

Module 23.2

Obesity: Assessment and Prevention

Assist. Professor Michael Chourdakis, BSc, Msc, MPH PhD MD RD
Dept. Of Medicine
School of Health Sciences
Aristotle University of Thessaloniki, Greece

Learning Objectives

- Prevalence of overweight / obesity;
- Early life strategies in the prevention of obesity;
- Obesity paradox;
- How lifestyle modification can prevent obesity;
- Effect of nutrients on obesity.

Contents

1. Where do we stand in regard to obesity?
 - 1.1 Introduction
 - 1.2 Prevalence of overweight / obesity
 - 1.2.1 Adult populations
 - 1.2.2 Children and adolescents
 - 1.3 Mortality risk in body mass index (BMI) trajectories among older populations
2. Definition of obesity
 - 2.1 Definition, usages and limitations of the use of Body Mass Index
 - 2.2 Assessing obesity
 - 2.2.1 Bioelectrical impedance analysis (BIA) in obesity
 - 2.2.2 DEXA in obesity
 - 2.2.3 CT-scanning in obesity
 - 2.2.4 Magnetic Resonance Imaging (MRI) in obesity
 - 2.2.5 REE in obesity
 - 2.2.6 More techniques
 - 2.3 Overcoming the limit of anthropometric assessment
3. Obesity paradox
 - 3.1 "Optimal" BMI and obesity
 - 3.2 Different patient groups
 - 3.2.1 Elderly
 - 3.2.2 Cardiovascular disease patients
 - 3.2.3 ICU patients
 - 3.2.4 Dialysis patients
 - 3.2.5 Cancer patients
 - 3.2.6 Patients with peripheral vascular disease
 - 3.2.7 Hospitalized individuals
 - 3.3 Explanatory for OP
4. Obesity prevention
 - 4.1 Early prevention of obesity

- 4.2 Obesity and early life strategies
- 4.3 Strategies for obesity prevention in children and adolescents
- 5. Lifestyle changes to prevent overweight/obesity and promote weight maintenance
 - 5.1 Body weight gain vs. body weight maintenance
 - 5.2 Effect of the macronutrients
 - 5.3 Fat intake
 - 5.4 Dairy Products
 - 5.5 Dietary Sugars
 - 5.6 Dietary salt
 - 5.7 Glycaemic index
 - 5.8 Lifestyle adaptation
 - 5.9 Hospital and community- based health services
- 6. Conclusions
- 7. Appendix
 - 7.1 Baseline assessment tool for NICE guideline on Obesity (update) (CG189)
 - 7.2 School based strategies (112)
- 8. References

Key Messages

- Obesity is one the major health problems of today and is associated with increased risk of several diseases;
- “Obesity paradox” (OP) is about an overall prognosis that is no worse and may even be better in some groups than non-obese patients;
- The OP could be explained by the fact that current classifications of obesity based on BMI may place together, in the same category, subjects with very different clinical and biochemical characteristics;
- Generally breastfed infants tend to have a lower BMI than formula-fed infants and behavioural and hormonal mechanisms may explain this difference;
- Sedentary behaviour (viewing television, playing video games, doing cognitive work, and listening to music) and reduced overall physical activity along with shorter sleep duration promote the overconsumption of dietary macronutrients leading to obesity;
- Physical activity or exercise in a sufficient dose seems to better facilitate long-term maintenance of new lower body weight;
- Low glycaemic index diets do not show any consistent effect on maintenance of lower weight after 12 months;
- A negative energy balance is the crucial parameter in regard to weight loss and prevention of obesity.

1. Where Do We Stand in Regard to Obesity?

1.1 Introduction

Being underweight, overweight, or obese during childhood and adolescence is associated with adverse health consequences throughout the life-course. In particular, overweight and obesity are global health problems contributing to an ever-increasing non-communicable disease burden. The expanded lifespan of the aging population imposes a challenge on the continuous increase of chronic disease. Obesity, in addition to having disease-specific effects, may accelerate the rate of aging affecting all aspects of physiology (e.g. cellular and molecular processes) and thus shortening life span and health span (1). Chronic overfeeding (**Table 1**) seems to be the most important factor leading to obesity (2). There are additional causal factors, such as genetic disposition, sedentary lifestyle and impairment of mechanisms that could protect an individual against excessive fat storage after overfeeding. Among such mechanisms are postprandial thermogenesis, non-exercise related thermogenesis, physical activity, composition of muscle fibres, thyroid hormone activity, etc. In addition, risk factors for obesity include parental fatness, social factors, birth weight, timing or rate of maturation, physical activity, dietary factors and other behavioural or psychological factors.

Furthermore, obesity may be explained by an interaction between genetic and environmental factors (e.g. diet) (3, 4). In addition, some individuals are more prone to gain body weight when exposed to a given diet, because of susceptibility determined by a specific genetic background. Research regarding the influence of genetics on obesity has expanded in the recent years; however sufficiently clear results are not yet available to complete our understanding of the interactions that impact on body weight and weight management (*for more information on the impact of genetics on obesity, see **Module 23.1***).

Table 1
The consequences of chronic overfeeding

Metabolic and organ changes connected with chronic overnutrition
<ul style="list-style-type: none">• Production of fat, steatosis of liver, muscles and pancreas• Obesity• Excessive secretion of fat cell hormones, fatty acids and cytokines• Insulin resistance• Metabolic syndrome incl. type 2 diabetes, hypertension and hyperlipidaemia• Impaired coagulation and fibrinolysis – thrombotic complications• Sterility and hormonal disorders• Sleep apnoea syndrome• Respiratory failure• Impaired regeneration and wound healing• Infections• Atherosclerosis, endothelial dysfunction, cardiovascular diseases• Oxidative stress• Obesity related tumours• Diabetes mellitus• Depression

Due to the high prevalence of overweight and obesity in the developed and developing world (see 1.2) weight control is a national and global priority. The steep rise in the incidence of obesity in the developed world, when the genetic background has not changed, is due to excess intake of energy and diminishing levels of activity. It is also probable that the thrifty gene hypothesis plays a part (5). It is obvious that those alive now are the descendants of those who could cope with shortage of food (during wars or famine), due to their highly efficient storage and energy conserving mechanisms. Nowadays, with shortage of food no longer a problem for developed and developing countries, humans easily tend to develop obesity and suffer from its consequences. In addition, this is due mainly to the fact that mammals have a variety of mechanisms to adapt to low intake of food (e.g. including reduced energy expenditure), but mechanisms to counteract excess energy intake are limited to increased physical activity. Nevertheless, it has been shown in studies both in animal models and humans, that after a period of excess intake of energy, body weight tends to return to its usual value.

Additionally, the type of obesity seems to be important, since the android type of obesity leads to significantly more frequent metabolic complications (e.g. development of diabetes and atherosclerosis). A ratio between waist and hip circumferences (WHR) of >1 can be used as an index of android obesity, although waist circumference alone correlates better with the amount of visceral fat. Therefore the risk of metabolic complications, i.e. the predisposition to develop android type of obesity, is related to the waist circumference and is usually classified as mild or severe.

Nonetheless, in addition to the weight or BMI value the level of activity seems also to be closely related to mortality for obese patients. In sumo wrestlers, who are chronically overfed while highly active, their increased muscle mass gets quickly replaced by adipose tissue when they quit exercising. Therefore it is without doubt better to be obese but physically fit (fit-fat) rather than slim but unfit (unfit-lean)(6).

Although obese individuals use fat as their main source of energy, this is also influenced by the pattern of substrate intake. Obese subjects have higher absolute energy expenditure due to their increased lean mass and the work of moving their heavier bodies, although oxygen consumption per kg body weight may be lower than in normal weight subjects, since adipose tissue has a lower metabolic rate than lean tissue. Some obese subjects also have lower diet-induced thermogenesis (*for more information on this see Topic 3*).

1.2 Prevalence of Overweight / Obesity

1.2.1 Adult Populations

During the last four decades, the percentage of obese people has quadrupled in males and doubled in females, reaching a global epidemic scale as the biggest health-related issue in developed and developing countries (7). The prevalence of overweight and obesity has increased dramatically in the United States and around the world (8). More than one-third of adults (35.5% men and 35.8% women) in the United States are obese, although the prevalence remained stable between 2003-2004 and 2009-2010 and recent data suggest a slowing or leveling off of these trends (8). In the USA the increase in the prevalence of obesity previously observed has not appeared to be continuing at the same rate over the past 10 years, particularly for females and possibly for males (9). However, the number of obese people in the European Union (EU) continues to increase (**Fig.1**). The dynamics of obesity growth in Europe are greater for males than for females (3.09% per year vs. 1.92% per year). With the growth rate remaining at the estimated level, in 2030 there will

probably be more obese males (38.1%) than females (32.7%) in Poland, and in the EU 36.6% and 32.0%, respectively (15). Therefore, obesity prevalence remains high and thus it is important to continue surveillance (10).

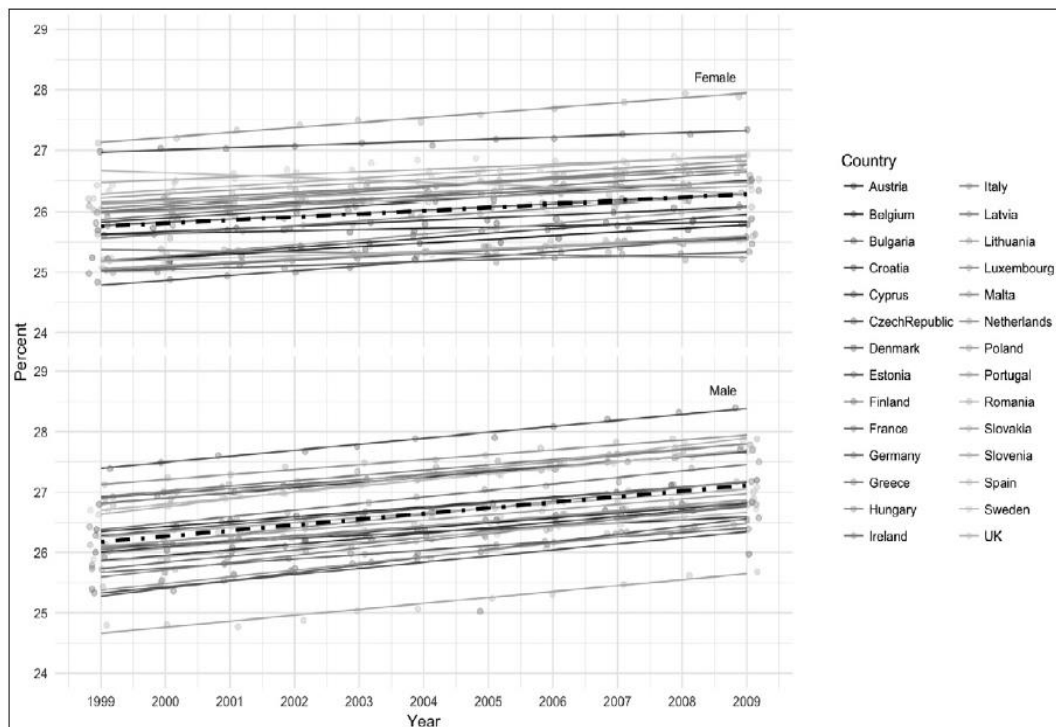


Fig. 1 Assessment of epidemiological obesity among adults in EU countries (7)

1.2.2 Children and Adolescents

Childhood and adolescent overweight and obesity are a major health problem not only in the United States and other western countries but also in developing countries throughout the world, and are thus a worldwide public health concern (**Fig. 2**). Around 30% of American adolescents and 22–25% of European adolescents are overweight or obese (11). The National Health and Nutrition Examination Surveys (NHANES) indicate that childhood obesity in the United States has approximately doubled during the past three decades and adolescent obesity has more than tripled during the same period (12). Mean BMI and prevalence of obesity increased worldwide in children and adolescents from 1975 to 2016, with the rate of change in mean BMI moderately correlated with that of adults until around 2000, but only weakly correlated afterwards (13). In the USA a third of the country's children are overweight or obese. Some low-income and middle-income countries have reported similar or more rapid rises in child obesity (14).

In the last years, the trend in children's and adolescents' mean BMI has plateaued, albeit at high levels, in many high-income countries since around 2000 (12), but has accelerated in east, south, and southeast Asia. However, it should be noted that some epidemiological studies indicate that the prevalence of childhood obesity is continuing to increase in certain sex, age, ethnic, and socioeconomic status groups within the United States and that the current childhood obesity epidemic will contribute to an increase in the number of obese adults (15).

It seems that socioeconomic status is inversely related to obesity in most Western countries (11, 16). These inequalities in obesity and health-related behaviours stem from the unequal distribution of social and economic resources (17).

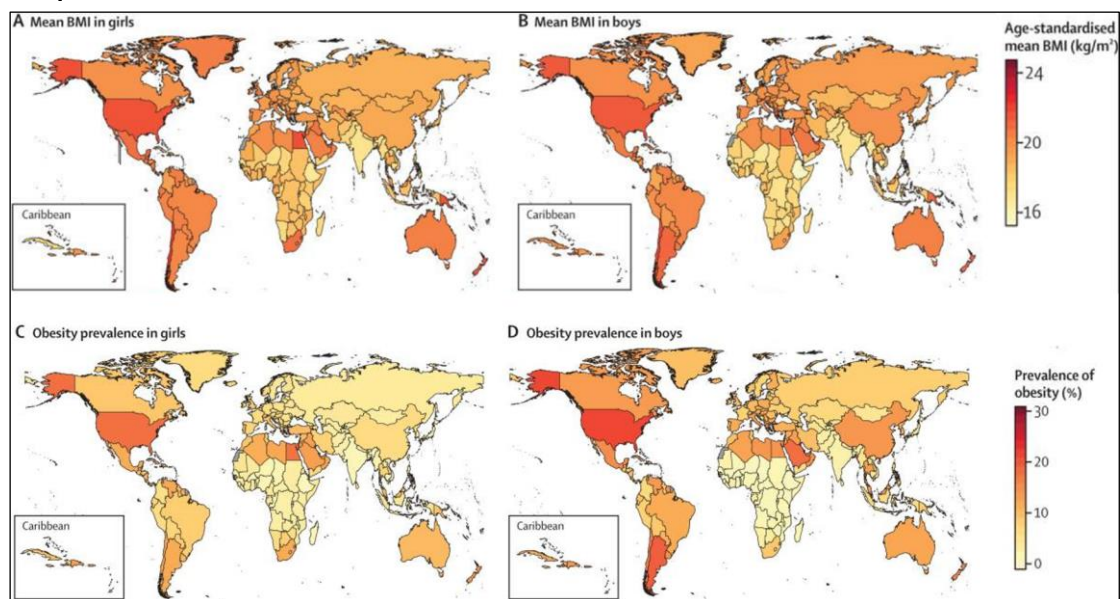


Fig. 2 from NCD Risk Factor Collaboration

1.3 Mortality Risk in Body Mass Index (BMI) Trajectories Among Older Populations

Little research has addressed the heterogeneity and mortality risk in body mass index (BMI) trajectories among older populations. Applying latent class trajectory models to 9,538 adults aged 51 to 77 years from the US Health and Retirement Study (1992-2008), 6 latent BMI trajectories were defined (normal weight downward, normal weight upward, overweight stable, overweight obesity, class I obese upward, and class II/III obese upward). People in the overweight stable trajectory had the highest survival rate, followed by those in the overweight obesity, normal weight upward, class I obese upward, normal weight downward, and class II/III obese upward trajectories. Further analysis suggested that BMI trajectories were more predictive of mortality risk than was static BMI status. This suggests that trajectories of increasing obesity past 51 years of age pose a substantive threat to future gains in life expectancy (18). Many previous studies have found that weight gain was associated with a higher mortality risk in overweight/obese individuals (18). These findings indicate the associations of weight gain with mortality risk depend on baseline BMI status.

Paradoxically, it appears that weight loss, even of a small degree (a decrease of approximately 1 BMI unit), in a person in the normal weight category at 51 years of age can potentially have a significant deleterious effect on health. Many studies have found that even small weight losses can exert a harmful effect on survival, regardless of the initial BMI level (19).

Persons in the underweight and class II/III obese categories had increased mortality risk compared with the reference category (overweight). Normal weight and class I obesity were not associated with significant increases in mortality risk. These findings are consistent with those from the analysis by Mehta and Chang (20).

2. Definition of Obesity

2.1 Definition, Usages and Limitations of the Use of Body Mass Index

The definition of obesity of the WHO identifies it as “a condition in which percentage body fat (PBF) is increased to an extent in which health and well-being are impaired” (21). It is clear that it is defined by the expansion of the adipose tissue, rather than defining it solely on the basis of the increase in body weight. However, as the fat mass is not easily measurable and there is no consensus regarding normal levels of body fat mass, this definition of obesity is not easy to use in everyday life. Therefore, a classification of overweight and obesity is being done most commonly according to the “Body Mass Index” (BMI). The BMI is a simple mathematical index, which aims to estimate standard weight versus underweight and overweight status, taking into account the actual weight and height of a person. The BMI equals the weight in kilograms divided by the square of the height in meters. The major drawback of the BMI is that it does not take into account the body composition, therefore it is a simple index, but provides only a crude estimation of obesity and undernutrition. The WHO categorized groups of BMI to define underweight, normal weight, overweight and obesity (in 3 grades) as follows (**Table 2**). Overall, the BMI is a practical tool for the rapid, age-, size- and gender-independent estimation of the nutritional status.

A BMI ≥ 30 kg/m² does not necessarily mean that obesity is present. Also a significant increase in muscle mass, as observed in the bodybuilder, or a significant increase in body water, e.g. oedema associated with heart or kidney failure or ascites in hepatic insufficiency may result in increased BMI. A simple measure for the assessment of the visceral fat depot is the measurement of the waist circumference (WCF), which is more difficult in the obese by the fact that no waist is recognizable. If the WCF is ≥ 88 cm in women and ≥ 102 cm in men, abdominal obesity is present (22). The measurement of WCF in overweight, and possibly also grade I obesity, allows a better estimation of metabolic risk than BMI alone, whereas at higher obesity waist circumference and metabolic risk are usually elevated (23). Therefore, it is recommended to measure and document the percentage Body Fat (% BF) when BMI is 25-35 kg/m² in addition to BMI.

Table 2
Classification of overweight and obesity

Category	BMI [kg/m²]	Risk for associated diseases
Very severely underweight	less than 15	very high
Severely underweight	15.0 to 15.9	elevated
Underweight	15.9 to 18,5	low
Normal weight	18.5 – 24.9	low
Overweight	25 – 29.9	slightly elevated
Obesity grade I	30 – 34.9	elevated
Obesity grade II	35 – 39.9	high
Obesity grade III	≥ 40	very high

2.2 Assessing Obesity

BMI is an acceptable tool for screening for obesity and tracking weight over time. However, in obese people BMI and %BF may not be strongly correlated and since BMI does not give

information about different body compartments (fat-free mass and fat mass) or identify the distribution of fat, it should not be used to further assess body composition in the obese beyond just classifying the level of obesity (24).

The use of BMI for obese subjects, or those of normal weight, with or without metabolic syndrome is a longstanding controversy. Research has overcome this paradox with the introduction to diagnosis of body composition, the evaluation of visceral fat, metabolic indices and genetic predisposition (25). It is well known, that the expansion of visceral and ectopic fat attribute a cardiovascular and metabolic risk that exceeds that of BMI. In clinical practice, measurements of visceral fat, adiposity, body composition and genetic/metabolic factors should be implemented to improve risk assessment and develop effective preventive and therapeutic strategies for high-risk obesity (26).

In particular, BMI results are limited and often unfit to discover hidden fat (27). In fact, as reported using BMI cutoff values for the diagnosis of obesity, approximately half of people with excess fat are missed, due to a low sensitivity of identifying adiposity (28). Therefore, it is necessary to use methods that accurately evaluate the amount of body fat (BF), fat free mass (FFM), skeletal muscle mass (29), the metabolically active body cell mass (BCM), bone mass, and the total amount of body water with the distribution of water compartments in large population samples (30).

2.2.1 Bioelectrical Impedance Analysis (BIA) in Obesity

BIA measures the body impedance using electrodes that are connected from one leg to the other, or to the arm, to form a circuit for the current to pass through. The impedance measure is used to predict total body water (TBW) and fat-free mass (FFM) and fat mass is calculated from the difference between weight and FFM (*for more information see Topic 3*).

The Third National Health and Nutrition Examination Survey (NHANES III) tested the accuracy of BMI for diagnosing obesity in an adult general population using data from 13,601 individuals (27). The results from using BIA to calculate BF and BMI $>30 \text{ kg/m}^2$ to define obesity, BMI had a very high specificity (97%) but poor sensitivity (42%) in the detection of obesity.

Single frequency BIA (SF-BIA) should not be used for body composition assessment in the obese because the theory that the human body is a single cylinder with constant resistivity which underlies the extrapolations made in BIA analysis cannot be applied to the obese (31).

Segmental BIA (tetra- and eight-polar-BIA) recognizes that the human body is complex in shape and combines several impedance measures together for a more accurate assessment. However, segmental-BIA has been found to significantly overestimate %BF in obese adults (31).

Multi frequency-BIA (MF-BIA) allows multiple frequencies to assess fluid distribution; low electric frequencies (e.g. 1 or 5 kHz) measure extra cellular water (ECW) and high frequencies (e.g. 100, 200, or 500 kHz) measure total body water (TBW). MF-BIA has been found to overestimate % BF in the overweight and obese groups (31), significantly underestimate both total and truncal fat in obese women, and offer accurate estimates of TBW and ECW in women with a BMI up to approx. 48 kg/m^2 (32).

Fatness-specific BIA equations, developed by Segal et al. have been validated for use in the obese (33) and more recent prediction equations specifically developed for the obese population are more accurate for prediction of BF. Overall, it seems that more research needs to be conducted to determine an agreement on the use of MF-BIA in the obese before recommendations can be made.

2.2.2 DEXA in Obesity

DEXA is a scanning technique that measures bone mineral, fat tissue, and fat-free soft tissue. Participants must lie completely still on the DEXA machine platform while X-rays at a high and low energy levels are passed over the body. DEXA can be used to determine abdominal obesity and is useful in predicting intraabdominal fat in obese males and females (34).

In addition, DEXA can also assess regional body composition, in that way allowing for the identification of gynoid or android obesity.

Limitations of DEXA include its high cost, need for trained technicians, and dedicated facilities. In obese participants, the DEXA scan is sensitive to difference in body thickness resulting in an overestimation of body fat (35). Traditional DEXA scan tables can only hold up to 150 kg and the width of the scanning area, average of 60 cm, does not accommodate the severely obese.

2.2.3 CT-scanning in Obesity

CT scanning uses X-ray beams to produce cross-sectional images of the body, allowing differentiation between measured muscle mass, visceral organ volumes, and measures of visceral adipose tissue in overweight and obese patients. CT scans at the whole body level involve high radiation exposure (36).

Undoubtedly, CT and magnetic resonance imaging (MRI) are currently the best methods for analyzing regional adiposity. However, they are expensive and are usually limited to the hospital setting. Overall, the high risk, combined with high cost, make the CT scan an unattractive option for routine clinical use for assessing body composition in all participant populations. It is also important to note that, single abdominal slice images provide good estimates of total body adiposity, visceral adiposity and skeletal muscle in group studies (37), but have limited applicability at the individual level due to individual variation (34).

2.2.4 Magnetic Resonance Imaging (MRI) in Obesity

MRI is a technique of generating images from interactions between the nuclei of hydrogen atoms in the body and magnetic fields generated by the MRI machine. Protons from the various tissues in the body resonate differently. The MRI recognizes these differences, generating an image of the tissues. The generated image can be used to measure body composition and to examine regional fat distribution (24). However, while single slices may be useful for cross-sectional estimation of volumes of relevant fat tissue compartments it is important to note that single slice imaging may not be sensitive or accurate in detecting small changes in abdominal adiposity (38).

There are no known long-term side effects from MRIs so they can be used for large coverage and repeated tests (39). Use in the obese was previously limited by the size of the MRI machines, which were not able to accommodate large body sizes. The developments of open-configuration MRI scanners have helped resolve this problem. MRI is a good option for assessing body composition in the obese (24).

2.2.5 REE in Obesity

Different established equations can be used for estimating REE at the population level in both sexes. However, the accuracy for all predictive equations may be very low, particularly among females and when BMI is high, limiting their use in clinical practice (24). Recent

findings suggest that validation of new predictive equations would improve the accuracy of REE prediction, especially for severely obese subjects ($BMI > 40 \text{ kg/m}^2$) (40, 41).

2.2.6 More Techniques

The discussion about different assessment techniques such as Bioimpedance spectroscopy (BIS), Dilution technique, Total body potassium (TBP), Air Displacement Plethysmography (ADP), Hydrostatic weighing, Three-Dimensional Photonic Scanning (3DPS), Quantitative Magnetic Resonance (QMR), Near-Infrared Interactance (NII), Multi-compartment methods is beyond the scope of this chapter but extended information can be found in the excellent paper by Beechy et al (24).

2.3 Overcoming the Limit of Anthropometric Assessment

A study including 3258 Italian subjects, noted that the percentage of obesity changed depending on the criterion adopted. According to the BMI, obesity affected 32.3% of population while, according to the cut-off acceptable percentage of fat mass (adjusted for sex and age), 64% of the population was in obesity status (28). In an effort to overcome the limit of anthropometric assessment, due to heterogeneity of obesity, the Edmonton Obesity Staging System (EOSS) was applied. This is a useful tool for a clinical staging system (42). EOSS divides the population with excess adiposity on an ordinal 5-point scale, taking into account the comorbidities linked to obesity: 1) no apparent risk factors; 2) presence of obesity related subclinical risk factors; 3) presence of established obesity-related chronic disease; 4) established end-organ damage; 5) severe disabilities. The possibility of predicting mortality according to the EOSS is independent from BMI values. However, since EOSS cannot be used for a direct or indirect measure of adiposity, it represents only a prognostic system capable of integrating anthropometric indices (43). On the other hand, the European Association for the Study of Obesity (EASO) has promoted various types of actions, and has proposed the revision of the diagnostic criteria of the International Classification of Diseases ICD-11 (44). In that context, the new definition of obesity as adiposity-based chronic disease (ABCD), for which the term "adiposopathy" means the whole represented by the total quantity of fat, its distribution, and the function of adipose tissue, seemed to fit well with the EASO's purposes (45). Therefore, a diagnosis of ABCD could allow a more specific analysis of the complications caused by the dysfunctional adipose tissue, with a greater possibility of effectiveness of the intervention.

3. Obesity Paradox

3.1 "Optimal" BMI and Obesity

The classification of obesity is accepted worldwide according to the BMI and is defined as a $BMI > 30$. However, as previously discussed, BMI as a unique measurement of obesity does not reflect the whole complexity of the disease (45). Nevertheless, it can be used as an indicator of life expectancy and of the risk of most of the complications of obesity, as a high BMI is associated with various morbidities and higher mortality, notably those related to cardiovascular diseases, hypertension, diabetes mellitus, stroke, cancer (e.g. colon cancer and endometrial cancer). The optimum BMI in terms of life expectancy seems to be 20-22, while health risks of increased body weight rise progressively with a $BMI > 25$. Morbid obesity ($BMI > 40$) is considered a serious disease and such patients rarely live longer than 60 years (6). Other studies have found that a dynamic measure of weight status (weight or BMI change) is more predictive of mortality than is a static measure of

weight status (i.e., baseline BMI), especially among older adults (46, 47). An exception to this rule is posed by overweight/obese patients in the ICU and/or with renal insufficiency and some other categories (see 3.2) among whom having a higher BMI is related to longer survival, which accounts for the so called "obesity paradox" (OP).

3.2 Different Patient Groups

3.2.1 Elderly

Recent evidence noticed a shift in OP with aging (**Fig. 3**). While some studies have reported a more pronounced "obesity paradox" (OP) in older patients, others have seen diminished cardiac benefits with overweight and obesity in elderly patients with CVD. These findings suggested that a complex relationship between aging, metabolism, and heart failure severity/chronicity, may explain the shift in OP in the elderly (48). In a meta-analysis Winter et al demonstrated that the optimal BMI for people ≥ 65 years is 28 (**Fig. 3**) (49). A study including 1614 residents (69.5% females) with mean age of 83.7 ± 8.4 found, taking into account background disease, that high BMI was significantly protective. In particular mortality reductions per 1 unit increase in BMI were 9% at 6 months, 10% at 1 year, 9% at 2 years, 7% at 4 years, and 5% at 9 years ($p < 0.001$) (50).

3.2.2 Cardiovascular Disease Patients

The obesity paradox was first demonstrated in patients suffering from various cardiac ailments. The paradoxical U-shaped relationship between obesity and mortality was found in patients with acute MI, cardiac failure, and atrial fibrillation (51-53). In multiple studies, obesity measured by BMI and various other indices has been linked to the survival from heart failure (HF) (54).

Obesity is associated with an increased risk of developing cardiovascular disease (CVD), particularly HF and coronary heart disease (CHD) (55). The mechanisms through which obesity increases CVD risk involve changes in body composition that can affect haemodynamics and alter heart structure. When obesity and HF or CHD coexist, individuals with class I obesity present a more favourable prognosis than to individuals who are normal or underweight. This phenomenon is partially attributed to the (often seen) increased amount of lean mass among obese people, as it is associated with improved cardiorespiratory fitness. The latter is a major determinant of clinical outcome in the general population, but particularly in those with CVD, including HF. However, it has to be noted that increased lean mass is a stronger prognosticator in HF than fat mass; especially in patients with CHD, excess FM can exert protective effects particularly when not associated with increased systemic inflammation (55).

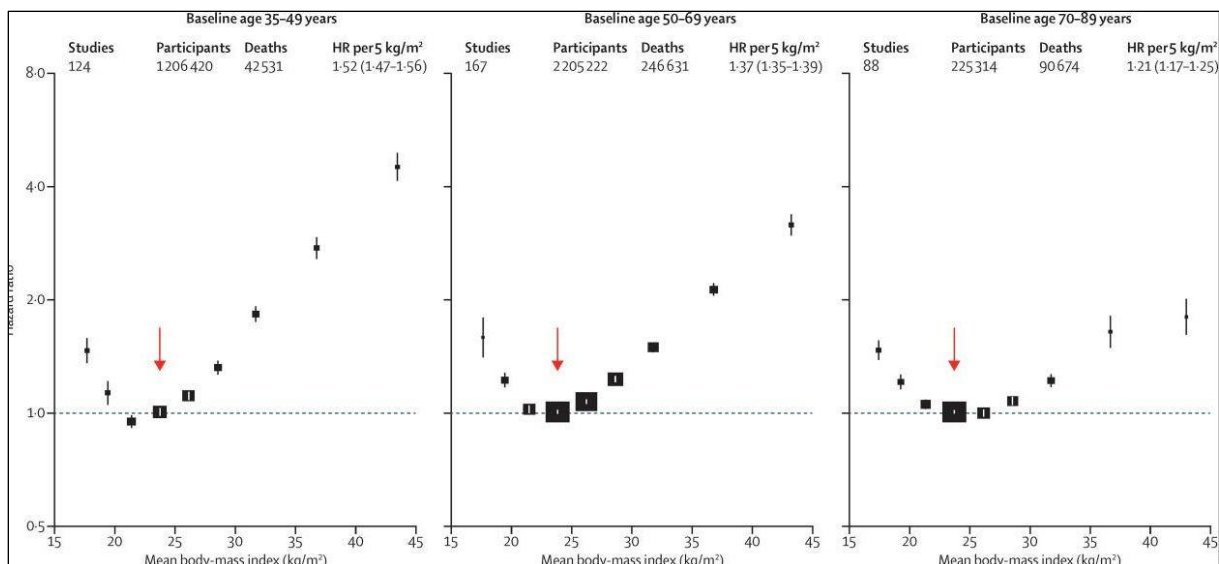


Fig. 3 Optimal BMI in people according to their age group (56)

2.3.3 ICU Patients

Obese patients hospitalized in the ICU have a more complicated course of hospitalization with higher rates of complications and longer hospitalization - overall and in the ICU; but mortality of the obese patients is lower than those patients whose BMI was defined as 'normal'. The optimal BMI for obese patients in ICU is 30 (**Fig. 4**) (57, 58).

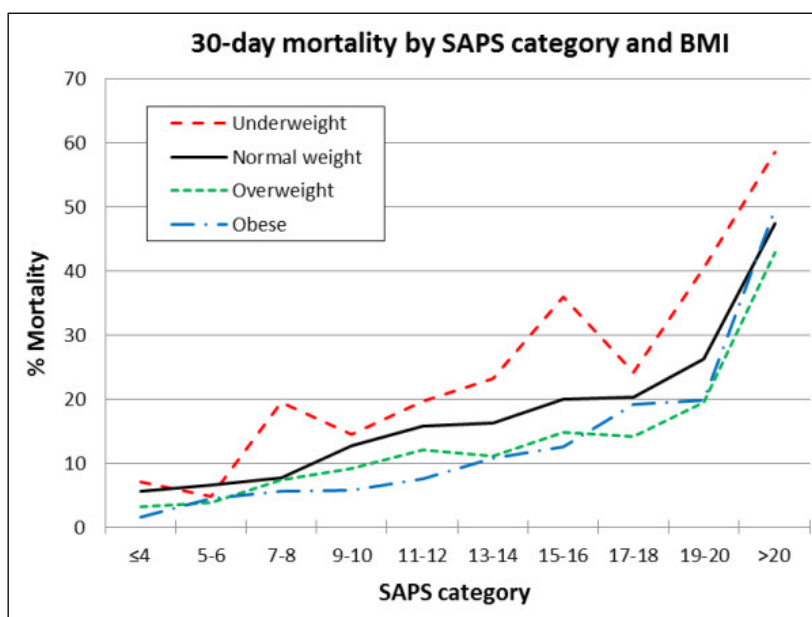


Fig. 4 BMI and mortality in ICU (57)

3.2.4 Dialysis Patients

Multiple studies of haemodialysis patients and peritoneal dialysis patients have demonstrated a protective effect of high BMI on survival in these groups of patients. Kim et al. published a study describing a two-year prospective follow-up of 900 peritoneal dialysis patients. Lower BMI was a significant risk factor for death, but increased BMI was not associated with mortality (59).

3.2.5 Cancer Patients

High BMI is associated with the development of cancers such as those of the colon and endometrium. Numerous studies have addressed the issue of nutritional status and prognosis in cancer patients. In particular, the relationship between nutrition and cancer has been addressed as a means of prevention of cancer, and with regards to prognosis, once cancer is present. Healthy lifestyle including healthy nutrition and normal BMI are related to lower risk of cancer development (60).

Mostly, the emphasis has been on malnutrition and prognosis, in which context malnutrition is definitely associated with reduced survival. However, Gonzalez studied 175 patients with various malignancies. The median survival time for overweight (2.64 y; range: 0.23–3.16 y) and obese (2.61 y; range: 0.26–3.20 y) patients was significantly higher than for patients with a normal (2.04 y; range: 0.06–3.05 y) or low (0.52 y; range: 0.19–0.98 y) BMI ($P < 0.001$). The worst prognosis was for sarcopenic patients (**Fig. 5**) (61).

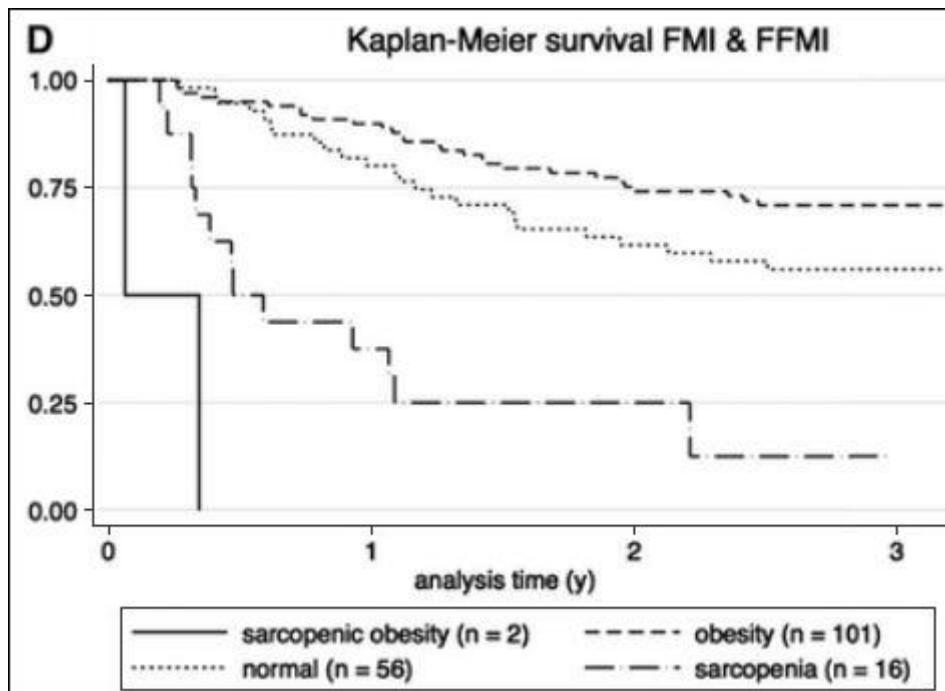


Fig. 5 BMI sarcopenia and survival in cancer patients

3.2.6 Patients with Peripheral Vascular Disease

In a prospective observational study of 1472 patients with peripheral vascular disease survival at 3 years was 37.5, 78.1, 86.8 and 87.0% for patients who were underweight, normal weight, overweight and obese at recruitment, respectively, $p < 0.001$. Patients who were underweight had approximately twice the risk of dying (RR 2.15, 95% CI 1.31–3.55, $p = 0.003$), while patients who were overweight (RR 0.67, 95% CI 0.49–0.91, $p = 0.011$) or obese (RR 0.59, 95% CI 0.41–0.85, $p = 0.005$) had approximately half the risk of dying, after adjustment for other risk factors and using normal weight subjects as the reference group (51).

3.2.7 Hospitalized Individuals

Overall, the optimal BMI for hospitalized patients was found to be higher than the WHO-defined healthy BMI. The optimal BMI for hospitalized patients, depending on the specific clinical setting was found to be 28-33.

3.3 Explanations for the Obesity Paradox

The OP could be explained by the fact that with the classification of obesity with BMI, subjects with different clinical and biochemical characteristics end up together in the same category without taking into account, e.g. the inflammatory status of their visceral adipose tissue, known to be related to the risk of CVD (62). In that context, when the diagnosis of malnutrition, due to excess of body fat mass and/or a defect of body lean mass, does not take into account the equilibrium and cross-talk between fat mass and muscle mass, patients with morbid or severe obesity tend to have lower morbidity and hospital mortality rates than patients with normal weight or who are underweight (63).

In summary, some additional possible mechanisms for protection conferred by obesity that have been suggested include: attenuation of reduction of lean body mass and physical function in patients suffering from chronic disease and aging, reduction of oxidative stress and inflammation, lowering of the levels of B-type natriuretic peptide thereby favourably altering regional sympathetic activity, provision of favourable functional lipid profiles, and secretion of adipokines (e.g. apelin), which might provide cardiovascular benefits (64).

4. Obesity Prevention

4.1 Early Prevention of Obesity

The prevention of obesity seems to be an easier and more effective target than losing weight after already becoming overweight or obese. There are data showing that factors contributing to obesity later in life are already present from the time of gestation. Additionally, the first months of life and in particular the quality and the quantity of the calories received during this first period of life seem to play an important role in the development of obesity later in life.

Offspring of obese parent(s) are consistently seen to be at increased risk of fatness, although few studies have looked at this relationship over longer periods of childhood and into adulthood. It is important to identify modifiable risk factors for early childhood overweight in order to develop effective prevention or intervention programs. Besides biological factors, familial interactions and parental behavioural patterns may influence children's weight development. Longitudinal investigation of children at overweight risk could help to detect significant risk and protective factors (65).

A study focusing on whether parental responsibility is an effective method in weight control interventions in children, concluded that reviewing intensity of parental involvement and behaviour change techniques are important issues in the effectiveness of long-term childhood weight control interventions (66).

Studies have shown that maintaining a lower weight after a negative caloric balance diet is a very difficult task, often resulting in a higher weight gain. For maintenance of the lower weight, adapting to a new diet seems not to be enough. An overall new approach, including increased physical activity, recording of dietary intake and a supportive social environment, seem to be the best way to achieve the expected result.

Therefore, it is quite important to avoid overweight or obesity, and this can start from the early stages of life. The Position Paper on the interventions for the prevention and treatment of paediatric overweight and obesity of the Academy of Nutrition and Dietetics addresses this very important topic (67). Prevention and treatment of paediatric overweight and obesity require systems-level approaches that include the skills of registered dietitians, as well as consistent and integrated messages and environmental support across all sectors of society to achieve sustained dietary and physical-activity behaviour change (67). Early childhood and school-based interventions should integrate behavioural and environmental approaches that focus on dietary intake and physical activity using a systems-level approach targeting the multilevel structure of the socioecological model as well as interactions and relationships between levels. Secondary prevention and tertiary prevention/treatment should emphasize sustained family-based, developmentally appropriate approaches that include nutrition education, dietary counselling, parenting skills, behavioural strategies, and physical-activity promotion. Policy and environmental interventions are recommended as feasible and sustainable ways to support healthful lifestyles for children and families.

The position of the Academy of Nutrition and Dietetics states that primary prevention is the most effective, affordable method to prevent chronic disease, and that dietary intervention positively impacts health outcomes across the life span (68). A study evaluated the influence of adulthood weight history on mortality risk (69). BMI at age 18 years was strongly positively related to total mortality in men and women (68). Weight gain was positively related to mortality, with stronger associations for gain between ages 18 and 35 years and ages 35 and 50 years than between ages 50 and 69 years. Mortality risks were higher in persons who attained or exceeded a BMI of 25.0 at a younger age than in persons who reached that threshold later in adulthood, and risks were lowest in persons who maintained a BMI below 25.0. Heavier initial BMI and weight gain in early to middle adulthood strongly predicted mortality risk in persons aged 50–69 years (69). These results suggest that excess weight-related mortality may be reduced among persons who enter adulthood with a low BMI, avoid gaining weight with age, and maintain a body mass within the healthy range over the course of adulthood. Public health efforts should encourage weight control in early and middle adulthood (69).

4.2 Obesity and Early Life Strategies

Generally breastfed infants are leaner than artificially (formula fed infants and behavioural and hormonal mechanisms may explain this difference. The theory is that a high nutrient diet in infancy adversely programmes the principal components of the metabolic syndrome in the child (body mass index, blood pressure and blood lipids) by promoting growth acceleration, whereas slower growth benefits later cardiovascular disease and its risk factors (70). The higher protein content of artificial baby milk compared to the lower protein content in breast milk might be responsible for the increased growth rate and adiposity of the formula-fed during this influential period of infancy.

During late infancy many infants have a protein intake which is more than three times as high as the physiological need. Several observational studies have shown an association between a high-protein intake (>15 energy %) early in life and an increased risk of developing obesity and thereby non-communicable diseases (NCDs) later in life.

It is plausible that an important reason for the slower growth in breast-fed infants is the lower content of protein in breast milk, but other qualities of breast milk could also play a role. A high intake of protein, especially dairy protein, stimulates the growth factors insulin-like growth factor (IGF-I) and insulin, and it has been suggested that the lower risk

of NCDs in breast-fed infants is mediated through a regulation of IGF-I. In conclusion, there is some evidence that a high protein intake during the complementary feeding period is associated with increased risk of NCDs and that avoidance of a high protein intake could reduce the risk of obesity (71, 72). Follow-up at 2 years of age shows that lower protein supply with formula normalizes early growth relative to a breast fed reference group and to the WHO growth reference. These results demonstrate that modification of infant feeding practice has an important potential for long-term health promotion and should prompt a review of the recommendations and policies for infant formula composition (73).

Future research on the relationship between breastfeeding, timely introduction of complementary feeding or rapid weight gain and obesity are warranted in developing countries. The focus of interventions to reduce risk of obesity in later life in developing countries could include: improving maternal nutritional status during pregnancy to reduce low birth weight; enhancing breastfeeding (including durations of exclusive and total breastfeeding); timely introduction of high-quality complementary foods (containing micronutrients and essential fats but not excessive in protein). Further evidence is needed to understand how the extent of weight gain and length gain during early childhood are related to body composition in later life (74).

4.3 Strategies for Obesity Prevention in Children and Adolescents

Much research has been devoted to finding successful interventions that can prevent obesity in children, as can be seen by the number of published papers on child obesity prevention which rose from about 20 per year in the late 1980s, to 60 per year in the late 1990s, and to more than 1000 in 2013 alone (14). Few of these papers are reports of actual intervention studies: a 2013 systematic review found 124 childhood obesity prevention studies in school, home, community, child care, primary care, and multiple other settings in high-income countries (75). Most existing studies were school-based, and the review called for further research to test interventions in other settings and to test more comprehensive and systems-oriented approaches.

A review which aimed to summarize the evidence on the effectiveness of obesity prevention and treatment programmes for adolescents from socioeconomically disadvantaged backgrounds found that only 2 out of 9 obesity prevention studies showed significant beneficial effects on BMI (11). Overall, the current admittedly inconclusive evidence suggests that involving adolescents in the development and delivering of interventions, the use of experiential activities and involvement of parents seem to be promising strategies; more high quality studies are needed (11).

In addition, a recent Cochrane review (76) concluded that current strategies to improve childhood obesity were weakly or inconsistently effective, a previous report suggested that a comprehensive approach including school life, home activities, environmental and cultural practices, and supportive parents and teachers would be promising policies and strategies (77).

Another review including 41 studies showed a strong association between convenience store access and weight related behaviours among children and adolescents. However, the association between convenience store access and children's weight status was found to be negative in Canada, rather mixed in the United States and the United Kingdom, and not significant in East Asia (78). In that study, inadequate intake of vegetables and fruits, excessive intake of high fat foods, sugary drinks, fast food, take-away foods, snacks, and insufficient physical exercise were shown to be important factors in weight control failure, leading to weight gain and overweight or obesity.

Current data generally suggest that, instead of single-component interventions, the most sustainable and beneficial effect on obesity prevention involves multiple strategies that focus on meals, classroom activities, sports, and play activities, and involve home, school or kindergarten, and community participants (75, 79). According to Lobstein et al, this conclusion should be no surprise: the more an environment consistently promotes healthy behaviour, the greater the likelihood that such behaviour will occur (14). In addition, it is crucial also to put in effort to ensure that interventions' results will be translated from one environment to another and across culturally diverse groups, recognizing that inequalities in outcomes might widen the existing differences in health status.

Furthermore, as pointed out by Huang and colleagues (80) both a strengthened evidence base for community interventions at the local and intermediate levels (in towns and cities) and an analysis of the effectiveness of activities that empower communities to build health-promoting communities are needed. It has to be noted that most intervention studies have been performed in Asia, Australia, Europe, or North America, and the most common settings for controlled interventions are in schools where the experimental designs can ensure a degree of scientific validity to the results. However, this focus on schools creates a strong settings bias in the scientific literature.

From a policy-making perspective, the dearth of societal policies for childhood obesity prevention is noticeable, and furthermore, of all reported intervention studies, only a few studies describe the probable costs of scaling up the intervention, despite such costs being a key issue for policy makers seeking policies and programmes to prevent child obesity (14). It seems that schools can be identified as an ideal place to implement prevention interventions, however the cost-effectiveness of a school-based intervention seems to be subject to substantial uncertainty and more research to explore obesity prevention within schools as part of a wider systems approach to obesity prevention is recommended (81).

- 1. Integrate education with supportive environmental change.** In school and child-care settings, the most successful interventions at achieving behavior change coupled educational messages with institutional change, so that children are taught about healthy eating and physical activity while provided healthy foods and more opportunities for physical activity.
- 2. Include both nutrition education and physical education.** The most successful interventions were those that included both nutrition and physical activity as integral parts of the intervention. Targeting obesity prevention through physical activity alone does not seem to be as effective without incorporating nutrition education. Younger children appear to learn best when exposed to behaviorally based or hands-on (rather than didactic) activities including ample opportunities for tasting, touching, and working with food. Providers, caregivers, and parents should be reminded that repeated exposure is typically required to promote acceptance of new foods by children.
- 3. Build in parent engagement for younger children.** Interventions that aimed to involve parents were generally more successful than those that did not, especially among preschool and elementary school-age children. Efforts to include parents are most effective when the parent not only receives information that reflects what the child is learning, but is also given guidance and at-home activities to aid in the progression of healthier lifestyle changes for the child at home.
- 4. Promote community engagement in schools and child care.** School— and child-care—based interventions show better results when coupled with community efforts that reinforce healthy eating and activity, as well as consistent messaging, both in and out of school and child care.
- 5. Policies that limit food availability show promise.** Policies that limit food availability, especially in schools, seem to be associated with lower body mass index.
- 6. Dose and continuity is important.** Children are inundated with messages promoting consumption of high-energy foods, so it is important to intensify and sustain the dose of nutrition education. More intensive interventions show better results. Although including health education in curricula is important, more innovative and "out of the box" messaging and other strategies should be explored, such as role model stories or novels, social media, and incorporation of health outcomes and consequences into all facets of society.

Fig. 6 Summary of recommendations from the review of child obesity primary prevention literature(67)

5. Lifestyle Changes to Prevent Overweight/Obesity and Promote Weight Maintenance

5.1 Body Weight Gain vs. Body Weight Maintenance

Obesity, one of the major health problems of today, is a condition in which fat is accumulated to excess, and body weight and fat percentage are increased. As obesity is associated with increased risk of several diseases (e.g. type 2 diabetes, certain cancers, and cardiovascular diseases) (3, 4, 82), it is relevant to target the excess of fat accumulation and prevent body-weight gain during lifetime.

Body weight increases as a result of a positive energy balance, which occurs when energy intake exceeds energy expenditure. The desired goal for the treatment and the reduction of development of body-weight gain or obesity is to decrease fat mass whilst preserving

or increasing fat free mass. The resultant higher ratio of fat free mass to fat mass plays an important role in the maintenance of energy balance and body weight, as fat free mass is the main determinant of basal energy expenditure (82), and the preservation of metabolic and overall health (84, 85).

Maintenance of body weight is achieved when energy intake is adjusted to energy expenditure. Usually, the pitfall appears to be body-weight regain after initial weight loss (4).

Snacking behaviour is a common practice among children and adolescents. Modifying current snack foods with nutrient-rich choices could lead to an improvement of their diet's nutritional quality (86).

5.2 Effect of the Macronutrients

Different macronutrients do not have the same satiating efficacy. Protein seems to have the greatest satiating effect, while fat the least intense one. Parallel to this is the sequence of priority with respect to metabolizing macronutrients (87). Therefore, postprandial energy expenditure of a mixed meal is mainly oxidation of carbohydrate and protein followed by fat oxidation in the fasted state.

High protein diets might have an effect on energy intake and body composition. Increasing the relative protein content of a diet (from 10-15% of energy to 20-30%) might lead to a reduction in food intake under ad libitum conditions, resulting in immediate body-weight loss. Increasing protein intake content of a diet might also increase the chance of maintaining the new body weight (after body-weight loss).

An increase in the relative protein content of the diet is promising to reduce the risk of a positive energy balance and the development of obesity.

The World Health Organization (WHO) recommends that dietary protein should account for around 10-15% of energy when individuals are in energy balance and weight stable. When summarizing the role of dietary protein in body-weight loss and body-weight maintenance however, absolute protein intake seems to be more important than the proportion of protein in the diet.

5.3 Fat Intake

The optimal intake of total fat was debated at the Joint Food and Agriculture Organization of the United Nations/World Health Organization expert consultation on fats and fatty acids in human nutrition held in November 2008. It was agreed that any effect of total fat intake on body weight was crucial to making global recommendations (in the context of increasing overweight and obesity, in particular in low and middle income countries undergoing rapid transition in nutrition).¹

To investigate the relation between total fat intake and body weight in adults and children 33 randomized controlled trials (73,589 participants) and 10 cohort studies were included, all from developed countries. There is high quality, consistent evidence that reduction of total fat intake has been achieved in large numbers of both healthy and at risk trial participants over many years. Lower total fat intake leads to small but statistically

¹ World Cancer Research Fund/American Institute for Cancer Research. Preventability of cancer by food, nutrition, and physical activity: Appendix A. Policy and Action for Cancer Prevention. Food, Nutrition, and Physical Activity: a Global Perspective. AICR, 2009
(<https://www.wcrf.org/dietandcancer/contents>)

significant and clinically meaningful, sustained reductions in body weight in adults in studies with baseline fat intakes of 28-43% of energy intake and durations from six months to over eight years. Evidence supports a similar effect in children and young people (88). Lowering total fat intake in adults compared with not lowering fat intake was associated with reductions in body weight, body mass index, and waist circumference. These effects were found in studies of more than eight years' duration, with baseline total fat intakes of 28% to 43% of energy, and in healthy adults and those with risk factors or current illness. However, these relations have not been tested in low or middle income countries. Although the evidence was slightly less strong in children, diets higher in total fat seem to be associated with higher body weight, body mass index, and waist circumference in both adults and children than diets lower in fat (88).

Calorie restriction can achieve short-term weight loss but the weight loss has not been shown to be sustainable in the long-term. An alternative approach to calorie restriction is to lower the fat content of the diet. However, the long-term effects of fat-restricted diets on weight loss have not been established. A review suggested that fat-restricted diets are no better than calorie restricted diets in achieving long term weight loss in overweight or obese people. Overall, participants lost slightly more weight on the control diets but this was not significantly different from the weight loss achieved through dietary fat restriction and was so small as to be clinically insignificant (89).

5.4 Dairy Products

Although several observational and experimental studies have investigated the effect of dairy consumption on weight and body composition, results are inconsistent. It has been postulated that the consumption of dairy products may facilitate body weight and fat loss because dairy products contain calcium, protein (casein and whey), and other bioactive compounds that may favourably affect energy balance. A meta-analysis does not support the beneficial effect of increasing dairy consumption on body weight and fat loss in long-term studies or studies without energy restriction. However, dairy products may have modest benefits in facilitating weight loss in short-term or energy-restricted RCTs (90). Additionally, increased dairy consumption without energy restriction might not lead to a significant change in weight or body composition; whereas inclusion of dairy products in energy-restricted weight loss diets significantly affects weight, body fat mass, lean mass and WC compared with results from the usual weight loss diets (91).

5.5 Dietary Sugars

Sugar has been a component of human diets since ancient times, with earliest reports of consumption coming from China and India, and only much later from Europe after the Crusades in the 11th century. The most obvious mechanism by which increasing sugars might promote weight gain is by increasing energy consumption to an extent that exceeds energy output and distorts energy balance (92). A series of meta-analyses provides evidence that intake of sugars is a determinant of body weight in free living people consuming ad libitum diets. The data suggest that the change in body fatness that occurs with modifying intake of sugars results from an alteration in energy balance rather than a physiological or metabolic consequence of monosaccharides or disaccharides (92). However, when considering the rapid weight gain that occurs after an increased intake of sugars, it seems reasonable to conclude that advice relating to sugar intake is a relevant component of a strategy to reduce the high risk of overweight and obesity in most countries.

Studies with respect to the association between intake of dietary sugars and body weight in adults and children report that among free living people involving ad libitum diets, intake of free sugars or sugar sweetened beverages is a determinant of body weight. The change in body fatness that occurs with modifying intakes seems to be mediated via changes in energy intakes, since isoenergetic exchange of sugars with other carbohydrates was not associated with weight change (92).

Regarding sugar-sweetened beverages (SSBs) which are the single largest source of added sugar and the top source of energy intake in the U.S. diet the scientific evidence that decreasing SSB consumption will reduce the prevalence of obesity and its related diseases is compelling (93, 94). Prevention of long-term weight gain through dietary changes such as limiting consumption of SSBs is more important than short-term weight loss in reducing the prevalence of obesity in the population. This is due to the fact that once an individual becomes obese, it is difficult to lose weight and keep it off (93).

Similar results, correlated with higher BMI z score, were found also for younger children aged 2 to 5 years drinking SSB. Paediatricians and parents should discourage SSB consumption to help avoid potential unhealthy weight gain in young children. From a public health standpoint, strong consideration should be made toward policy changes leading to decreases in SSB consumption among children (95).

It has to be noted that the World Health Organization (WHO) recommends a reduced intake of free sugars (96) throughout the life course (strong recommendation) with a reduction of free sugar intake to less than 10% of the total energy intake (strong recommendation) and preferably below 5% of the total energy intake (conditional recommendation) in both adults and children (97). Available data clearly show that people already consume significantly more sugar than they should, increasing the risk for dental caries, overweight and obesity and therefore WHO recommendations are intended for use by the policy makers as a benchmark for assessing intake of sugars by populations and as a driving force for policy change. It is in general advised to undertake public health interventions in an effort to create a favourable environment, enabling the overall amount of free sugar intake to be as low as possible and to reduce the frequency of consumption of sugar-rich foods.

5.6 Dietary Salt

Another study tried to determine the association among dietary salt, fluid, and sugar-sweetened beverage (SSB) consumption and weight status in a nationally representative sample of 4283 Australian children aged 2 to 16 years. Dietary salt intake was positively associated with fluid consumption ($r=0.42$, $P < 0.001$); each additional 1 g/d of salt was associated with a 46 g/d greater intake of fluid. In those consuming SSBs, salt intake was positively associated with SSB consumption; each additional 1 g/d of salt was associated with a 17 g/d greater intake of SSB. The study concluded that dietary salt intake predicted total fluid consumption and SSB consumption in consumers of SSBs. Furthermore, SSB consumption was associated with obesity risk. In addition to the known benefits of lowering blood pressure, salt reduction strategies may be useful in childhood obesity prevention efforts (98).

5.7 Glycaemic Index

It has been suggested that low glycaemic index (LGI) or load diets may stimulate greater weight loss than higher GI or load diets or other weight reduction diets. Overweight or obese people on LGI lose more weight and have more improvement in lipid profiles than

those receiving regular diets. In studies comparing ad libitum LGI diets to conventional restricted energy low-fat diets, participants fared as well or better on the LGI diet, even though they could eat as much as desired. Lowering the glycaemic load of the diet appears to be an effective method of promoting weight loss and can be simply incorporated into a person's lifestyle (99).

5.8 Lifestyle Adaptation

Healthy lifestyle comprises different aspects of living including healthy eating, physical activity, keeping a healthy BMI and refraining from smoking and excess consumption of alcohol.

A number of recent studies suggest that both sedentary behaviour (viewing television, playing video games, doing cognitive work, and listening to music) and reduced overall physical activity along with shorter sleep duration promote the overconsumption of dietary macronutrients, particularly fats and refined carbohydrates (100-103).

Sedentary behaviour is implicated in youth and adult overweight and obesity. However, the relationship between sedentary behaviour and weight status is often small or inconsistent, with few studies controlling for confounding factors such as diet and physical activity. Diet has been hypothesized to be related to some sedentary behaviours. It is opportune, therefore, to review whether dietary intake is associated with sedentary behaviour in young people and adults. This may allow for better interpretation of the diversity of findings concerning sedentary behaviour and weight status. The association drawn mainly from cross-sectional studies is that sedentary behaviour, usually assessed as screen time and predominantly TV viewing, is associated with unhealthy dietary behaviours in children, adolescents, and adults. Interventions need to be developed that target reductions in sedentary time to test whether diet also changes (104).

It is recommended that each individual maintains moderate physical activity equivalent to ≥ 30 minutes brisk walking, building up to ≥ 60 minutes of moderate walking per day.

Taxation is commonly proposed as a mechanism to reduce consumption of poor food choices and hence reduce rates of obesity and overweight in the community (105). However, results have been not as promising as expected.

Overall, a healthy diet includes the eating of variety of vegetables and fruit each day, consuming energy-dense foods sparingly, avoiding simple sugars, consuming fibre-rich foods, and keeping a balanced diet. Different healthy food protocols have been published as models for a healthy diet. The American Healthy Eating Index is an example (<http://www.cnpp.usda.gov/healthyeatingindex.htm>). Healthy eating, as part of healthy lifestyle, was found to correlate with reduced morbidity and mortality of up to $\sim 30\%$ in a large longitudinal cohort study (378,864 participants) in Europe (60).

5.9 Hospital and Community- based Health Services

A recent review(105) considered the role of hospital and community- based health services in adult obesity prevention as well as the potential enablers and barriers to the delivery of preventive health services. The review acknowledged that the health care system alone is not the answer to reducing the population impact of obesity. However, it suggested, there is evidence that health services can significantly contribute to obesity prevention commencing with screening all patients for risk factors and providing brief advice. This should be followed up with referral to a service, which provides long term follow-up with a focus on lifestyle change rather than just weight loss and should include consideration of an individual's health literacy.

Further research into the role of health services in obesity prevention should take a systems approach to examine the impacts of changing models of care whilst also taking into account the perceptions of health staff towards obesity and obesity prevention and the breadth of issues impacting on each individual's ability to make lifestyle changes (106).

6. Conclusions

Obesity has become a major epidemiological problem and an undisputable health problem in European countries and worldwide, and forecasts for the future are very disturbing. In terms of population health and public health, preventative measures are necessary, such as individual treatment strategies and population-wide measures aimed at preventing the development of obesity.

As adolescence is a key period of biological, social and behavioural change, this is a particularly important age period for adopting a healthy lifestyle. Prevention and treatment of paediatric overweight and obesity require synergy between personal and public responsibility in an integrated systems-level approach that includes consistent messages and environmental support across all sectors of society to achieve sustainable behaviour change for life (67). Moreover, adolescent health affects adult health. Therefore, adolescence is an especially important period in which to start preventive efforts. Physical activity levels tend to decline during adolescence; therefore, it is important to prevent such a decline and stimulate a physically active lifestyle.

Hospital- and community-based health services might also help by enabling screening at every primary care visit and tracking the results longitudinally (107).

The previous sections outline the importance of keeping a healthy BMI, but as the individual ages, it must be acknowledged that there is more to keeping healthy than just maintaining optimal BMI. Moreover, in some instances the optimal BMI is higher than described for the general younger (healthy) population. Pooled data from prospective cohort studies with 20,672 Asian American adults revealed that a high BMI, was associated with increased risk of total mortality among individuals aged 35 to 69 years, but the BMI was not related to total mortality among individuals aged 70 years and older (108).

All this does not mean that patients with chronic diseases have to gain weight. But, it is rather important to evaluate body composition, the person's weight history, the type of previous or current medication and behavioural therapy (i.e., diet and physical activity changes). All this because it is possible that higher mortality in normal weight subjects may be associated with low muscle mass and not low adiposity (109).

Prevention and management of obesity ultimately targets the negative energy balance by eating less and engaging in physical activities. Although this strategy seems simple, long-term weight reduction and/or maintenance of reduced weight is difficult to achieve. In adult studies, the actual maximum weight loss may be only 10%, which again increased within a year,(110) even if some studies yield better results (111).

7. Appendix

7.1 Baseline assessment tool for NICE guideline on Obesity (update) (CG189)

<https://www.nice.org.uk/guidance/cg189/resources>

Recommendation 1.2.7	
Classification	BMI (kg/m ²)
Healthy weight	18.5–24.9
Overweight	25–29.9
Obesity I	30–34.9
Obesity II	35–39.9
Obesity III	40 or more

Recommendation 1.2.9			
BMI classification	Waist circumference		
	Low	High	Very high
Overweight	No increased risk	Increased risk	High risk
Obesity 1	Increased risk	High risk	Very high risk
For men, waist circumference of less than 94 cm is low, 94–102 cm is high and more than 102 cm is very high.			
For women, waist circumference of less than 80 cm is low, 80–88 cm is high and more than 88 cm is very high			

Recommendation 1.2.11				
BMI classification	Waist circumference			Comorbidities present
	Low	High	Very high	
Overweight	1	2	2	3
Obesity I	2	2	2	3
Obesity II	3	3	3	4
Obesity III	4	4	4	4

1	General advice on healthy weight and lifestyle
2	Diet and physical activity
3	Diet and physical activity; consider drugs
4	Diet and physical activity; consider drugs; consider surgery

7.2 School Based Strategies (112)

A school-based low-cost nutrition and lifestyle education intervention on behaviour modification and risk profile can be comprised by several components. As suggested by Singhal et al (112) 7 seem to be of a greater importance: (i) lectures and focus group discussions; (ii) promotion of physical activity; (iii) activities to promote healthy lifestyle; (iv) individual counselling; (v) policy-level changes in the school; (vi) involvement of teachers and parents and (vii) training of student volunteers to sustain the programme.

- Behaviour substitution – listing healthy alternatives to junk food
- Behavioural rehearsal/practice – demonstration of healthy snacks
- Restructuring the physical environment – healthier school menu
- Restructuring the social environment – involvement and counselling of parents and teachers
- Health consequences – nutrition education (e.g. harmful effects of consuming junk food every day)
- Problem solving – individual counselling on perceived problems
- Social support (general) – encouragement and counselling
- Demonstration of the behaviour – demonstration of healthy snacks
- Instruction on how to perform a behaviour – demonstration of healthy snacks
- Knowledge transfer – lectures
- Parental involvement – nutritional and telephone counselling
- Peer leaders – training of students to disseminate health messages to their peers

Recommendations include (67):

1. make physical activity an integral and routine part of life;
2. create food and beverage environments that ensure that healthy food and beverage options are the routine, easy choices;
3. market healthy messages about physical activity and nutrition;
4. expand the role of health care providers, insurers, and employers in obesity prevention; and
5. make schools a national focal point for good practice.

8. References

1. Salvestrini, V., C. Sell, and A. Lorenzini, *Obesity May Accelerate the Aging Process*. Front Endocrinol (Lausanne), 2019. 10: p. 266.
2. Kopelman, P.G., I.D. Caterson, and W.H. Dietz, *Clinical Obesity in Adults and Children*. Vol. 3rd. 2009: Wiley-Blackwell.
3. Stunkard, A.J., *Current views on obesity*. Am J Med, 1996. 100(2): p. 230-6.
4. Pi-Sunyer, F.X., *The obesity epidemic: pathophysiology and consequences of obesity*. Obes Res, 2002. 10 Suppl 2: p. 97S-104S.
5. Wells, J.C., *Thrifty: a guide to thrifty genes, thrifty phenotypes and thrifty norms*. Int J Obes (Lond), 2009. 33(12): p. 1331-8.
6. Svačina, Š. and M. Haluzík, *Overnutrition - functional and clinical consequences*, in *Basics in Clinical Nutrition*, L. Sobotka, Editor. 2011, GALEN: Prague.
7. Krzysztozek, J., I. Laudanska-Krzeminska, and M. Bronikowski, *Assessment of epidemiological obesity among adults in EU countries*. Ann Agric Environ Med, 2019. 26(2): p. 341-349.
8. Flegal, K.M., et al., *Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010*. JAMA, 2012. 307(5): p. 491-7.
9. Flegal, K.M., et al., *Prevalence and trends in obesity among US adults, 1999-2008*. JAMA, 2010. 303(3): p. 235-41.
10. Ogden, C.L., et al., *Prevalence of childhood and adult obesity in the United States, 2011-2012*. JAMA, 2014. 311(8): p. 806-14.
11. Kornet-van der Aa, D.A., et al., *The effectiveness and promising strategies of obesity prevention and treatment programmes among adolescents from disadvantaged backgrounds: a systematic review*. Obes Rev, 2017. 18(5): p. 581-593.
12. Ogden, C.L., et al., *Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010*. JAMA, 2012. 307(5): p. 483-90.
13. *Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults*. Lancet, 2017. 390(10113): p. 2627-2642.
14. Lobstein, T., et al., *Child and adolescent obesity: part of a bigger picture*. Lancet, 2015. 385(9986): p. 2510-20.
15. Wang, Y., et al., *Controversies regarding reported trends: has the obesity epidemic leveled off in the United States?* Adv Nutr, 2012. 3(5): p. 751-2.
16. Brug, J., et al., *Differences in weight status and energy-balance related behaviors among schoolchildren across Europe: the ENERGY-project*. PLoS One, 2012. 7(4): p. e34742.
17. Ball, K., *Traversing myths and mountains: addressing socioeconomic inequities in the promotion of nutrition and physical activity behaviours*. Int J Behav Nutr Phys Act, 2015. 12: p. 142.
18. Zheng, H., D. Tumin, and Z. Qian, *Obesity and mortality risk: new findings from body mass index trajectories*. Am J Epidemiol, 2013. 178(11): p. 1591-9.
19. Myrskylä, M. and V.W. Chang, *Weight change, initial BMI, and mortality among middle- and older-aged adults*. Epidemiology, 2009. 20(6): p. 840-8.
20. Mehta, N.K. and V.W. Chang, *Mortality attributable to obesity among middle-aged adults in the United States*. Demography, 2009. 46(4): p. 851-72.
21. Jadad, A.R. and L. O'Grady, *How should health be defined?* BMJ, 2008. 337: p. a2900.
22. Lean, M.E., T.S. Han, and C.E. Morrison, *Waist circumference as a measure for indicating need for weight management*. BMJ, 1995. 311(6998): p. 158-61.

23. Nazare, J.A., et al., *Usefulness of measuring both body mass index and waist circumference for the estimation of visceral adiposity and related cardiometabolic risk profile (from the INSPIRE ME IAA study)*. Am J Cardiol, 2015. 115(3): p. 307-15.
24. Beechy, L., et al., *Assessment tools in obesity - psychological measures, diet, activity, and body composition*. Physiol Behav, 2012. 107(1): p. 154-71.
25. Iacobini, C., et al., *Metabolically healthy versus metabolically unhealthy obesity*. Metabolism, 2019. 92: p. 51-60.
26. Neeland, I.J., P. Poirier, and J.P. Despres, *Cardiovascular and Metabolic Heterogeneity of Obesity: Clinical Challenges and Implications for Management*. Circulation, 2018. 137(13): p. 1391-1406.
27. Oliveros, E., et al., *The concept of normal weight obesity*. Prog Cardiovasc Dis, 2014. 56(4): p. 426-33.
28. De Lorenzo, A., et al., *Adiposity rather than BMI determines metabolic risk*. Int J Cardiol, 2013. 166(1): p. 111-7.
29. Bourgeois, B., et al., *Improved strength prediction combining clinically available measures of skeletal muscle mass and quality*. J Cachexia Sarcopenia Muscle, 2019. 10(1): p. 84-94.
30. Sun, Q., et al., *Comparison of dual-energy x-ray absorptiometric and anthropometric measures of adiposity in relation to adiposity-related biologic factors*. Am J Epidemiol, 2010. 172(12): p. 1442-54.
31. Shafer, K.J., et al., *Validity of segmental multiple-frequency bioelectrical impedance analysis to estimate body composition of adults across a range of body mass indexes*. Nutrition, 2009. 25(1): p. 25-32.
32. Neovius, M., et al., *Bioelectrical impedance underestimates total and truncal fatness in abdominally obese women*. Obesity (Silver Spring), 2006. 14(10): p. 1731-8.
33. Stolarczyk, L.M., et al., *The fatness-specific bioelectrical impedance analysis equations of Segal et al: are they generalizable and practical?* Am J Clin Nutr, 1997. 66(1): p. 8-17.
34. Kamel, E.G., G. McNeill, and M.C. Van Wijk, *Usefulness of anthropometry and DXA in predicting intra-abdominal fat in obese men and women*. Obes Res, 2000. 8(1): p. 36-42.
35. Panotopoulos, G., et al., *Dual x-ray absorptiometry, bioelectrical impedance, and near infrared interactance in obese women*. Med Sci Sports Exerc, 2001. 33(4): p. 665-70.
36. Duren, D.L., et al., *Body composition methods: comparisons and interpretation*. J Diabetes Sci Technol, 2008. 2(6): p. 1139-46.
37. Lee, S.J., et al., *Relation between whole-body and regional measures of human skeletal muscle*. Am J Clin Nutr, 2004. 80(5): p. 1215-21.
38. Kanaley, J.A., I. Giannopoulou, and L.L. Ploutz-Snyder, *Regional differences in abdominal fat loss*. Int J Obes (Lond), 2007. 31(1): p. 147-52.
39. Kullberg, J., et al., *Whole-body adipose tissue analysis: comparison of MRI, CT and dual energy X-ray absorptiometry*. Br J Radiol, 2009. 82(974): p. 123-30.
40. Marra, M., et al., *Prediction and evaluation of resting energy expenditure in a large group of obese outpatients*. Int J Obes (Lond), 2017. 41(5): p. 697-705.
41. Achamrah, N., et al., *Validity of Predictive Equations for Resting Energy Expenditure Developed for Obese Patients: Impact of Body Composition Method*. Nutrients, 2018. 10(1).
42. Sharma, A.M. and R.F. Kushner, *A proposed clinical staging system for obesity*. Int J Obes (Lond), 2009. 33(3): p. 289-95.

43. Padwal, R.S., et al., *Using the Edmonton obesity staging system to predict mortality in a population-representative cohort of people with overweight and obesity*. CMAJ, 2011. 183(14): p. E1059-66.
44. Hebebrand, J., et al., *A Proposal of the European Association for the Study of Obesity to Improve the ICD-11 Diagnostic Criteria for Obesity Based on the Three Dimensions Etiology, Degree of Adiposity and Health Risk*. Obes Facts, 2017. 10(4): p. 284-307.
45. Fruhbeck, G., et al., *The ABCD of Obesity: An EASO Position Statement on a Diagnostic Term with Clinical and Scientific Implications*. Obes Facts, 2019. 12(2): p. 131-136.
46. Mikkelsen, K.L., et al., *Independent effects of stable and changing body weight on total mortality*. Epidemiology, 1999. 10(6): p. 671-8.
47. Somes, G.W., et al., *Body mass index, weight change, and death in older adults: the systolic hypertension in the elderly program*. Am J Epidemiol, 2002. 156(2): p. 132-8.
48. Wang, S. and J. Ren, *Obesity Paradox in Aging: From Prevalence to Pathophysiology*. Prog Cardiovasc Dis, 2018. 61(2): p. 182-189.
49. Winter, J.E., et al., *BMI and all-cause mortality in older adults: a meta-analysis*. Am J Clin Nutr, 2014. 99(4): p. 875-90.
50. Lee, J.S., et al., *Obesity can benefit survival-a 9-year prospective study in 1614 Chinese nursing home residents*. J Am Med Dir Assoc, 2014. 15(5): p. 342-8.
51. Golledge, J., et al., *Body mass index is inversely associated with mortality in patients with peripheral vascular disease*. Atherosclerosis, 2013. 229(2): p. 549-55.
52. Lavie, C.J., et al., *Impact of obesity and the obesity paradox on prevalence and prognosis in heart failure*. JACC Heart Fail, 2013. 1(2): p. 93-102.
53. Zafrir, B., N. Salman, and O. Amir, *Joint impact of body mass index and physical capacity on mortality in patients with systolic heart failure*. Am J Cardiol, 2014. 113(7): p. 1217-21.
54. Horwich, T.B., G.C. Fonarow, and A.L. Clark, *Obesity and the Obesity Paradox in Heart Failure*. Prog Cardiovasc Dis, 2018. 61(2): p. 151-156.
55. Carbone, S., et al., *Obesity paradox in cardiovascular disease: where do we stand?* Vasc Health Risk Manag, 2019. 15: p. 89-100.
56. Global BMIMC, Di Angelantonio E, Bhupathiraju Sh N, Wormser D, Gao P, Kaptoge S, et al. *Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents*. Lancet. 2016; 388(10046): 776-86.
57. Abhyankar, S., et al., *Lower short- and long-term mortality associated with overweight and obesity in a large cohort study of adult intensive care unit patients*. Crit Care, 2012. 16(6): p. R235.
58. Miehsler, W., *Mortality, morbidity and special issues of obese ICU patients*. Wien Med Wochenschr, 2010. 160(5-6): p. 124-8.
59. Kim, Y.K., et al., *The association between body mass index and mortality on peritoneal dialysis: a prospective cohort study*. Perit Dial Int, 2014. 34(4): p. 383-9.
60. Vergnaud, A.C., et al., *Adherence to the World Cancer Research Fund/American Institute for Cancer Research guidelines and risk of death in Europe: results from the European Prospective Investigation into Nutrition and Cancer cohort study1,4*. Am J Clin Nutr, 2013. 97(5): p. 1107-20.
61. Gonzalez, M.C., et al., *Obesity paradox in cancer: new insights provided by body composition*. Am J Clin Nutr, 2014. 99(5): p. 999-1005.

62. Lavie, C.J., et al., *Obesity and cardiovascular diseases: implications regarding fitness, fatness, and severity in the obesity paradox*. J Am Coll Cardiol, 2014. 63(14): p. 1345-54.
63. Kramer, A.A., *A Different Type of "Obesity Paradox"*. Crit Care Med, 2019. 47(2): p. 300-301.
64. Ghoorah, K., et al., *Obesity and cardiovascular outcomes: a review*. Eur Heart J Acute Cardiovasc Care, 2016. 5(1): p. 77-85.
65. Grube, M., et al., *Obese parents--obese children? Psychological-psychiatric risk factors of parental behavior and experience for the development of obesity in children aged 0-3: study protocol*. BMC Public Health, 2013. 13: p. 1193.
66. van der Kruk, J.J., et al., *Obesity: a systematic review on parental involvement in long-term European childhood weight control interventions with a nutritional focus*. Obes Rev, 2013.
67. Hoelscher, D.M., et al., *Position of the Academy of Nutrition and Dietetics: interventions for the prevention and treatment of pediatric overweight and obesity*. J Acad Nutr Diet, 2013. 113(10): p. 1375-94.
68. Fitzgerald, N., K.T. Morgan, and D.L. Slawson, *Practice paper of the Academy of Nutrition and Dietetics abstract: the role of nutrition in health promotion and chronic disease prevention*. J Acad Nutr Diet, 2013. 113(7): p. 983.
69. Adams, K.F., et al., *Body mass and weight change in adults in relation to mortality risk*. Am J Epidemiol, 2014. 179(2): p. 135-44.
70. Oddy, W.H., *Infant feeding and obesity risk in the child*. Breastfeed Rev, 2012. 20(2): p. 7-12.
71. Koletzko, B., et al., *Infant feeding and later obesity risk*. Adv Exp Med Biol, 2009. 646: p. 15-29.
72. Michaelsen, K.F., A. Larnkjaer, and C. Molgaard, *Amount and quality of dietary proteins during the first two years of life in relation to NCD risk in adulthood*. Nutr Metab Cardiovasc Dis, 2012. 22(10): p. 781-6.
73. Koletzko, B., et al., *Can infant feeding choices modulate later obesity risk?* Am J Clin Nutr, 2009. 89(5): p. 1502S-1508S.
74. Yang, Z. and S.L. Huffman, *Nutrition in pregnancy and early childhood and associations with obesity in developing countries*. Matern Child Nutr, 2013. 9 Suppl 1: p. 105-19.
75. Wang, Y., et al., *Childhood obesity prevention programs: comparative effectiveness review and meta-analysis*. Comparative effectiveness review no. 115. Rockville, MD: Agency for Healthcare Research and Quality ed. 2013.
76. Wolfenden, L., et al., *Strategies to improve the implementation of healthy eating, physical activity and obesity prevention policies, practices or programmes within childcare services*. Cochrane Database Syst Rev, 2016. 10: p. CD011779.
77. Waters, E., et al., *Interventions for preventing obesity in children*. Cochrane Database Syst Rev, 2011(12): p. CD001871.
78. Xin, J., et al., *Association between access to convenience stores and childhood obesity: A systematic review*. 2019.
79. Khambalia, A.Z., et al., *A synthesis of existing systematic reviews and meta-analyses of school-based behavioural interventions for controlling and preventing obesity*. Obes Rev, 2012. 13(3): p. 214-33.
80. Huang, T.T., et al., *Mobilisation of public support for policy actions to prevent obesity*. Lancet, 2015. 385(9985): p. 2422-31.

81. Canaway, A., et al., *Economic evaluation of a childhood obesity prevention programme for children: Results from the WAVES cluster randomised controlled trial conducted in schools*. PLoS One, 2019. 14(7): p. e0219500.
82. Pi-Sunyer, F.X., *Medical hazards of obesity*. Ann Intern Med, 1993. 119(7 Pt 2): p. 655-60.
83. Baba, N.H., et al., *High protein vs high carbohydrate hypoenergetic diet for the treatment of obese hyperinsulinemic subjects*. Int J Obes Relat Metab Disord, 1999. 23(11): p. 1202-6.
84. Harber, M.P., et al., *Effects of dietary carbohydrate restriction with high protein intake on protein metabolism and the somatotrophic axis*. J Clin Endocrinol Metab, 2005. 90(9): p. 5175-81.
85. Wolfe, R.R., *The underappreciated role of muscle in health and disease*. Am J Clin Nutr, 2006. 84(3): p. 475-82.
86. Mitsopoulou, A.V., et al., *Association of meal and snack patterns with micronutrient intakes among Greek children and adolescents: data from the Hellenic National Nutrition and Health Survey*. J Hum Nutr Diet, 2019.
87. Veldhorst, M., et al., *Protein-induced satiety: effects and mechanisms of different proteins*. Physiol Behav, 2008. 94(2): p. 300-7.
88. Hooper, L., et al., *Effect of reducing total fat intake on body weight: systematic review and meta-analysis of randomised controlled trials and cohort studies*. BMJ, 2012. 345: p. e7666.
89. Pirozzo, S., et al., *Advice on low-fat diets for obesity*. Cochrane Database Syst Rev, 2002(2): p. CD003640.
90. Chen, M., et al., *Effects of dairy intake on body weight and fat: a meta-analysis of randomized controlled trials*. Am J Clin Nutr, 2012. 96(4): p. 735-47.
91. Abargouei, A.S., et al., *Effect of dairy consumption on weight and body composition in adults: a systematic review and meta-analysis of randomized controlled clinical trials*. Int J Obes (Lond), 2012. 36(12): p. 1485-93.
92. Te Morenga, L., S. Mallard, and J. Mann, *Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies*. BMJ, 2013. 346: p. e7492.
93. Kosoglou, T., et al., *Hypothalamic-Pituitary-Adrenal Axis Effects of Mometasone Furoate/Formoterol Fumarate vs Fluticasone Propionate/Salmeterol Administered Through Metered-Dose Inhaler*. Chest, 2013. 144(6): p. 1795-802.
94. Malik, V.S., et al., *Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis*. Am J Clin Nutr, 2013. 98(4): p. 1084-102.
95. DeBoer, M.D., R.J. Scharf, and R.T. Demmer, *Sugar-sweetened beverages and weight gain in 2- to 5-year-old children*. Pediatrics, 2013. 132(3): p. 413-20.
96. Collaboration, N.C.D.R.F., *Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults*. Lancet, 2017. 390(10113): p. 2627-2642.
97. Breda, J., J. Jewell, and A. Keller, *The Importance of the World Health Organization Sugar Guidelines for Dental Health and Obesity Prevention*. Caries Res, 2019. 53(2): p. 149-152.
98. Grimes, C.A., et al., *Dietary salt intake, sugar-sweetened beverage consumption, and obesity risk*. Pediatrics, 2013. 131(1): p. 14-21.

99. Thomas, D.E., E.J. Elliott, and L. Baur, *Low glycaemic index or low glycaemic load diets for overweight and obesity*. Cochrane Database Syst Rev, 2007(3): p. CD005105.
100. Stroebele, N. and J.M. de Castro, *Listening to music while eating is related to increases in people's food intake and meal duration*. Appetite, 2006. 47(3): p. 285-9.
101. Temple, J.L., et al., *Television watching increases motivated responding for food and energy intake in children*. Am J Clin Nutr, 2007. 85(2): p. 355-61.
102. Westerlund, L., C. Ray, and E. Roos, *Associations between sleeping habits and food consumption patterns among 10-11-year-old children in Finland*. Br J Nutr, 2009. 102(10): p. 1531-7.
103. Gubbels, J.S., P. van Assema, and S.P. Kremers, *Physical Activity, Sedentary Behavior, and Dietary Patterns among Children*. Curr Nutr Rep, 2013. 2(2): p. 105-112.
104. Pearson, N. and S.J. Biddle, *Sedentary behavior and dietary intake in children, adolescents, and adults. A systematic review*. Am J Prev Med, 2011. 41(2): p. 178-88.
105. Comans, T.A., et al., *The cost-effectiveness and consumer acceptability of taxation strategies to reduce rates of overweight and obesity among children in Australia: study protocol*. BMC Public Health, 2013. 13: p. 1182.
106. Pearce, C., et al., *Obesity prevention and the role of hospital and community-based health services: a scoping review*. BMC Health Serv Res, 2019. 19(1): p. 453.
107. Mackey, E.R., et al., *Obesity Prevention and Screening*. Prim Care, 2016. 43(1): p. 39-51, vii.
108. Park, Y., et al., *Body mass index and risk of death in Asian Americans*. Am J Public Health, 2014. 104(3): p. 520-5.
109. Batsis, J.A., et al., *Low Lean Mass With and Without Obesity, and Mortality: Results From the 1999-2004 National Health and Nutrition Examination Survey*. J Gerontol A Biol Sci Med Sci, 2017. 72(10): p. 1445-1451.
110. Goodrick, G.K., W.S. Poston, 2nd, and J.P. Foreyt, *Methods for voluntary weight loss and control: update 1996*. Nutrition, 1996. 12(10): p. 672-6.
111. Metzgar, C.J. and S.M. Nickols-Richardson, *Effects of nutrition education on weight gain prevention: a randomized controlled trial*. Nutr J, 2016. 15: p. 31.
112. Singhal, N., et al., *Effects of controlled school-based multi-component model of nutrition and lifestyle interventions on behavior modification, anthropometry and metabolic risk profile of urban Asian Indian adolescents in North India*. Eur J Clin Nutr, 2010. 64(4): p. 364-73.