Dietary Intervention and Nutritional Counseling

21

Alessandro Pinto, Lucia Toselli, and Edda Cava

21.1 Introduction

The best dietary approach, recommended by health organizations such as the National Institutes of Health (NIH) and the American Heart Association (AHA), is a low-fat diet providing 20–30 % of energy as fats, 55–70 % as carbohydrates and 15–20 % as proteins. This approach is in accord with the population Dietary Reference Values (DRVs) that advise 20–35 % as Reference Intake (RI) range for lipids. Despite this recommendation, the poor efficacy of the traditional dietary treatment together with the high prevalence of *overweight* and *obesity* allowed the growing number of alternative dietary proposals, with some differences in energy percentage as macronutrients, not always in agree with the RI or supported by scientific evidences [1–5].

Several systematic reviews and meta-analysis comparing the different dietary models have been published but, considering the variability in study design, including sample size, duration, population, and macronutrient composition, there is no conclusive evidence especially in regard to a long-lasting *weight loss* and comorbidity improvement. Conversely, there is increasing evidence that the treatment of overweight or obesity should be a multidisciplinary approach, joining together caloric restriction, exercise, and behavior modification [6].

Scientific literature deals with dietary models usually defined "high protein" when the proteins provide ≥25 E% of total daily energy intake (TDEI); "low fat" if fats provide ≤30 E% or "high fat" >30 E%; and "low-carbohydrate diets" when CHOs intake is ≤45 E%; different combinations of these models are also described. According to *Freedman M.R.* et al. [7], a comprehensive comparison of different dietary models should include a careful analysis of the results on the following

A. Pinto (✉) • L. Toselli • E. Cava
Food Science and Human Research Unit, Medical Pathophysiology, Food Science and Endocrinology Section, Experimental Medicine Department, "Sapienza" University of Rome, P.le Aldo Moro, 5, Rome CAP 00141 RM, Italy
e-mail: alessandro.pinto@uniroma1.it; lucia.toselli@uniroma1.it; eddaka@yahoo.it

A. Lenzi et al. (eds.), *Multidisciplinary Approach to Obesity: From Assessment to Treatment*, DOI 10.1007/978-3-319-09045-0_21

outcomes: size and composition of weight loss (loss of body fat, BF, and lean body mass, LBM), long-term maintenance, nutritional quality of a *diet* (vitamin and mineral adequacy), metabolic parameters (e.g., blood glucose, insulin sensitivity, lipid levels, uric acid, and ketone bodies), influence on hunger, appetite, and subsequent food intake, psychological well-being, risk for chronic disease, and changes in long-term hormonal regulators of energy intake and expenditure (e.g., insulin and leptin). Specific relevant topics for the definition of general criteria to develop a suitable dietary protocol will be discussed hereinafter.

21.2 High-Protein Versus Standard-Protein Diets for Weight Loss: The Issue of Adequate Protein Intake

High-protein diets (*HPD*) (protein intake ≥25 E%) are among the most popular diets, although there is no consensus about the long-term efficacy and the potential harms. Compared to isocaloric standard-protein (SPD) and to low-fat diets (LFD), HPDs provide a limited advantage in reduction of body weight (BW), BF, and triacylglycerols (TAG) and in mitigating the loss of *LBM* and basal metabolic rate (BMR), but there are no significant differences in total cholesterol (TC), LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), blood pressure (BP), fasting plasma insulin, and glucose [8, 9]. It is difficult to provide the evidence whether the effects of HPD are due to the increased dietary proteins or to the mutual variation of CHOs and/or fat intake [10]. Several hypotheses have been suggested to explain the HPD results: a greater satiety effect, a higher dietary-induced thermogenesis (DIT), the *ketone* synthesis (in low CHOs-HPD) which improves the LBM preservation by a hypothesized anabolic effect on the muscle protein metabolism, and a higher sensitivity of the central nervous system to leptin [11, 12]. However, the *USDA/ARS* [13] review concludes that BW loss is not directly related to the macronutrients proportion in the diet when energy intake is reduced. As regards to the potential adverse effects induced by a too-high protein intake, acute adverse effects have been reported for protein intakes ≥45 E%, but not up to 35 E%, and the European Food Safety Authority (EFSA) suggests that an intake of twice the population reference intake (PRI) is safe in adults [10].

Beyond the size of BW loss, the preservation of LBM is another issue of the HPD, but this hypothesis has not been yet unequivocally demonstrated. Physiologically, the total BW loss is made up of about 75 % BF and 25 % LBM [14]. A daily protein intake of 0.8–1.2 g/kg/day should be sufficient to sustain satiety, BMR, and LBM, regardless of dietary CHO content with a greater effect in studies of >3 months [15, 16]. Several observations should be carried out about the protein intake cutoff to distinguish HPD and SPD. First, the "safe level of protein intake" (PRI) is 0.83 g/kg BW per day [17, 10]; as a result any protein intake lower than the PRI cannot be considered adequate even if results are between 10 and 15 % of the TDEI. Second, the caloric restriction results in a significant decrease in *nitrogen* balance [18]. Protein: energy ratio should be an essential issue to evaluate the adequacy of protein intake during low-calorie diets to satisfy protein metabolism and avoid the metabolic

shift of protein to gluconeogenesis or ATP synthesis. Analyzing studies on nitrogen balance in adults has been established that 1 mg nitrogen/kg/day is gained per extra intake of 1 kcal/kg/day [19]. Third point, obesity is associated with a chronic low-grade inflammation, which induces insulin resistance. Insulin resistance, inflammation, and oxidative stress could have a role in the obesity-related impairment in protein metabolism and turnover, as they are associated not only with increased BF but also with low muscle mass and muscle strength (sarcopenia) [20].

Westerterp-Plantenga MS et al. [21] reviewed the effects of relatively HPD during BW loss and maintenance and concluded that an intake of 1.2 g/kg BW is beneficial to body composition, improves BP, and reduces the risk of BW regain, and no kidney problems occur in healthy subjects. Contradictory results are described for the effects of HPD on insulin sensitivity and glucose tolerance [10].

Concluding, these observations support an average *protein* intake ≈1.2 g/ kg ideal BW per day during caloric restriction [21–24] and underline the need to define the individual adequate protein intake as absolute amount (g/kg of ideal BW), rather than as percentage of TDEI. This issue must be taken into account running meta-analyses or systematic reviews of literature comparing HPD with well-balanced standard diets, especially regarding the preservation of LBM.

21.3 Low Versus Standard Carbohydrate Diets for Weight Loss: The Matter of Glycemic Index/Load in Weight Management

Commonly, *low-carbohydrate diets* (*LChoDs*) are considered to contain <100 g/day or <30 % of energy from CHOs. Since LChoDs usually contain a relatively increased proportion of the other macronutrients, these diets are often "high protein" or "high fat." LChoDs rule out or significantly reduce the intake of some foods, such as cereals and fruits, resulting in a low fiber intake and increase the assumption of animal foods to achieve an adequate protein and energy intake. However, LChoDs do not have a fixed cutoff for CHOs intake or macronutrients ratio and not necessarily are high in protein or fat, depending on the level of caloric restriction and the food sources [25]. Several studies report that LChoDs result in a more rapid short-term BW loss and in greater improvements in TAG and HDL-C, but not in TC and LDL-C, than conventional low-calorie diets at 3 and 6 months, with no differences observed after 12 months [26–29]. Conversely, a recent meta-analysis [30] reported persistent although small effects of LCho-HPD on BW, BF, and fasting TAG also after 12 months; moreover, the effect on fasting insulin was small and probably related to the decreasing BF rather than macronutrient intake differences; no differences were observed in other plasma *lipids*, glucose, and LBM. Therefore, the apparent preservation of *LBM* in the short term [15] should be lost during BW regain in the follow-up period when the subjects having a normal protein intake regain the LBM lost. Furthermore, a systematic review of *LChoDs* found that the BW loss is associated with the length of the diet and the energy restriction rather than the CHO restriction [31]. There are some concerns about potential adverse

effects, and there is a need for long-term studies to measure changes in nutritional status and body composition during the LChoD and to assess fasting and postprandial cardiovascular risk factors and adverse effects. Without these informations LChoD cannot be recommended [26, 32]. Conversely, many studies demonstrated the beneficial effects of the *Mediterranean diet*, characterized by a balanced intake of macronutrient, albeit the CHO intake is at the lower tail of the RI and the fat intake, with monounsaturated fatty acids (MUFA) as the main source, at the higher tail of the RI; it was effective in reducing adiposity and other metabolic features of the metabolic syndrome [33, 34]. Unlike the LChoD but yet related to the dietary intake of CHOs, the role of *low glycemic index or load diets* (*LGIDs*) has been recently proposed as useful tool in BW management. It has been suggested that LGID may promote a greater loss of BW and BF and a better improvement in lipid profile (TC and LDL-C) than other dietary models [35]. The mechanisms involved in BW management might include the ability to promote satiety and delay hunger, reducing fluctuations in glycemia and insulinemia, promoting higher rates of fat oxidation and minimizing decreases in metabolic rate during energy restriction, and at last increasing the intake of whole grains with greater food volume [36].

Schwingshackl L. and Hoffmann G. [37], in a systematic review and meta-analysis, provided evidence about beneficial effects of long-term interventions with a LGID in regard to fasting *insulin* and proinflammatory markers, such as C-reactive protein as primary prevention of obesity-associated diseases; no significant changes were observed for blood lipids, anthropometric measures, HbA1c, and fasting glucose, while the decrease in LBM was significantly higher in LGID. This result could be explained assuming that a key requisite of a diet aimed to preserve LBM could be the CHOs to proteins ratio. An increasing proportion of protein to CHOs (in particularly an 1:2 protein/CHO ratio) in BW loss diet is more feasible and satiating, and it is associated to a better improvement in BF%, WC, and waist/hip ratio, plus it supports the preservation of LBM compared with the other diets and may be more effective in reducing long-term chronic disease risk improving blood lipids and glucose homeostasis [38, 39]. Therefore, it could be concluded that LGID is not more effective than traditional low-fat diet for BW loss or BW maintenance in general but may be beneficial for patients with certain risk factors such as insulin resistance [26].

21.4 Low-Fat Diet or Normal-Fat Diet: One or More Reference Model for Weight Loss?

The NIH clinical guidelines [14] provide a strong and consistent evidence that an average BW loss of 8 % of initial BW, with a decrease in abdominal fat, can be obtained over 3–12 months by an individually planned *low calorie diet* (*LCD*), creating a deficit of 500–1,000 kcal/day, aimed at achieving a BW loss of 0.5–1 kg/week. The NIH panel highlights that reducing fats as part of an LCD is a practical way to reduce calories and, in addition, *lower-fat diets* (*LFDs*), low in saturated fatty acids (SFA), reduce serum cholesterol levels and consequently the cardiovascular risk (CVR). The RCTs selected in NIH review tested the effects of LFD ranging from 20 to 30 % of calories from fat and between 1,200 and 2,300 cal. These

studies, taken together, show how LFD can contribute to lower the caloric intake even when caloric reduction is not the focus of the intervention, and if LCD is low in fat contribution, better BW loss is achieved. However, there is little evidence that LFDs, per se, cause BW loss independently from caloric reduction.

Beyond the NIH statement evidence [14], some remarks have to be expressed. *Schwingshackl L.* et al. [40] compared in a systematic review and meta-regression analysis the long-term (≥12 months) effects of LFD (lipid intake ≤30 % of TDEI) versus high-fat diets (HFD, lipid intake >30 % of TDEI) on blood lipid levels in overweight and obese patients. The results of the meta-regressions support the hypothesis that the heterogeneity in TC and HDL-C outcomes might be explained by a wide range in total *fat* (>30–60 %) and mainly in the percentage distributions of SFA, MUFA, and polyunsaturated fatty acids (PUFA): increases in TC levels were associated with higher SFA and lower PUFA, whereas a rise in HDL-C level was related to higher amounts of total fat especially in MUFA content, such as in the *Mediterranean diet*. A meta-analysis of short-term studies suggested that LFD results in higher TAG levels and lower HDL-C levels compared to diets in which SFA are completely replaced by unsaturated fatty acids. No significant differences in the BW change were observed between LFD and HFD, but this observation was no longer valid if only trials adopting a LCD were selected. The authors underline that BW loss might represent a potential confounder in the interpretation of blood lipid levels variation, indeed, and there was a significantly greater decrease in BW in the HFD compared with the LFD counterparts; this may explain the seeming observation that LFDs do not exert any beneficial effects on TC and LDL-C levels in studies aiming at BW loss (in contrast to the observations made when all studies were included in the meta-analyses). Supporting these findings, *Dattilo AM* et al. [41] demonstrated that 1Kg of BW loss is associated with a 1.93 mg/dL decrease in TC and a 0.77 mg/dL decrease in LDL-C level, respectively. It must be remarked that a diet high in MUFA, as the Mediterranean-style diet, compared with LFD improves more CVR factors, markers of vascular inflammation, and glycemic control [40, 42] without significant differences in glucose or insulin concentrations during the OGTT, in the Matsudas index, in BW, or in body composition [43].

21.4.1 The Very-Low-Energy Diets (VLED) and the Ketogenic Diet

According to NIH clinical guidelines [14], diets are categorized on the energy intake basis as:

- *Low-calorie diet* (*LCD*): a calorie restriction ranging between about 800 and 1,500 cal (approximately 12–15 kcal/kg BW) per day
- *Very-low-calorie diet* (*VLCD*): a diet of 800 or fewer calories (approximately 6–10 kcal/kg BW) per day

The VLCD are designed for subjects with a BMI ≥30 kg/m^2, with increased CVR, amendable by BW loss. These diets should only be used for a short time, about 12 weeks, and the patients should be monitored by a physician every 2 weeks

during the period of rapid BW loss (e.g., 1.5–2.5 kg/week). The *side effects* are usually mild and easily managed: cholelithiasis, cold intolerance, hair loss, headache, fatigue, dizziness, dehydration with electrolyte abnormalities, muscle cramps, nausea, constipation, or diarrhea. The risk of cholelithiasis can be decreased by ursodeoxycholic acid, including a moderate amount of fat in the diet and limiting the rate of BW loss to 1.5 kg/week. The majority of the side effects, including death, occurred when dieters consumed products that contained low-quality protein (e.g., hydrolyzed collagen) and were deficient in vitamins and minerals. Presently VLCD are considered safe and effective using high-quality proteins (e.g., milk, egg, or soy) or VLCD formulas, designed to provide all the nutrients needed and in appropriately selected individuals dieting for 8 weeks or fewer under careful medical supervision [43, 45]. The NIH clinical guidelines [14] report the results of studies that compared the amount of BW loss obtained by VLCD versus LCD in short (end of VLCD active phase: 12–16 weeks) and long term (24 weeks–5 years) concluding that VLCD produce greater initial BW loss than LCD; in the long term (>1 year), on the other hand, no significant differences are observed because a rapid BW reduction does not allow a gradual acquisition of changes in eating behavior and hence more BW is usually regained later on. Indeed a rapid BW regain between 6 and 12 months is observed if a maintenance program (dietary and behavioral support with increased physical activity) is not included [26, 47]. A significant improvement in BP, WC, and lipid profile is also reported but the results are more likely to be associated with the extent of BW loss, rather than the dietary model. More recent studies have confirmed this observation highlighting the high risk of nutritional inadequacies unless VLCD are supplemented with vitamins and minerals and hypothesizing an increased risk of BED (binge eating disorder) following the "yo-yo" effect of rapid BW loss and regain. The long-term evidence remains unclear; however, the intermittent VLCD use does not seem to be associated to any detrimental effect on metabolic parameters such as BMR, fasting insulin, insulin resistance, leptin, inflammatory markers, lipids, or BP [44, 46]. Formerly, *Fricker J*. et al. [47] studied the changes in LBM during the VLCD by nitrogen balance, demonstrating that the BMR-LBM ratio quickly decreases of about 15 % during the first week of a VLCD and this decrease persists but tapers off in the following weeks. This result suggests a metabolic adaptation of obese women to a VLCD, which leads to an increase in LBM energy metabolism efficiency, and it concurs with the decrease in the BMR-LBM ratio found in lean healthy men after 24 weeks of an LCD and in chronic human malnutrition.

The use of *ketogenic diets* (*KD*) as BW-loss therapy is an old proposal and became popular in the 1970s with the "Atkins Diet" in particular [48, 49]. Moreover the treatment of the epilepsy resistant to pharmacological therapy with KD has been well established, but in the last years an increasing amount of evidences suggests that KDs could have a therapeutic role in several diseases. It should be stated that not all *VLCD* are ketogenic, and the degree of CHOs restriction required to achieve ketosis remains unclear [25], although the term "ketogenic diet" is usually referred to diets containing ≤50 g/day CHOs with a relative increase in the proportions of protein and fat [50]. Increasing serum or urinary ketones is reported in subjects on daily intakes

of CHOs >50 g, and conversely, not everyone assuming ≤50 g of CHOs has urinary ketone levels on trace or greater. The *macronutrient* composition of the diet is an important determinant of ketosis, e.g., LChoD high in protein may not cause ketosis, as up to 57 g glucose can be produced from 100 g of dietary protein, and KD used in the treatment of pediatric epilepsy typically restricts protein as well as CHOs (using a ratio of fat to CHO and protein of 3:1 or 4:1, respectively). According to *Stock AL and Yudkin J* [51], ketosis will occur when fat intake exceeds twice the CHOs intake plus half the protein intake. Together, data indicate that CHO restriction is a more important determinant of ketosis than restriction of total calories. *Ketones* are normally present in small amounts in the blood of healthy individuals following an overnight fasting or prolonged exercise, with plasma levels reported in the ranges of 0.2–0.5 mM [52]. Circulating ketone levels can increase up to 50-fold during periods of caloric deprivation, with β-hydroxybutyrate levels reported to be 4–5 mM in the blood following a 5–8 day fast. An excess of circulating ketones, which are strong organic acids that fully dissociate at physiological pH and overload the buffering capacity of serum and tissues, may result in metabolic acidosis, a potentially life-threatening condition. Moreover, increased ketone levels may affect the brain microvascular endothelium permeability and cerebral edema has been associated with diabetic ketoacidosis. However, such effects are observed only when ketones overreach physiological levels (10–20 mM or higher) during pathological states [53]. About the short-term safety, serious adverse events are reported to occur in adults on a low-calorie ketogenic diet (LCKD), including acute pancreatitis, exacerbation of panic disorder, severe metabolic acidosis, dehydration, severe electrolyte impairments, and hypokalemia, possibly associated with sudden cardiac death. Very few studies examined the effects of LCKD for periods longer than 12 months in adults: the risk of nutritional inadequacy depends on several factors, including the overall composition of the diet, nutrient sources, degree of CHOs restriction, and diet duration. Since dietary CHO restriction often results in increased protein intake, it is difficult to separate the renal and bone effects of *LCKD* from the effects of increased dietary protein. Therefore, the long-term effects of LCKD on renal and bone health are unknown [25, 54]; nonetheless, examining body composition, studies have generally found a reduction in BF with preservation of *LBM* [25]. There are contrasting theories regarding the mechanisms LCKDs work on, and, anyway, some of the initial BW loss is due to increased diuresis (renal sodium and water loss), both as a result of glycogen depletion and ketonuria. *Paoli A* et al. [50] summarized and listed in order of importance and available evidence the hypothesized effects of VLCKDs:

1. Reduction in appetite due to higher *satiety* effect of proteins, effects on appetite control hormones, and to a possible direct appetite-suppressant action of the KBs, although there is conflicting evidence regarding this issue in the published literature
2. Reduction in lipogenesis and increased *lipolysis*
3. Reduction in the resting respiratory quotient (RRQ) and, therefore, greater metabolic efficiency in consuming fats
4. Increased metabolic costs of gluconeogenesis and thermic effect of proteins

It has also been hypothesized an up-regulation of mitochondrial uncoupling proteins with a resultant wasting of ATP as heat [25].

However, the US Department of Agriculture concludes that diets, reducing calories, will result in effective BW loss independent of the macronutrient composition, which is considered less important or even irrelevant [50], and the studies evaluating long-term outcomes of LCKD found a greater BW loss at 3–6 months, with no difference at 12 months. In studies comparing LCKDs and LFDs, the LCKDs are significantly associated with a greater reduction in TAG and an increase (or less of a decrease) in HDL, although this result seems to be explained by differences in BW loss. Conversely, LFDs generally have a more beneficial effect on LDL levels than LCKD. Despite their unfavorable effect on total LDL levels, several studies have found a beneficial effect of LCKD on certain *lipoprotein* subclasses, with a reduction in VLDL, an increase in large LDL, and a reduction in small LDL particles [25]. In conclusion, the classic LCKD containing high fat, low CHOs, and low protein are difficult to manage, are unpalatable, and may present an increased atherogenic risk as serum levels of cholesterol and triglycerides are often elevated [55].

21.5 Criteria for the Formulation of a Balanced Diet in Obese Patient

It is clear that any low-calorie *diet* resulting in a negative energy balance produces BW loss in the short term (3–12 months); nevertheless, the optimal macronutrient composition of the diets continues to be controversial and object of ongoing researches.

A useful algorithm to develop a well-balanced dietary plan involves the following steps:

1. Setting the energy intake to obtain a suitable BW loss
2. Setting the protein intake
3. Splitting the nonprotein kcal (NP-kcal) between CHOs and lipids
4. Verifying the adequacy of micronutrients (vitamins and minerals) and fiber intake
5. Scheduling meals

The first step must take into account the broad interindividual variability (15 % on average) of the *BMR* normalized to the LBM in subjects of same sex, age, BW, and body size. Regarding the 24 h-energy expenditure (24-EE), this variability reaches 30 %, related to the individual energy expenditure for any physical activities (affected by muscle tone, ergonomic efficacy, intensity, etc.) [56]. It should be stressed that in obese subjects the BMR is higher than in lean subjects, since both LBM and BF are increased [57].

In clinical practice the BMR is calculated by predictive regression equations using sex, age, BW, and height (e.g., Harris-Benedict equation or Schofield et al., quoted in FAO/WHO/ONU report, 1985) with an average standard error of 10 % in the single

subject and 2 % in population groups [56]. More recent predictive equations give slightly lower errors in overweight subjects [58], e.g., the Mifflin St. Jeor equation or the Livingston equation, which are useful to predict the BMR in adults of various BMI levels, although the accuracy is lower in obese than nonobese people [59, 60].

A higher accuracy in estimating the BMR can be reached taking into account the *LBM*, by means of prediction equations such as the J. Cunningham equation [61]. Obviously it is critical an accurate assessment of the BC to avoid a source of bias. Several limits are embedded to the usual inexpensive and noninvasive methods in the outpatient practice (anthropometrics and Biolectrical Impedance Analysis, BIA), that are only partly overtaken by dual energy x-ray absorptiometry (DEXA). Evaluating BC allows to identify the "desirable BW," essential to establish the individual adequate energy and protein intake, indeed:

1. In a "*normative* approach" aimed to BW loss, BMR must be esteemed using "desirable BW," while a "*conservative* approach" uses the subject actual weigh.
2. "Desirable BW" is in accord to an optimal ratio between BF and LBM, taking into account that these are physiologically increased in obese subjects and assuming that the *exceeding BW* consist of BF 75 % and LBM 25 %.
3. LBM is the mean factor affecting BMR, therefore, predicting equations that include LBM have higher accuracy; in order to preserve the LBM, energy intake should not be lower than BMR −10 E%.
4. Protein PRI is about 1 g/kg or better 1.2 g/kg of "desirable BW" in agreement with the findings of the literature previously reported; in calculating the "desirable BW" it is essential to take into account the 25 % of the exceeding BW as LBM, in order to preserve the LBM during the BW loss; likewise, the LBM loss should not exceed the 25 % of the BW loss, when it occurs [14].

When it is not possible to assess the BC, "desirable BW" could be esteemed adding a 25 % of the exceeding BW (as hypothetical LBM) to the "ideal BW" resulting by the common literature equations.

Afterwards, *physical activity level* (*PAL*) should be assessed. Subject must collect a record of any activity performed in a 24 h period (sleeping, eating, walking, working, all other activity such as personal hygiene, sport, hobbies, etc.). Alternatively but less accurately, it is possible to carry out a physical activity recall [56, 62]. A specific energetic value is assigned to every activity and the PAL is calculated through a factorial procedure as weighted average of all these activity, adjusted for the time dedicated to each [63, 64]. Finally, to calculate the 24 h-EE in Kcal/day, BMR is multiplied by the PAL esteemed (Table 21.1). It is possible to simplify this procedure, with a satisfying accuracy, using average PAL values codified by lifestyle ranges as "light," "moderate," or "heavy" (Fig. 21.1) [56, 62].

To lose 1 kg/week of BF, it is necessary a daily negative energy balance of about 1,000 kcal, and the guidelines usually suggest a moderate reduction of the TDEI, around 500–1,000 kcal/die cut down from the estimated *24 h-EE* or from the usual TDEI, whether the subject's BW is steady and anyway never going below the BMR more than 10 %; this is aimed to a gradual and long-lasting weight loss: losing 3–5 kg

Table 21.1 Criteria for the formulation of a dietary plan

Parameters	Determinant factors		
Desirable weight	Body composition: lean body mass (LBM)/fatty mass (FM)		
Basal metabolic rate (BMR)	Sex	Conservative approach	Normative approach
	Age		
	Height	BMR is calculated utilizing the actual weight	BMR is calculated utilizing the desirable weight
	Weight		
	LBM		
Physical activity level (PAL)	Frequency, intensity, and duration of the different activities during the 24 h, included the sleeping time	Physical activity energy expenditure is affected by the actual weight	
Daily energy expenditure (24 h-EE)	BMR × PAL		

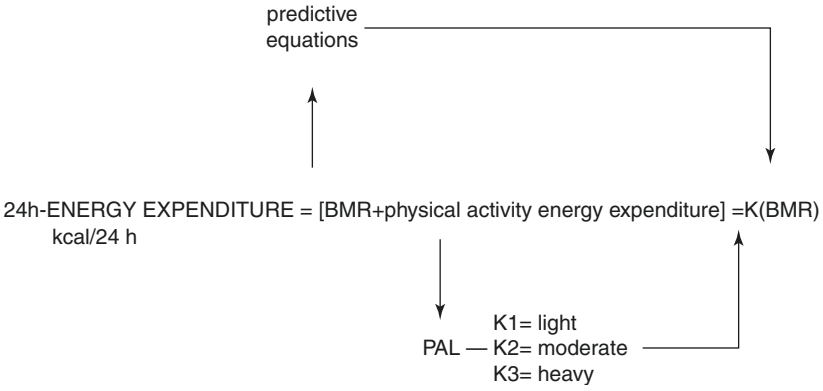

Fig. 21.1 Algorithm to calculate the 24 h-daily expenditure

of BW is an excellent achievement, allowing to preserve the LBM and prevent the dehydration, without raising the risk of eating disorders. Cognitive-behavioral therapy together with physical activity program gives the best and long-lasting results [14, 65].

To define an adequate dietary energy intake, it is also necessary to take into account the patient's eating habit (quality and quantity of food consumption, meals planning, and eating disorders suspected by psychodynamic tests). Food surveys could be retrospective, with the use of memory such as the 24/48 h recall, food frequency, dietary history, or perspective by recording the weight or the estimated quantity of the foods consumed.

Usually, food frequency questionnaire, dietary history, and food diary allow to collect all the data required. Then, the calculated 24 h-EE can be compared to the actual TDEI taking into account the BW changes. Energy balance is the difference

between food intake and energy expenditure [*24 h-EE* = BMR + DIT (diet-induced thermogenesis) + PA (physical activity)]. This balance is positive in obese subjects actively gaining BW; instead, if the BW is steady, the TDEI is equivalent to 24 h-EE and the subject is in energetic balance with a TDEI not necessarily too high.

Established the most adequate energy intake to obtain a healthy BW loss, it should be set the *protein* intake level as above stated (1–1.2 g/kg "desirable BW") and the protein-kcal must be apart from the NP-kcal which, in the end, have to be split between CHOs and lipids, pointing out that proteins and *CHOs* provide 4 kcal/g while lipids 9 kcal/g. Fifty percent of protein intake should come from animal source and the other 50 % from plant to avoid a high intake of animal *lipids* and meanwhile providing an adequate intake of vegetable protective factors (phytochemicals). Assuming the *nitrogen* (*N*) content of the proteins to be on average 16 %, it is easy to state that 6.25 g of proteins are equivalent to 1 g of N. It must be emphasized that in order to achieve an efficient protein synthesis, 100–150 nonprotein kcal (NP-kcal) are needed for every intake of 1 g N or 6.25 g proteins. In a dietary plan providing for the actual energy expenditure, protein amount could not be higher than 13–15 % of the TDEI, but in formulating a low-calorie diet, protein requirements must be counted in grams/kg of "desirable BW" to meet the proteins need [66].

Once satisfied the protein requirement, NP-kcal should be split as CHOs, 65–70 E%, and lipids, 30–35 E%. The distribution of NP-kcal requires a careful screening of comorbidities: this is a critical issue for the metabolic effects of the dietary models described above, to avoid the possible adverse effects or in order to exploit their metabolic properties. Total CHOs should be composed between complex, 80 %, and simple, 20 %. The amount of fats should consist of SFAs (1/3), MUFAs (1/2), n PUFAs (1/6), and the RI of EFAs (essential fatty acids) and of the liposoluble vitamins (α-tocopherol, β-carotene, vitamin D) have to be assured; to respect this proportion, it is sufficient to have an intake of 30 % as animal fats and 70 % as vegetable fats. Animal fats, indeed, consist of 2/3 SFAs and 1/3 MUFA with a low PUFA content, and instead plant source are made up of 1/3 SFA and 2/3 MUFA and PUFA [67] (Fig. 21.2). With respect to the TDEI, total CHOs intake should provide 45–60 % of TDEI, with maximum 15 % of simple sugar, while fats should provide 20–35 % of TDEI [67]. The highest values of the range (RI) should be considered only in the low-carbohydrate diets, when required. In other cases, the intake of total lipids must be ≤30 %, SFAs 7–10 %, and trans-fatty acids ≤1 % of TDEI [26, 67]. On the other hand, a too-low-fat diet has poor organoleptic properties, resulting bland and tasteless. Olive oil should not be removed since its composition in MUFA helps to keep an adequate HDL cholesterol level. To achieve a good level of *ω*-3-fatty acids, 150 g of any kind of fish twice a week are enough, better if chosen among *anchovy*, *sardine*, *mackerel*, or similar. In the end, an eating plan well balanced and consistent with dietary guidelines endorses to consume at least five servings of fruits and vegetables per day, emphasizing the use of whole grains, with a daily *fiber* intake of 35 g or more. BW-loss diet that excludes one or more foods or food groups and/or substantially restrict macronutrients intake below the PRI could produce nutrient deficiencies and increase health risks. The *micronutrient* intake level should be evaluated on a weekly basis or on a longer term for liposoluble

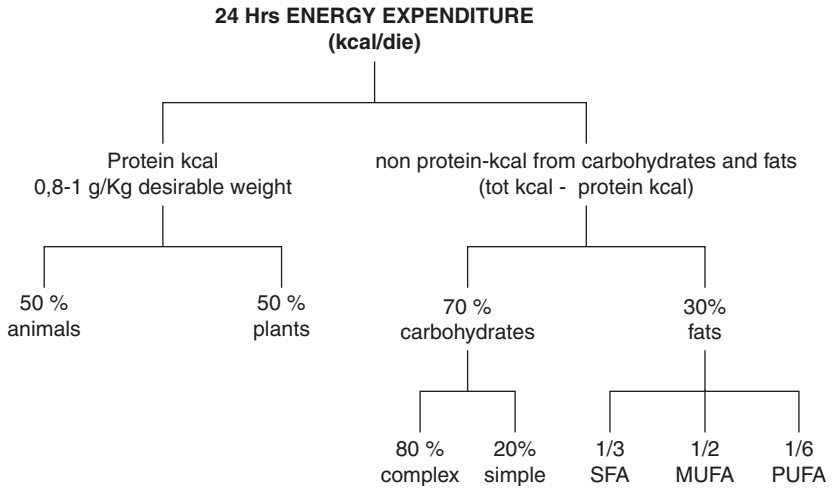

Fig. 21.2 Flow chart to allocate the macronutrients in a balanced diet

vitamins and β-carotene; in a moderate low-calorie diet, it is possible to meet PRI by the weekly consumption of the different food groups as suggested by the guidelines for a healthy diet [67]. The supply of essential fatty acids, minerals, vitamins, and fiber has to be checked in relation to the DRVs (Table 21.2).

21.6 Nutritional Counseling and Conclusion

The World Health Organization defined obesity as a serious chronic disease, largely preventable through lifestyle changes [75]. This definition means that although the weight loss is essential for reducing the risk of obesity-associated comorbidities and mortality, the acquisition of a healthy lifestyle should be the main objective of the whole therapeutic intervention. Dietary treatment should instruct patients on how to modify their diets in order to lower the caloric intake, obtaining a slow and progressive BW loss, reducing CVR, and other comorbidities. It was described an inverse relation between adherence to a *Mediterranean* dietary pattern and the prevalence of obesity in a free-eating, population-based sample of men and women, irrespective of various potential confounders [68]; several studies support the evidence that promoting eating habits consistent with Mediterranean diet (MD) nutrients pattern may be a useful and safe strategy for the treatment of obesity [69]. The MD features were recently revised by Bach-Faig A et al. [72]: the MD is rich in plant foods (cereals, fruits, vegetables, legumes, tree nuts, seeds, and olives), with olive oil as the principal source of added fat, along with high-to-moderate intakes of fish and seafood; moderate consumption of eggs, poultry, and dairy products (cheese and yogurt); low consumption of red meat; and a moderate intake of alcohol (mainly wine during meals) [70].

Table 21.2 Criteria to develop a balanced diet in obese subjects

Criteria			Parameters
1	Intake energy	Energy intake: reducing 500–1,000 kcal from the usual intake and anyway never < BMR −10 %	Estimate basal metabolic rate (BMR)
			Estimate 24 h energy expenditure (24 h-EE)
		Aim: ↓ 3–5 Kg/month	Assessing nutritional habits (usual energetic intake)
			Body weight changes in the last month (steady state or dynamic)
2	Protein intake	1 g protein/Kg desirable weight	Desirable weight
		100–150 kcal nonprotein/g nitrogen intake	Metabolic impairments and/or pathological conditions, e.g., kidney failure, microalbuminuria of nephrotic syndrome, etc.)
3	Nonprotein kcal Allocation (carbohydrates and lipids)	Total fats: 20–35 % total kcal	Dietary reference intakes
		Saturated fatty acids: ≤ 7–10 % tot. kcal trans-fatty acid ≤1 % tot. kcal	Nutritional status assessment
			Metabolic impairments (dyslipidemia, diabetes, etc.)
		Monounsaturated fatty acids: ≤15 % tot kcal	
		Polyunsaturated fatty acids: ≤10 % tot. kcal Essential fatty acids: ω-6 = 2 % and ω-3 = 0,5 % tot kcal	
		Cholesterol: ≤300 mg/die	
		Carbohydrates: ≥ 45 % tot kcal; ≥100 g/die	
		Simple sugars: ≤15 % tot. kcal	
		Calcium: 1,000–1,500 mg/die	
		NaCl: ≤6 g o Na 2,4 g/die	
4	Verifying fiber and micronutrients intake (minerals and vitamins)	Evaluation of the need to use nutritional supplements	Dietary reference intakes
		Fiber: 35 g	Nutritional status assessment
			Energy intake of the diet
5	Meal scheduling	Frequency complying with recommended requirements in guidelines	Usual day schedule
			Meal consumption modalities
		Food choices variety	Nutritional habits
		Regular meals	Limits (family, socials, working, food preferences, etc.)

Corbalán MD et al. [69] assert that "although there is no all-inclusive diet for the treatment of *obesity* and metabolic syndrome, a Mediterranean-style diet has most of the desired attributes, including lower refined carbohydrate content, high fiber

content, moderate fat content (mostly unsaturated), and moderate to high vegetable protein content." According to the recommendations of the Spanish Society of Community Nutrition, the distribution of macronutrient components in MD is: 35 % fat (<10 % SFA and 20 % MUFA), 50 % CHOs, and 15–20 % protein [69].

Educational efforts should highlight the following topics as reported NIH clinical *guidelines* [14]:

- Energy value of different foods
- Food composition: fats, CHOs (including dietary fiber), and proteins
- Reading nutrition labels to determine caloric content and food composition
- New habits of purchasing with preference to low calorie foods
- Food preparation avoiding adding high-calorie ingredients during cooking (e.g., spreads and oils)
- Avoiding overconsumption of high-calorie foods (both high-fat and high-CHO foods)
- Maintaining adequate water intake
- Reducing portion sizes
- Limiting alcohol consumption

Whatever else is reported by healthy eating guidelines and effectively depicted in the *diet* pyramid or "eatwell plate" showing the proportions of food groups that should be eaten daily in a well-balanced diet completes these topics.

However, a successful BW loss is more likely to occur when patients' food preferences are considered to tailor an individual diet, adapted to the specific realities of different countries and to the variations in the dietary pattern related to geographical, socio-economic, and cultural contexts, taking into account the traditional, local, eco-friendly, and biodiverse products, thereby contributing to a higher and long-term sustainable compliance.

In the traditional framework, the patient is in a state of almost total dependence by the physician, and hence this model has been defined prescriptive, directive, paternalistic, or authoritarian. On the other side, the obese patients live in a dichotomous relationship with food, friend, or foe, and they think that the diet is not a means to improve their health status but a way to prove their willpower. In this perspective, when the patient transgresses the diet, he experiences a failure resulting in reduced self-esteem. Conversely, the nutritional *counseling* aims to "enable" the patient to make a decision about personal choices or problems or issues directly concerning themselves. The counseling procedure emphasizes the importance of the self-perception, self-determination, and self-control, taking the shape of helping a relationship finalize to return to autonomy, a greater sense of dignity and self-esteem to the person [70]. As in all chronic diseases, the objective is not the full recovery, but in the case of *obesity* can represent a way aimed at not only the weight loss but also in the ability to self-manage risk situations, to develop active lifestyle, and knowing how to choose what is really important to live fully their own existence and thus enhance the quality of life. The aim should be not only to improve the knowledge of the patients but especially their skills, know-how, and their ability to master events, known as how to be. The main tools at the basis of

nutritional *counseling* are common with the cognitive behavioral therapy: therapeutic alliance, therapeutic adherence, motivation, problem solving, empowerment, and narrative medicine. The latest experience bears the *cognitive behavioral therapy* (*CBT*) as a key tool to achieve a lifestyle change and thus a long-lasting and stable BW loss. It has been designed to improve diet and physical activity compliance in the patients combining the behavioral method of influencing and reinforcing a positive behavior to the cognitive approach of conditioning emotions and human behavior by thoughts [71].

In summary, dietary intervention should respect physiological and metabolic bases. Any exception should take in account coexistent metabolic impairments and is allowed only if supported by clinical scientific evidences. The effectiveness of the dietary *therapy* should be evaluated in risk reduction for mortality and morbidity and in the ability of maintaining the results achieved rather than considering the BW loss only. The dietary intervention must follow a thorough *multidimensional* assessment of the biological (nutritional status), psychological, and social indices that could affect the BW gain and the unhealthy food habits. Since among the "dieters" there is a dropout rate of 40 % after 12 months [72], while a long-term success occurs only in ≤15 % [73], it is necessary to promote an active involvement of the patients, planning realistic solutions and goals to comply with, and trying to avoid unreachable achievements. Although the basis to formulate a balanced diet is strict scientific evidence, a high degree of *flexibility* is required to reach a good compliance of the patient [74, 75]. A good experience and knowledge by the professional operators can turn the dietary prescription into a guideline for a nutritional "reeducational" intervention.

References

Introduction

1. Wing RR, Phelan S (2005) Long-term weight loss maintenance. Am J Clin Nutr 82(1 Suppl):222S–225S. Review. PubMed PMID: 16002825
2. Elte JW, Castro Cabezas M, Vrijland WW, Ruseler CH, Groen M, Mannaerts GH (2008) Proposal for a multidisciplinary approach to the patient with morbid obesity: the St. Franciscus Hospital morbid obesity program. Eur J Intern Med 19(2):92–98. doi:10.1016/j.ejim.2007.06.015. Epub 2008 Jan 11. Review. PubMed PMID: 18249303
3. World Health Organisation (2004) Global strategy on diet, physical activity and health. WHO, Geneva, 700
4. Veerman JL, Barendregt JJ, van Beeck EF, Seidell JC, Mackenbach JP (2007) Stemming the obesity epidemic: a tantalizing prospect. Obesity (Silver Spring) 15(9):2365–2370. PubMed PMID: 17890506
5. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y, Armstrong R, Prosser L, Summerbell CD (2011) Interventions for preventing obesity in children. Cochrane Database Syst Rev (12):CD001871. doi:10.1002/14651858.CD001871.pub3. Review. PubMed PMID: 22161367
6. Clifton PM (2008) Dietary treatment for obesity. Nat Clin Pract Gastroenterol Hepatol 5(12): 672–681. doi:10.1038/ncpgasthep1283. Epub 2008 Oct 14. Review. PubMed PMID: 18852729
7. Freedman MR, King J, Kennedy E (2001) Popular diets: a scientific review. Obes Res 9(Suppl 1):1S–40S. Review. PubMed PMID: 11374180

High Protein Versus Standard Protein Diets for Weight Loss: The Issue of Adequate Protein Intake

8. Wycherley TP, Moran LJ, Clifton PM, Noakes M, Brinkworth GD (2012) Effects of energy-restricted high-protein, low-fat compared with standard-protein, low-fat diets: a meta-analysis of randomized controlled trials. Am J Clin Nutr 96(6):1281–1298. doi:10.3945/ajcn.112.044321. Epub 2012 Oct 24. Review. PubMed PMID: 23097268
9. Santesso N, Akl EA, Bianchi M, Mente A, Mustafa R, Heels-Ansdell D, Schünemann HJ (2012) Effects of higher- versus lower-protein diets on health outcomes: a systematic review and meta-analysis. Eur J Clin Nutr 66(7):780–788. doi:10.1038/ejcn.2012.37. Epub 2012 Apr 18. Review. PubMed PMID: 22510792; PubMed Central PMCID: PMC3392894
10. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA)(2012) Scientific opinion on dietary reference values for protein. EFSA Journal 10(2):2557 [66 pp.]. doi:10.2903/j.efsa.2012. 2557. Available online: www.efsa.europa.eu/efsajournal
11. Weigle DS, Breen PA, Matthys CC, Callahan HS, Meeuws KE, Burden VR, Purnell JQ (2005) A high-protein diet induces sustained reductions in appetite, ad libitum caloric intake, and body weight despite compensatory changes in diurnal plasma leptin and ghrelin concentrations. Am J Clin Nutr 82(1):41–48. PubMed PMID: 16002798
12. Paddon-Jones D, Westman E, Mattes RD, Wolfe RR, Astrup A, Westerterp-Plantenga M (2008) Protein, weight management, and satiety. Am J Clin Nutr 87(Suppl):S1558–S1561
13. USDA/ARS (United States Department of Agriculture Agricultural Research Service) (2009) USDA National Nutrient Database for Standard Reference, Release 22. Nutrient Data Laboratory Home Page. Available from: http://www.ars.usda.gov/ba/bhnrc/ndl
14. National Heart, Lung, and Blood Institute in cooperation with The National Institute of Diabetes and Digestive and Kidney Diseases (1998) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. The evidence report. NIH publication no. 98-4083 National Institutes of Health
15. Krieger JW, Sitren HS, Daniels MJ, Langkamp-Henken B (2006) Effects of variation in protein and carbohydrate intake on body mass and composition during energy restriction: a meta-regression 1. Am J Clin Nutr 83(2):260–274. PubMed PMID: 1646998
16. Martens EA, Westerterp-Plantenga MS (2014) Protein diets, body weight loss and weight maintenance. Curr Opin Clin Nutr Metab Care 17(1):75–79. doi:10.1097/MCO.0000000000000006. PubMed PMID: 24310056
17. WHO/FAO/UNU (World Health Organization/Food and Agriculture Organization of the United Nations/United Nations University) (2007) Protein and amino acid requirements in human nutrition. Report of a joint WHO/FAO/UNU expert consultation. WHO technical report series, no 935, 284 pp
18. Smith WJ, Underwood LE, Clemmons DR (1995) Effects of caloric or protein restriction on insulin-like growth factor-I (IGF-I) and IGF-binding proteins in children and adults. J Clin Endocrinol Metab 80(2):443–449. PubMed PMID: 7531712
19. Pellett PL, Young VR (1992) The effects of different levels of energy intake on protein metabolism and of different levels of protein intake on energy metabolism: a statistical evaluation from the published literature. In: Scrimshaw NS, Schürch B (eds) Protein energy interactions. International Dietary Energy Consultancy Group Switzerland, Lausanne, pp 81–136
20. Guillet C, Masgrau A, Walrand S, Boirie Y (2012) Impaired protein metabolism: interlinks between obesity, insulin resistance and inflammation. Obes Rev 13(Suppl 2):51–57. doi:10.1111/j.1467-789X.2012.01037.x
21. Westerterp-Plantenga MS, Lemmens SG, Westerterp KR (2012) Dietary protein – its role in satiety, energetics, weight loss and health. Br J Nutr 108(Suppl 2):S105–S112. doi:10.1017/S0007114512002589. Review. PubMed PMID: 23107521
22. Pasiakos SM, Cao JJ, Margolis LM, Sauter ER, Whigham LD, McClung JP, Rood JC, Carbone JW, Combs GF Jr, Young AJ (2013) Effects of high-protein diets on fat-free mass and muscle protein synthesis following weight loss: a randomized controlled trial. FASEB J 27(9):3837–3847. doi:10.1096/fj.13-230227. Epub 5. PubMed PMID: 23739654

23. Tang M, Armstrong CL, Leidy HJ, Campbell WW (2013) Normal vs. high-protein weight loss diets in men: effects on body composition and indices of metabolic syndrome. Obesity (Silver Spring) 21(3):E204–E210. doi:10.1002/oby.20078. PubMed PMID: 23592676
24. Soenen S, Martens EA, Hochstenbach-Waelen A, Lemmens SG, Westerterp-Plantenga MS (2013) Normal protein intake is required for body weight loss and weight maintenance, and elevated protein intake for additional preservation of resting energy expenditure and fat free mass. J Nutr 143(5):591–596. doi:10.3945/jn.112.167593. Epub 2013 Feb 27. PubMed PMID: 23446962

Low Versus Standard Carbohydrate Diets for Weight Loss: The Matter of Glycemic Index/Load in Weight Management

25. Sumithran P, Proietto J (2007) Ketogenic diets for weight loss: a review of their principles, safety and efficacy. Obes Res Clin Pract. 2008;2(1):I–II. doi: 10.1016/j.orcp.11.003. PubMed PMID: 24351673
26. Fitch A, Everling L, Fox C, Goldberg J, Heim C, Johnson K, Kaufman T, Kennedy E, Kestenbaun C, Lano M, Leslie D, Newell T, O'Connor P, Slusarek B, Spaniol A, Stovitz S, Webb B (2013) Institute for Clinical Systems Improvement. Prevention and management of obesity for adults. Updated. https://www.healthpartners.com/ucm/groups/public/@hp/@public/documents/documents/cntrb_037112.pdf
27. Tian H, Mills KT, Yao L, Demanelis K, Eloustaz M, Yancy WS, Jr TN, Kelly JH, Bazzano LA (2012) Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors: a meta-analysis of randomized controlled clinical trials. Am J Epidemiol 176(Suppl):S44–S54
28. Focster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S (2003) A randomized trial of a low-carbohydrate diet for obesity. N Engl J Med 348(21):2082–2090. PubMed PMID: 12761365
29. Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF (2004) The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. Ann Intern Med 140(10):778–785. PubMed PMID: 15148064
30. Clifton PM, Condo D, Keogh JB (2014) Long term weight maintenance after advice to consume low carbohydrate, higher protein diets – a systematic review and metaanalysis. Nutr Metab Cardiovasc Dis 24(3):224–235. doi:10.1016/j.numecd.2013.11.006. Epub 20. PubMed PMID: 24472635
31. Saris WH (2003) Sugars, energy metabolism, and body weight control. Am J Clin Nutr 78(4):850S–857S. Review. PubMed PMID: 14522749
32. Astrup A, Meinert Larsen T, Harper A (2004) Atkins and other low-carbohydrate diets: hoax or an effective tool for weight loss? Lancet 364(9437):897–899. Review. PubMed PMID: 15351198
33. Mobbs CV, Mastaitis J, Yen K, Schwartz J, Mohan V, Poplawski M, Isoda F (2007) Low-carbohydrate diets cause obesity, low-carbohydrate diets reverse obesity: a metabolic mechanism resolving the paradox. Appetite 48(2):135–138. Epub 2006 Dec 1. Review. PubMed PMID: 17141367; PubMed Central PMCID: PMC2714161
34. Schröder H, Marrugat J, Vila J, Covas MI, Elosua R (2004) Adherence to the traditional mediterranean diet is inversely associated with body mass index and obesity in a spanish population. J Nutr 134(12):3355–3361. PubMed PMID: 15570037
35. Thomas DE, Elliott EJ, Baur L (2007) Low glycaemic index or low glycaemic load diets for overweight and obesity. Cochrane Database Syst Rev (3):CD005105. Review. PubMed PMID: 17636786
36. McMillan-Price J, Brand-Miller J (2006) Low-glycaemic index diets and body weight regulation. Int J Obes 30(Suppl 3):S40–S46
37. Schwingshackl L, Hoffmann G (2013) Long-term effects of low glycemic index/load vs. high glycemic index/load diets on parameters of obesity and obesity-associated risks: a systematic review and meta-analysis. Nutr Metab Cardiovasc Dis 23(8):699–706. doi:10.1016/j.numecd.2013.04.008. Epub 2013 Jun 17. Review. PubMed PMID: 23786819

38. Layman DK, Boileau RA, Erickson DJ, Painter JE, Shiue H, Sather C, Christou DD (2003) A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women. J Nutr 133(2):411–417. PubMed PMID: 12566476
39. Campbell DD, Meckling KA (2012) Effect of the protein: carbohydrate ratio in hypoenergetic diets on metabolic syndrome risk factors in exercising overweight and obese women. Br J Nutr 108(9):1658–1671. doi:10.1017/S0007114511007215. PubMed PMID: 22243943

Low Fat Diet or Normal Fat Diet: One or more Reference Model for Weight Loss?

40. Schwingshackl L, Hoffmann G (2013) Comparison of effects of long-term low-fat vs high-fat diets on blood lipid levels in overweight or obese patients: a systematic review and meta-analysis. J Acad Nutr Diet 113:1640–1661 (B)
41. Dattilo AM, Kris-Etherton PM (1992) Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. Am J Clin Nutr 56(2):320–328. PubMed PMID:1386186
42. Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, Golan R, Fraser D, Bolotin A, Vardi H, Tangi-Rozental O, Zuk-Ramot R, Sarusi B, Brickner D, Schwartz Z, Sheiner E, Marko R, Katorza E, Thiery J, Fiedler GM, Blüher M, Stumvoll M, Stampfer MJ, Dietary Intervention Randomized Controlled Trial (DIRECT) Group (2009) Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. N Engl J Med 359(3):229–241. doi:10.1056/NEJMoa0708681; Erratum in: N Engl J Med. 31;361(27):2681. PubMed PMID: 18635428
43. Due A, Larsen TM, Hermansen K, Stender S, Holst JJ, Toubro S, Martinussen T, Astrup A (2008) Comparison of the effects on insulin resistance and glucose tolerance of 6-mo high-monounsaturated-fat, low-fat, and control diets. Am J Clin Nutr 87(4):855–862. PubMed PMID: 18400707

The Very-Low-Energy Diets (VLED) and the Ketogenic Diet

44. Tsai AG, Wadden TA (2006) The evolution of very-low-calorie diets: an update and meta-analysis. Obesity (Silver Spring) 14(8):1283–1293. Review. PubMed PMID: 1698807
45. U.S. Department of Health and Human Services (August 2008 Updated December 2012) Very low-calorie diets. WIN Weight-control Information Network. NIH publication no. 03–3894
46. Mulholland Y, Nicokavoura E, Broom J, Rolland C (2012) Very-low-energy diets and morbidity: a systematic review of longer-term evidence. Br J Nutr 108(5):832–851. doi:10.1017/S0007114512001924. Epub 2012 Jul 17. Review. PubMed PMID: 22800763
47. Fricker J, Rozen R, Melchior JC, Apfelbaum M (1991) Energy-metabolism adaptation in obese adults on a very-low-calorie diet. Am J Clin Nutr 53(4):826–830. PubMed PMID: 2008860
48. Yang MU, Van Itallie TB (1976) Composition of weight lost during short-term weight reduction. Metabolic responses of obese subjects to starvation and low-calorie ketogenic and nonketogenic diets. J Clin Invest 58(3):722–730. PubMed PMID: 956398; PubMed Central PMCID: PMC333231
49. Bistrian BR (1978) Recent developments in the treatment of obesity with particular reference to semistarvation ketogenic regimens. Diabetes Care 1(6):379–384. PubMed PMID: 729452
50. Paoli A, Rubini A, Volek JS, Grimaldi KA (2013) Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets. Eur J Clin Nutr 67(8):789–796. doi:10.1038/ejcn.2013.116. Epub 2013 Jun 26. Review. PubMed PMID: 23801097, PubMed Central PMCID: PMC3826507

51. Stock AL, Yudkin J (1970) Nutrient intake of subjects on low carbohydrate diet used in treatment of obesity. Am J Clin Nutr 23(7):948–952. PubMed PMID: 5455557
52. Laffel L (1999) Ketone bodies: a review of physiology, pathophysiology and application of monitoring to diabetes. Diabetes Metab Res Rev 15(6):412–426. Review. PubMed PMID: 10634967
53. Clarke K, Tchabanenko K, Pawlosky R, Carter E, Todd King M, Musa-Veloso K, Ho M, Roberts A, Robertson J, Vanitallie TB, Veech RL (2012) Kinetics, safety and tolerability of (R)-3-hydroxybutyl (R)-3-hydroxybutyrate in healthy adult subjects. Regul Toxicol Pharmacol 63(3):401–408. doi:10.1016/j.yrtph.2012.04.008. Epub 2012
54. Bergqvist AG, Schall JI, Stallings VA, Zemel BS (2008) Progressive bone mineral content loss in children with intractable epilepsy treated with the ketogenic diet. Am J Clin Nutr 88(6):1678–1684. doi:10.3945/ajcn.2008.26099. PubMed PMID: 19064531
55. McPherson PA, McEneny J (2012) The biochemistry of ketogenesis and its role in weight management, neurological disease and oxidative stress. J Physiol Biochem 68(1):141–151. doi:10.1007/s13105-011-0112-4. Epub 2011 Oct 8. Review.PubMed PMID: 21983804

Criteria for the Formulation of a Balanced Diet in Obese Patient

56. Società Italiana di Nutrizione Umana (S.I.N.U.) (2000) Livelli di assunzione raccomandati di energia e nutrienti per la popolazione italiana. LARN. Revisione 1996. Ed. EDRA, Milano
57. Elia M (1992) Organ and tissue contribution to metabolic rate. In: Kinney JM, Tucker HN (eds) Energy metabolism. Tissue determinants and cellular corollarie. Raven Press, New York, pp 61–79
58. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO (1990) A new predictive equation for resting expenditure in healthy individuals. Am J Clin Nutr 51:241–247
59. Frankenfield D, Roth-Yousey L, Compher C (2005) Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc 105(5):775–789. Review. PubMed PMID: 15883556
60. Frankenfield DC (2013) Bias and accuracy of resting metabolic rate equations in non-obese and obese adults. Clin Nutr 32(6):976–982. doi:10.1016/j.clnu.2013.03.022. Epub. PubMed PMID: 23631843
61. Cunningham J (1980) A reanalysis of the factor influencing basal metabolism rate in normal adults. Am J Clin Nutr 33:2372–2374
62. Polito A, Ferro Luzzi A (2006) Energia: misura e bisogni. In: Costantini MA, Cannella C, Tomassi G. Il Pensiero scientifico editore, Rome (2nd eds): pp 215–250
63. Report of a Joint FAO/WHO/UNU expert consultation on energy and protein requirements. WHO technical report series 724, Geneva 1985 – ISBN 92 4 120724 8, Reprinted 1987, 1991
64. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Bassett DR Jr, Tudor-Locke C, Greer JL, Vezina J, Whitt-Glover MC, Leon AS (2011) Compendium of physical activities: a second update of codes and MET values. Med Sci Sports Exerc 43(8):1575–1581. doi:10.1249/MSS.0b013e31821ece12. PubMed PMID: 21681120
65. Committee to develop criteria for evaluating the outcomes of approaches to prevent and treat obesity, food and nutrition board, institute of medicine, National Academy of Sciences. Weighting the options: criteria for evaluating weight-management programs. Edizione italiana a cura della Task Force Obesità Italia (TFOI). ed. Pendragon, 1998
66. Donini LM, Pinto A, Cannella C (2004) Diete iperproteiche ed obesità. Ann Ital Med Int 19:36–42
67. Società Italiana di Nutrizione Umana (S.I.N.U.) (2012) LARN – Livelli di Assunzione di Riferimento di Nutrienti ed energia per la popolazione italiana. Revisione 2012. http://www.sinu.it/documenti/20121016_LARN_bologna_sintesi_prefinale.pdf

Nutritional Counseling and Conclusion

68. Panagiotakos DB, Chrysohoou C, Pitsavos C, Stefanadis C (2006) Association between the prevalence of obesity and adherence to the Mediterranean diet: the ATTICA study. Nutrition 22(5):449–456. Epub 2006 Feb 2. PubMed PMID: 16457990
69. Corbalán MD, Morales EM, Canteras M, Espallardo A, Hernández T, Garaulet M (2009) Effectiveness of cognitive-behavioral therapy based on the Mediterranean diet for the treatment of obesity. Nutrition 25(7–8):861–869. doi:10.1016/j.nut.2009.02.013. PubMed PMID: 19539176
70. Mucchielli R. Apprendere il counseling (2001) Manuale di autoformazione al colloquio d'aiuto. Erickson, Trento. ISBN: 88-7946-053-6
71. Società Italiana dell'obesità (SIO)– Associazione Italiana di Dietetica e Nutrizione Clinica (ADI) (2012/2013) Standard Italiani per la cura dell'obesità. http://www.sio-obesita.org/Standard.pdf
72. Bach-Faig A, Berry EM, Lairon D, Reguant J, Trichopoulou A, Dernini S, Medina FX, Battino M, Belahsen R, Miranda G, Serra-Majem L, Mediterranean Diet Foundation Expert Group (2011) Mediterranean diet pyramid today. Science and cultural updates. Public Health Nutr 14(12A):2274–2284. doi:10.1017/S1368980011002515. Review. PubMed PMID: 22166184
73. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ (2005) Comparison of the atkins, ornish, weight watchers, and zone diets for weight loss and heart disease risk reduction: a randomized trial. JAMA 293:43–53
74. Ayyad C, Andersen T (2000) Long-term efficacy of dietary treatment of obesity: a systematic review of studies published between 1931 and 1999. Obes Rev 1:113–119
75. World Health Organization (WHO) (2000) Obesity: preventing and managing the global epidemic, WHO technical report series, no 894. Consultation on obesity, Geneva, 3–5 June 1997 (Switzerland), 252 p. ISBN 9241208945. Languages: English (reprinted 2004), French (2003)