Obesity in the past was only seen in great numbers in Western countries but is now gaining prevalence in India with a growing Indian economy. Historically a favourable phenotype, obesity has been associated with multiple diseases affecting almost all organ systems. In addition, the cost of obesity to a health care system is enormous. Weight loss improves many of the outcomes and yet is a difficult process for most patients with even the medical community baffled about methods for weight loss. Our group and others have looked at weight loss in obese individuals using a low carbohydrate diet and have shown some success at six months and one year. Many studies have also shown a decrease in insulin resistance and improvement in lipid profiles when obese individuals are placed on a low carbohydrate diet. The reason for weight loss on this diet was initially thought to be diuresis from increased ketone production but it may be due to decreased insulin concentrations causing a decrease in appetite. In addition, lowering carbohydrates limits food choice and in the process decreases overall calorie consumption. In the United States, an increase in calories, predominantly from carbohydrates, appears to parallel the obesity epidemic. However, it is not known if guidelines or policies lowering carbohydrates in normal or slightly overweight individuals may help prevent obesity and its complications. Equally less known, is the applicability of a low carbohydrate diet to populations like India where many main dishes are carbohydrate-based.

**Key words** Diets - low carbohydrate - obesity - weight loss

Obesity is a growing problem in the Western world and is steadily growing in Asia as well\(^1\). Obesity burdens the health care system, costing an estimated 100 billion dollars a year in the United States alone\(^2\). Obesity is an independent risk factor for overall mortality\(^3\), as well as, for heart disease, sleep apnoea, diabetes mellitus, arthritis and cancer\(^2,4\). Obese patients typically find it very difficult to lose weight and low carbohydrate diets may be a method of weight loss for some. In this review, Western and Indian data on the epidemiology, history, and causes of obesity are
discussed. The main focus will be on newer data by our group and others regarding the use of low carbohydrate diets for weight loss. In addition, the mechanisms of action, potential effects, and future needed studies will also be examined.

Epidemiology of obesity and historical context

Obesity has been arbitrarily defined by body mass index (BMI) and waist circumference. Current classification of obesity by the World Health Organization (WHO) is shown in Table I. In addition to weight, increased risk of metabolic disorders is found in men with waist circumferences greater than or equal to 102 cm and in women with 88 cm. Although these are current guidelines of obesity, they give primarily a Western population perspective. Overweight in Asians has been suggested to start at 23 kg/m² and also proposed are lower waist circumference cut-offs of ≥90 cm in men and ≥80 cm in women.

The number of people in the United States who are categorized as overweight or morbidly obese (BMI ≥40 kg/m²) has increased. A retrospective study showed a 4 yr incidence of overweight of up to 16 per cent in women and 30 per cent in men. This study also showed that long-term risk of a normal weight individual developing overweight was 50 per cent and obesity 25 per cent. Obesity in India has been found to be as prevalent as 15 per cent of the population in urban areas.

Obesity is a disease of a prosperous and wealthy society and has been valued historically. Artwork depicting obese women such as the prehistoric idol Venus of Wollendorf and the Baroque paintings of Rubens illustrate this idea. Obesity as a disease entity has been described since the 1700s. One of the first written records of this was by Malcolm Flemyng who described in his 1757 Discourse on Corpulency the four main causes of obesity: (1) too much intake, (2) too easily distended cells, (3) too much ability of fat to be separated from blood, and (4) deficient evacuation of sweat from the fat. This early obesity statement shows the core deficit in obese individuals of a chronic imbalance between energy intake and expenditure. Low resting energy expenditure is still a questionable cause of obesity and much research has focused on single hormone deficits in appetite that would explain obesity. There are several recently discovered central and peripheral modulators of energy homeostasis such as ghrelin, polypeptide Y, and leptin. However, only a few monogenetic defects, such as those found in Prader Willi syndrome and melanocortin receptor mutations, explain obesity. Although 50 to 90 per cent of obesity is genetic, it is most likely a polygenic background of an individual that is influenced by the environment. Environment may take the form of concomitant illness such as hypothyroidism or medications that increase appetite such as corticosteroids, insulin, or psychiatric medications. However, there is a profound societal influence on obesity that was initially commented upon by William Wadd in 1816: “If the increase of wealth and the refinement of modern times, have tended to banish plague and pestilence from our cities, they have probably introduced the whole train of nervous disorders, and increased the frequency of corpulence.” Greater wealth and lower activity in the setting of “desk” jobs has created a population that has a net increase in available energy stored as fat. This is evidenced by the fact that in developing nations the ratio of obese individuals to underfed individuals is increasing. The transformation of India into an affluent nation has brought these changes in lifestyle and diet that have led to an increase in obesity. Not only do environmental changes in adults create obese adults.
but also environmental changes in children and adolescents eventually create obese adults\textsuperscript{14}. One study has found a prevalence of 11 per cent of Indian children with obesity\textsuperscript{22}. Again as in adults, childhood obesity has been linked to an environment of poor portion control, high intake of energy dense foods and lack of physical activity\textsuperscript{1}.

**Low carbohydrate diets**

People in the United States have increased calories as well as the percentage of calories derived from carbohydrates\textsuperscript{23}. However, even early man’s prehistoric diets may have been low carbohydrate diets\textsuperscript{24}. Low carbohydrate diets have primarily been used in the treatment of diabetes prior to the discovery of insulin\textsuperscript{25,26}. High fat, low carbohydrate diets have also been used in the early 1900s as a paediatric anti-epileptic therapy\textsuperscript{27}. In the United States, it has been popularized as a weight loss method by Robert C Atkins\textsuperscript{28}. Other popular diets that emphasize a lower carbohydrate diet include The South Beach Diet\textsuperscript{29} and The Zone Diet\textsuperscript{30}. Dr Atkins diet has been a model for many of the recent low carbohydrate diet studies and calls for a 30 g daily intake of carbohydrates initially followed by 60 g daily for maintenance with unlimited fat and protein intake\textsuperscript{28}. The induction phase is slightly more than the carbohydrates found in two 1-ounce slices of white bread\textsuperscript{31}. In the United States, the medical establishment had been opposed to this method of weight loss for many years\textsuperscript{32}. Much of the animosity towards the diet has been toward the “salesmanship” of the diet by Dr Atkins as well as the lack of good studies to back his claims\textsuperscript{32}. The reasons he initially stated in favour of using the diet surrounded data by a study from Kekwick and Pawan\textsuperscript{33}. They observed in three different trials a significant decrease in weight in obese subjects eating daily 1000 kcal 90 per cent fat diet for seven days\textsuperscript{33}. Similar data were obtained with five obese women taking a 1000 kcal, 84 per cent fat diet where an average of 6.6 kg was lost over a 10 day span\textsuperscript{34}. These studies used crude methods of calculating body fat mass and estimated that much of the weight lost was fat. Other studies of this era indicated that the weight loss seen in low carbohydrate diets was due to water loss\textsuperscript{35}, and others found that only variation in total calorie intake, not fat percentage led to weight loss\textsuperscript{36}. Criticism is still present and centers around a potential for eventual weight gain with long term low carbohydrate diets and the potential effect of these diets on coronary disease risk\textsuperscript{37}.

Large studies of low carbohydrate diets for weight loss in obesity had not taken place until nearly 20 yr after the critical review of Dr Atkins’ diet revolution appeared in 1973\textsuperscript{32}. Westman’s group studied 51 (total starting number) healthy overweight and obese individuals who were instructed to follow a 25 g/day low carbohydrate diet that was increased to 50 g/day after a 40 per cent target weight loss\textsuperscript{38}. The subjects were allowed an unlimited amount of fat and protein intake. Subjects had an initial BMI of approximately 31 kg/m\textsuperscript{2}, and after 6 months of dieting the subjects on an average significantly decreased BMI by 3 kg/m\textsuperscript{2} and also decreased fat mass as measured by skin fold thickness\textsuperscript{38}. They also found significant decreases in total cholesterol, low density lipoprotein (LDL), and triglycerides and increases in high density lipoprotein (HDL)\textsuperscript{38}. This was the first large study which used a low carbohydrate diet to treat overweight and obesity but did not address the dietary habits of subjects pre-study and also did not compare low carbohydrate dieters to traditional low fat dieters. A meta-analysis evaluating the weight change with low carbohydrate diets from 1966 to 2003 demonstrated that weight loss on a low carbohydrate diet was due to a decrease in dietary caloric intake\textsuperscript{39}. They could not conclude that the diet was a favourable approach to weight loss. Following this, Brehm and colleagues looked at cardiovascular risk factors in obese women who were given either a low fat or a low carbohydrate diet for 6 months\textsuperscript{40}. The low carbohydrate dieters were told to eat ad libitum fat and protein but limiting themselves to...
20 g carbohydrate for two weeks\textsuperscript{34}. The low fat dieters were told to restrict calories predominantly from fat in a proportion of 55 per cent carbohydrate, 15 per cent protein and 30 per cent fat\textsuperscript{40}. At baseline both diet groups had similar intakes of macronutrients and calories. Over six months time, the low carbohydrate dieters and low fat dieters decreased their intake by approximately 450 kcal with the low carbohydrate dieters taking in 46 per cent fat versus 29 per cent fat in low fat dieters. However, low carbohydrate dieters lost a significant amount of body fat compared to their low fat counterparts\textsuperscript{40}, though there was no significant difference in changes in lipids, insulin, or glucose. This was the first comparative study to show that the low carbohydrate diet approach may work to achieve weight loss without much anticipated deleterious lipid effects. However, no other advantage was found with this dietary method besides weight loss.

Studies by our group\textsuperscript{41} and others\textsuperscript{42} looked at the weight loss effects of a low carbohydrate diet versus a conventional low fat diet in larger populations (Table II). In our study of 20 per cent females, low carbohydrate dieters were taught a 30 g carbohydrate, \textit{ad libitum} fat and protein intake\textsuperscript{41}, while in other study most female subjects were taught a 20 g carbohydrate diet with \textit{ad libitum} protein and fat intake\textsuperscript{42}. In both studies conventional dieters were to decrease their caloric intake by 500 kcal with between a 25 and 30 per cent intake of fat. Both the groups found a significantly decreased weight and triglycerides in low carbohydrate dieters versus conventional low fat dieters at the 6 month interval. Foster’s group also found significant increases in the low carbohydrate group of HDL cholesterol versus conventional low calorie diets at the six month and one year intervals\textsuperscript{42}. Neither diet showed deleterious changes in LDL cholesterol. Our one-year follow up analysis\textsuperscript{13} and Foster’s study\textsuperscript{42} showed that weight was not significantly different in low carbohydrate dieters versus low fat dieters at one year. These studies demonstrated a possible benefit of triglyceride lowering of low carbohydrate

<table>
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<th>Group</th>
<th>Subjects</th>
<th>Months</th>
<th>Comparison</th>
<th>Weight loss (kg)</th>
<th>Lipid findings</th>
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<tbody>
<tr>
<td>Westman et al</td>
<td>41 healthy overweight and</td>
<td>6</td>
<td>25-50 g carb/day for 6 months</td>
<td>-9.0 ± 5.3</td>
<td>Sig ♦ Tchol, ♦ LDL, ♦ TG, ♦ HDL</td>
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<td>Brehm et al</td>
<td>53 healthy obese women</td>
<td>6</td>
<td>20 g carb/day vs. 30 % fat dec.</td>
<td>- 8.5 ± 1.0</td>
<td>No sig. change</td>
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<td>Foster et al</td>
<td>37 healthy obese men and</td>
<td>12</td>
<td>30 g carb/day vs. dec. 500 kcal w/</td>
<td>-4.4 ± 6.7%</td>
<td>♦ TG, ♦ HDL</td>
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<td>and women</td>
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<td>25 % fat</td>
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<td>Samaha et al</td>
<td>79 unhealthy obese men and</td>
<td>6</td>
<td>30 g carb/day vs dec. 500 kcal w/</td>
<td>-5.8 ± 8.6</td>
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<td>and women</td>
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Columns focus on type of subjects who completed the study, length of study, study design, weight lost in the low carbohydrate arm, and lipid changes associated with the low carbohydrate arm. Foster’s analysis stated weight change in terms of per cent from baseline. All weight loss and lipid changes noted reached a significance of $P<0.05$ from baseline values. Dec.=decreased from baseline diet, Tchol= Total cholesterol, LDL= low density lipoprotein, HDL= high-density lipoprotein, TG=Triglycerides.
diet that was apart from weight loss. The major difference between our study population and others was a high BMI of 40 kg/m² and large numbers of subjects with multiple sequelae of obesity such as sleep apnoea (29%), diabetes (37%) and metabolic syndrome (72%)\(^4\). Our sub-population of diabetic subjects experienced a significant decrease in starting hemoglobin A₁c (HbA₁c) on the low carbohydrate diet but this change was not significant compared to low fat dieters\(^4\). These studies indicate that weight loss in the obese in the short-term may be possible with low carbohydrate dieting and in some instances may lower triglycerides, blood sugar, and insulin resistance (Fig.).

The long-term atherogenic consequences of eating a low carbohydrate diet are still unknown. One hypothesis is that such diets may actually be cardioprotective because pre-historic low carbohydrate hunter-gatherer diets were cardioprotective. Cordain et al suggested that these pre-historic diets were high in dietary protein and fat (up to 58%) and despite this, these hunter-gatherer societies were relatively free of cardiovascular disease\(^2\). The authors also specified that their fat intake was predominantly monounsaturated fats and combined with pre-historic man’s exercise throughout the day were key contributors to the lower incidence in cardiovascular disease. Although there are as of yet no long-term studies performed in patients eating a low carbohydrate diet, many studies have been performed to look specifically at lipid concentrations when individuals eat a low carbohydrate diet\(^4\). In general, they showed lowering of pre- and

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**Fig.** Proposed mechanism of action of low carbohydrate diets. A low carbohydrate diet decreases food choices and lowers insulin concentrations that combine to decrease caloric intake and results in weight loss. Lower insulin concentrations and decreased caloric intake also help improve lipid profiles.
post-prandial triglycerides and increase (or less of decrease compared with low fat diets) in high-density lipoprotein (HDL) without changes in total low-density lipoprotein (LDL) concentration. There is essentially no difference in changes in lipid subfractions and oxidized LDL between low carbohydrate dieters and low fat overweight and obese dieters. However, decreases in more atherogenic large VLDL and post-prandial triglycerides have been seen in low carbohydrate dieters than in low fat dieters. In addition to lipids, inflammatory markers such as C-reactive protein (CRP), tumour necrosis factor alpha (TNF-\(\alpha\)), adiponectin, and leptin associated with cardiac risk have also been studied. Many of these markers do not change when comparisons are made between low fat and low carbohydrate diets. However, our group found that low carbohydrate dieters with the highest levels of CRP decreased their levels more than low fat dieters. These studies demonstrated either no change or improvement in lipid and inflammatory marker profiles, and at least in terms of lipid effects should not be discouraged as a method of weight loss.

**Mechanism of action**

Our group and others have suggested that low carbohydrate diets work primarily by decreasing food choice in diets where fat and carbohydrate are so tightly associated. Also there is a suggestion that there is increased satiety with low carbohydrate diets. Others have felt the weight lost is primarily total body water due to an increased production of diuretic ketone bodies.

The best study to date looking at mechanism of weight loss in low carbohydrate diets was by Boden et al. This study looked at 10 obese type 2 diabetic individuals who were taking their usual diet for seven days and then took a low carbohydrate diet for 14 days. The major difference in this study versus previous analysis was that although subjects were allowed an *ad libitum* low carbohydrate diet, all subjects were given modified hospital diets and were confined to the hospital. This allowed the group to know exactly what foods were ingested. They found that body fat was lost primarily because of a decrease in food intake. Satisfaction surveys did not show significant changes in hunger, queasiness, or energy. There were significant changes in fasting insulin glucose and HbA1c. Insulin clamp studies on these subjects also showed decreased insulin resistance. It was concluded that the weight loss experienced in these dieters was caused by caloric restriction and that decreased insulin concentrations contributed to a potential decrease in appetite. The analysis suggests that many of the *ad libitum* studies, using caloric diaries and 24 h dietary recall methods, miss significant decreases in caloric intake.

Lastly, the lipid improvements have been proposed to be due to an upregulation of lipoprotein lipase in muscle and downregulation of hepatic lipase and adipocyte lipoprotein lipase. These changes may be due to an increase in post-prandial chylomichrons from an increased fat intake and a decrease in insulin secretion from a decreased carbohydrate intake. This mechanism has not been proven experimentally.

**Recommendations for usage**

In the United States, the medical community has not endorsed the low carbohydrate diet for weight loss. However, some previous critics of the diet have suggested using it as a tool for weight loss. The long-term consequences of these diets are still unknown. What we can determine is that the diets appear to be relatively safe at six months and one year and may help in lowering of triglycerides and blood sugar. Although there is a potential for weight regain on this diet, it may be a diet to consider in those obese hyperlipidaemic individuals who would like a “jump start” into lifestyle modification. Even an attempt at weight loss may be helpful as there are data to suggest that there is a decreased mortality in overweight
adults who have tried to lose weight versus those who have not tried at all\textsuperscript{55}.

With regard to prevention of obesity, low carbohydrate diets have not been studied. There is a suggestion by the lay press that besides the usual culprits of high-fat foods found in fast food restaurants the subsidy of the corn industry in America has promoted obesity\textsuperscript{56,57}. Policies instituted since the 1970s may have promoted the use of corn as an essential ingredient of high fructose corn syrup found in soft drinks\textsuperscript{58}. Although these associations are speculative, these facts should be taken in the context of an increase in carbohydrate consumption noted by the Centers for Disease Control\textsuperscript{23} and the subsequent increase in obesity described by the Nutrition Health and Examination Study\textsuperscript{7}.

The ability for Indians to lower carbohydrate intake will be limited due to the high amount of carbohydrates in traditional foods. Unlike in North America, where carbohydrates are typically a side dish, Indians, especially vegetarians, have carbohydrates as the main dish\textsuperscript{59}. In India, greater wealth and subsequently greater amount of carbohydrate intake may generate an enormous obesity and diabetes epidemic in the future.

Future studies

Many of the future studies should focus on the potential consequences of obesity and a low carbohydrate diet over long periods of time. Follow up is necessary for people who have already taken part in the low carbohydrate interventions to see if the weight and diet are maintained. Also, a specific analysis on subjects with disorders related to obesity such as sleep apnoea and type 2 diabetes mellitus will need to be conducted. Lastly, studies are needed that look at overweight individuals instructed on a low carbohydrate diet over a five to ten year stretch of time to determine if obesity may be prevented.

Reference


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