CHAPTER THREE

Dietary Strategies to Increase Satiety

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Abstract

Obesity has a multifactorial etiology. Although obesity is widespread and associated with serious health hazards, its effective prevention and treatment have been challenging. Among the currently available treatment approaches, lifestyle modification to induce a negative energy balance holds a particularly larger appeal due to its wider reach and relative safety. However, long-term compliance with dietary modifications to reduce energy intake is not effective for the majority. The role of many individual nutrients, foods, and food groups in inducing satiety has been extensively studied. Based on this evidence, we have developed sample weight-loss meal plans that include multiple satiating foods, which may collectively augment the satiating properties of a meal. Compared to a typical American diet, these meal plans are considerably lower in energy density and probably more satiating. A diet that exploits the satiating properties of multiple foods may help increase long-term dietary compliance and consequentially enhance weight loss.

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1. INTRODUCTION

Overweight and obese individuals are susceptible to several medical conditions that contribute to morbidity and mortality including diabetes and cardiovascular disease (Guh et al., 2009). According to the results of the National Health and Nutrition Examination Survey, 69.2% of adults in the United States and 16.9% of children and adolescents were overweight or obese in 2009–2010 (Flegal, Carroll, Kit, & Ogden, 2012; Ogden, Carroll, Kit, & Flegal, 2012). The prevalence of obesity among both males (35.5%) and females (35.8%) has not significantly changed in the two most recent years (2009–2010), as compared with the previous 6 years (Flegal et al., 2012). Although there is evidence for a leveling off in the prevalence of obesity (Rokholm, Baker, & Sorensen, 2010), the rise in health care costs attributable to overweight and obesity has been predicted to account for 16–18% of total U.S. health care costs (860.7–956.9 billion US dollars) by 2030 (Wang, Beydoun, Liang, Caballero, & Kumanyika, 2008). It is estimated that an obese individual in the United States is associated with approximately $1723 of additional medical spending per year (Tsai, Williamson, & Glick, 2011). The United States has a high per capita expenditure on health care; yet, life expectancy in the United States ranked 34th in the world in 2009 (WHO, 2011). Obesity has reduced longevity in several industrialized countries; however, the effects are more severe in the United States, which may be due to the unusually high rate of obesity in younger age groups and higher rates of severe obesity (Preston & Stokes, 2011).

Obesity, is a multifaceted problem with contributing factors that are undoubtedly complex and include genetics (Schwarz, Rigby, La Bounty, Shelmadine, & Bowden, 2011), endocrine function, behavioral patterns, and their environmental determinants (Gortmaker et al., 2011). Besides the well-known factors, several other putative contributors of obesity have been considered (McAllister et al., 2009). There is little argument about the health hazards of obesity. However, it is challenging to produce large and meaningful weight loss, and easy to regain weight. Although some research studies show weight loss and its benefits, there is an inability to successfully translate weight-loss strategies from a research setting to the general public. This issue was illustrated in a recent editorial as follows: "Consider a recent trial of weight loss and another of weight maintenance, which lasted an impressive two and three years, respectively (Sacks et al., 2009; Svetkey et al., 2008). These trials conducted by some of the world's foremost experts, on highly motivated individuals, who were closely monitored and supervised, resulted in about 4 kg reduction from about 100 kg starting body weight. While the results were statistically significant, their biological significance may be questioned. Will an approach that costs millions of dollars to produce less than 4% weight loss or maintenance, succeed in combating the global obesity epidemic in the free living population, where the facilities, the expertise of health care professionals, and the motivation of subjects is likely to be inferior to that in these studies?" (Dhurandhar, 2012).

Due to a lack of better alternatives, lifestyle modification, and obesity drugs or surgery in appropriately selected cases form the cornerstone of obesity management despite their limitations. Among these approaches, lifestyle modification to induce a negative energy balance holds a particularly larger appeal due to its wider reach and relative safety. Theoretically, negative energy balance could be achieved by increasing physical activity and reducing energy intake. However, recent studies demonstrate the inability of exercise by itself to make a significant contribution to weight loss (Thomas et al., 2012). This implies that the majority of the responsibility to induce a long-term negative energy balance that is adequate to reduce weight must be shouldered by dietary manipulations. However, modifying diet to reduce the long-term energy intake may work well for some responders but does not achieve adequate and lasting benefits for the majority. In most cases, the nonresponders or poor responders often outnumber the responders. It is time to recognize this limitation so that novel weight management approaches could be designed that are practical and effective.

Success of a weight-loss dietary regimen is closely related to compliance, which, in turn, is largely dependent on hunger, appetite, and satiety. As indicated by extensive research in the psychology, physiology, behavior, endocrinology, and pharmacology of hunger, appetite, and satiety, the significance of these factors is clearly well recognized. Much of the research about the effect of nutrients, foods, or food groups focuses on individual items. The role of many individual nutrients, foods, and food groups in inducing satiety is known, but not much attention has been focused on exploiting the combined effect of multiple satiating foods in a dietary regimen. Here, we have extensively and selectively reviewed the information available about the satiating and weight-loss properties of various common foods that may be relevant to a weight-loss regimen. The available evidence could be categorized into three groups: (a) effect on perceptions of satiety, (b) effect on actual food intake, and (c) weight loss as a result of a diet containing a satiating food. Based on this evidence, we have developed sample weight-loss meal plans that include multiple satiating foods, which may collectively augment the satiating properties of a meal.

1.1. Appetite and satiety

Appetite results from a convergence of several factors related to biology and the environment, referred to as the psychobiological system. It reflects the synchronous operation of events occurring on three levels: (1) psychological events and behavior, (2) peripheral physiology, and (3) the central nervous system (Blundell, 1999). However, apart from the desire to satisfy appetite sensations, in humans, sensory hedonics, sensory stimulation, tension reduction, social pressure, and boredom can also trigger an eating episode (de Graaf, Blom, Smeets, Stafleu, & Hendriks, 2004). Thus, a comprehensive definition of appetite would encompass the whole field of food intake, selection, motivation, and preference (Blundell et al., 2010).

Satiety is the process that inhibits further eating, causes a reduction in hunger, and an increase in fullness after a meal is eaten; whereas, the inhibitory processes that lead to the termination of a meal cause satiation. Though satiation and satiety are distinct concepts, they act together along with a host of other factors to determine eating behavior (Blundell et al., 2010). The satiety cascade proposed several years ago provides a framework for examining the processes (sensory, cognitive, post-ingestive, and post-absorptive) that mediate the satiating effect of foods (Blundell, 1999). A more recent modification to the satiety cascade includes the concepts of "liking" which is the pleasure derived from the orosensory stimulation of food and "wanting" which refers to the desire or motivation to actually engage in eating (Mela, 2006).

Eating behavior is controlled by metabolic factors that drive appetite and satiety, and sensory factors that influence food choice. In the brain, the sensory signals of food are associated with the metabolic consequences leading to a conditioning of eating and nutrition patterns. Cognitive factors such as an estimation of the satiating effect of foods and the timing of the next meal contribute to making eating a learned behavior (Blundell et al., 2010). It has been argued that food intake is controlled by an integrated set of signals at a momentary level. These signals are liable to change with the environment in which food is available, as each combination of items is consumed, and with every change in the physical and social context (Booth, 2008). By this argument, food intake tests and appetite-rating scales fall short in their assessment of satiety as they do not consider the changing influences over eating within, before, or after the test period, reactive responses, and the mental state of the individual at the precise moment that the quantitative judgment is being expressed (Booth, 2009).

Short-term control of eating is influenced by episodic signals that arise mainly from the gastrointestinal (GI) tract and are generated at regular intervals as food intake occurs. The peptides involved in GI signaling include cholecystokinin, glucagon-like peptide-1 (GLP-1), peptide YY (PYY), and ghrelin. Long-term control of eating, also referred to as tonic signaling, reflects the metabolic state of adipose tissue. Tonic or enduring effects influence traits (stable predispositions), whereas episodic or transient control influences states (dispositions subject to rapid fluctuations) (Blundell et al., 2008). Psychometric tests such as the Eating Inventory (Stunkard & Messick, 1985) are used to identify traits that predispose individuals to restrained eating or a lack of control over eating or susceptibility to hunger. States reflect the drive to eat, are expressed as appetite ratings, and are measured using visual analog scales (VAS) (Blundell et al., 2008).

In a review of studies (de Graaf et al., 2004) that assessed satiety using subjective ratings of appetite, or by measuring actual energy intake, the vast majority of the studies showed that appetite ratings correlated with food intake in a standardized setting. Further, subjective appetite ratings and food intake were associated with changes in hormone concentrations (de Graaf et al., 2004). Subjective satiety responses usually correlate with the time of occurrence and magnitude of the effect of physiological processes such as increases in gastric volume and the absorption of nutrients (Blundell et al., 2010). Nevertheless, satiety claims do not need to be substantiated by physiologic data (Blundell, 2010). Appetite scores measured through VAS can be reproduced and are therefore feasible tools to measure appetite and satiety sensations (Blundell et al., 2010; Cardello, Schutz, Lesher, & Merrill, 2005; Flint, Raben, Blundell, & Astrup, 2000). Moreover, appetite and satiety have been found to be good determinates of food intake (Drapeau et al., 2005; Drapeau et al., 2007).

Macronutrient composition, energy density, and physical structure influence satiety (Blundell et al., 2010). Different amino acids, fatty acids, and carbohydrates have differing effects on the markers of appetite regulation (de Graaf et al., 2004). The differing effects imply that each nutrient interacts with the processes that mediate satiety in different ways (Blundell, 1999). Dietary protein may promote weight loss by increasing energy expenditure and stimulating satiety (Lejeune, Westerterp, Adam, Luscombe-Marsh, & Westerterp-Plantenga, 2006; Smeets, Soenen, Luscombe-Marsh, Ueland, & Westerterp-Plantenga, 2008; Westerterp-Plantenga, Nieuwenhuizen, Tome, Soenen, & Westerterp, 2009; Westerterp-Plantenga, Rolland, Wilson, & Westerterp, 1999). Carbohydrates that evade digestion, or which have a pronounced effect on glucose metabolism, have the potential to produce changes in appetite and affect satiety (van Dam & Seidell, 2007). Similarly, novel oils designed to prolong their presence in the GI tract can potentially produce beneficial effects on appetite and satiety (Halford & Harrold, 2012). The challenge lies in understanding which components of food interact optimally with the mediating processes to influence food intake. While the focus of this chapter is the effect of macronutrient composition, and energy density, of foods on appetite and satiety, the effect on body weight will also be explored.

2. DIETARY PROTEIN AND THE REGULATION OF FOOD INTAKE AND BODY WEIGHT

The most satiating macronutrient appears to be protein (Westerterp-Plantenga et al., 2009). High-protein diets with their potential to act on metabolic targets regulating body weight have become the subject of a body of research spanning 40 years (Halford & Harrold, 2012). Results from intervention studies suggest that an increase in the relative protein content of the diet reduces the risk of positive energy balance and the progress to weight gain (Clifton, Keogh, & Noakes, 2008; Weigle et al., 2005). Dietary protein may promote weight loss by increasing energy expenditure and by inducing satiety (Lejeune et al., 2006). In short-term studies (lasting for 24 h up to 5 days), evaluating subjective satiety sensations, high-protein diets have been shown to induce greater satiety than isoenergetic intakes of carbohydrate and fat (Lejeune et al., 2006; Westerterp-Plantenga et al., 1999).

Protein intake influences energy expenditure primarily through its effects on diet-induced thermogenesis. The thermic effect of nutrients is related to the adenosine triphosphate required for metabolism, storage, and oxidation (Westerterp-Plantenga et al., 2009). Three phosphate bonds are utilized for the incorporation of each amino acid into protein (Tome, Schwarz, Darcel, & Fromentin, 2009). The body is unable to store protein under conditions of high-protein intake and has to metabolize it which increases thermogenesis. Ultimately, resting metabolic rate also increases as a result of protein synthesis and protein turnover (Westerterp-Plantenga et al., 2009). In a study (Crovetti, Porrini, Santangelo, & Testolin, 1998) evaluating energy expenditure after consuming isoenergetic high-carbohydrate, high-protein, and high-fat meals, protein was found to be the most thermogenic nutrient and resulted in the greatest increase in sensations of fullness. Additionally, the study demonstrated a correlation between the thermic effect of food and eating behavior.

In a crossover trial (Lejeune et al., 2006), normal-weight women were prescribed an adequate protein diet consisting of 10% of energy from protein or a high-protein diet consisting of 30% of energy from protein. It was found that energy expenditure measured in a respiration chamber was greater during the higher protein intervention as a result of increased diet-induced thermogenesis and resting metabolic rate. In another respiratory chamber experiment, satiety and thermogenesis were shown to increase with a high-protein diet. Further, satiety was positively related to 24-h diet-induced thermogenesis (Westerterp-Plantenga et al., 1999).

Gluconeogenesis or the *de novo* synthesis of glucose from noncarbohydrate sources including amino acids is stimulated in the fed state by a high-protein diet (Veldhorst, Westerterp, & Westerterp-Plantenga, 2011). The energy expenditure that it requires as well as modulation of glucose homeostasis may have a role to play in protein-induced energy expenditure (Tome et al., 2009; Westerterp-Plantenga et al., 2009). Hepatic gluconeogenesis may be an alternative biochemical pathway to metabolize amino acids consumed in excess of requirements (Westerterp-Plantenga et al., 2009). However, not all amino acids can be used in gluconeogenesis; hence, the amino acid composition of the diet may have an effect on postprandial gluconeogenesis (Westerterp-Plantenga et al., 2009). In a comparison between a high-protein (30% of energy) and low-protein (12% of energy) diet, an increase in gluconeogenesis with the high-protein diet was demonstrated. However, there was no correlation between appetite ratings and gluconeogenesis (Veldhorst et al., 2011).

It has been hypothesized that protein-induced satiety is related to increased concentrations of the anorexigenic hormones GLP-1 and PYY and a decrease in the orexigenic hormone ghrelin (Batterham et al., 2006; Lejeune et al., 2006). In the respiratory chamber experiment (Lejeune et al., 2006), GLP-1 concentrations were measured nine times throughout the day, on the fourth day of consuming each of the diets. After dinner, GLP-1 concentrations were significantly higher on the high-protein diet when compared with the adequate protein diet. Energy expenditure, protein balance, and fat oxidation were also significantly higher on the high-protein diet in comparison with the adequate protein diet. Although ghrelin concentrations decreased, it could not be clearly attributed to the protein content of the diet, as the adequate protein diet with a relatively high-carbohydrate content also resulted in a decrease in ghrelin concentrations. Additionally, the increase in GLP-1 was related to the increase in satiety (Lejeune et al., 2006).

In a 3-week crossover trial, an effect of protein consumption on PYY concentrations was demonstrated with significantly higher plasma PYY and greater satiety responses to a high-protein meal in normal weight and obese individuals as compared with isoenergetic high-fat and high-carbohydrate meals (Batterham et al., 2006). However, in another study (Smeets et al., 2008), there were no differences in ghrelin and PYY responses between a high-protein (25% of energy) and average-protein (10% of energy) diet. GLP-1 response was in fact lower following the high-protein meal as compared with the average-protein (but higher carbohydrate) meal.

Specific amino acids may influence satiety by virtue of the fact that they are precursors for certain neurotransmitters involved in the regulation of appetite and body weight. Tryptophan is a precursor for the neurotransmitter serotonin. Tyrosine can be converted into the neurotransmitters dopamine and norepinephrine, and histadine can be converted into the neurotransmitter histamine. Each of these neurotransmitters has been linked with food intake regulation, although there is no direct evidence for their role in protein-induced satiety (Westerterp-Plantenga et al., 2009).

In a hypothesis propounded by Stock (1999), overeating a low- or high-protein diet is less metabolically efficient than overeating an average-protein diet (10–15% of energy). The metabolic inefficiency of a diet low or high in dietary protein stems from the energy cost involved in sparing lean body mass with a low-protein diet and building lean body mass with a high-protein diet (Westerterp-Plantenga et al., 2009). Energy intake required to build 1 kg of fat-free mass is much higher than the energy intake needed to build 1 kg of body weight with 60% fat mass and 40% fat-free mass (Stock, 1999). The data published in a recent study (Bray et al., 2012) demonstrated the metabolic inefficiency of low-protein diets. Weight gain in the low-protein group (3.16 kg) was less than in the normal (6.05 kg) and high-protein (6.51 kg) groups when the same number of extra calories was eaten over 56 days. Failure to increase lean body mass in the low-protein group accounted for their smaller weight gain.

By regulating food intake, diets high in protein have been shown to cause weight loss and affect body composition. The Recommended Dietary Allowance (RDA) for protein is 0.8 g/kg of body weight per day (NAP, 2005). In a meta-regression (Krieger, Sitren, Daniels, & Langkamp-Henken, 2006) classifying high-protein diets as >1.05 g/kg and low-protein diets as <1.05 g/kg of body weight per day, it was found that there was a greater association between fat-free mass retention and a high-protein diet as compared with a diet closer to the RDA (mean intake: 0.74 g/kg of body weight per day). Interestingly, in this meta-regression, while protein was found to be a good predictor of fat-free mass retention, it was not found to be a predictor of weight loss.

When subjects received a high-protein diet (30%, 50%, and 20% of energy from protein, carbohydrate, and fat, respectively), under controlled feeding conditions, satiety increased but body weight remained stable (Weigle et al., 2005). When the same diet was continued and subjects were advised to eat as much as they wanted (*ad libitum*), there was a sustained decrease in energy intake resulting in a weight loss, 76% of which comprised fat loss. In this study, protein was increased at the expense of the more energy-dense fat. In a cross-over trial (Blatt, Roe, & Rolls, 2011b), energy density was controlled, but the protein content of the diets was varied (10%, 15%, 20%, 25%, and 30% of energy). *Ad libitum* energy intake over 24 h did not vary significantly between the different protein levels. Additionally, satiety ratings were not significantly different across conditions.

Long-term (64 weeks) effects of a high-protein weight-loss diet (34%, 46%, and 20% of energy from protein, carbohydrate, and fat, respectively) were compared with an isoenergetic high-carbohydrate diet (17%, 64%, and 20% of energy from protein, carbohydrate, and fat, respectively) (Clifton et al., 2008). Weight loss was greater in participants who reported consuming a diet higher in protein. In a meta-analysis comparing high-protein–low-carbohydrate diets with high-carbohydrate–low-fat diets, weight loss was significantly greater with the high-protein diets in studies lasting up to 6 months, but the differences were not significant in studies spanning 12 months (Hession, Rolland, Kulkarni, Wise, & Broom, 2009).

Consumption of high-protein diets is not without some health risks. High levels of proteins can cause hypercalciuria and increase the rate of progression in renal dysfunction (Knight, Stampfer, Hankinson, Spiegelman, & Curhan, 2003). There appears to be evidence to indicate that high-protein diets enhance satiety and influence weight loss. However, the evidence that high-protein diets support weight maintenance over time is lacking, hence the need for long-term controlled trials on the safety and efficacy of high-protein diets.

2.1. Milk and milk products

Dairy products have been shown to induce satiety and reduce food intake (Dove et al., 2009; Gilbert et al., 2011; Harper, James, Flint, & Astrup, 2007). Additionally, dietary patterns that emphasize increased consumption of milk have been associated with the prevention of body weight gain (Drapeau et al., 2004). The regulatory effects on food intake and body weight associated with dairy intake have been attributed to the physiologic actions of its protein and calcium components (Gilbert et al., 2011; Lorenzen, Frederiksen, Hoppe, Hvid, & Astrup, 2012).

Casein and whey protein comprise 80% and 20%, respectively, of cow, sheep, goat, and buffalo milk (Luhovyy, Akhavan, & Anderson, 2007). Differences in the physical properties of casein and whey protein result in differing preabsorptive physiological effects. While whey protein remains soluble in the stomach and is emptied rapidly into the duodenum, casein forms a solid clot in the gastric acidic environment and is released slowly from the stomach (Boirie et al., 1997; Dangin, Boirie, Guillet, & Beaufrere, 2002). After whey protein ingestion, the plasma appearance of dietary amino acids is quick and high but of short duration. Further, it is associated with increased protein synthesis and oxidation with no change in protein catabolism. Casein ingestion results in a slower, more prolonged plasma appearance of amino acids. In comparison with whey, the metabolic response is different; it is accompanied by a considerable inhibition of protein breakdown, with a slight increase in synthesis and a moderate increase in oxidation (Boirie et al., 1997; Dangin et al., 2002). Both proteins, however, contribute to satiety following ingestion of dairy products (Luhovyy et al., 2007).

Milk proteins are a major source of a wide range of biologically active peptides. These peptides are inactive within the sequence of parent proteins and are released by enzymatic hydrolysis during digestion *in vivo*, or during processing of dairy products. Bioactive peptides in dairy products which have pharmacological similarities to opium are called opioid peptides. Opioid peptides are opioid receptor ligands with agonistic or antagonistic activities (Haque, Chand, & Kapila, 2009). Although endogenous opioid receptor agonists typically enhance feeding whereas antagonists inhibit feeding (Froetschel, 1996), their effects depend upon the macronutrient, individual food preference, and palatability of the food (Gosnell & Levine, 2009). Opioid peptides influence GI functions by affecting smooth muscles and reducing intestinal transit time (Haque et al., 2009). Opioid peptides known as β-casomorphins hydrolyzed from casein have been shown to delay gastric emptying and intestinal transit (Froetschel, 1996). The evidence linking opioid receptors to control of eating is supported largely by animal studies. From the evidence in human studies, it appears that opioid receptor antagonists have a small effect on hedonic taste preferences and short-term food intake. A number of clinical trials using naltrexone, an opioid receptor antagonist, have found no effects on body weight (Nathan & Bullmore, 2009).

As compared with other foods, whey proteins contain the highest concentrations of branched-chain amino acids (BCAA), especially leucine. Intracerebroventricular administration of either an amino acid mixture or leucine alone was shown to suppress 24-h food intake. It was determined that a rapamycin-dependent inhibition of Agouti-related protein gene expression contributed to the effect. Thus, increasing the amino acid concentrations within the brain suppressed food intake (Morrison, Xi, White, Ye, & Martin, 2007). BCAA, in general, and leucine, in particular, are first used for new protein synthesis, and diets that provide BCAA in excess of these requirements can support the intracellular leucine concentrations required to support other signaling pathways (Zemel, 2004).

Milk proteins stimulate the release of hormones involved in appetite regulation. Milk consumption modifies the insulinemic and glycemic response to carbohydrate-rich foods, with the whey fraction being the more efficient insulin secretagogue than casein (Nilsson, Stenberg, Frid, Holst, & Bjorck, 2004). Increased plasma concentrations of insulin have been associated with short-term satiety and decreased food intake (Samra, Wolever, & Anderson, 2007). The satiating effect of milk proteins has also been linked to the release of the anorexigenic hormones cholecystokinin, GLP-1, PYY, and suppression of the orexigenic hormone ghrelin (Luhovyy et al., 2007). Caseinomacropeptide, a digestion product of casein, collects in the whey fraction during cheese making. In rats, the glycosylated form of caseinomacropeptide, glycomacropeptide has been shown to increase pancreatic secretion (a marker of cholecystokinin) in a dose-dependent manner. Although glycomacropeptide may have potential as an appetite suppressant (Anderson & Moore, 2004), its effects on food intake remain uncertain (Luhovyy et al., 2007).

Whey protein has been found to be more satiating in some studies (Hall, Millward, Long, & Morgan, 2003; Veldhorst, Nieuwenhuizen, Hochstenbach-Waelen, van Vught, et al., 2009), while other studies have found no difference in the satiating effects of whey and casein protein (Bowen, Noakes, Trenerry, & Clifton, 2006; Lorenzen et al., 2012) or that casein protein leads to a greater satiating effect than whey proteins (Acheson et al., 2011). However, the effect of protein source is modulated by several factors including dose, form (solid or liquid), duration to the next meal, and the presence or absence of other macronutrients (Luhovyy et al., 2007). Nevertheless, energy intake was 9% lower after intake of milk than after intake of casein or whey (Lorenzen et al., 2012). Thus, complete milk proteins might elicit an intermediate yet optimal satiating effect, or other bioactive components in milk influence its satiating power.

The role of calcium intake on energy and fat balance has been explored, and studies that reviewed the available data provide some evidence for a beneficial effect of calcium on energy metabolism and the control of obesity (Major et al., 2008; Zemel, 2004). The "calcium appetite" theory (Tordoff,

2001) states that vertebrates develop a taste for and seek out minerals that they lack. Calcium-deficient individuals are more prone to increase their intake of foods high in fats and sugar since these nutrients are often found together with calcium (as in dairy foods). This preference for fat and sugar is the result of a learnt association between these nutrients and calcium.

Very few studies have assessed the impact of calcium consumption alone on the regulation of food intake. In women ($n = 13$) consuming <600 mg/day of calcium, supplementation with calcium + vitamin D reduced fat and total energy intake at an *ad libitum* food intake test. However, the sample size was small (Major, Alarie, Dore, & Tremblay, 2009). Subjects participating in a 6-month energy restriction program were assigned to either milk (1000 mg of calcium) or placebo (0 mg calcium) supplemented groups. Milk supplementation was accompanied by an increase in measured fullness that was significantly different from the decrease predicted by weight loss. Additionally, weight loss was found to induce stimulatory effects on appetite that were attenuated in the group receiving the milk supplementation (Gilbert et al., 2011). However, dairy supplementation increased protein intake; hence, it was difficult to distinguish between the effects of protein and calcium on food intake regulation.

In a crossover trial, four isoenergetic meals consisting of three meals with dairy products as the calcium source (low calcium [68 mg], medium calcium [350 mg], and high calcium [793 mg]) and one meal with a calcium supplement + calcium from dairy (total calcium: 850 mg) were compared. The four meals contained equivalent amounts of dairy protein. There was no significant effect of a high calcium intake from either dairy products or the supplement on appetite sensations, appetite hormones, or energy intake at a subsequent *ad libitum* meal (Lorenzen et al., 2007).

Satiety has been reported after consumption of dairy foods. Chocolate milk and a carbonated soft drink matched for energy density and energy were compared in a study to assess the satiety effects of the two beverages. Increased short-term satiety was observed after consumption of the chocolate milk when compared with the soft drink, but *ad libitum* energy consumption at lunch served 30 min later was not significantly different (Harper et al., 2007). The addition of 600 ml of skim milk to a fixed energy breakfast induced greater satiety than a fruit drink and reduced energy intake at a buffet sandwich meal 4 h later (Dove et al., 2009).

In a comparison of isoenergetic servings of yogurt, cheese, and milk, although there was no effect on energy intake, yogurt produced the greatest suppressive effect on appetite. Hunger ratings were 8%, 10%, and 24% lower after intake of yogurt as compared with cheese, milk, and water, respectively

(Dougkas, Minihane, Givens, Reynolds, & Yaqoob, 2012). However, in a comparison between isoenergetic, isovolumetric, servings of milk, high-fructose corn syrup sweetened, and sucrose-sweetened beverage preloads matched for energy density, no significant effect on satiety or food intake at a subsequent meal was observed (Soenen & Westerterp-Plantenga, 2007). Similarly, measures of satiety or food intake at a subsequent meal did not significantly differ between isoenergetic, isovolumetric preloads of orange juice, 1% milk, or a cola drink matched for energy density (Almiron-Roig & Drewnowski, 2003).

It is not only important that dairy products enhance satiety but it must be accompanied by a reciprocal compensation in energy intake through a reduction in nondairy foods. A cheesy snack containing a mixture of casein or whey + casein resulted in partial compensation in energy intake at an *ad libitum* lunch meal 1 h later and full compensation over the 24-h period following the preload. There was no difference in the satiety ratings between the two snacks (Potier et al., 2009). In a crossover trial, participants who were instructed to consume a high-dairy diet (three servings/day) or a low-dairy diet (one serving/day) for 7 days, separated by a 7-day wash-out period, increased their energy intake by 209 kcal during the high-dairy intervention. Further, there was no difference in appetite ratings between the two interventions (Hollis & Mattes, 2007b). However, a comparison of 200 kcal preloads consisting of semi-solid yogurt, liquid yogurt, dairy fruit beverage, and a fruit drink served 90 min prior to a meal resulted in increased satiety with the yogurt preloads as compared with the fruit beverages. Although the reduction in energy intake at the meal was not significant between the yogurt and fruit drinks, there was no energy compensation (Tsuchiya, Almiron-Roig, Lluch, Guyonnet, & Drewnowski, 2006).

There is not enough evidence to indicate that dietary calcium alone has regulatory effects on satiety and food intake. Dairy products include dairy proteins and other bioactive components that may have an effect on appetite control. The regulatory effects on satiety and food intake associated with increased dairy consumption have only been observed in the short term. Nevertheless, it demonstrates the need for investigating the various components of milk and milk products in the development of functional foods aimed at regulating appetite.

In a review of the epidemiological evidence on the effect of dairy consumption on body weight (Dougkas, Reynolds, Givens, Elwood, & Minihane, 2011), it was concluded that the data from cross-sectional studies supported an association between reduced adiposity and consumption of yogurt or milk, whereas cheese had the opposite effect. An inverse association was also observed between calcium intake from dairy and other sources with weight status in cross-sectional studies (Dougkas et al., 2011). However, in a systematic review of prospective cohort studies (Louie, Flood, Hector, Rangan, & Gill, 2011), it was concluded that the association between dairy consumption and weight status was inconsistent among children/adolescents as well as adults. Nevertheless, dairy consumption did not have an adverse effect on weight status, but the high heterogeneity of the studies and the inconsistent exposure and outcome measures precluded the conduct of a meta-analysis (Louie et al., 2011).

In two reviews (Dougkas et al., 2011; Lanou & Barnard, 2008) and a meta-analysis (Abargouei, Janghorbani, Salehi-Marzijarani, & Esmaillzadeh, 2012) of randomized controlled trials (RCTs) investigating the effects of dairy consumption on body weight, the authors concurred in their conclusion that body weight does not change due to increased consumption of dairy products in the context of diets without an energy restriction. Fat mass, lean body mass, and waist circumference were also unaffected by consumption of dairy products in the recommended amounts (three to four servings/day) when energy intake was not restricted (Abargouei et al., 2012). While one review of 11 studies investigating the effects of dairy or supplemental calcium intake on body weight in the context of energy-restricted diets found no effect of dairy or supplemental calcium intake on body weight (Lanou & Barnard, 2008), another review of five studies in which energy intake was restricted found inconsistent results (Dougkas et al., 2011). However, the conclusion from a meta-analysis of 11 studies imposing an energy restriction was that high-dairy energy-restricted diets may result in greater weight loss, fat loss, and higher reduction in waist circumference compared with energy-restricted diets that do not emphasize increased dairy consumption (Abargouei et al., 2012).

A more pronounced effect with equivalent calcium intakes from dairy products as opposed to the supplemental form in energy-restricted diets was also observed, suggesting that dairy components other than calcium may in part mediate the beneficial impact on body weight and composition (Dougkas et al., 2011). Thus, the inclusion of dairy products in the recommended amounts may prevent weight loss in weight maintenance diets, and high-dairy intake may facilitate weight loss in energy-restricted diets.

2.2. Meat, fish, and eggs

Dietary proteins provide amino acids responsible for major metabolic functions in the body. Protein quantity as well as quality influences its physiologic effects. Protein quality describes the characteristics of protein as they relate to the attainment of definite metabolic actions. The amino acid composition is the major determinant of protein quality (Millward, Layman, Tome, & Schaafsma, 2008). A complete protein provides all the essential amino acids (EAA) which include histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine. Meat, poultry, fish, eggs, milk, cheese, and yogurt are considered complete proteins (Brown, 2008). Postprandial protein synthesis is enhanced when the composition of dietary protein matches the optimal amino acid needs of the body for protein synthesis. Thus, a well-balanced amino acid mixture as would be found in a complete protein would produce a higher thermogenic response than would an amino acid mixture of lower biological value (Westerterp-Plantenga et al., 2009).

The quality of a protein is influenced by the characteristics of the protein and the food matrix in which it is consumed, as well as the requirements of the individual consuming the food (Millward et al., 2008). The intake of protein over the whole day must provide as closely as possible the substrate needed for protein synthesis and any other biosynthetic pathways including a provision for sufficient signal amino acids (e.g., leucine) required for optimizing metabolism and stimulating anabolism (Millward et al., 2008; Young & Pellett, 1994). However, according to current methods of assessing protein quality, protein synthesis is limited by the available (digested and absorbed) EAA without considering regulatory amino acids (Millward et al., 2008).

While protein-induced satiety has been demonstrated in several studies (Harper et al., 2007; Lejeune et al., 2006; Smeets et al., 2008; Westerterp-Plantenga et al., 1999), there is less evidence for the role of protein from different sources on the regulation of appetite and food intake. Consumption of pork, beef, or chicken meals matched for energy and macronutrient content did not differ in their effects on satiety, food intake, or appetite hormones (cholecystokinin, PYY, ghrelin, and insulin) (Charlton et al., 2011).

Among protein-rich foods, fish was found to be the most satiating item, followed by beef, baked beans, eggs, cheese, and lentils in a study to assess the satiating capacity of several foods (Holt, Miller, Petocz, & Farmakalidis, 1995). In a comparison of beef, chicken, and fish meals containing 50 g protein, there was no difference in satiety between the chicken and beef meals; however, after consuming the fish meal, satiety increased as compared with the chicken and beef meals. The increase in the ratio of tryptophan to large neutral amino acids following consumption of the fish meal led the researchers to conclude that the neurotransmitter serotonin was one of the signals that induced satiety (Uhe, Collier, & O'Dea, 1992).

Liquid shakes containing protein from tuna, turkey, whey, or egg albumin that were matched for flavor, texture, and taste were compared.

Although the whey containing shake elicited the greatest satiety response, the tuna meal produced a significantly higher increase in satiety and a reduction in food intake than the turkey or egg shakes. The concentrations of protein, fat, and carbohydrate were matched in all the meals (Pal & Ellis, 2010). In another study, isoenergetic fish and beef meals matched for macronutrient composition, taste, and appearance were compared. The fish meal reduced energy intake at a subsequent meal compared with the beef meal; however, there were no significant differences in the appetite and satiety ratings (Borzoei, Neovius, Barkeling, Teixeira-Pinto, & Rossner, 2006).

The protein content of eggs is 35% of their total energy content (Pombo-Rodrigues, Calame, & Re, 2011). Isoenergetic egg (23% of energy from protein) and bagel (16% of energy from protein) breakfasts were compared. The egg breakfast significantly reduced the insulin, glucose, and ghrelin concentrations. Additionally, hunger was reduced and satisfaction increased after the egg breakfast as compared with the bagel breakfast, which resulted in a reduction in food intake at a subsequent meal (Ratliff et al., 2010). In another study comparing isoenergetic egg (18.3 g protein) and bagel (13.5 g protein) breakfasts matched for weight, consumption of the egg breakfast increased satiety and reduced energy intake at lunch. There was no compensation for the reduction in energy intake in the 24-h period following breakfast, as assessed by self-reported food intake (Vander Wal, Marth, Khosla, Jen, & Dhurandhar, 2005). Following consumption of three isoenergetic test lunches omelet, jacket potato, and chicken sandwich, the omelet meal was found to elicit a higher satiety response than the potato and chicken meals, but energy intake at dinner was not significantly different between the three conditions (Pombo-Rodrigues et al., 2011). In a comparison of isoenergetic egg and bagel breakfast meals matched for energy density, and consumed daily for 2 months, it was found that the egg breakfast did not promote weight loss. However, when subjects were placed on a reduced energy diet, eating the egg breakfast rather than the bagel breakfast resulted in greater weight loss (Vander Wal, Gupta, Khosla, & Dhurandhar, 2008).

Using NHANES data collected during the period from 1999 to 2004, it was determined that total meat consumption and "other meat products" (including frankfurter, sausage, organ meats, and food mixtures composed of meat poultry and fish) were positively associated with BMI and waist circumference. Half of the total meat consumption comprised other meat products. Of the remaining total meat consumption, red meat was consumed the most, followed by poultry and seafood (Wang & Beydoun, 2009). The European Prospective Investigation into Cancer and Nutrition

(EPIC) was a large-scale multicenter cohort study including subjects from 10 countries. Weight gain over 5 years among meat eaters, fish eaters, vegetarians (no meat or fish but including eggs and dairy products), and vegans (no foods of animal origins) from one arm of the EPIC study was assessed, and a statistically significant reduction in weight gain was found in vegans and fish eaters when compared with meat eaters. During the follow-up period, subjects who reduced the intake of meat had the lowest weight gain (Rosell, Appleby, Spencer, & Key, 2006).

Prospective studies investigating the effects of meat consumption on weight management using data from the EPIC study have yielded consistent results. Whereas an inverse association with red meat consumption and waist circumference was found among both males and females, a positive association between waist circumference and processed meat and poultry was found only in females. Fish and egg consumption was not associated with waist circumference in both groups (Halkjaer, Tjonneland, Overvad, & Sorensen, 2009). Using data from the EPIC study, an increase in meat intake of 250 g/day was determined to lead to a 2 kg weight gain in 5 years. In this cohort, red meat, poultry, and processed meat were positively associated with weight gain (Vergnaud et al., 2010). Data from six cohorts participating in the EPIC study showed that weight gain was positively associated with consumption of protein from red meat and processed meat, and poultry rather than fish and dairy (Halkjaer et al., 2011). However, fish consumption did not prevent an increase in waist circumference (Jakobsen et al., 2011). In another 7-year follow-up study, animal protein intake was found to be positively associated with overweight and obesity as assessed by BMI (Bujnowski et al., 2011).

Contrary results showing no significant associations between meat consumption and body weight or changes in adiposity measures over time were obtained in a 6-year follow-up study (Drapeau et al., 2004). Although protein intake was inversely associated with waist circumference, the effects were not significantly different between protein from animal and plant sources in subjects followed prospectively for 5 years (Halkjaer, Tjonneland, Thomsen, Overvad, & Sorensen, 2006).

A 2-year intervention trial comparing the effectiveness of a low-fat, energy-restricted, or Mediterranean diet determined that one of the leading predictors for 2-year successful weight loss was increased intake of meat and decreased intake of eggs (Canfi et al., 2011). In a RCT, participants who followed an energy-restricted diet containing lean or fatty fish had a greater weight loss than participants who followed an isoenergetic diet without fish

(Thorsdottir et al., 2007). A 12-week randomized controlled weight-loss trial among overweight women showed no significant difference in weight loss between subjects consuming an energy-restricted diet containing either lean beef or chicken (Melanson, Gootman, Myrdal, Kline, & Rippe, 2003).

RCTs investigating the effect of meat consumption on body weight are singularly lacking. The evidence associating meat intake with body weight is provided primarily by observational studies which precludes the establishment of a cause and effect relationship. There is a need for controlled trials investigating the effects of meat intake from various sources on appetite, satiety, food intake, and body weight.

2.3. Legumes

Legumes include alfalfa, clover, green beans and peas, peanuts, soy beans, dry beans, broad beans, dry peas, chickpeas, and lentils. Pulses are a type of legume exclusively harvested for the dry grain and include dry beans, chickpeas, lentils, and peas. Pulses are high in fiber which contributes to lowering the energy density and reducing the glycemic response, but they are also a good source of protein (McCrory, Hamaker, Lovejoy, & Eichelsdoerfer, 2010). A comparison of isoenergetic servings of chickpeas (16.1 g protein, 11.3 g fiber), lentils (18.3 g protein, 13.5 g fiber), navy beans (18.7 g protein, 16.6 g fiber), yellow peas (17.2 g protein, 9.1 g fiber), and a control of white bread (9.9 g protein, 2.8 g fiber) all served in a tomato-based sauce did not result in a significant difference in appetite and satiety ratings or food intake (Wong, Mollard, Zafar, Luhovyy, & Anderson, 2009). In the same study, canned beans and homemade beans were compared with glucose matched for carbohydrate content but differing in energy content. Satiety increased following consumption of homemade beans as compared with glucose, but canned beans consumption as compared with glucose reduced food intake at a pizza meal 2 h later (Wong et al., 2009).

Breads made by replacing 24.3% of wheat flour with chickpea flour (5 g fiber) or extruded chickpea flour (6 g fiber) were compared with wheat bread (3 g fiber) served as part of a breakfast meal. The meals were of equal food weight and had similar energy content. There were no significant differences in the satiety responses or energy intake at a buffet meal 2 h after the three breakfast meals, each containing one type of bread (Johnson, Thomas, & Hall, 2005). Pasta and tomato sauce meals matched for energy density and containing chickpeas, lentils, navy beans, yellow peas, or larger amounts of pasta and sauce were compared. The lentil treatment led to lower food intake compared to chickpeas and pasta with sauce, whereas navy beans led to lower intake compared only to chickpeas. However, there were no significant differences in food intake at a pizza meal 4 h later, although lentil treatment resulted in a significant decrease in cumulative intake (Mollard, Zykus, et al., 2011).

Isoenergetic preloads consisting of lentils and yellow peas but not chickpeas resulted in an increase in satiety and a reduction in food intake at a subsequent meal as compared with a "macaroni and cheese" meal (Mollard, Wong, Luhovyy, & Anderson, 2011). In another study assessing the effects of chickpea supplementation in the diet, participants were required to consume four 300 g cans of chick peas each week for 4 weeks. The increase in satiety at the end of 12 weeks was attributed to the increase in fiber intake (Murty, Pittaway, & Ball, 2010). Thus, the increase in satiety observed with pulse consumption may at least in part be attributed to the fiber content. However, the influence of pulses on satiety and food intake, particularly in comparison with other protein foods, and in the long term needs further substantiation through controlled studies.

The protein digestibility-corrected amino acid score is the currently approved method for protein quality assessment (Millward et al., 2008), and soy has been given a score of 1.0 which is the same as casein and egg protein. Thus, soy protein is considered a complete protein (Cope, Erdman, & Allison, 2008; Velasquez & Bhathena, 2007). In a study comparing isoenergetic breakfasts, matched for color, viscosity, and taste as assessed by VAS, satiety ratings were significantly higher after the breakfast meal containing 25% of energy from soy protein as compared with the meal containing 10% of energy from soy protein. However, food intake at a subsequent meal was not significantly different (Veldhorst, Nieuwenhuizen, Hochstenbach-Waelen, Westerterp, et al., 2009).

The satiating effects of three isoenergetic meals, which provided 50% of energy as whey, casein, or soy protein, were compared with a high-carbohydrate meal (95.5% energy from carbohydrate). The thermic effects of the protein-rich meals were significantly greater than the carbohydrate meal. Whey protein produced the highest thermogenic response, but casein and soy were more satiating than whey. However, satiety resulting from consumption of soy or casein proteins was not significantly different from the carbohydrate meal (Acheson et al., 2011). In a comparison of pretzels made with wheat flour and pretzels in which 27.3% of the wheat flour was replaced with soy ingredients, no significant effect on satiety was observed (Simmons, Miller, Clinton, & Vodovotz, 2011). In a comparison of soy protein, whey protein, and carbohydrate supplementation for

23 weeks, no significant effects on measures of satiety were observed (Baer et al., 2011). Although there appears to be some support for the satiety-enhancing effects of soy protein, a reduction in food intake remains to be established.

Using data from NHANES, an inverse association between high-pulse consumption and body weight was determined (Papanikolaou & Fulgoni, 2008). In a review (McCrory et al., 2010) of the effect of pulse consumption on weight management, five intervention trials that assessed the effectiveness of incorporating pulses in energy-restricted diets were identified (Abete, Parra, & Martinez, 2009; Hermsdorff, Zulet, Abete, & Martinez, 2011; Karlstrom et al., 1987; McCrory et al., 2008; Sichieri, 2002). In one study, a beneficial effect of legume consumption on weight loss at 1 month but not at 2 months was observed (Sichieri, 2002). Greater weight loss occurred with a medium intake (0.5 cup/day) as compared with a low intake (1 tablespoon/day) of pulses (McCrory et al., 2008). In a comparison of a high-legume diet (17% of energy from protein), a high-protein diet (30% of energy from meat, eggs, lean dairy), a fatty fish diet (17% energy from protein, mainly from fatty fish), and a control diet (17% of energy from protein, excluding legumes and fish), similar effects on weight loss were observed with the high-protein and high-legume diet. Both diets produced weight loss which was significantly greater than the control diet (Abete et al., 2009).

The consumption of legumes (four servings/week) within a low-energy diet resulted in a greater reduction in weight than a control diet that excluded legumes (Hermsdorff et al., 2011). However, in one study, no significant effect of legume consumption on body weight was observed (Karlstrom et al., 1987). The provision of pulses and whole grains for incorporation into the diet (during the first 6 months) also did not result in a significantly different weight loss after 6 or 18 months, as compared with a control group that was provided refined cereals and high-glycemic index (Gi) foods (Venn et al., 2010).

In a review (Cope et al., 2008) of the effect of soy protein on body weight, it was determined that epidemiological data provided inconsistent evidence for an inverse relationship between soy protein consumption and body weight. Clinical data did not indicate a clear advantage for soy protein over other sources of protein for weight and fat loss when consumed at isoenergetic levels. However, the comparator in most of the studies reviewed was milk (Cope et al., 2008). Subsequently, in a RCT assessing the effects of soy protein, whey protein, or carbohydrate supplementation for 23 weeks on measures of body weight and composition, there were no significant differences between soy and whey protein or soy and carbohydrate supplemented groups (Baer et al., 2011). Thus, the consumption of pulses and other legumes may enhance weight loss, but there does not appear to be sufficient evidence to support their role in weight management at this time.

2.4. Nuts

Nuts are fruits with one seed in which the ovary wall becomes hard at maturity. Common edible nuts include almonds, hazelnuts, walnuts, pistachios, pine nuts, cashews, pecans, macadamia, and Brazil nuts. Peanuts (groundnuts) are botanically a legume but are widely included in the nuts food group (Ros, 2010). Nuts are nutrient-dense foods rich in unsaturated fat and other bioactive compounds but are also high in energy (Ros, 2009). Nevertheless, evidence from epidemiological Albert, Gaziano, Willett, & Manson, 2002; Bes-Rastrollo et al., 2007; Fraser, Sabate, Beeson, & Strahan, 1992; Hu et al., 1998) as well as intervention trials (Natoli & McCoy, 2007; Rajaram & Sabate, 2006) suggest that nut consumption is not associated with weight gain. The mechanisms by which nuts impact body weight include increased satiety, increased energy expenditure, and incomplete digestion or absorption leading to increased fecal fat (Mattes, 2008; Rajaram & Sabate, 2006).

Nuts are energy-dense, and are high in fiber and protein, components of the diet associated with satiety (Mattes, Kris-Etherton, & Foster, 2008). The physical properties of nuts have also been implicated in their effects on satiety. Nuts require an effort at mastication, which may promote satiety (Slavin & Green, 2007). Nuts are high in fat and the prolonged mastication may influence satiety, since the metabolic effects of consumption of high-fat foods are modulated by oral exposure (Smeets & Westerterp-Plantenga, 2006). Nuts which have to be shelled have been shown to reduce energy intake as compared with nuts which have been shelled (Honselman et al., 2011). The act of shelling the nuts may slow the rate of consumption by permitting greater metabolic feedback during ingestion (Mattes, 2008) or may increase satiety by increasing perception of the quantity consumed (Kennedy-Hagan et al., 2011).

A diet containing walnuts was found to increase satiety ratings over a 3–4 day period as compared with a placebo diet (Brennan, Sweeney, Liu, & Mantzoros, 2010). However, no differences in satiety or energy intake were observed in a comparison of isoenergetic meals rich in polyunsaturated fats from walnuts, monounsaturated fat from olive oil, and saturated fat from dairy products (Casas-Agustench et al., 2009). Whole almonds (42.5 g) were found to increase fullness during the day when added to a breakfast meal matched for carbohydrate, protein, fat, and fiber when compared with meals containing almond flour, almond butter, almond oil, or no almonds (Mori, Considine, & Mattes, 2011).

It has been suggested that nut consumption leads to a spontaneous reduction in energy intake from other food sources during the day leading to an overall reduction in energy intake (Mattes, 2008). Almond supplementation for 6 months led to a 54–78% lack of compensation for the energy consumed from almonds (Fraser, Bennett, Jaceldo, & Sabate, 2002). From reported values of energy reduction, it has been estimated that approximately 65–75% of the energy provided from nuts is offset by lower energy intake at subsequent meals (Mattes et al., 2008).

An increase in resting energy expenditure (REE) following nut consumption has been observed. Regular consumption of peanuts for 19 weeks resulted in an 11% increase in REE when compared with the baseline measurement (Alper & Mattes, 2002). The high-unsaturated fat and protein content of nuts may increase fat oxidation and thereby influence diet-induced thermogenesis and REE (Mattes et al., 2008; Rajaram & Sabate, 2006). However, almond supplementation for 6 months did not increase REE (Fraser et al., 2002). In another study, diet-induced thermogenesis or REE did not change significantly after 10 weeks of almond supplementation (Hollis & Mattes, 2007a).

It is likely that limited fatty acid availability results from incomplete digestion or absorption. The parenchymal cell wall of nuts is resistant to microbial and enzymatic degradation. Thus, cells that are not ruptured as a result of insufficient mastication may pass through the GI tract without releasing the oils they contain (Mattes et al., 2008; Rajaram & Sabate, 2006). Using electron microscopy, it has been demonstrated that the cell walls of almonds remain intact in fecal samples, decreasing the bioaccessibility of intracellular fatty acids contained in the almonds and leading to a threefold increase in percent fecal fat excretion (Ellis et al., 2004). When participants chewed almonds 10 times, fecal fat losses significantly increased as compared with fecal fat excretion measured after they had chewed the almonds 25 or 40 times (Cassady, Hollis, Fulford, Considine, & Mattes, 2009). In a comparison of usual diets containing 70 g/day of whole peanuts, peanut oil, or peanut flour, fecal fat excretion was significantly higher with consumption of whole peanuts (Traoret et al., 2008). Further, other nutrients that contribute to energy are also less bioavailable which may cause further declines in energy intake (Mattes et al., 2008).

The Seguimiento Universidad de Navarra study is the only epidemiologic study that has prospectively examined the direct effect of nut consumption on body weight. The study included approximately 8800 adult men and women and found that those who ate nuts frequently (≥two times/week) had a 40% reduced risk of weight gain. During a follow-up period of 28 months, frequent nut consumers gained 350 g less weight than did those who did not eat nuts (Bes-Rastrollo et al., 2007). In a recent review of the epidemiologic evidence on the effect of nut consumption on weight gain and obesity, it was concluded that nut consumption up to four servings/week does not lead to any appreciable weight gain in the long term (Martinez-Gonzalez & Bes-Rastrollo, 2011).

The impact on body weight of consumption of almonds, peanuts, pistachios, and walnuts has been examined in intervention trials. Six months supplementation of almonds averaging (320 kcal/day) resulted in a weight gain of 0.4 kg. The predicted weight gain as a result of the extra energy intake from the almonds was 6.4 kg (Fraser et al., 2002). In another study, subjects consuming an almond-enriched (84 g/day), low-calorie diet for 24 weeks lost 62% more weight and showed significant improvements in measures of body composition than subjects assigned to a low-calorie complex carbohydrate diet (Wien, Sabate, Ikle, Cole, & Kandeel, 2003). In contrast, reduced energy almond-enriched (56 g/day) or nut-free diets resulted in a greater reduction in weight loss in the nut-free diet condition at 6 months in overweight and obese individuals. There was, however, no difference in weight loss between the diets at 18 months (Foster et al., 2012). It has also been reported that almond supplementation (approximately 344 kcal) for 10 weeks caused no significant change in body weight or body composition (Hollis & Mattes, 2007a).

In other studies, peanut supplementation for 6 months increased energy intake; however, the actual weight gain was less than the predicted weight gain as a result of partial compensation for energy provided by peanuts (Alper & Mattes, 2002). A low-calorie weight-loss trial comparing the effects of pistachios or pretzels served as an afternoon snack for 12 weeks found a significantly greater reduction in the BMI of participants consuming pistachios (Li et al., 2010). Daily consumption of either 42 g or 70 g of pistachios for 12 weeks did not lead to weight gain or an increase in waist-to-hip ratio in Chinese subjects with metabolic syndrome (Wang, Li, Liu, Lv, & Yang, 2012). Participants following their usual diet were provided with walnuts corresponding to 12% of their daily energy intake (28–56 g). At the end of 6 months, the theoretical weight gain was predicted to be 3.1 kg. Although daily energy intake increased by 133 kcal, the weight gain was only 0.4 kg, neither of which were significant changes (Sabate, Cordero-Macintyre, Siapco, Torabian, & Haddad, 2005).

Thus, increased satiety with nut consumption, the displacement of foods from the habitual diet, and increased fecal fat excretion appear to be plausible mechanisms by which nut consumption does not adversely influence body weight despite the fact that nuts are energy dense. However, the effects of nut consumption on diet-induced thermogenesis and REE need further substantiation. Inclusion of nuts in energy-restricted diets may help adherence to the diet, facilitate weight loss, and improve measures of body composition.

3. CARBOHYDRATES AND THE REGULATION OF FOOD INTAKE AND BODY WEIGHT

Carbohydrates influence satiety and food intake through several mechanisms related to their hormonal effects, intrinsic properties, and intestinal fermentation (Beck, Tapsell, Batterham, Tosh, & Huang, 2009; Beck, Tosh, Batterham, Tapsell, & Huang, 2009; Greenway et al., 2007; Hamedani, Akhavan, Abou Samra, & Anderson, 2009; Ludwig, 2002; Vitaglione, Lumaga, Stanzione, Scalfi, & Fogliano, 2009). The hormonal effects of carbohydrates on satiety are mediated by insulin (Ludwig, 2002) and GI hormones (Beck, Tapsell, et al., 2009; Vitaglione et al., 2009), whereas the intrinsic properties relate to the bulking and viscosity effects of dietary fiber (Beck, Tosh, et al., 2009; Hamedani et al., 2009). Carbohydrates that elude small intestinal digestion enter the large bowel and are fermented by colonic microorganisms into short-chain fatty acids which have been shown to enhance satiety (Greenway et al., 2007). The manipulation of the carbohydrate content of the diet has been explored as a means of regulating body weight.

3.1. Glycemic index

The Gi was developed as a method for classifying the carbohydrates in different foods according to their post-ingestion glycemic effect (Jenkins et al., 1981). It is defined as the incremental area under the curve (AUC) for the blood glucose response after consumption of a 50 g carbohydrate portion of a test food expressed as a percent of the response to an equivalent carbohydrate amount from a reference food ingested by the same subject (Wolever, Jenkins, Jenkins, & Josse, 1991), with glucose or white bread as the reference food. Based on a commonly accepted classification system, foods are categorized as low Gi (<55) or high Gi (>70) (Venn & Green, 2007) For a meal, and by implication, a diet, the Gi of individual food items may be used to predict the Gi of a mixed meal (Wolever et al., 1991). However, the quantity and source of carbohydrate are important components affecting the glycemic response (Venn & Green, 2007).

The concept of glycemic load (GL) has been developed to take into account the amount of carbohydrate consumed. The GL value of a food may be determined indirectly as a product of the Gi of a food and the amount of available carbohydrate (carbohydrate that is absorbed by the small intestine and used in metabolism (Livesey, 2005)) in the portion of food consumed. The glycemic equivalence is a method of directly determining the GL. It involves constructing a standard curve for each subject based on the AUC for glucose calculated for a range of doses of the reference food measured on different days. The AUC for a given food consumed at any portion size is compared to the individual's standard curve. Theoretically, it is the amount of glucose that would produce the same blood glucose AUC as that particular portion size of food consumed. It is a time consuming and costly method which in any event agrees well with the GL measured indirectly (Venn & Green, 2007).

The potential physiological mechanisms relating Gi to the regulation of food intake are based on the postprandial metabolic environment precipitated by hyperglycemia and hyperinsulinemia. It has been suggested that a high-GL meal elicits high insulin and low glucagon responses that promotes uptake of glucose in the muscle, liver, and fat tissue. Hepatic glucose production is thereby restrained and lipolysis is inhibited. Thus, denial of full access to the two major metabolic fuels in the post-absorptive state may lead to a quicker hunger response and overeating, as the body attempts to restore the concentration of metabolic fuels to normal (Ludwig, 2002). Low-Gi foods are characterized by a slow rate of digestion and absorption, thereby eliciting a low glycemic response (Wolever et al., 1991).

Increased short-term satiety with low-Gi foods or meals as compared with high-Gi foods or meals has been demonstrated in a vast majority of the studies investigating the effects of the diets based on the Gi (Livesey, 2005). A systematic review of the effect of low-Gi diets on satiety and body weight in the long term (several days or weeks duration) found inconsistent results (Bornet, Jardy-Gennetier, Jacquet, & Stowell, 2007). More recently, a 10-week parallel study (Krog-Mikkelsen et al., 2011) to investigate the effects of low-Gi or high-Gi diets found no differences in postprandial plasma leptin and ghrelin to support the satiating effect of either Gi diet. Subjective appetite sensations, energy expenditure, or substrate oxidation were also not significantly different.

The association between Gi or GL and obesity has been the focus of numerous studies, and the subject of controversy, yet many best-selling diet books including the South Beach diet, the Zone diet, and the New Glucose Revolution advocate consumption of a low-Gi diet. Although some epidemiological studies indicate that increasing the Gi of a diet is associated with an increase in BMI, a collective analysis of the data does not support the view that a high-Gi diet is predictive of a higher BMI when compared with a lower Gi diet. The majority of epidemiological evidence also does not support the belief that high-GL diets are predictive of adiposity (Gaesser, 2007).

A meta-analysis of six RCTs (Thomas, Elliott, & Baur, 2007) that compared low-Gi/GL diets with either high-Gi/GL or low-fat diets in overweight or obese individuals concluded that low-Gi or -GL diets resulted in a decrease in body mass by 1.1 kg, fat mass by 1.1 kg, and body mass index by 1.3 kg/m^2. Another meta-analysis of 23 studies (Livesey, Taylor, Hulshof, & Howlett, 2008) that measured weight loss following low-Gi/GL diets showed that a reduction in body weight occurred with a reduction in dietary GL when food intake was either *ad libitum* or relatively uncontrolled. The beneficial effect, however, was not observed in studies where the food intake was controlled. Low-GL diets prescribed *ad libitum* have also been shown to be effective in promoting weight loss in overweight adolescents in comparison with reduced-fat diets (Ebbeling, Leidig, Sinclair, Hangen, & Ludwig, 2003).

A RCT (Maki, Rains, Kaden, Raneri, & Davidson, 2007) to evaluate the effects of an *ad libitum* reduced-GL diet on body weight and body composition in overweight or obese adults found that subjects assigned to the reduced GL diet lost significantly more weight than those assigned to a low-fat diet at 12 weeks. However, there was no significant difference in body weight between the groups at 36 weeks despite continuation of the diet in the weight maintenance phase following weight loss. In the CALERIE trial, a 1-year RCT designed to examine the effects of high- or low-GL, calorie-restricted diets on weight and fat loss in overweight women, there was no significant change in body weight, body fat, resting metabolic rate, hunger, and satiety between the groups (Das et al., 2007). Thus, it appears that reduced energy intake with low-GL diets may not persist in the long term. Analysis of 4-day food records, in a randomized crossover intervention (Aston, Stokes, & Jebb, 2008), comparing two diets differing in the Gi, suggested that 19 overweight or obese women consumed comparable amounts of energy and macronutrients on both diets and more dietary fiber on the low-Gi diet. There was, however, no significant difference in body weight, body composition, and waist circumference between the two intervention periods.

The clinical relevance of diets based on the Gi or GL remains unclear. The inconsistencies in the data appear to be largely responsible for the debate surrounding the concept. The Gi is influenced by the nature of the starch, the physical form, the amount of fiber, fat, and protein, as well as the cooking times and methods (Thorne, Thompson, & Jenkins, 1983). Other dietary factors influencing food digestibility, GI motility, or insulin secretion also determine the Gi of a food (Ludwig, 2003). The GI relates to a food and not the individual; therefore, there exists the possibility of intra- and inter-individual variances in the Gi and GL. Even in repeated experiments of the same food under standardized conditions, a seemingly inexplicable variation occurs in the glycemic response (Venn & Green, 2007). Such variability makes following a low-Gi diet complex.

The question remains as to whether the individual glycemic indices of foods can be summed to reliably derive the Gi of the meal. While some researchers contend that the Gi predicts the glycemic response of foods eaten as part of a mixed meal, others have shown no association between the calculated Gi and the measured Gi of the meal as a whole (Venn & Green, 2007). Nevertheless, the most important point of issue is the practicality of recommending Gi diets.

None of the studies reviewed in this chapter have suggested any adverse effects from consuming a low-Gi diet. In general, nonstarchy vegetables, fruits, legumes, and minimally processed grain products have a low Gi (Ludwig, 2003). A diet that includes these food groups meets the recommendations of the Dietary Guidelines for Americans, 2010 and could contribute to increasing the fiber and lowering the energy density of a diet. Central to the concept of low Gi is the emphasis on carbohydrate quality, which presents less of a challenge to glucose homeostasis than high-Gi diets. Low-Gi and -GL diets have been recommended in the treatment of diabetes and prevention of chronic diseases including diabetes, cancer, and cardiovascular disease (Jenkins et al., 2002). Moreover, benefits have been shown from *ad libitum* consumption of low-Gi diets (Ebbeling et al., 2003; Livesey et al., 2008) which suggests that these diets may be less restrictive and more acceptable as a dietary approach to the regulation of body weight. Nevertheless, the data do not present an unequivocal association between low-Gi and -GL diets, and reductions in body weight.

3.2. Dietary fiber

The World Health Organization and the Food and Agriculture Organization consider dietary fiber to be a polysaccharide with 10 or more monomeric units which is not hydrolyzed by endogenous hormones in the small intestine (Lattimer & Haub, 2010). Dietary fiber may be classified into soluble and insoluble fiber on the basis of water solubility. Colonic fermentation of soluble fiber yields short-chain fatty acids. Insoluble fiber generally has low fermentability, but it has water-attracting properties that promote fecal bulk (Papathanasopoulos & Camilleri, 2010).

Several mechanisms have been proposed to explain the effects of dietary fiber on the regulation of appetite and satiety. (1) Dietary fiber traps nutrients and retards their passage through the GI tract. Exposure of the intestinal mucosa to nutrients induces the release of appetite-regulating peptides which function as hormones or activate neural pathways involved in appetite regulation (Kristensen & Jensen, 2011). (2) Dietary fiber lowers the energy density of a food (Howarth, Saltzman, & Roberts, 2001). Energy density is inversely associated with satiety (Drewnowski, 1998). Thus, by implication, fiber enhances satiety. (3) It requires time and effort to eat the fiber-containing food, increasing mastication. Additionally, fiber stimulates the secretion of saliva and gastric secretions that cause stomach distension, thereby activating satiety signals (Slavin & Green, 2007). (4) Last, colonic fermentation of undigested carbohydrate to short-chain fatty acids has been hypothesized to increase satiety; however, data from human intervention studies do not appear to support a role for intestinal fermentation in appetite regulation (Darzi, Frost, & Robertson, 2011; Hess, Birkett, Thomas, & Slavin, 2011; Peters, Boers, Haddeman, Melnikov, & Qvyjt, 2009).

Highly viscous soluble dietary fiber by increasing the viscosity of GI contents delays gastric emptying which can increase stomach distension (Marciani et al., 2001). Some afferents in the gastric mucosa are mechanoreceptors, while others may stimulate chemical or other signals of satiation (Powley & Phillips, 2004). While gastric satiety is for the most part mechanical in origin, intestinal satiety is nutrient dependent; nevertheless, there exists evidence for a synergy of the two types of stimulation (Maljaars, Peters, & Masclee, 2007; Powley & Phillips, 2004). In the small intestine, transit time is increased and the absorption rate of nutrients is reduced as a result of the increased viscosity of GI contents (Maljaars, Peters, Mela, & Masclee, 2008). A thickening of the unstirred water layer poses an additional barrier to absorption (Johnson & Gee, 1981). Thus, viscous dietary fiber triggers an interaction between neural and hormonal signals that mediate satiety by enhancing the possibility of interaction between nutrients and the cells that release these hormones (Kristensen & Jensen, 2011). Hunger and satiety sensations originate in the central nervous system; however, hormonal secretion from the gut plays a pivotal role in the regulation of food intake (Chaudhri, Field, & Bloom, 2008).

The influence of dietary fiber on body weight is related to its effects not only on satiety and food intake but also on metabolizable energy, which is gross energy minus energy lost in feces, urine, and combustible gases (Lattimer & Haub, 2010). While dietary fiber has been shown to decrease metabolizable energy by decreasing fat digestibility and replacing some simple carbohydrates, the effects of soluble and insoluble fiber on metabolizable energy are the subject of debate. The fat content of the diet and the type of fiber may influence the results (Lattimer & Haub, 2010).

Epidemiologic data suggest that an increase in fiber intake is associated with lower weight gain over the 10- to 12-year period (Liu et al., 2003; Ludwig et al., 1999). An analysis of the data from the Health Professional's Follow-up Study which included 27,082 men followed for 8 years showed that an inverse association existed between dietary fiber intake and weight gain independent of the intake of whole grains. Significant inverse associations were observed between weight gain and fiber from cereals and fruits, but not vegetables. Fiber from fruits displayed the strongest dose–response relationship. Fiber from fruits was associated with reduction in weight gain by 2.51 kg for every 20 g/day increase. However, total dietary fiber was associated with a reduction in long-term weight gain by 5.5 kg, for every 20 g/day increase (Koh-Banerjee et al., 2004). An analysis of data from the EPIC study showed that a 10-g/day intake of dietary fiber was associated with an annual reduction in weight by 39 g and waist circumference by 0.08 cm. Ten grams per day of cereal fiber was associated with an annual weight loss of 77 g/year and a reduction in waist circumference of 0.1 cm/year. Fiber from fruits and vegetables was not associated with an appreciable weight change but was associated with a similar reduction in waist circumference as total fiber (Du et al., 2010).

The effect of dietary fiber on appetite and satiety, energy intake, and body weight as assessed in RCTs has been systematically reviewed (Wanders et al., 2011). Fiber consumption, regardless of the type of fiber, was found to cause an average reduction in appetite by 5% over a 4-hour interval. Further, appetite was determined to reduce by 0.18% per gram of fiber. For more viscous fibers, the reduction increased to 0.41%. Fiber intake also reduced energy intake by 2.6% in studies, wherein fiber supplementation was given over a period of 1 week or more. Irrespective of the type, fiber supplementation was determined to decrease body weight by 0.4% per month (Wanders et al., 2011).

Epidemiologic as well as experimental studies have demonstrated that intake of dietary fiber is inversely associated with satiety, food intake, body weight, and abdominal obesity. While evidence for the effects of fiber from cereals on body weight is supported in epidemiologic studies, the data evaluating the effects of fiber from fruits and vegetables on body weight are inconsistent.

3.2.1 Breads and cereals

Cereals are defined as the fruit of plants that belong to the Gramineae family of grasses and include wheat, rice, barley, corn, rye, oats, millets, sorghum, tef, triticale, canary seed, Job's tears, Fonio, and wild rice. Amaranth, buckwheat, and quinoa function as cereals. However, they are seeds from non-Gramineae families and are referred to as pseudocereals (Harris & Kris-Etherton, 2010). Whole-grain products are derived from cereals and are a good source of dietary fiber. According to the U.S. Food and Drug Administration, to be considered a whole-grain product, the endosperm, germ, and bran components of the grain must be present in the same relative proportion as they are present naturally in the seed. Additionally, whole-grain foods are defined as foods that contain 51% or more whole-grain ingredients (DHHS, 2009).

The 2010 Dietary Guidelines for Americans recommend that at the 2000 kcal level, grain products should comprise six servings of which at least three servings should come from whole grains (USDA-DHHS, 2010). In the United States, total grain servings are typically overconsumed; however, most Americans are not consuming adequate amounts of whole grain (USDA, 2012). An analysis of NHANES data from 1999–2004 indicated that mean whole-grain consumption among adults aged 19–50 and $\geq$51 years was 0.63 and 0.77 servings/day, respectively. Less than 5% of adults in the age group 19–50 years consumed the recommended servings of whole grains (O'Neil, Zanovec, Cho, & Nicklas, 2010). Since the 2005 Dietary Guidelines for Americans, consumers have increased purchases of whole grains, especially cereals, breads, and pasta. Competition among manufacturers leading to more products with whole grains being made available may have triggered the increase in consumption (Mancino, Kuchler, & Leibtag, 2008).

The vast majority of whole grains (56.9%) are consumed at breakfast (Whole-Grain-Council, 2009). Breads (32%) and breakfast cereal products (30%) are the major sources of whole-grain consumption in the United States (Cleveland, Moshfegh, Albertson, & Goldman, 2000), and they have been shown to enhance satiety (Abou Samra, Keersmaekers, Brienza, Mukherjee, & Mace, 2011; Hamedani et al., 2009; Holt, Delargy, Lawton, & Blundell, 1999; Rosen, Ostman, & Bjorck, 2011b; Rosen, Ostman, Shewry, et al., 2011). However, not all whole-grain breads increased satiety. In a comparison of whole-grain wheat bread and refined-grain wheat bread, subjective satiety and food intake following consumption of the whole-grain bread providing 10.5 g of fiber/day for 3 weeks were not significantly different as compared with refined-grain bread providing 5.8 g of fiber/day (Bodinham, Hitchen, Youngman, Frost, & Robertson, 2011). Fiber components, when added to refined-grain flours used in the production of breads and breakfast cereals, have also been found to have beneficial effects on the regulation of appetite (Lee et al., 2006; Vitaglione et al., 2009). Moreover, enriched and fortified grains provide important nutrients, especially folate (USDA, 2012). Thus, it is vital to encourage consumption of both enriched grains and whole grains in the recommended proportion.

Rye is a good source of soluble and insoluble dietary fiber (Andersson, Fransson, Tietjen, & Aman, 2009). The main fiber components of the cell wall in rye are arabinoxylan, β-glucan, and cellulose. Arabinoxylan is the dominant fiber, and the water-extractable component of arabinoxylan exhibits a high viscosity when dispersed in water (Ragaee, Campbell, Scoles, McLeod, & Tyler, 2001). Arabinoxylan is resistant to the bread-making process and retains its average molecular weight, unlike β-glucan which tends to degrade (Andersson et al., 2009). The molecular weights of the individual fiber types affect their physiologic properties, including viscosity.

Rye flour is usually made from a blend of different rye varieties. Several whole-grain rye breads each made with a different rye variety, including a commercial blend of rye varieties, were compared with bread made from refined-wheat flour. Subjective satiety was significantly higher following consumption of the commercial blend which had the highest insoluble fiber content (10.3 g) as compared with the wheat bread (2.4 g insoluble fiber). However, not all varieties of rye increased satiety (Rosen, Ostman, & Bjorck, 2011b; Rosen, Ostman, Shewry, et al., 2011).

In an assessment of a dose–response relationship, it was found that rye bread (60% rye bran flour, 40% wheat flour) with 5 or 8 g of fiber served as part of isoenergetic breakfasts increased satiety as compared with a wheat bread breakfast. However, there was no significant difference in satiety between the two rye bread breakfasts (Isaksson, Fredriksson, Andersson, Olsson, & Aman, 2009). Varying the structure of rye flour (whole-rye kernels or milled rye kernels) used to make bread did not result in a significantly different effect on satiety (Isaksson et al., 2011).

Rye porridge and rye bread made from different parts of the rye grain (endosperm, whole grain, and bran) were compared with bread made from refined wheat. It was found that the porridge made from whole-grain and bran fractions increased satiety as compared with the bread made from the same parts of the grain; however, all rye products increased satiety when compared with wheat bread (Rosen et al., 2009). The same investigators also compared the effects of similar rye breads on appetite and satiety, with meals made by boiling rye kernels. Besides being more satiating than the breads, the rye kernel meal also resulted in a reduction in food intake at a subsequent meal (Rosen, Ostman, & Bjorck, 2011a).

Whole-grain rye porridge breakfast (followed by whole-grain wheat pasta lunch or refined-wheat pasta lunch) and refined-wheat bread breakfast (followed by refined-wheat pasta lunch) were compared. The meals were matched for macronutrient content. Satiety ratings were significantly higher after the rye porridge breakfast when compared with the refined-wheat bread breakfast. After consuming the refined-wheat pasta lunch meal, subjects who ate the rye porridge breakfast meal continued to have greater sensations of satiety as compared with those who ate the refined-wheat bread breakfast meal (Isaksson, Sundberg, Aman, Fredriksson, & Olsson, 2008). In another study comparing whole-grain rye porridge with an isoenergetic refined-wheat breakfast, satiety was found to be greater after consumption of the rye porridge. Although the effect on satiety was sustained during 3 weeks of regular intake, it was only maintained up to 4 h and energy intake at subsequent meals were not significantly different (Isaksson et al., 2012).

Lupin-kernel flour, derived from the endosperm of lupin seeds contains 40–45% protein and 25–30% fiber with negligible amounts of sugar and starch (Lee et al., 2006). A lupin-kernel fiber-enriched sausage patty has been shown to produce greater effects on satiety than both a conventional patty and an inulin fiber-enriched patty (Archer, Johnson, Devereux, & Baxter, 2004). Partial substitution of lupin-kernel flour for wheat flour used in bread making increases the protein and fiber content of bread. Bread made by a substitution of 40% of wheat flour with lupin-kernel flour was compared with bread made with 100% wheat flour. Served as isoenergetic breakfasts with margarine, and jam, the lupin-kernel fiber bread resulted in greater satiety and lower energy intake at lunch when compared with the wheat bread (Lee et al., 2006).

β-Glucan, which is a soluble fiber found in significant amounts in oats and barley, exhibits a high viscosity at relatively low concentrations (Sadiq Butt, Tahir-Nadeem, Khan, Shabir, & Butt, 2008). The satiating effect of β-glucan has been demonstrated in several studies using β-glucan in doses ranging from 2.2 to 9 g (Beck, Tapsell, et al., 2009; Beck, Tosh, et al., 2009; Lyly et al., 2009; Schroeder, Gallaher, Arndt, & Marquart, 2009; Vitaglione et al., 2009; Vitaglione et al., 2010). However, the results have been inconsistent. Some studies found no effect of β-glucan on satiety (Hlebowicz, Darwiche, Bjorgell, & Almer, 2008; Hlebowicz et al., 2007; Kim, Behall, Vinyard and Conway 2006).

Bread made with 100% wheat flour was compared with bread in which 4.5% of the wheat flour was replaced with 3 g of concentrated extract of barley β-glucan. The bread containing barley β-glucan increased satiety and reduced food intake at a subsequent meal by 19% as compared with the bread made with 100% wheat flour (Vitaglione et al., 2009). In contrast, inclusion of barley β-glucan into breakfast and lunch meals (including barley cereal at breakfast and barley bread at lunch) did not increase satiety as compared with wheat-containing meals (including bran flakes at breakfast and refined-wheat bread at lunch) with similar energy and nutrient contents. Although barley-containing meals were associated with higher energy intake during the remainder of the day, intake was assessed through self-reported food records (Keogh, Lau, Noakes, Bowen, & Clifton, 2007) which are susceptible to misreporting and altered feeding behavior (Stubbs, Johnstone, O'Reilly, & Poppitt, 1998).

Breakfast cereals are often produced from crushed or rolled oats (Sadiq Butt et al., 2008). The content of β-glucan in commercial grade oats in North America varies from 35 to 50 g/kg (Malkki & Virtanen, 2001). When present in cereal-based foods, the physiological response is affected by the amount, solubility, molecular weight, and structure of the β-glucan in the products. These physicochemical properties are in turn affected by the source, processing treatments such as milling, temperature, pH, and shear effects, as well as the interactions with other components in the food matrix (Skendi, Biliaderis, Lazaridou, & Izydorczyk, 2002).

Viscosity is controlled by concentration in solution and molecular weight (Wood, 2007). Oat β-glucan is more soluble in hot water than in water at room temperature, so processing steps that involve moisture and heat will in all likelihood increase the solubility of β-glucan (Tosh et al., 2010). β-Glucan is connected with the cellulose and other noncellulosic polysaccharides in the cell wall and cooking releases it from this matrix (Johansson, Tuomainen, Anttila, Rita, & Virkki, 2006). When used as an ingredient in muffins, the cooking of oats has been shown to increase the percentage of β-glucan solubilized by threefold (Wood, 2004). Thus, food structure and matrix of the product delivering the β-glucan affects its functionality (Skendi et al., 2002).

Breakfast cereals containing oat β-glucan in amounts ranging from 2.2 to 5.7 g and a corn-based breakfast cereal (0 g β-glucan) were compared. The breakfast meals were isoenergetic. Subjective satiety increased with each of the breakfast meals containing oat β-glucan when compared with the corn-based breakfast meal, but there was no difference in the overall satiety responses between the breakfasts containing oat β-glucan in varying amounts. Subsequent food intake decreased only with β-glucan doses in excess of 5 g (Beck, Tosh, et al., 2009). In a separate study, the same investigators examined the effects by varying the dose of oat β-glucan from 2.2 to 5.5 g, delivered through breakfast cereals, on appetite and satiety. They concluded that the optimal dose of β-glucan affecting satiety and other markers of appetite regulation were between 4 and 6 g. Increasing the dose of β-glucan resulted in a greater release of PYY. The hormonal effects were mediated through increased viscosity (Beck, Tapsell, et al., 2009).

In contrast, some studies have shown that oat β-glucan had no effect on satiety. Muesli containing 4 g of oat β-glucan served in yogurt did not increase satiety when compared with an isoenergetic meal consisting of cornflakes served in yogurt (Hlebowicz et al., 2008). Satiety ratings were compared following ingestion of wheat bran flakes (7.5 g fiber), whole-meal oat flakes (4 g fiber: 0.5 g β-glucan), and cornflakes (1.5 g fiber) of equal weight served with milk. Neither bran flakes nor oat flakes resulted in significantly higher satiety when compared with corn flakes (Hlebowicz et al., 2007).

In a study investigating effects of barley β-glucan on satiety, hunger was found to be lower with barley products (9 g β-glucan) when compared with whole-wheat and rice products. The products were served at breakfast as a hot cereal and at mid-morning as a snack mix (Schroeder et al., 2009). In other studies, 1.2 g barley β-glucan in a meal replacement bar (Peters et al., 2009) had no effect on satiety, and a hot cereal containing 2 g of barley β-glucan did not affect short-term satiety in overweight individuals (Kim, Behall, Vinyard, & Conway, 2006).

The insoluble fiber found in breakfast cereals made with whole-grain wheat has also been demonstrated to increase satiety as compared with cornflakes of equal weight (Hamedani et al., 2009) or equal energy content (Samra & Anderson, 2007). The amount of insoluble fiber used in these studies was fairly high, ranging from 26 to 33 g/meal (Hamedani et al., 2009; Samra & Anderson, 2007). In a comparison of isoenergetic breakfasts, increased fullness was observed with a meal high in insoluble fiber (whole-grain wheat bran breakfast cereal: 18.1 g fiber) when compared with a breakfast of bacon and eggs (Holt et al., 1999), higher in fat and comparable in protein.

Whole-grain wheat breakfast cereals that provide fiber in excess of 18 g have been shown to increase satiety, but the evidence is limited. Novel fiber and protein combinations such as that obtained from lupin-kernel flour hold promise, but again the evidence is limited. While the effects of rye on subjective satiety appear to be unequivocal, the results of studies investigating the satiating effects of oats and barley are inconsistent. Additionally, rye porridge appears to be more satiating than rye bread; however, it is not clear if the effects rye exerts on satiety translate into a reduction in food intake.

Epidemiologic as well as intervention trials for the most part evaluated the effects of bread consumption on weight and body composition as part of a food group rather than focusing on bread alone. A comprehensive review (Bautista-Castano & Serra-Majem, 2012) of the epidemiologic data indicated that in a majority of the studies the food group containing bread was not associated with weight status. However, whole-grain bread consumption was found to have beneficial effects on weight status (Cleveland et al., 2000; Greenwood et al., 2000; Koh-Banerjee et al., 2004) and abdominal fat distribution (Halkjaer et al., 2006; Jacobs, Meyer, Kushi, & Folsom, 1998) when compared with bread made with refined grains.

In the Physician's Health Study, a 13-year prospective study, BMI, and weight gain were found to be inversely related to consumption of breakfast cereals regardless of type (whole grain or refined) and independent of other risk factors (Bazzano et al., 2005). In another prospective study, weight gain was inversely associated with the intake of high-fiber, whole-grain foods but positively related to the intake of refined-grain foods (Liu et al., 2003). Using data from NHANES, it was found that eating a breakfast cereal was associated with a significantly lower BMI in adults when compared with other types of breakfast or not eating breakfast (Cho, Dietrich, Brown, Clark, & Block, 2003). Further, consuming a ready-to-eat breakfast cereal (RTEC) was also associated with a macronutrient profile conducive to prevention of obesity in women but not in men (Song, Chun, Obayashi, Cho, & Chung, 2005).

In a RCT, individuals with self-reported night snacking behaviors reduced their postdinner energy intake while consuming a RTEC as an after-dinner snack when compared with a control group consuming their usual snacks (Waller et al., 2004). However, in a study among obese individuals participating in partial meal replacement program, a postdinner RTEC snack did not enhance weight loss when compared with the control group not consuming the RTEC snack (Vander Wal et al., 2006). In another study, while energy intake reduced in the group consuming a RTEC as an evening snack for 6 weeks, there was no significant change in body weight as compared with the control group consuming their usual snacks (Matthews, Hull, Angus, & Johnston, 2012). A RCT tested the effectiveness of a partial meal replacement including breakfast cereal products on weight status. At the end of 4 weeks, greater reductions in BMI, waist, hip, and thigh measurements, as well as percent body fat were seen in subjects consuming the meal replacements containing breakfast cereal products when compared with subjects consuming their normal diet (Wal, McBurney, Cho, & Dhurandhar, 2007). In another study, subjects consuming a RTEC at breakfast and as a meal replacement at lunch or dinner for 2 weeks lost significantly more weight than subjects consuming their usual diet (Mattes, 2002).

Incorporation of oatmeal into a hypocaloric diet for 6 weeks to increase the soluble fiber content (3.9 g) did not result in any significant changes in satiety, body weight, or body composition as compared with a control diet low in soluble fiber (1.9 g) (Saltzman et al., 2001). In an intervention trial to test the effectiveness of weight control measures based on increasing cereal consumption (especially breakfast cereals) or increasing consumption of vegetables, no significant differences in food intake, BMI, or fat mass were observed between the two treatment groups (Rodriguez-Rodriguez et al., 2008). In a 16-week trial to compare the effects of replacing bread, rice, pasta, and breakfast cereals in the usual diet with 100% wheat bread or bread in which 40% of the wheat flour was replaced with lupin-kernel flour, there were no significant effects on body weight or fat mass (Hodgson et al., 2010).

While epidemiologic data suggest that bread consumption does not adversely affect weight status, the paucity of experimental data precludes the establishment of a cause and effect relationship. Observational studies also support whole-grain consumption for improvements in body composition measures. RTECs may have a beneficial role in weight management when used as a partial meal replacement and may reduce energy intake when consumed as a night snack.

3.2.2 Fruits and vegetables

The energy density of a food is primarily a function of its water content; however, fiber does play a lesser role (Drewnowski, 2003). Fat is the most energy-dense nutrient, which provides 9 kcal/g versus 4 kcal/g provided by carbohydrate or protein. Most fruits and vegetables have a high-water content, a low-fat content, and are associated with increased consumption of fiber, making them low in energy density (Rolls, Ello-Martin, & Tohill, 2004). Consumption of fruits and vegetables has been associated with increases in satiety (Gustafsson, Asp, Hagander, & Nyman, 1994, 1995;

Holt et al., 1995) and beneficial effects on weight management (de Oliveira, Sichieri, & Venturim Mozzer, 2008; He et al., 2004; Vioque, Weinbrenner, Castello, Asensio, & Garcia de la Hera, 2008). The energy density and fiber content may have a role to play in the effects of fruit and vegetable consumption on the regulation of food intake and body weight (Alinia, Hels, & Tetens, 2009; Rolls, Ello-Martin, et al., 2004).

The effects of different vegetables on satiety were investigated in a series of experiments (Gustafsson, Asp, Hagander, & Nyman, 1993; Gustafsson et al., 1994, 1995). In the first study (Gustafsson et al., 1993), carrots, peas, Brussels sprouts, or spinach were added to a typical Swedish lunch meal in portions of 96–164 g. The meals were similar in energy and macronutrient content providing 4.4 g of fiber from the added vegetables. No effects of vegetable intake were observed on ratings of satiety as compared with a control lunch. In a subsequent study (Gustafsson et al., 1995) of similar design, 150 and 200 g of spinach providing 4.3 and 7.2 g of fiber, respectively, increased satiety as compared with a control meal without spinach. Further, the satiety ratings positively correlated with the dietary fiber and water content of the meal. When carrots in portions of 100, 200, and 300 g providing 2.9, 5.8, and 8.7 g dietary fiber, respectively, were added to a mixed lunch, satiety increased in a dose–response manner when compared with an isoenergetic meal without carrots (Gustafsson et al., 1994). Thus, it appears that the addition of spinach or carrots to meals in portions of 200 g or more can provoke a satiety response.

In a study comparing the effects of bean purée with potato purée on satiety, it was found that the bean purée delayed the return of hunger and decreased ratings for desire to eat (Leathwood & Pollet, 1988). Another study assessed the effects of a first course comprised of salads (iceberg and romaine lettuce, carrots, cherry tomatoes, celery, and cucumber tossed with Italian dressing and shredded mozzarella and parmesan cheese) in three versions of energy density each served in two different portion sizes. It was found that the portion size of the salad served as a first course was the major factor determining subsequent intake of pasta served as the main course. Energy density had no statistically significant effect on subsequent intake. However, when intake of the entire meal was analyzed, energy intake decreased as the energy density of the salad was decreased, regardless of the portion size. However, portion sizes but not energy density affected satiety ratings with the larger salads eliciting a greater satiety response (Roe, Meengs, & Rolls, 2012). In another study, the covert incorporation of pureed vegetables to lower the energy density (three versions) of main entrees resulted in a decreasing energy intake over the day as the energy density of the entrées was reduced. However, ratings of hunger and fullness did not significantly differ across the conditions (Blatt, Roe, & Rolls, 2011a).

In a study to assess the satiating capacities of isoenergetic portions of 38 different foods categorized into six groups (fruits, bakery products, confectionery, protein-rich foods, carbohydrate-rich foods, and breakfast cereals), fruits were found to elicit the greatest average satiety score (Holt et al., 1995). Although the consumption of dried fruit in the United States is low, it has been associated with improved nutrient intakes, and lower body weight, BMI, and waist circumference (Keast, O'Neil, & Jones, 2011). However, daily consumption of fruits and nut bars (80 g) for 8 weeks did not significantly affect measures of BMI, weight, and waist circumference (Davidi et al., 2011). Nevertheless, a preload of prunes was associated with a greater effect on satiety and a reduction in energy intake at a subsequent meal when compared with an isoenergetic bread and cheese snack (Farajian, Katsagani, & Zampelas, 2010).

Studies have assessed the effects of the physical form of fruits on satiety and energy intake (Bolton, Heaton, & Burroughs, 1981; Flood-Obbagy & Rolls, 2009; Haber, Heaton, Murphy, & Burroughs, 1977). Whole apples were associated with higher satiety ratings than apple purée which in turn was more satiating than apple juice (Haber et al., 1977). Similarly, whole oranges provided greater satiety than orange juice, and whole grapes increased satiety as compared with grape juice (Bolton et al., 1981). A study assessed the effect of a preload of apple juice with added fiber, applesauce, and whole apples matched for weight, energy density, energy, and fiber content on subsequent energy intake. It was found that subjects consumed significantly less energy from the test meal after eating apple segments compared to the applesauce or apple juice, and that the applesauce preload reduced energy intake as compared with the apple juice preload. Eating apple segments also resulted in higher ratings of fullness and lower ratings of hunger compared to other forms of fruits (Flood-Obbagy & Rolls, 2009).

In a review of the epidemiologic data on the relationship between fruit and vegetable intake and body weight, including 16 studies in adults, eight were found to report a significant inverse association between fruit and vegetable intake and weight status which did not vary regardless of the category being fruits and vegetables, fruits only, or vegetables only (Tohill, Seymour, Serdula, Kettel-Khan, & Rolls, 2004). However, in a subsequent review of epidemiologic data, it was concluded that fruit and nonstarchy vegetable consumption was not associated with levels of subsequent weight gain and obesity (Summerbell et al., 2009).

In a prospective investigation involving three separate cohorts, 4-year weight change was found to be inversely related to fruit intake (Mozaffarian, Hao, Rimm, Willett, & Hu, 2011). In the Diet, Obesity, and Genes (DiOGenes) study, including 89,432 individuals from five countries participating in the EPIC study, an inverse association was observed between fruit and vegetable intake and annual weight change (Buijsse et al., 2009). Including participants from 16 centers in addition to those included in the DiOGenes study, the EPIC-PANACEA study found that baseline fruit and vegetable intakes were not associated with weight change after an average of 5 years of follow-up (Vergnaud et al., 2012).

A computer-assisted dieting intervention trial found that although fruit consumption did not increase, fruit intake and body weight were inversely related (Schroder, 2010). In a systematic review, it was determined that among adult experimental studies, increased fruit and vegetable consumption reduced adiposity but the relationship was due to multiple weight-related behaviors. Additionally, in this review, longitudinal studies suggested only a weak relationship between fruit and vegetable consumption and adiposity (Ledoux, Hingle, & Baranowski, 2011). A review of intervention, prospective, observational, and cross-sectional studies on fruit intake and body weight in adults indicated that a majority of the evidence points to an inverse relationship between fruit intake and body weight in the adult population (Alinia et al., 2009).

There appears to be some evidence associating increased fruit consumption with a reduction in adiposity. Fruits and vegetables have a high water, low protein, and low-fat content yet differ greatly in their nutritional profiles, sensory properties, and culinary usage (Dauthy and Food and Agriculture Organization of the United Nations, 1995). The United States Department of Agriculture (USDA) provides separate recommendations for fruits and vegetables (USDA, 2011). Nevertheless, a vast majority of the studies have assessed the combined effects of fruit and vegetable intake on body weight. The data, however, do not support an unequivocal association between fruit and vegetable intake and body weight.

4. FATS AND THE REGULATION OF FOOD INTAKE

The regulation of fat intake involves an integration of physiological events that begins with perception through the nose or mouth of fat-soluble volatile flavor molecules. Food texture defined as the mechanical perception of the oral sensations stimulated by the placement of food in the mouth is then sensed by the oral cavity during chewing and swallowing (Drewnowski, 1997).

However, evidence suggests that the sensing of fat could also be mediated through chemoreception (Drewnowski & Almiron-Roig, 2010). Although the transduction pathways for fatty acid (FA) taste are not clearly understood, numerous receptor systems have been isolated from animal and human tissue (Stewart, Feinle-Bisset, & Keast, 2011).

One mechanism proposed to explain the detection of FAs is through the inhibition of potassium channels (Stewart et al., 2011). It has been shown that long-chain *cis*-polyunsaturated FAs inhibited potassium channels and prolonged stimulus-induced depolarization of rat taste receptor cells. However, saturated, monounsaturated, and *trans*-polyunsaturated fatty acids had no effect on potassium channels (Gilbertson, Fontenot, Liu, Zhang, & Monroe, 1997). Additionally, a FA transporter (CD36) which binds long-chain fatty acids (LCT) in human taste receptors aids in calcium-mediated signaling of taste. The threshold of taste detection is, however, dependent on FA chain length (Mattes, 2009). Using a modified sham feeding technique, in a comparison of high-fat meals containing olive oil, linoleic acid, and oleic acid, it was found that feelings of satiety increased with modified sham feeding of all oils. Thus, the metabolic effects of consumption of high-fat foods may at least in part be modulated by oral exposure (Smeets & Westerterp-Plantenga, 2006).

Lipases present in digestive juices, inside cells, and in endothelial cells aid the chemosensory process. Oxidized FAs or FAs in high concentrations have an unpleasant taste (Stewart et al., 2011). However, in adults, the levels of lingual lipase are low (Drewnowski & Almiron-Roig, 2010). Thus, the levels of fatty acids imputed to stimulate the sensation of taste are low enough to not be sensed as unpleasant, but sufficient to activate taste receptors (Stewart et al., 2011). The ability to taste food containing oxidized fat may be an evolutionary adaptation designed to avoid ingestion of undesirable or toxic compounds (Drewnowski & Almiron-Roig, 2010).

Fat-soluble compounds that contribute to the odor accompanying fats stimulate receptor cells that send signals to brain structures also involved in the processing of emotions and memories. This overlap of neuroanatomical structures offers a biological explanation for feelings of pleasure or disgust that are produced in response to the odorous compounds from fats. Neuroimaging studies have identified the areas of the brain that are activated by fat in the mouth and by viscosity (De Araujo & Rolls, 2004). Further, it appears that the areas of the brain that coordinate the neuronal responses to satiety coincide with the areas of the brain that coordinate neuronal activity related to whether a food tastes pleasant and whether it should be eaten (Rolls, 2004).

Fats are higher in energy density than carbohydrates and proteins, but fats have the added distinction of a characteristic taste and texture that contributes to the palatability of foods (Drewnowski, 1997). One school of thought suggests that palatability reflects an underlying biological need for a nutrient predicted by the sensory properties of the food, while the other relates palatability to reward processes (Yeomans, Blundell, & Leshem, 2004). Distinct neural substrates for homeostatic and hedonic systems have been identified, which implies that the processes of reward can operate free of biological deficits (Blundell & Finlayson, 2004). Palatable foods by influencing appetite sensations can stimulate overconsumption (Yeomans et al., 2004). However, based on this view, unpalatable foods as an appetite-reducing strategy do not present a plausible course of action (Mela, 2006).

Foods that are both energy dense and high in fat are typically the most palatable foods. High palatability is associated with increased food intake, whereas satiety and satiation are associated with a decrease in food intake. Fat by virtue of its palatability stimulates an increase in intake (Drewnowski & Almiron-Roig, 2010). Thus, fat does not satiate but may increase satiety. The combination of taste and smell sensations (using vanilla) has been shown to enhance satiety following consumption of a high-fat meal (Warwick, Hall, Pappas, & Schiffman, 1993). However, the role of palatability in fat-induced overeating is unclear. While the initial food selection may be based on orosensory qualities, postingestive nutritional factors may determine how much energy is consumed (Sclafani, 2004). Children given repeated exposures to distinctly flavored high-fat and low-fat yogurt drinks matched for orosensory characteristics increased their preference for the high-fat flavor (Johnson, McPhee, & Birch, 1991).

Fats have been shown to reduce hunger when present in the GI tract by eliciting satiety signals (Little & Feinle-Bisset, 2011). Fat in the duodenum stimulates the release of cholecystokinin directly and other GI peptides such as PYY and GLP-1 by an indirect neurohumoral pathway to affect satiety (Maljaars et al., 2007). Exposure of the ileum to fat stimulates an even larger satiety response than exposure to the duodenum (Maljaars et al., 2008). Fat reaching the ileum stimulates the ileal brake, a distal to proximal feedback mechanism that slows gastric emptying and delays the transit of food through the GI tract. Nutrients in the small intestine influence satiety and food intake by activation of neural afferents or by inducing the release of gut hormones involved in appetite regulation (Maljaars et al., 2007; Van Citters & Lin, 1999).

Bariatric surgery is arguably the most effective weight-loss treatment for the morbidly obese (Karra, Chandarana, & Batterham, 2009). The

Roux-en-Y gastric bypass surgery results in a speedy delivery of partially digested nutrients to the distal parts of the GI tract. Meal-stimulated increases in PYY, and GLP-1, gut hormones with anorectic effects, implicated in the ileal brake activation, have been observed in subjects who have undergone the Roux-en-Y gastric bypass (Field, Chaudhri, & Bloom, 2010; Karra et al., 2009). Thus, the reduction in body weight and the physiological responses observed following bariatric surgery provide evidence that a sustained appetite-reducing effect is possible through a recurring activation of the ileal brake (Maljaars et al., 2008).

Infusion of triglycerides into the ileum has been shown to alter duodenal motility and delay gastric emptying (Fone, Horowitz, Read, Dent, & Maddox, 1990). Ileal fat infusion has also been shown to cause a dose-dependent delay in gastric emptying and has been related to increased plasma concentrations of PYY (Pironi et al., 1993; Read et al., 1984). Following an ileal infusion of corn oil, feelings of satiety increased and *ad libitum* food intake decreased at a meal 30 min after the start of the infusion. Although in this study, the rate of infusion of fat can be compared to what one may find in normal subjects after eating a heavy meal (Welch, Saunders, & Read, 1985; Welch, Sepple, & Read, 1988), even a low physiological dose of fat (6 g) into the ileum elicited a significant reduction in hunger and food intake when compared with an oral ingestion of the same amount of fat (Maljaars et al., 2011).

In a pooled analysis of studies investigating the effects of fat on gastric emptying and GI hormone release, it was determined that the magnitude of stimulation of pyloric pressures and release of cholecystokinin, a hormone with anorexigenic effects, are independent predictors of subsequent energy intake (Seimon et al., 2010). A high-fat breakfast meal has been shown to delay gastric emptying at lunch as compared with low-fat meals matched for energy or mass of the high-fat meal; however, the high-fat breakfast meal resulted in increased food intake 7 h later (Clegg & Shafat, 2010).

The regulation of GI motor function, gut hormone release, and satiety by fat is affected by its physicochemical properties. These effects are more pronounced with LCT ($\geq$12 carbons) than shorter chain fatty acids (Feltrin et al., 2004; French et al., 2000; Little & Feinle-Bisset, 2011). Food intake was reduced by over 200 kcal following a duodenal infusion of long-chain fat emulsions (180 kcal) when compared with a saline infusion (French et al., 2000). Duodenal infusion of 12-carbon fatty acids reduced appetite and energy intake at a subsequent meal when compared with 10-carbon fatty acids. The effects on gastroduodenal motility that were observed are typically associated with delayed gastric emptying (Feltrin et al., 2004). It has been suggested that accelerated gastric emptying decreases gastric distension, thereby promoting hunger (Little, Horowitz, & Feinle-Bisset, 2007). Thus, hunger and gastric emptying are closely related. For fat to affect gastric emptying, however, digestion of fats and consequent release of free fatty acids appear to be crucial (Little et al., 2007).

Medium chain triglycerides (MCT) (6–12 carbons) have been shown to influence satiety through increased energy expenditure. Unlike LCT, MCT are directly absorbed into portal circulation and are more rapidly metabolized. The faster rate of oxidation increases thermogenesis (St-Onge & Jones, 2002). Three isoenergetic breakfasts matched for fat content, but differing in fatty acid chain length was compared with respect to their effects on satiety. LCT from beef tallow, MCT from coconut oil, and short-chain triglycerides from dairy fat were added to savory muffins. There was no significant difference in satiety ratings following consumption of the three breakfasts differing in the type of lipid when measured over 6 h (Poppitt et al., 2010).

In a comparison of meals differing in the degree of saturation of the fat content, no significant differences in satiety were observed. The meals were high in polyunsaturated fatty acids from walnuts, or monounsaturated fatty acids from olive oil, or saturated fatty acids from dairy fat (Casas-Agustench et al., 2009). The role played by the degree of saturation in modulating the effects of fat on the GI tract has yet to be resolved (Maljaars, Romeyn, Haddeman, Peters, & Masclee, 2009; Strik et al., 2010).

Pinnothin™ is a natural oil pressed from Korean pine nuts and contains linoleic acid (C18:2), pinolenic acid (C18:3), and oleic acid (C18:1). Consumption of Pinnothin™ triacylglycerols and free fatty acids have been shown to produce an increase in cholecystokinin and GLP-1 in postmenopausal overweight women when compared with olive oil. However, appetite ratings did not significantly differ (Pasman et al., 2008). In overweight women, although consumption of Pinnothin™ free fatty acids reduced food intake by 7% at a subsequent meal, appetite ratings were not significantly different after consumption of Pinnothin™ triacylglycerols or free fatty acids compared to olive oil (Hughes et al., 2008). In both the studies (Hughes et al., 2008; Pasman et al., 2008), participants consumed Pinnothin™ in a capsule form. Added to yogurt, Pinnothin™ triacylglycerol consumption did not result in appetite sensations and energy intake that were significantly different when compared with milk fat (Verhoef & Westerterp, 2011).

Delaying lipid digestion is an important factor in stimulating the ileal brake. The digestion of fat can be slowed down by manipulating the oil emulsion interfacial composition using galactolipids. It has been shown that galactolipids reduce the rate and extent of lipolysis by sterically hindering the penetration of pancreatic colipase and lipase or preventing the formation of a colipase–lipase complex at the oil–water interface in the duodenum (Chu et al., 2009). Olibra™ is a fat emulsion comprising fractionated palm and oat oil in the proportion of 95:5. The palm oil is emulsified by hydrophilic galactolipids derived from oat oil (Knutson et al., 2010). One study using a method of delivering Olibra™ directly into the GI tract demonstrated a delay in GI transit (Knutson et al., 2010). Although oral administration in another study showed a 45-min delay in orocecal transit time (Haenni, Sundberg, Yazdanpandah, Viberg, & Olsson, 2009), the computation of orocecal transit time has been questioned (Peters, Beglinger, Mela, & Schuring, 2010). However, when ingested orally, the GI responses manifested by an intragastric administration may differ. Unless the emulsion is resistant to digestion in the dynamic environment of the GI tract, an increase in satiety and a reduction in food intake are unlikely to occur.

Early studies (Burns, Livingstone, Welch, Dunne, & Rowland, 2002; Burns et al., 2000; Burns et al., 2001), all crossover designs, reported a reduction in energy, macronutrient, and total weight of food intake following consumption of yogurt containing the Olibra™ emulsion. Subsequent studies failed to show a reduction in energy intake (Chan et al., 2012; Diepvens, Steijns, Zuurendonk, & Westerterp-Plantenga, 2008; Logan et al., 2006; Rebello, Martin, Johnson, O'Neil, & Greenway, 2012). Olibra™ has been shown to positively impact body composition and weight maintenance after weight loss (Diepvens, Soenen, Steijns, Arnold, & Westerterp-Plantenga, 2007), but in another study although body fat mass decreased by 0.9%, there was no change in body weight at the end of 12 weeks (Olsson, Sundberg, Viberg, & Haenni, 2011). In these studies by Diepvens et al. and Olsson et al., the calorie restriction imposed during the weight-loss period may have had a role to play in the beneficial effects. In a recent review, it was concluded that Olibra had no efficacy as a satiety-enhancing weight-loss strategy (Rebello et al., 2012).

In humans, exposure to a high-fat or high-energy diet has been shown to decrease sensitivity to the GI mechanisms that regulate appetite (Clegg et al., 2011; Little & Feinle-Bisset, 2011; Little et al., 2007). It has been suggested that dietary restriction may cause a reversal of these effects resulting in enhanced nutrient sensing and appetite suppression (Little & Feinle-Bisset, 2011). A modification of appetite perceptions with an increase in hunger and a decrease in fullness has been observed following a high-fat diet

(58% of energy intake) for 2 weeks. Further, a significant increase in energy intake of about 160 kcal/day was observed for the following 2-week period (French, Murray, Rumsey, Fadzlin, & Read, 1995). Placing subjects on a high-fat diet derived from sunflower oil for only 3 days resulted in an acceleration of gastric emptying (Clegg et al., 2011). However, the acceleration in GI transit and reduction in satiety following a high-fat diet that occurred over a 1-week period returned to prediet levels by the end of 4 weeks (Clegg et al., 2011).

Fat is the most energy-dense macronutrient, contributing to pleasantness and thereby perceived palatability of foods, which may induce overconsumption. Fats bestow on foods a wide range of taste and texture properties, making it difficult to determine which particular oral sensations contribute to the perception of fat content. Fat perception is also influenced by physical form and other taste sensations, such as sweetness. Important textural properties include viscosity and lubricity (Drewnowski & Almiron-Roig, 2010). By adding hydrocolloid thickeners or other components that influence viscosity, it is possible to create an illusion of fat content (Drewnowski & Almiron-Roig, 2010). While fat may not reduce meal termination (satiation), fat in the GI tract generates satiety (meal initiation). The physicochemical properties of fat influence the postingestive effects of fat on satiety, but these properties can be manipulated. Thus, the paradoxical effects of fats on energy density and satiety notwithstanding, they could be manipulated to produce a desired directional change in feeding behavior.

5. TEAS, CAFFEINE, AND PUNGENT FOODS

Certain food components do not provide energy but have been shown to increase energy expenditure. Tea is made from the leaves of the *Camellia sinensis* L. species of the Theaceae family. Oolong tea is partially fermented and oxidized, while green tea is not fermented or oxidized. Both oolong and green tea contain several polyphenolic components such as epicatechin, epicatechin gallate, epigallocatechin, epigallocatechin gallate (EGCG), and caffeine. Of these polyphenols, EGCG is the most abundant and is highly active pharmacologically (Hursel & Westerterp-Plantenga, 2010; Kovacs & Mela, 2006).

Catechins inhibit catechol *O*-methyltransferase an enzyme that degrades norepinephrine, and caffeine inhibits phosphodiesterase, an enzyme that degrades c-AMP. A reduction in degradation causes an increase in the levels of norepinephrine and c-AMP. Norephinephrine controls biochemical mechanisms that either result in an increased use of ATP or an increased rate of mitochondrial oxidation with inefficient coupling of ATP synthesis, leading to increased heat production (Hursel & Westerterp-Plantenga, 2010; Westerterp-Plantenga, Diepvens, Joosen, Berube-Parent, & Tremblay, 2006). Thus, the thermogenic effects of caffeine and tea catechins are related to prolonged or increased stimulatory effects of norepinephrine and c-AMP on energy and lipid metabolism (Kovacs & Mela, 2006).

The results of a meta-analysis of studies investigating the effects of green tea on weight loss and weight maintenance suggest that an EGCG–caffeine mixture has a beneficial effect on weight loss and weight maintenance after a period of energy restriction. Subjects in the treatment groups lost an average of 1.31 kg or gained 1.31 kg less weight than subjects in the control groups over a 12-week period. However, habitually low-caffeine consumers reacted with greater sensitivity than habitually high consumers. Ethnicity appeared to be a moderator of the thermogenic effect as Asian subjects lost more weight than Caucasians. There was no dose–response relationship between intake of catechins and body weight (Hursel, Viechtbauer, & Westerterp-Plantenga, 2009).

Caffeine belongs to a class of compounds called methylxanthines and is present in coffee, tea, cocoa, chocolate, and some cola drinks (Westerterp-Plantenga et al., 2006). The effect of caffeine intake on energy expenditure has been demonstrated in several short-term studies (Acheson, Zahorska-Markiewicz, Pittet, Anantharaman, & Jequier, 1980; Acheson et al., 2004; Arciero, Gardner, Calles-Escandon, Benowitz, & Poehlman, 1995; Astrup et al., 1990; Bracco, Ferrarra, Arnaud, Jequier, & Schutz, 1995; Dulloo, Geissler, Horton, Collins, & Miller, 1989; Hollands, Arch, & Cawthorne, 1981). However, a RCT found that there was no effect on body weight over a 16-week period (Pasman, Westerterp-Plantenga, & Saris, 1997) although epidemiologic data from a 12-year prospective study supported an inverse association between caffeine intake and long-term weight gain (Lopez-Garcia et al., 2006). It appears that although caffeine intake may result in increasing energy expenditure in the short term, the evidence to support its effects on weight loss is lacking. The available evidence supports a role for green tea in weight loss; however, a meta-analysis determined that the magnitude of the effect may lack clinical relevance (Phung et al., 2010).

Capsaicin is the major pungent ingredient in red hot pepper. Capsaicin has been reported to increase thermogenesis by enhancing catecholamine secretion and inducing β-adrenergic stimulation (Yoshioka et al., 1995). Capsaicin is perceived as pungent because it activates the transient receptor potential vanilloid receptor 1 (TRPV1) found in neurons on the tongue.

The activation of the TRPV1 receptor stimulates the release of catecholamines which leads to an increase in energy expenditure by stimulation of the sympathetic nervous system and the upregulation of uncoupling proteins (Hursel & Westerterp-Plantenga, 2010).

Studies have assessed the effects of capsaicin on energy metabolism in humans and demonstrated an increase in energy expenditure (Yoshioka et al., 1995), diet-induced thermogenesis, and fat oxidation (Yoshioka, St-Pierre, Suzuki, & Tremblay, 1998). In a comparison of high-fat and high-carbohydrate breakfast meals with and without red pepper, it was found that the red pepper-containing meals reduced appetite before lunch. Differences in diet composition at the breakfast meal did not affect energy and macronutrient intake at lunch, but protein and fat intake at lunch was reduced with intake of red pepper-containing meals consumed at breakfast (Yoshioka et al., 1999). The effects were more pronounced in the high fat as opposed to the high-carbohydrate diet (Yoshioka et al., 1998; Yoshioka et al., 1999). The addition of red pepper to an appetizer at lunch time resulted in reduced intake of carbohydrate and energy during the rest of the lunch and a snack served several hours later (Yoshioka et al., 1999). The effect of red pepper on reducing energy intake was found to be greater when administered in tomato juice than when administered in capsule form (Westerterp-Plantenga, Smeets, & Lejeune, 2005). In another study, although a capsaicin containing meal resulted in an increase in GLP-1, there were no effects on appetite and energy expenditure as compared with a control meal without capsaicin (Smeets & Westerterp-Plantenga, 2009).

The data on the long-term consumption of capsaicin are scarce. Capsaicin supplementation for 3 months after a modest weight loss had no effect on weight maintenance as compared with a control group that was not supplemented. In this study, compliance with the diet appeared to pose a problem (Lejeune, Kovacs, & Westerterp-Plantenga, 2003). Capsinoids are nonpungent capsaicin-related substances found in the CH-19 sweet pepper and have been investigated for their thermogenic effects (Snitker et al., 2009). Capsinoid supplementation for 12 weeks was well tolerated but did not affect energy expenditure or body weight, although a significant increase in fat oxidation and reduction in abdominal adiposity was observed (Snitker et al., 2009).

There appears to be some evidence to support an increase in satiety and a reduction in food intake following consumption of foods containing capsaicins but the data are inconsistent. Long-term studies investigating the effects of capsaicin consumption on body weight are lacking which may perhaps be due to the difficulty in adhering to a diet containing pungent foods. The CH-19 sweet pepper may promote greater compliance, but its effects on satiety, food intake, and body weight need further substantiation through controlled studies.

6. ENERGY DENSITY

Energy density is defined as the amount of energy per unit weight of a food or beverage (most commonly expressed as kilocalories per gram or kilojoules per gram). The amount of water present in a food is a major influencer of energy density because water adds weight without adding calories. Macronutrient composition also influences the energy density of a food with fat providing 9 kcal/g compared to 4 kcal/g for carbohydrates and protein. Dietary fiber adds weight while contributing minimal energy. Thus, food that is high in water and/or fiber is often low in energy density.

Several epidemiologic studies have found a positive association between the energy density of the diet and measures such as weight gain, BMI, and waist circumference (Perez-Escamilla et al., 2012). While the effects of energy density on satiety have been mixed, numerous short-term studies have found that increasing the energy density of a test food or meal decreases energy intake at subsequent meals. Some studies have shown that adding a low-energy density preload, such as salad or soup, before a meal decreases the amount of food consumed at the meal (Flood & Rolls, 2007; Rolls, Bell, & Waugh, 2000; Rolls, Roe, & Meengs, 2004; Rolls et al., 1998). Flood and Rolls found that when subjects consumed vegetable soup prior to their lunch, meal-time energy intake decreased by 20% or 134 ± 25 kcal (Flood and Rolls, 2007). Consuming salad before or with a meal resulted in an 11% (57 ± 19 kcal) decrease in meal-time energy intake (Roe et al., 2012). These studies demonstrate that adding low-energy density foods to a meal can reduce the amount of calories consumed at a single meal.

One strategy to lower the energy density of foods is to increase their water content. Work from Barbara Rolls' lab has shown that incorporating water into food decreases meal-time energy consumption (Rolls, Bell, & Thorwart, 1999). Adding water to a chicken rice casserole so that it became a chicken rice soup resulted in a 16% reduction in energy intake at lunch. Subjects did not compensate for their reduced lunch-time food intake at dinner. However, water served as a beverage with a meal did not alter energy intake suggesting that water as a beverage is perceived differently than water in a food such as soup.

Another strategy for reducing the energy density of foods is the incorporation of puréed vegetables into recipes. Blatt et al. covertly substituted puréed carrots, squash, and cauliflower into various recipes and tested subjects' energy intake compared to the normal recipes. When the energy density of meals was lowered by 15%, daily energy intake decreased by 202 ± 60 kcal. When the energy density of meals was lowered by 25%, daily energy intake decreased by 357 ± 47 kcal (Blatt et al., 2011a). While ingredient substitution may be challenging to implement for the home cook, it has been shown to be effective for decreasing short-term energy intake and increasing the amount of vegetables eaten. Various other studies have found that decreasing the energy density of a meal leads to decreased energy intake at the meal itself and subsequent food intake later in the day (Bell, Castellanos, Pelkman, Thorwart, & Rolls, 1998; Bell & Rolls, 2001; Chang, Hong, Suh, & Jung, 2010; Cheskin et al., 2008; Latner, Rosewall, & Chisholm, 2008; Rolls, Bell, Castellanos, et al., 1999; Rolls, Roe, & Meengs, 2006). Collectively these studies show that lowering the energy density of meals can successfully reduce short-term energy intake, which suggests that maintaining a low-energy density diet may result in weight loss. While the evidence from short-term food intake studies is very strong, longer-term weight-loss studies have produced mixed results.

A small number of RCTs have been performed to examine the role of energy density in weight loss. Two studies manipulated the diet of subjects by having them add snacks of varying energy density to their diets. De Oliveira et al. had overweight or obese subjects ($n = 49$) add three apples, pears, or oat cookies to their usual diet and measured weight change over 10 weeks (de Oliveira et al., 2008). Both the apple and pear groups lost weight (−0.93 and −0.84, respectively), while the oat cookie group gained a small amount of weight (+0.21 kg). Viskaal-van Dongen et al. had normal-weight participants add low or high-energy density snacks to their usual diets but found no differences in weight change between the two groups after 8 weeks (Viskaal-van Dongen, Kok, & de Graaf, 2010).

Alternatively, other trials have provided counseling on increasing the energy density of participants' diets but provided no study foods. A short 4-week study found no differences between a calorie-restricted high-energy density diet and a calorie-restricted low-energy density diet (Song, Bae, & Lee, 2010). The low-energy density group reported less hunger but weight loss was not different between the groups. A 12 week study found that subjects on a low-energy density diet lost an average of 9.3 kg similar to results achieved by subjects consuming an energy-restricted low-fat diet (−7.7 kg). Combining these two sets of dietary advice did not result in enhanced weight loss (Raynor, Looney, Steeves, Spence, & Gorin, 2012). Similarly, a 6-month weight-loss trial for obese women produced an average weight change of −6.4 kg with a reduced-fat diet and −7.9 kg with a reduced-fat diet plus increased fruits and vegetable consumption (Ello-Martin, Roe, Ledikwe, Beach, & Rolls, 2007). Though the 1.5 kg difference is modest, these values were statistically significant ($P=0.019$). The longest study which measured weight change with reduced dietary energy density is a 4-year trial with female breast cancer survivors. Groups received counseling on increasing fruit and vegetable consumption or general dietary guideline materials. After 4 years, there were no differences in weight between the two groups (Saquib et al., 2008). Overall, the evidence suggests that reducing dietary energy density may be an effective tool for promoting weight loss. However, there currently is no evidence to suggest that it is superior to other weight-loss strategies.

Choosing foods with lower energy density may be one way to promote reduced energy intake and enhance weight loss, but there are some exceptions to choosing solely based on energy density. Sugar sweetened beverages such as soda generally have a low-energy density value, but they have been linked to the promotion of excess energy intake and weight gain (Malik, Popkin, Bray, Despres, & Hu, 2010). Indeed, several studies have found that beverages are only weakly satiating compared to solid food, and it may be that calories consumed from beverages are not sensed by the body in the same way as calories from foods (Mattes, 2006; Mattes & Campbell, 2009). The other exception appears to be nuts, which were discussed earlier in this review. Nuts have very high-energy density values but do not appear to cause excess weight gain when consumed regularly. The energy densities of some common foods are presented in Table 3.1.

Table 3.1 Energy density of selected foods based on the United States Department of Agriculture National Nutrient Database for Standard Reference, Release 24

Food	Energy density (kcal/g)
Butter	7.17
Walnuts	6.54
Almonds	5.95
Peanuts	5.85
Pistachio nuts	5.67
Potato chips	5.42

Table 3.1 Energy density of selected foods based on the United States Department of Agriculture National Nutrient Database for Standard Reference, Release 24—cont'd

Food	Energy density (kcal/g)
Chocolate chip cookie	4.54
Cheddar cheese	4.03
Prunes	3.39
Pork sausage	3.39
Cheesecake	3.21
Pizza	2.76
Ground beef	2.70
Rye bread	2.58
Bagel	2.57
Whole-wheat bread	2.47
Chicken breast	1.65
Chickpeas	1.64
Eggs	1.55
Navy beans	1.40
Tuna	1.28
Lentils	1.16
Low-fat yogurt	1.02
Banana	0.89
Tofu	0.70
Apple	0.52
Orange juice	0.49
1% milk	0.42
Carrots	0.41
Butternut squash	0.40
Grapefruit	0.32
Cauliflower	0.25
Spinach	0.23
Lettuce	0.15

7. MEAL PLANS

The effects of select foods on satiety, food intake, and body weight using evidence from RCTs are presented in Table 3.2. Meal plans that provide 1200, 1600, or 2000 kcal were developed (Tables 3.3–3.5). These meal plans meet 100–113% of the USDA recommendations in the dairy, fruit, vegetable, and grain food groups and provide about 22–24% of energy from protein. Despite the relatively high-protein contents, the meal plans do not exceed one egg/day and 6 ounces/day in servings from meat and fish. Dairy products are either low fat or fat free in keeping with the recommendations

Table 3.2 The effects of individual foods on satiety, food intake, and body weight using evidence from randomized controlled trials

Food	Satiety	Food intake	Body weight
Protein	+	+/−	+/−
Dairy products	ND	ND	+/−
Milk	+/−	+/−	+ (with energy restriction)
Yogurt	+	−	+
Cheese	ND	ND	ND
Meat and meat products	ND	ND	−
Beef/pork/chicken	−	−	−
Fish	+	+	+
Eggs	+	+/−	+ (with energy restriction)
Pulses	+	ND	+/−
Chickpeas	−	−	ND
Lentils	+/−	+/−	ND
Navy beans	−	−	ND
Yellow peas	+/−	+/−	ND
Soybean	+/−	−	−
Walnuts	+/−	−	+

Table 3.2 The effects of individual foods on satiety, food intake, and body weight using evidence from randomized controlled trials—cont'd

Food	Satiety	Food intake	Body weight
Almonds	+	+	+/−
Peanuts	ND	ND	+
Pistachios	ND	ND	+
Carbohydrates			
Breads and cereals	ND	ND	+/−
Whole-wheat bread	–	–	ND
Rye bread	+	ND	ND
Lupin bread	+	+	ND
Barley bread	+/−	+/−	ND
Rye porridge	+	+/−	ND
Oat breakfast cereal	+/−	–	ND
Barley breakfast cereal	+/−	–	ND
Whole-wheat breakfast cereal	+	ND	ND
Ready-to-eat-cereal	ND	+	+/−
Fruits and vegetables	ND	ND	–
Fruits	+	ND	+
Apple/pear/grapefruit	ND	ND	+
Dried fruit/prunes	+	+	+
Vegetables	+	ND	ND
Spinach ≥ 200 g	+	ND	ND
Carrots ≥ 200 g	+	ND	ND
Salad (raw vegetables)	ND	+	ND
Teas, caffeine, and pungent foods			
Green tea	ND	ND	+
Oolong tea	ND	ND	+
Caffeine	ND	ND	–
Capsaicin	+/−	+/−	–

+, Beneficial effect; − no effect; +/− inconsistent effect; ND, not determined.

Table 3.3 Meal plan that provides approximately 1200 kcal/day

Meal	1200 kcal	Weight (g)
Breakfast	1 cup cooked rye porridge or oatmeal (a, b)	234
	¼ cup dried fruit (a, b)	34
	1 cup fat-free milk (a, b, c)	245
Snack	6 ounces low-fat yogurt (a, c)	183.75
	1 cup green tea (c)	
Lunch	1 egg omelet (a, b, c)	61
	Sautéed green beans ¾ cup cooked green beans (a)	93.75
	1 teaspoon olive oil	4.5
	¼ cup sliced almonds (a, b, c)	26.25
	1 slice bread (rye, lupin, or barley) (a, b)	32
Snack	½ cup fruit (a, c)	77.63
	1 cup fat-free milk (a, b, c)	245
Dinner	1½ cups chicken and rice soup (d)	361.5
	1 slice bread (rye, lupin, or barley) (a, b)	32
	½ cup steamed carrots sliced (a)	78

The meal plan meets 100–112% of the USDA recommendations in the dairy, fruit, vegetable, and grain food groups and provides 24% of energy from protein. Energy density of the diet is 0.70 kcal/g. Analyzed using the USDA National Nutrient Database for Standard Reference, Release 24.
a, Satiety; b, food intake; c, body weight: evidence provided in randomized controlled trials. d, Energy density studies: Rolls et al. (1998, 2000b), Rolls, Roe, and Meengs (2004), Flood and Rolls (2007). Green tea is not included in the energy density calculation.

Table 3.4 Meal plan that provides approximately 1600 kcal/day

Meal	1600 kcal	Weight (g)
Breakfast	1 egg (a, b, c)	61
	2 slices bread (lupin, rye, or barley) (a, b)	64
	2 teaspoons light *trans*-fat-free margarine	9.6
	1 slice low-fat cheese	28.35
	1 cup fruit (a, c)	155.27
Snack	6 ounces low-fat yogurt (a, c)	183.75
	¼ cup dried prunes (a, b, c)	43.5
	1 cup green tea (c)	

Table 3.4 Meal plan that provides approximately 1600 kcal/day—cont'd

Meal	1600 kcal	Weight (g)
Lunch	Grilled chicken salad (d)	
	2 cups lettuce/tomatoes/celery/cucumber (b)	237
	2 tablespoons low-fat salad dressing	32
	2 ounces grilled chicken breast	56.7
	¼ cup sliced almonds (a, b, c)	26.25
	1 slice bread (lupin, rye, or barley) (a, b)	32
Snack	1 cup whole-wheat cereal (a)	60
	1½ cups fat-free milk (a, b, c)	367.5
Dinner	4 ounces fish (steamed or grilled) (a, b, c)	113.4
	1 cup sautéed spinach (a)	180
	1 teaspoon olive oil	4.5
	1 slice bread (lupin, rye, or barley) (a, b)	32

The meal plan meets 100–109% of the USDA recommendations in the dairy, fruit, vegetable, and grain food groups and provides 24% of energy from protein. Energy density of the diet is 0.95 kcal/g. Analyzed using the USDA National Nutrient Database for Standard Reference, Release 24.
a, Satiety; b, food intake; c, body weight: evidence provided in randomized controlled trials. d, Energy density studies: Rolls et al. (1998, 2000b), Rolls, Roe, and Meengs (2004, 2007). Green tea is not included in the energy density calculation.

of the 2010 Dietary Guidelines for Americans. The fiber content ranges from 20 g/day in the 1200 kcal diet to 42 g/day in the 2000 kcal diet. Nonnutritive sweeteners may be added to increase the palatability of foods, if desired.

The most distinctive aspect of these meal plans is the evidence-based consideration given to satiating properties of the foods included, and the overall energy density of the diet. We would like to term these meals as a "high satiety" (HS) plan. To illustrate the basis for choosing various foods to include in the HS meals, we have used a scoring system. The letters a–d following the foods indicate the underlying available research evidence about that food. For instance, the letter a denotes that the food was reported to induce a subjective feeling of satiety, the letter b indicates evidence for a reduction in food intake, c refers to evidence for a role in weight loss, and the letter d indicates evidence for using that food to increase satiety by lowering

Table 3.5 Meal plan that provides approximately 2000 kcal/day

Meal	2000 kcal	Weight(g)
Breakfast	1 cup rye porridge or oatmeal (a, b)	234
	¼ cup sliced almonds (a, b, c)	28.35
	1 egg (a, b, c)	61
	1 slice bread (lupin, rye, or barley) (a, b)	32
	1 teaspoon light *trans*-fat-free margarine	4.8
	1 cup fat-free milk (a, b, c)	245
Snack	½ cup bean dip (a, c)	131
	6 baked tortilla chips	8.4
	1 cup green tea (c)	
Lunch	Roast beef sandwich	
	2 ounces roast beef	56.7
	½ cup lettuce + 2 tomato slices (b)	68
	1 tablespoon light mayonnaise	15.6
	2 slices bread (lupin, rye, or barley) (a, b)	64
	1 cup lentil soup (a, b, d)	248
	½ cup grapefruit (c)	115
Snack	1 medium pear (c)	178
	1½ cups fat-free milk (a, b, c)	367.5
Dinner	4 ounces fish (baked or broiled) (a, b, c)	113.4
	Spinach salad (d)	
	2 cups raw spinach (a, b)	60
	¼ cup dried cranberries (a, b)	27.5
	2 tablespoons low-fat salad dressing	32
	¼ cup low-fat cheese crumbled	37.5
	1 slice bread (lupin, rye, or barley) (a, b)	32
	1 teaspoon light *trans*-fat-free margarine	4.8

The meal plan meets 100–113% of the USDA recommendations in the dairy, fruit, vegetable, and grain food groups and provides 22% of energy from protein. Energy density of the diet is 0.92 kcal/g. Analyzed using the USDA National Nutrient Database for Standard Reference, Release 24.
a, Satiety; b, food intake; c, body weight: evidence provided in randomized controlled trials. d, Energy density studies: Rolls et al. (1998, 2000b), Rolls, Roe, and Meengs, (2004, 2007).

the energy density. Thus, at a glance, these HS meal plans convey the scientific basis for the foods included. The value of the HS-meal plans is apparent, particularly when compared with the typical American meal plan (Table 3.6). Based on the evidence, almost every food item included in the HS plans has satiating properties compared to only two items (raw vegetables and whole milk) in the typical American plan. The energy density of the HS meals ranges from 0.70 to 0.95 kcal/g compared to 1.54 kcal/g for the typical American diet.

Table 3.6 Sample menu of the typical American diet (2100 kcal/day) based on the control diet used in the DASH trial[a]

Meal	2100 kcal	Weight (g)
Breakfast	Apple juice unsweetened	126
	Blueberry muffin	50
	Butter without salt	10
	Jelly	14
	Whole milk	120
Lunch	Turkey breast meat	100
	Lettuce, iceberg (raw)	20
	Mayonnaise salad dressing	20
	Bread (white)	55
	Yellow cake	50
	Chocolate frosting	25
Dinner	Pork stir fry	227
	Olive oil	6
	Rice (cooked, white)	200
	Bread (French)	40
	Butter	20
	Gelatin dessert	135
	Dessert topping (nondairy)	20
Snack	Applesauce (canned)	110
	Saltine crackers	20

Energy density of the diet is 1.54 kcal/g.
[a]Appel et al. (1997).

We would like to emphasize a few points. Most of the foods included in these HS-meal plans have been shown to increase satiety when studied individually. The evidence relating to the satiety-enhancing effect is neither unequivocal in each case nor is there evidence to demonstrate that when consumed collectively, the satiating foods will have a synergistic or additive effect on satiety. We have postulated this logical extension of the available information. Perhaps, future research may test these concepts.

In summary, based on the available data and information, we have created HS-meal plans. Compared to a typical American diet, these meal plans are considerably lower in energy density and are probably more satiating. A diet that exploits the satiating properties of multiple foods may help increase long-term dietary compliance, and consequentially enhance weight loss.

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