Review Article

FAD DIET

Mariam Omar*[1], Faiza Nouh[1], Manal Younis[2], Nesma Nabil[1],Bushra Elamshity[1], Hajar Ahmad[1], Ibraheem Elhadad[1], Abdelraouf Elmagri[1]

[1]Department of Nutrition, Faculty of Public Health, Benghazi University, Benghazi, Libya

[2]Royal College of Obstetrics and Gynecologist, Cork University Maternity Hospital (CUMH), Cork, Ireland.

*Corresponding author

Faiza Nouh

Email: faiza.nouh@uob.edu.ly

Abstract

This paper reviewed the common types of fad diets. Fad diets have an effective role in promoting weight loss, beneficial effects on body composition. Fad diets may protect against the development of obesity and related chronic diseases such as type two diabetes and coronary heart disease. Fad diets work simply because they restrict calorie intake, showing that the most important dietary concept of weight loss and maintenance is a decrease in caloric intake. Based on the contemporary studies on fad diets, the future concept for successful weight loss could run on the concept of energy density, which refers to the amount of energy in a given weight of food.

Key Words: fad, diet, low, carbohydrates, fat

Introduction

Obesity is an important public health problem during the recent years. Obesity has reached epidemic proportions. The prevalence of obesity has increased gradually throughout this century. The prevalence of obesity has reached alarming levels, affecting virtually both developed and developing countries across all socio-economic groups irrespective of age, sex or ethnicity (1). Childhood obesity has been estimated that worldwide. Over 22 million children under the age of 5 are severely overweight. One in every ten children are overweight (1-2). The prevalence of overweight in Africa and Asia is below 10% and in the Americas and Europe, above 20% (3, 4) (Table 1).

Table 1: Prevalence and projection of overweight /obesity in children and adolescents in various regions of the world

Region	Overweight *	Obesity**	Overweight*	Obesity**
Africa (1987-2003)	1.6	0.2		
Americas(1988-2002)	27.7	9.6	46.4	15.2
Eastern Med (1992-2001)	23.5	5.9	41.7	11.5
Europe (1992-2003)	25.5	5.4	38.2	10
South East Asia (1997-2002)	10.6	1.5	22.9	5.3
West pacific (1993-2000)	12.0	2.3	27.2	7.0

*Body mass index more than 25 kg/m^2 - **Body mass index more than 30 kg/m^2

The proportion of school-age children affected will almost double by 2025 compared with the most recently available surveys from the late 1990s up to 2003 (5-7). Obesity is an independent risk factor for overall mortality (2). Obesity is associated with heart diseases, sleep apnea, diabetes mellitus, arthritis, and cancer (8). In 2014, the World Health Organization acknowledged that obesity was a "global epidemic" (4), and it was based on classifications depending on the BMI cut-offs (Table 2). The BMI itself is the weight in kilograms divided by the square of the height in meters.

Diet is one of the factors contributing to obesity as excessive calorie intake leads to weight gain (9). Increased availability of fast foods and televised entertainment could contribute to obesity by making eating more attractive and physical activity less. Previous research on the relationship between television viewing and body weight has shown positive associations in studies involving children and adults (6).

Table 2: Cut –off points by a WHO expert committee for the classification of overweight

BMI(kg/m^2)	WHO classification	Popular description
<18.5	Underweight	Thin
18.5-24.9	-	Healthy "normal", "Acceptable"
25.0-29.9	Grade 1 Overweight	Overweight
30.0-39.9	Grade 2 Overweight	Obesity
>40.0	Grade 3 Overweight	Morbid Obesity

Fad diet

Studies have shown that fad diet is a weight loss eating plan that promises dramatic results over short term period (8). In fact, to some extent, the word "fad" is not a scientific term (17). Conversely, the widely used term "healthy" is also not scientific (17). However, an attribute of a fad diet is that those who consume it do not like it (18). It is generally considered that a fad diet is the other person's diet (9, 17). In practical terms there are two kinds of fad diets; firstly, those that have some quirky feature which hardly anybody adheres to (unlike fads in fashion), and secondly, the bête noire of the nutritional establishment. The operational definition of fad therapies includes the following; "therapies used for a limited period of

time", "therapies used without proven efficacy", "therapies used by a limited under of patients", and "therapies that are not mainstream" (10).

People have always been obsessed with dieting. They will try the latest fad diets that defy logic, basic biochemistry, and even appetite appeal. Fad diets are popular because they promise quick results, are relatively easy to implement, and claim remarkable improvements in how their followers will look or feel. Unfortunately, there are some types of attributes fad diets have in common, and that is, they seldom promote sound weight loss(9). The common features of fad diets can be summarized according to (19) as follows. "They promise rapid weight loss"; "there is a restriction of one food item or food group"; "They promote intake of a particular food item or food group" and that "they claim to cure numerous health problems". Other attributes include these; "they have simplistic theories that are presented in a scientifically sound manner" though "they are not supported by scientific evidence".

Glycemic index

There are general concepts that must carefully be understood when such studies relating to low glycemic are made. Glycemic index (GI) is a physiological assessment of foods carbohydrate content through its effect on postprandial blood glucose concentrations. GI has been defined as the outcome of a test of glucose levels of a person in the morning, after an overnight fast (37). However, it must be understood that it is the property of carbohydrate-containing foods that describe the rise of blood glucose post-prandial. There are foods that are rapidly digested and absorbed or transformed metabolically into glucose, and thus have a high GI index (40). GI is also defined as the incremental area under the glucose response curve after a standard amount of carbohydrate from a test food relative to that a control food (either white bread or glucose) is consumed. Another attribute is that the GI of a specific food or meal is determined primarily by the nature of the carbohydrate consumed as well as other dietary factors that affect nutrient digestibility or insulin secretion. GI values for common foods differ by more than 5 fold and glycemic response to mixed meals can be predicted with reasonable accuracy from the glycemic index of constituent foods when standard methods are used (36). On the other hand, the term of glycemic load (GL) is defined as the weighted average GI of individual's food multiplied by the percentage of dietary energy.

Low fat diet

Another very important terminology that must be understood is "low fat diet" which is defined as limiting food sources of fat. Dietary guidelines recommend a reduction in total fat content to less than 30% of the energy intake to help reduce the prevalence of obesity, ischemic heart disease and certain cancer (50). In 2001, the American Heart Association published its therapeutic lifestyle changes (TLC) diets and recommended that fat should account for no more than 30% of a person's daily intake of calories. It was also recommended that saturated fats should make up no more than 10% of total calories (51). The therapeutic lifestyle changes are aimed at an energy intake of 1500 kcal per day for women and 1800 kcal per day for men. The publication also recommended that 30% of calories must be obtained from fat, 10% of calories from saturated fat, and an intake of a maximum of 300 mg of cholesterol per day (52). The American Heart Association recommended keeping total fat intake to less than 35% of total calories (20 g per day based on a 2,000- calorie diet) and limiting trans-fat consumption to less than 1% (or about 2 g based on a 2,000-calorie diet). The consumption of saturated fat to less than 7% of total daily calories has also been recommended.

A very low fat diet is also defined as one in which 15% of total calories are derived from fat (33 g for a 2000-calorie diet and 50 g for a 3000-calorie diet) with fat calories distributed approximately equally among saturated, monounsaturated and polyunsaturated fatty acids. Approximately 15% of total daily calories consumed should be derived from protein and more than or equal 70% from carbohydrates. Similarly, current dietary guidelines from both the American Heart Association and the National Cholesterol Education Program recommend restricting consumption of fat to an upper limit of 30% of daily caloric intake. This limit translates into 67 g of fat for small or sedentary individuals who need 2000 calories per day and 100 g of fat for larger or more active individuals who need 3000 calories per day. World Health Organization Study Group recommends that 15% of total calories be derived from fat. However, current guidelines do not specify a lower limit on fat intake (54).

METHODOLOGY

Google search engine was used to gather information relating to obesity and associated diets. The studies were written in English. The study period has occurred from January to the end

of June 2018. Keywords included “fad diet”, “low carbohydrates”, and “fat”. The search generated about 165 sources, of which 70 sources were used. These 70 articles were considered relevant because they answered the objectives of the review. The library databases such as PubMed and MEDLINE were also used during the study.

Factors influencing obesity

Obesity is not a single disorder but a heterogeneous group of conditions with multiple causes (8). Body weight is the result of the interaction between genetic, environmental and psychosocial factors, acting through the physiological mediators of energy intake and expenditure (9-10).

Fatness runs in families but the influence of the genotype on the etiology of obesity may be attenuated or exacerbated by non-genetic factors (11). The genetic influences seem to operate through susceptible genes for obesity which are inherited and impact adversely on body fat composition, anatomical distribution of fat, food intake, and energy expenditure. It has been shown that environmental factors drive the susceptible gene into action (11, 12). Research hypothesized that environmental factors play a key role in unmasking tendencies to develop obesity (12). The biggest component of energy expenditure is a physical activity which represents 20-50% of total energy expenditure (5-7).

It is surprising that no direct correlation has been reported between the prevalence of obesity and increased energy intake in developed nations, given the ready availability of highly palatable foods (13). The evidence for the critical role of environmental factors in the development of obesity comes from migrant studies and the ' westernization' of diet and lifestyles of other cultures previously from developing countries (13). For example, there are reports that those migrants that move to live in the United States are on average 25 kg heavier than Pima Indians living in Mexico (7). Similarly, in Nigeria the mean BMI for men and women has been reported as 21.7 and 22.6 respectively, relative to 27.1 and 30.8 in the United States (7) For instance, the prevalence of hypertension in adult Nigerians living in Africa is 15%, relative to as high as 30% among those living in the United States. A higher prevalence of overweight and obesity is observed in those with lower educational attainments and low income (7, 14-16). There is evidence that over-nutrition of the fetus during

intrauterine development may determine the later onset of obesity, hypertension and type 2 diabetes independent of genetic inheritance. (15)

Types of fad diet

According to (20), there are many types of fad diets. The most popular fad diets are low carbohydrate diets, high fat diets, and very low carbohydrate diets. Some diets are a combination of high carbohydrates, low fat or moderate fat. There are diets that are also low in glycemic index, vegetarian diet, high protein diet, cappage soup diet and grape fruit diet. In this review, only three types of fad diet were discussed; low carbohydrates diet, low glycemic index diet and low fat diet.

Low carbohydrate diet

Low carbohydrate diets have primarily been used in the treatment of diabetes prior to the discovery of insulin (10). Low carbohydrate/high fat diets have also been used in the early 1900s as a pediatric anti-epileptic therapy (11). Low carbohydrate diet has effective role in weight loss and in reduction of cardiovascular diseases risk factors (13). The low carbohydrate, high-protein diet is named "Atkins diet" (14, 15). This diet is low in carbohydrate, high in protein. it is one of the most popular alternative weight loss approaches. These diets derive a large proportion of energy intake from protein and fat (16). Low carbohydrate diet less than 130 gram/day or less than 26% of a nominal 2000 kcal/day diet is the American Diabetes Association definition which seems appropriate (17). Carbohydrate restriction is not well defined. Anything less than 50% of the diet is considered by some to be a low carbohydrate diet from this perspective (17). The definition of low-carbohydrate diets is even more widespread. Some of the diets known to be low in carbohydrate are the Atkins diet, protein powder diet, the South Beach Diet, and the Zone diet (18). However, Low carbohydrate diets appear to have some adverse effects. A low carbohydrate diet has common short-term side-effects such as constipation, fatigue, halitosis, headache, thirst, polyuria, and nausea (19) muscle cramps, diarrhea general weakness, rash, chest pain, and hyperosmolar coma (12). Long term disadvantages of low carbohydrate diet include increased fat intake and consequently high cholesterol level; accelerate the progression of pre-existing kidney disease, increased urinary calcium excretion (16).

Mode of action of low carbohydrate diet

Low carbohydrate diet shows success at six months to one-year period. Low carbohydrate diet decreases insulin resistance and improves lipid profiles. (21, 22). Ketosis occurs during the first few days of fasting or a low carbohydrate diet when breakdown of fat (oxidation) outstrips breakdown of carbohydrate (glycolysis) (23). Low carbohydrate diets work primarily by decreasing food choice in diets where fat and carbohydrate are so tightly associated (24). Also, there is an increased satiety with low carbohydrate diets (25). The systolic pressure did not change significantly but diastolic pressure decrease after following the low carbohydrate diet for six months (25, 26) Low carbohydrates diet relatively increases HDL cholesterol concentration and relatively decreases triglyceride concentration (27). Carbohydrate restriction leads to a marked reduction in triglycerides (TAG). This is one of the most reliable features of any dietary intervention. Changes in total cholesterol and LDL tend to be variable on low carbohydrate diet (28). Low carbohydrate diets are nutritionally inadequate. They are low in vitamins E, A, thiamin, B6, folate, calcium, magnesium, iron, potassium, and dietary fiber. This diet is usually high in saturated fat and cholesterol (29).

Randomized trial of low carbohydrate diet in literature

The study was approved by the institutional review board at the Philadelphia Veterans Affairs Medical during an enrollment period that lasted from May to November 2001, involving one hundred and thirty–two (132) subjects from the Philadelphia Veterans Affairs Medical center. In this study they had randomly assigned 132 severely obese subjects (including 77 blacks 23 women) with a mean body-mass index of 43kg/m^2 and high prevalence of diabetes (39 percent). All subjects, who are exposed to the low-carbohydrate diet lost more weight than those on the low –fat. Subjects on low carbohydrates diet had greater decreases in triglyceride. The amount of weight lost and assignment to the low-carbohydrate diet were independent predictors of improvement in triglyceride levels and insulin sensitivity (30).

In 2001 in Philadelphia they conducted a one-year multicenter, controlled trial involving 63 obese men and women who were randomly assigned to either a low carbohydrate, high-protein, high fat diet. All subjects on the low-carbohydrate diet had lost more weight than subjects on the conventional diet at 3 months (3.7% of body weight) and 6 months (5.6 % of body weight), but the difference at 12 months was not significant. After three months, no

significant difference was found between the groups in total or low-density lipoprotein cholesterol concentrations (32).

Low glycemic index

The glycemic index was proposed in 1981 as an alternative system for classifying carbohydrate-containing food. Several hundred scientific articles and books have been published on the topic (36). One of the main dietary factors that thought to influence body weight is the glycemic index (GI). The glycemic index was initially conceived by Jenkins as a tool for the dietary management of type 1 diabetes and, later dyslipidaemia (37). Several studies have described a link between satiety and body metabolism. A large number of these studies have focused on the role of glycemic index and glycemic load (GI\GL) in weight loss. Evidence suggests that low GI and low glycemic load GL diets may be protective against the development of obesity-related disease (type 2 diabetes and coronary heart disease (CHD) (38). The GI of food will vary depending on the rate of digestion. The faster the digestion of food, the higher is the GI value (>70). Food with a low GI (≤70) is considered to be favorable in terms of health, particularly for the prevention of obesity, T2DM, and CVD (39).

Mode of action

The GI of a food is impacted by the nature of the starch, particle size, pH, the amount of fiber, fat and protein, in addition to cooking method. Low GI diets are thought to be metabolically advantageous because of their potential in improving glycemic control. GI is determined by comparing the postprandial glycemic response of food with response to the postprandial glycemic food (white bread or glucose) in the same individual to the same amount of available carbohydrate from a standard (38). On the other hand, glycemic load (GL) includes both the GI and total carbohydrate intake; thus, approximates the total glycemic effect of the diet, which gives an adequate assessment of the total diet (41). The GI value of a food is tested on the food when eaten on its own, and there are published lists of high, medium and low GI foods (40). The able represents the precooked weight "weight of oats used were adjusted to reflect differences in hydration as described in ‘methods’. Treated with lactase to increase the GI of milk sugar (40).

Metabolic effects of low glycemic-index diets

Low GI diets and metabolic syndrome

GI has been shown to be positively associated with the prevalence of metabolic syndrome and insulin resistance. Weight loss is an additional potential mechanism by which low-GI diets may contribute to a reduced risk of metabolic syndrome (42). It is the result of a great limitation of food choices. Low GI diets give a more stable diurnal profile, reducing postprandial rebounds in circulating free fatty acids, all factors that exacerbate various components of the metabolic syndrome (39). A reduced rate of glucose absorption after the consumption of low glycemic index carbohydrate foods will reduce the postprandial rise in gut hormones such as insulin (42). By reducing insulinemia, low GI foods may provide greater access to fatty acids as a source of fuel, promoting greater fat oxidation. (43) The prolonged absorption of carbohydrate seen over time will maintain suppression of the free fatty acids (FFA) and the glucose will be withdrawn from the circulation at a faster rate. In addition, LDL-cholesterol concentrations were reduced with the low-glycemic-index diet. (42) This improved blood glucose control is of importance to individuals with insulin resistance (e.g. in obese, sedentary subjects), pre-diabetes, and diabetes.

Trial studies

There are a number of studies have shown trials with low glycemic index diets. In a study conducted in University of Minnesota, 20 subjects were randomly assigned to 1 of 3 hypo-caloric test diets: high glycemic index (HGI), low glycemic index (LGI) or high fat (HF); with varying macronutrient composition. The study was conducted in 2 continuous phases; a 12-weeks feeding phase, followed immediately by a 24 weeks "free-living" phase. Each diet group lost body weight during the 12 weeks feeding phase of the study, but the amount lost did not differ among the groups. The improvement in the LGI group was significantly greater than the improvement in the HF group at week 12. This dietary trial demonstrates that energy restriction over a 36-weeks period promotes weight loss and improves insulin sensitivity in obese individuals, irrespective of the dietary substrate. The hypothesis that a low glycemic load diet would enhance weight loss, relative to other diets, was not supported in either study phase (44).

In another trial, twenty-one obese or overweight volunteers (5 men 16 women), aged between 25 and 65 years, subjects with hypercholesterolemia were recruited. The study design is a randomized crossover single- blinded study examining the effects of low GI or GI breakfast on satiety, lipid and glucose metabolism. A total of 21 subjects were recruited to ensure adequate numbers in the event of subjects choosing to withdraw from the trial, and showed that total daily energy intake (calculated from 3-day dietary records) were not significantly different between the low and high dietary interventions after 21 days. Low GI intervention resulted in lower absolute intake in grams of all measured macronutrient compared with high GI intervention. However, the differences between interventions did not reach statistical significance (45).

A third study involving, a total of 23 (25 women and 7 men) of 34 enrolled participants completed the 6-month intervention period and necessary measurements. Participants were randomized for 24 weeks to either a high-glycemic load diet or a low glycemic load diet at 30 % calorie restriction compared with baseline individual energy needs. The main finding from this pilot study was that healthy overweight women and men with relatively greater insulin secretion in response to a standard oral glucose tolerance test lost more weight when assigned to a low-glycemic load hypocaloric diet than to a high glycemic load diet. However, there was no differential effect of the two diets on weight loss in individuals who had relatively lower insulin secretion (46).

In yet another trial, six patients with Non- Insulin Dependent Diabetes Miletus "NIDDM" were studied on both high and low GI diets of six weeks duration with metabolic diets with randomized crossover design. Both diets of similar composition (57% carbohydrate, 23% fat, and 34% g/day dietary fiber), but the low GI diet had a GI of 58 compared with 86 for the high GI diet. They find that small and similar amounts of weight were lost on both diets: 2.5 kg on high-GI diet and 1.8 kg on low GI diet. On the low GI diet, the mean level of serum fructosamine, as an index of overall blood glucose control, was lower than on the high-GI diet by 8% by 7%. The total serum cholesterol was lower. In overweight patients with NIDDM, reducing diet GI improves overall blood glucose and lipid control (47).

Mode of action of low fat diet

Genetic and environmental factors play a role in the development of obesity, and diet is one of the main environmental factors that contribute to this disease. Human studies have shown that increased fat intake is associated with body weight gain which can lead to obesity and other related metabolic diseases (55). In animals, increasing dietary fat increases body fat, and it is believed that human beings show similar important biological trend. In epidemiological studies, increasing dietary fat is associated with increased prevalence of obesity probably because of increased intake of energy dense foods. There is a strong relationship between dietary fat and physical activity which is most evident as the speed of adaption to dietary fat is increased by exercise. When dietary fat is reduced, weight is lost, but weight loss eventually plateaus. Studies have shown that the rate of weight loss during the initial phase is about 1.6 g/day for each 1% decrease in fat intake (54-56). It has been observed that when dietary fat was replaced with Olestra, to reduce fat intake from 33% to 25% in obese men, weight loss continued for about 9 months, reaching a maximum of nearly 6% of body weight and a loss of 18% of initial body fat (55). The study also showed that in the control group with a 25% reduced fat-diet, weight loss ceased for 3 months only to be regained over the next 6 months (56), indicating the difficulty of adhering to a conventional low- fat diet. Thus, dietary fat is an important contributor to obesity in some people. Several intervention trials have examined the effect of a low fat diet with or without energy restriction in overweight subjects and these have been reviewed (57, 58). In a meta-analysis performed by Astrup et al (2001) state that the rate of weight loss was generally greater when the low fat diet was combined with reduction in total energy intake (59). These studies show that apart from energy restriction; a low fat diet alone is effective in inducing weight loss in overweight subjects, with an observed mean weight loss about 1.8 kg/month (60). Accepting the favorable effect of lowering blood lipid concentration, the studies on the quantitative and qualitative effects of dietary fatty acids on serum lipids in man are of interest (61). However, considerable confusion still exists whether a reduction of saturated fatty acid or an increase of polyunsaturated fatty acid concentration in the diet is the more effective approach in lowering human blood lipid concentrations. It has been reported that shorter carbon chain length fatty acids; lauric and myristic acids, each have a stronger effect relative to palmitic acid, oleic acid, stearic acid and other fatty acids with longer chain lengths (61). Furthermore, it has been recently observed that elaidic acid in the presence of cholesterol, has a definite serum

cholesterol increasing effect relative to a mixture of lauric acid and myristic acid, consumed in equal amounts (62).

Trials on low fat diet

A study has been reported on dietary intervention that used a randomized controlled trial group in a 2-year trial involving 322 moderately obese subjects (63). Other characteristics of the subject include a mean age 52 years and a mean body index of 31 kg/m^2. About 86% males in sex and under three types of low fat diets, restricted calorie Mediterranean restricted calorie or low carbohydrate, non-restricted calorie. At this study the Mediterranean diet group consumed their diet in form of dietary fiber and had the highest ration of monounsaturated to saturated fat. In contrast, the low carbohydrate group consumed fat, protein and cholesterol as largest amount of diet but carbohydrate was consumed as the smallest amount of diet. The effect of this study on weight loss could be seen as the mean weight loss of 2.9 kg for the low fat group, 4.4 kg for the low Mediterranean-diet group and 4.7 kg for the low carbohydrate group. Thus, it is believed that Mediterranean and low carbohydrate diets may be effective alternatives to lower fat diet. Another study was done to report data on body weight in a long-term, low fat diet trial for which the primary end points were breast and colorectal cancer and also to examine the relationships between weight changes and changes in dietary components (64). The results showed that women in the intervention group lost weight in the first year (mean of 2.2 kg) and maintained lower weight relative to the controlled women during an average 7.5 years of follow up. It was evident that there was a difference of 1.9 kg per year for the test group relative to 0.4 kg for 7.5 years in the control group respectively. In other words, there was no tendency towards weight gain in the intervention women group overall, either when stratified by age, ethnicity or body mass index. Weight loss was greatest among women in either group who decreased their percentage of energy from fat. A similar but lesser trend was observed with increased vegetable and fruit servings. However, non-significant trend towards weight loss occurred with increasing intake of fiber, thus, a low fat diet pattern does not result in weight gain in postmenopausal women.

CONCLUSION

Fad diets promote quick weight loss which are easy to be implemented and do not take longer time to give the result. Low carbohydrate diet has shown success in weight reduction and

treatment for pathologies such as diabetes and cardiovascular stresses, while it may provide weight loss in the short term. Regarding low fat diet, there is a direct correlation between fat and disease, thus, people can avoid related diseases by decreasing the amount of fat intake in the food. It was also evident that strategies such as a decrease in the total daily energy consumption; reduction of saturated fatty acid or an increase of polyunsaturated fatty acid in the diet is a more effective approach in lowering human blood lipid concentrations.

REFERENCES

1. Kosti RI, Panagiotakos DB. The epidemic of obesity in children and adolescents in the world. Cent Eur J Public Health. 2006;14(4):151-9. Review.
2. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. Jama. 2005;293(15):1861-7.
3. 3-wellman NS, Friedberg B. Causes and consequences of adult obesity: health, socialand economic impacts in the United States. Asia Pacific J Clin Nutr (2002); 1.N Engl J Med. 2003;348:1625-38.
4. Volpe, Stella Lucia, RD, LDN, FACSM, popular weight reduction diets. Journal of Cardiovascular Nursing. 2006;21(1):34-39.
5. Lew EA. Mortality and weight: Insured lives and the American Cancer Society studies. Ann Intern Med. 1985;103(6 (Pt 2)):1024-9.
6. Liu T, Howard RM, Mancini AJ, Weston WL, Paller AS, Drolet BA, Esterly NB, Levy ML, Schachner L, Frieden IJ. Kwashiorkor in the United States: fad diets, perceived and true milk allergy, and nutritional ignorance. Archives of dermatology. 2001;137(5):630-6.
7. Hales CN, Barker DJ, Clark PM, Cox LJ, Fall C, Osmond C, Winter PD. Fetal and infant growth and impaired glucose tolerance at age 64. BMJ. 1991;303(6809):1019-22.
8. Gui G, Diets FAD. Fats & weight management. Department of nutrition &dietetics, Alexandra Hospital, SEP. 2008;34(4):14-9.
9. American Heart Association: No –fad diet . A personal plan for healthy weight loss, New York: Clarkson Potter; 2005.
10. Rollo J. Tow cases of the diabetes mellitus, the discovery of insulin. JAMA. 2003; 289(14):1837-50.
11. Vamecq J, Vallée L, Lesage F, Gressens P, Stables JP. Antiepileptic popular ketogenic diet: emerging twists in an ancient story. Prog Neurobiol. 2005;75(1):1-28. Epub 2005 Jan 27. Review.
12. Atkins RC. Dr. Atkins New diet Revolution. New York: Harper Collins; 1999.
13. Han TS, Van Leer EM, Seidell JC, Lean ME. Waist circumference action levels in the identification of cardiovascular risk factors: Prevalence study in a random sample. BMJ. 1995;311(7017):1401-5.
14. Atkins RC, Dr. Atkins new diet revolution. Avon Books, New York, NY; 2002.
15. Bravata DM, Sanders L, Huang J, Krumholz HM, Olkin I, Gardner CD, Bravata DM. Efficacy and safety of low-carbohydrate diets: A systematic review. JAMA. 2003;289(14):1837-50.
16. Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS Jr, Brehm BJ, Bucher HC. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk

factors: A meta-analysis of randomized controlled trials. Arch Intern Med. 2006;166(3):285-93.
17. Richard D F fad diet in the treatment of diabetes American association. Nutrition recommendations and interventions for diabetes- 2008. Diabetes Care. 2008;31 (suppl):s61-s78.
18. Stella LV. Popular weight reduction diets journal of cardiovascular nursing. 2006;21(1):34YxB.
19. Freedman MR, King J, Kennedy E. popular diets: A scientific review. Obese Res 2001;9:1s-40s Ann int Med. 2004;140:796-777.
20. Eric C. Westman, MD, MHS, John Mavropoulos, MPH, William S. Yancy, Jr., MD, MHS, and Jeff S. Volek, PhD, RD. low carbohydrate diet effect. J Pediatr. 2003;142:253-258.
21. Frederick F. Samaha, M.D, N. Iqbal, M.D, Prakashseshadri, M.D. Low carbohydrate as compared with low fat diet in severe obesity. The New England Journal of Medicine. 2006;355:1991-2002.
22. Seshadri P, Iqbal N. low carbohydrate diets for weight loss: Historical &environmental perspective Indian J Med Res. 2006;123:739-747.
23. Cardillo S, Seshadri P, Iqbal N. The effects of a low-carbohydrate versus low-fat diet on adipocytokines in severely obese adults: Three-year follow-up of a randomized trial. Eur Rev Med Pharmacol Sci. 2006;10(3):99-106.
24. Andrew J. Brown. Low-carb diets, Fasting and Euphoria Medical Hypotheses. 2007; 68:268-271.
25. Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams T, Williams M, Gracely EJ, Stern L. A low-carbohydrate as compared with a low-fat diet in severe obesity. N Engl J Med. 2003;348(21):2074-81.
26. Havel RJ. Lipoprotein and lipid transport. Metabolic Control and Disease. 1980;393-494.
27. Jackson RL, Morrisett JD, And A.M gotto, Jr. lipoprotein structure and metabolism. Physiol. Rev. Journal of Lipid Research. 1976;56259-316.
28. Krauss RM, Blanche PJ, Rawlings RS, Fernstrom HS, Williams PT. Separate effects of reduced carbohydrate intake and weight loss on atherogenic dyslipidemia. Am J Clin Nutr. 2006;83(5):1025-31. Quiz 1205. Erratum in: Am J Clin Nutr. 2006;84(3):668.
29. Marjorie R. Freedman, janet king, and Eileen Kennedy crowe, Timothy 2005, safety of low carbohydrate diets , Obes Rev. 2005;6(3):235-45. Review.
30. Wisconsin Nutrition Education Program. Family living programs. Nutrition for Family Living; 2004 .
31. Gary D. Foster, Ph.D, Holly R. Wyatt, M.D, James O. Hill, Ph. Brian. Randomized trial of low carbohydrate diet for obesity. The New England Journal of Medicine. 2003;348:2082-90.
32. Yancy WS, Olsen MK, Guyton JR, Bakst RP, Westman EC. A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: A randomized, controlled trial. Annals of Internal Medicine. 2004;140(10):769-77.
33. Brody, Keller, Degen, Cox, Scha Chinger. Efeect of 6month adherence to a low carbohydrate diet program. Am J Med. 2002;113:30-6.
34. Seshadri P, Iqbal N. Indian J Med Res. 2006;123:739-747.
35. Xavier F, Sunyer Pi. Glycaemic index and disease. Am J Clin Nutr. 2012;76(Suppl):290s-8s.
36. Esfahani A, Wong JM, Mirrahimi A, Villa CR, Kendall CW. The application of the glycemic index and glycemic load in weight loss: A review of the clinical evidence. IUBMB Life. 2011;63(1):7-13. DOI: 10.1002/iub.418. Review.

37. Radulian G, Rusul E, Dragomir A, Posea M. Metabolic effects of low glycaemic index diets. Nutrition Jornal. 2009;38:75.
38. David S. Ludwig, Joseph A. Majzoub, Ahmad Al-Zahrani, Gerard E. Dallal, Isaac Blanco, Susan B. Roberts. High Glycemic Index Foods, Overeating, and Obesity. Pediatrics.march1999:vol-103 No-3
39. Glucose, Insulin and Non Esterified Fatty Acid Responses to Ladies Finger and Pointed Gourd in Type 2 Diabetes Mellitus. International journal of nutrition and metabolism. 2011; 3(1):1-6.
40. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL, Axelsen M. Glycemic index: Overview of implications in health and disease. Am J Clin Nutr. 2002;76(1):266S-73S. Review.
41. Brand-Miller J, McMillan-Price J, Steinbeck K, Caterson I. Dietary glycemic index: Health implications. J Am Coll Nutr. 2009;28 Suppl:446S-449S. Review.
42. Raatz SK, Torkelson CJ, Redmon JB, Reck KP, Kwong CA, Swanson JE, Liu C, Thomas W, Bantle JP. Reduced glycemic index and glycemic load diets do not increase the effects of energy restriction on weight loss and insulin sensitivity in obese men and women. J Nutr. 2005;135(10):2387-91.
43. Pal S, Lim S, Egger G. The effect of a low glycaemic index breakfast on blood glucose, insulin, lipid profiles, blood pressure, body weight, body composition and satiety in obese and overweight individuals: A pilot study. J Am Coll Nutr. 2008;27(3):387-93.
44. Pittas AG, Das SK, Hajduk CL, Golden J, Saltzman E, Stark PC, Greenberg AS, Roberts SB. A low-glycemic load diet facilitates greater weight loss in overweight adults with high insulin secretion but not in overweight adults with low insulin secretion in the CALERIE Trial. Diabetes Care. 2005;28(12):2939-41. No abstract available.
45. Brand J. Effect of low glycaemic index diet in overweight niddm subject. Diabetes Care. April 1992;15:4.
46. Strychar I. Diet in the management of weight loss . CMAJ. 2006;174(1).
47. Govindji A, Glycaemic index. 6 September 2011;3:5.
48. Katanmb, Grandysm, Willettwc, beyond low fat diet, Newengl J. (1international Journal of Obesity). 1997;337:563-566.
49. Mendelson, Scott. Metabolic syndrome and psychiatric IIness: interactions, pathophysiology, assessment and treatment: Academic Press. 2008;146-337.
50. Krauss RM, Eckel RH, howard B, et al. AHA dietary guidelines: Revision 2000: A statement for healthcare professionals from the nutrition committee of the American heart Association. Circulation. 2000;102:2284-99.
51. Krauss RM, Deckelbaum RJ, Ernst N, Fisher E, Howard BV, Knopp RH, Kotchen T, Lichtenstein AH, McGill HC, Pearson TA, Prewitt TE, Stone NJ, Van Horn L, Weinberg R. Dietary guidelines for healthy American adults. Circulation. 1996; 94:1795-1800.
52. **S**ummary of the Second Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). Journal of American Medical Association. 1993;269:3015-3023.
53. World Health Organization Study Group on Diet, Nutrition, and Prevention of Non-communicable Diseases. Diet, Nutrition and the Prevention of Chronic Diseases: Report of a World Health Organization Technical Report Series. 1990;797.
54. Buettner R, Schölmerich J, Bollheimer LC. High-fat diets: modeling the metabolic disorders of human obesity in rodents. Obesity (Silver Spring). 2007;15(4):798-808. Review.

55. Van Heek M, Compton DS, France CF, Tedesco RP, Fawzi AB, Graziano MP, Sybertz EJ, Strader CD, Davis HR Jr. Diet-induced obese mice develop peripheral, but not central, resistance to leptin. J Clin Invest. 1997;99(3):385-90.
56. Physiology & behavior 83 (2004) 549-555 George A. bray*, Sahaspornpaeratakul, Barry M. popkin (pennington Biomedical Research center , Baton Rouge, LA 70808, united States the center for nutritional epidemiology, university of north Carolina, Chapel Hill, NC , united states Received 12 August 2004; Accepted 12 Agust 2004.
57. Prewitt TE, Schmeisser D, Bowen PE, Aye P, Dolecek TA, Langenberg P, Cole T, Brace L. Changes in body weight, body composition, and energy intake in women fed high- and low-fat diets. Am J Clin Nutr. 1991;54(2):304-10.
58. Pritchard JE, Nowson CA, Wark JD. Bone loss accompanying diet-induced or exercise-induced weight loss: A randomised controlled study. Int J Obes Relat Metab Disord. 1996;20(6):513-20.
59. Bray GA, Popkin BM. Dietary fat intake dose affect obesity. Am Clin Nutr. 1998;68:1157-73.
60. Astrup A. The role of dietary fat in the prevention and treatment of obesity. Efficacy and safety of low-fat diets. Int J Obes Relat Metab Disord. 2001;25(Suppl 1):S46-50. Review.
61. Willet WC. Dietary fat plays a major role in obesity: no. Obese Rev. 2002;3:59.
62. Astrup A, Ryan L, Grunwald GK, Storgaard M, Saris W, Melanson E, Hill JO. The role of dietary fat in body fatness: evidence from a preliminary meta-analysis of ad libitum low-fat dietary intervention studies. Br J Nutr. 2000;83(Suppl 1):S25-32.
63. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO. The role of low-fat diets in body weight control: A meta-analysis of ad libitum dietary intervention studies. Int J Obes Relat Metab Disord. 2000;24(12):1545-52.
64. Rolls DJ, Rowe EA, Turner RC. Persistent obesity in rats following a period of consumption of a mixed, high energy diet. J Physiol (Lond). 1980;298:415-27.
65. Shah M, Govern P, French S, Baxter J. Comparison of a low fat, ad libitum complex-carbohydrate diet with a low energy diet in moderately obese women. Am J Clin Nutr. 1994;59:980-4.
66. Jeffery RW, Hellerstedt WL, French SA, Baxter JE . A randomized trial of counseling for fat restriction versus calorie restriction in the treatment of obesity. Int. J. 1995;19:132-7.