23321486

23. Lennerz BS, Alsop DC, Holsen LM, Stern E, Rojas R, Ebbeling CB, et al. (2013). Effects of dietary glycemic index on brain regions related to reward and craving in men. Am J Clin Nutr. 98(3):641–7. http://dx.doi.org/10.3945/ajcn.113.064113 PMID:23803881

24. United Kingdom Scientific Advisory Committee on Nutrition (2015). Carbohydrates and health. London, UK: The Stationery Office. Available from: https://www.gov.uk/government/publications/sacn-carbohydrates-and-health-report.

25. Mirza NM, Palmer MG, Sinclair KB, McCarter R, He J, Ebbeling CB, et al. (2013). Effects of a low glycemic load or a low-fat dietary intervention on body weight in obese Hispanic American children and adolescents: a randomized controlled trial. Am J Clin Nutr. 97(2):276–85. http://dx.doi.org/10.3945/ajcn.112.042630 PMID:23255569

26. Swinburn BA, Caterson I, Seidell JC, James WP (2004). Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr. 7(1A):123–46. PMID:14972057

27. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO (2000). The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. Int J Obes Relat Metab Disord. 24(12):1545–52. http://dx.doi.org/10.1038/sj.ijo.0801453 PMID:11126204

28. Harvie MN, Pegington M, Mattson MP, Frystyk J, Dillon B, Evans G, et al. (2011). The effects of intermittent or continuous energy restriction on weight loss and metabolic disease risk markers: a randomized trial in young overweight women. Int J Obes (Lond). 35(5):714–27. http://dx.doi.org/10.1038/ijo.2010.171 PMID:20921964

29. Chen L, Appel LJ, Loria C, Lin PH, Champagne CM, Elmer PJ, et al. (2009). Reduction in consumption of sugar-sweetened beverages is associated with weight loss: the PREMIER trial. Am J Clin Nutr. 89(5):1299–306. http://dx.doi.org/10.3945/ajcn.2008.27240 PMID:19339405

30. Tobias DK, Chen M, Manson JE, Ludwig DS, Willett W, Hu FB (2015). Effect of low-fat diet interventions versus other diet interventions on long-term weight change in adults: a systematic review and meta-analysis. Lancet Diabetes Endocrinol. 3(12):968–79. http://dx.doi.org/10.1016/S2213-8587(15)00367-8 PMID:26527511

31. Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, et al.; Dietary Intervention Randomized Controlled Trial (DIRECT) Group (2008). Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. N Engl J Med. 359(3):229–41. http://dx.doi.org/10.1056/NEJMoa0708681 PMID:18635428

32. Allison KR, Dwyer JJM, MacGillivray A, Hawrychuk S; Ontario Agency for Health Protection and Promotion (2011). A review of workplace and community interventions for adults: effects of physical activity, eating behaviours and BMI – risk factors for diabetes. Toronto, Canada: Queen's Printer for Ontario. Available from: https://www.publichealthontario.ca/en/eRepository/A Review of Workplace and Community Interventions for Adults Effects on physical activity, eating behaviours, and BMI.pdf.

33. Romero-Martínez M, Shamah-Levy T, Franco-Núñez A, Villalpando S, Cuevas-Nasu L, Gutiérrez JP, et al. (2013). National Health and Nutrition Survey 2012: design and coverage [in Spanish]. Salud Publica Mex. 55(Suppl 2):S332–40. PMID:24626712

34. Aburto TC, Cantoral A, Hernández-Barrera L, Carriquiry AL, Rivera JA (2015). Usual dietary energy density distribution is positively associated with excess body weight in Mexican children. J Nutr. 145(7):1524–30. http://dx.doi.org/10.3945/jn.114.206359 PMID:25926409

35. Wang YC, Ludwig DS, Sonneville K, Gortmaker SL (2009). Impact of change in sweetened caloric beverage consumption on energy intake among children and adolescents. Arch Pediatr Adolesc Med. 163(4):336–43. http://dx.doi.org/10.1001/archpediatrics.2009.23 PMID:19349562

36. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB (2011). Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 364(25):2392–404. http://dx.doi.org/10.1056/NEJMoa1014296 PMID:21696306

37. Fung TT, Pan A, Hou T, Chiuve SE, Tobias DK, Mozaffarian D, et al. (2015). Long-term change in diet quality is associated with body weight change in men and women. J Nutr. 145(8):1850–6. http://dx.doi.org/10.3945/jn.114.208785 PMID:26084363

38. United States Department of Agriculture (2015). Scientific report of the 2015 Dietary Guidelines Advisory Committee. Washington (DC), USA: Department of Agriculture and Department of Health and Human Services. Available from: https://health.gov/dietaryguidelines/2015-scientific-report/.

39. Schwarzfuchs D, Golan R, Shai I (2012). Four-year follow-up after two-year dietary interventions. N Engl J Med. 367(14):1373–4. http://dx.doi.org/10.1056/NEJMc1204792 PMID:23034044

40. Chajès V, Biessy C, Ferrari P, Romieu I, Freisling H, Huybrechts I, et al. (2015). Plasma elaidic acid level as biomarker of industrial trans fatty acids and risk of weight change: report from the EPIC study. PLoS One. 10(2):e0118206. http://dx.doi.org/10.1371/journal.pone.0118206 PMID:25675445

41. Richter LM, Victora CG, Hallal PC, Adair LS, Bhargava SK, Fall CH, et al.; COHORTS Group (2012). Cohort Profile: the Consortium of Health-Orientated Research in Transitioning Societies. Int J Epidemiol. 41(3):621–6. http://dx.doi.org/10.1093/ije/dyq251 PMID:21224276

42. Lajous M, Ortiz-Panozo E, Monge A, Santoyo-Vistrain R, García-Anaya A, Yunes-Díaz E, et al. (2017). Cohort Profile: the Mexican Teachers' Cohort (MTC). Int J Epidemiol. 46(2):e10. http://dx.doi.org/10.1093/ije/dyv123 PMID:26337903

43. Romieu I, Escamilla-Núñez MC, Sánchez-Zamorano LM, Lopez-Ridaura R, Torres-Mejía G, Yunes EM, et al. (2012). The association between body shape silhouette and dietary pattern among Mexican women. Public Health Nutr. 15(1):116–25. http://dx.doi.org/10.1017/S1368980011001182 PMID:21875454

44. Emmett PM, Jones LR (2015). Diet, growth, and obesity development throughout childhood in the Avon Longitudinal Study of Parents and Children. Nutr Rev. 73(Suppl 3):175–206. http://dx.doi.org/10.1093/nutrit/nuv054 PMID:26395342

45. Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, et al.; Maternal and Child Undernutrition Study Group (2008). Maternal and child undernutrition: consequences for adult health and human capital. Lancet. 371(9609):340–57. http://dx.doi.org/10.1016/S0140-6736(07)61692-4 PMID:18206223

46. Yang Z, Huffman SL (2013). Nutrition in pregnancy and early childhood and associations with obesity in developing countries. Matern Child Nutr. 9(Suppl 1):105–19. http://dx.doi.org/10.1111/mcn.12010 PMID:23167588

47. Ramirez-Silva I, Rivera JA, Trejo-Valdivia B, Martorell R, Stein AD, Romieu I, et al. (2015). Breastfeeding status at age 3 months is associated with adiposity and cardiometabolic markers at age 4 years in Mexican children. J Nutr. 145(6):1295–302. http://dx.doi.org/10.3945/jn.114.198366 PMID:25926414