

Chapter 1

OVERVIEW OF LOW CARBOHYDRATE AND KETOGENIC DIETS

Historical Perspective

Who invented the low carbohydrate diet? Was it Dr. Robert Atkins' weight loss revolution in 1972? Or Wilder and Peterman's anti-seizure diet at the Mayo Clinic in the 1920's? Or perhaps Banting's pamphlet in Britain in 1863?

The answer: none of the above. But for sure, it was long, long before these recorded efforts to codify and monetize carbohydrate restriction. This does not in any way discount the contributions of these contrarian pioneers who attempted to steer us away from our sometimes fatal romance with agricultural carbohydrates. But to understand the origins of low carbohydrate metabolism and to appreciate how deeply it is rooted in our basic human physiology, we need to go back hundreds of thousands of years, if not a million or two.

Current evidence suggests that our human ancestors evolved in Africa and then spread across the globe in successive waves of migration. And while that original African ancestral group may have developed in a tropical environment where fruit and tubers could be foraged year-round, our ability as humans to migrate into barren or temperate regions depended

upon our ability to survive prolonged periods of fasting, and to adapt to hunting and gathering of less carbohydrate-rich fare. And eventually, this evoked tolerance of a low carbohydrate diet allowed some humans to become highly specialized hunters and herders, living as mobile cultures in rhythm with the animals that fed them. Recent examples of these low carbohydrate nomadic cultures were the Masai herdsmen in Central Africa[6], the Bison People of the North American Great Plains[7], and the Inuit in the Arctic[8].

But long before these last low carbohydrate cultures were finally suppressed by the agricultural imperative, much of the world's populace subsisted (if not thrived) on continuous or intermittent carbohydrate restriction. For example, agricultural carbohydrates such as wheat and rye did not come north of the Alps until brought by the Romans after the time of Christ. The Irish, Scandinavians, and Russians had no agricultural carbohydrates suitable to their climate until the potato emigrated to Europe from the Andes in the 16th century AD. What this means is that many of our ancestors had little exposure to high proportions of dietary carbohydrate until 1-2 thousand years ago; and for many aboriginal cultures, their choice of a low carbohydrate lifestyle persisted to within the last few hundred years.

Now fast forward to the present. The United States is currently re-assessing a 3-decade, uncontrolled experiment in which carbohydrates were lauded and fats demonized. Concurrently we have become one of the most obese countries in the world. And across the globe, tragically, indigenous peoples with historically low carbohydrate intakes now have extremely high prevalence rates of obesity and type-2 diabetes (e.g., the Gulf States in the Middle East, Pacific Islanders, First Nations in Canada, and Australian Aborigines).

What these observations suggest is that for many humans, from an evolutionary perspective, a high carbohydrate diet is a metabolic challenge that some find difficult as early as adolescence and many fail to meet in the middle years of life. Equally apparent is that these negative effects of a high carbohydrate intake can be forestalled or reduced by vigorous exercise, high intakes of micronutrients and/or fiber from vegetables and fruit, avoidance of simple sugars, and constant energy restriction. For many

of us with severe obesity, metabolic syndrome, or overt type-2 diabetes, however, these ‘healthy lifestyle’ choices are not enough to fully counteract the negative effects of a substantial contribution of carbohydrate to our daily energy intake.

This condition, in which a collection of diseases characterized by insulin resistance are driven by consumption of a single nutrient class, deserves to be identified as “carbohydrate intolerance”. And as with other single nutrient intolerances (e.g., lactose, gluten, fructose), the preferred intervention is to reduce one’s dietary intake below the threshold level that produces symptoms.

What Does “Low Carbohydrate” Mean?

There are two ways to define the threshold below which you are eating a “low carbohydrate” diet. The first is defined by what you as an individual perceive – it is that level of carbohydrate intake (be it 25 grams per day or 125 grams per day) below which your signs and symptoms of carbohydrate intolerance resolve. At one end of this experiential range, someone with early signs of metabolic syndrome (e.g., high serum triglycerides and 10 extra pounds around the middle) might permanently banish these harbingers of ill-health by holding total dietary carbohydrate intake in the range of 100-125 grams per day.

At the other end of this spectrum might be a type-2 diabetic who, on a “balanced diet” providing 300 grams per day of carbohydrate, requires 2 shots of insulin plus two other oral drugs to keep fasting glucose values even marginally controlled under 150 mg/dl. For this person to achieve an optimum initial response that allows reduction (and hopefully withdrawal) of diabetic medications, clinical experience has shown that holding dietary carbohydrate at 20-to-25 grams per day is often necessary. For many type-2 diabetics, a few weeks at this level allows them to reduce or stop both insulin and oral medication while at the same time achieving better overall glucose control. A few months later, following substantial weight loss, some individuals might be able to increase daily carbohydrate

intake above 50 grams per day and still maintain excellent glucose control, whereas others might need to remain below the 50 gram level to keep their type-2 diabetes in complete remission.

In either case, whether it is being able to lose weight and keep it off, or putting a frank case of type-2 diabetes into remission, how much you choose to limit your dietary carbohydrate intake should be driven by your personal experience. As a result, the amount of carbohydrate that you decide to eat might vary considerably depending on your individual metabolic condition and the level of benefit you wish to derive.

Defining ‘Nutritional Ketosis’

The second way to define ‘low carbohydrate’ is physiologic – specifically that level below which there is a fundamental shift in your body’s fuel homeostasis (i.e., energy regulation) away from glucose as a primary fuel. This shift is the adaptation of the body’s hormonal set and inter-organ fuel exchange to allow most of your daily energy needs to be met by fat, either directly as fatty acids or indirectly by ketone bodies made from fat. This process, which is discussed more fully in Chapter 7, begins for most adults when total carbohydrate is restricted to less than 60 grams per day along with a moderate intake of protein. After a few weeks at this level, the primary serum ‘ketone’ (beta-hydroxybutyrate, or B-OHB), rises above 0.5 millimolar (mM). At this ketone level, which is 10-fold higher than that in someone with a daily intake of 300 grams of carbohydrate, the brain begins to derive a substantial portion of its energy needs from B-OHB, resulting in a commensurate reduced need for glucose.

With further restriction of carbohydrate below 50 grams per day, the serum B-OHB rises in response to reduced insulin secretion. However, because dietary protein prompts some insulin release, and serum B-OHB itself stimulates insulin release by the pancreas (albeit subtly), adults eating 20 grams of carbohydrate and 75-150 grams per day of protein rarely run serum B-OHB levels above 3 mM. This is in contrast to the response

to total starvation (i.e., no dietary carbs or protein) where the serum B-OHB levels run as high as 5 mM.

This 10-fold range of serum ketones, from 0.5 to 5 mM, is your body's normal physiological response to varying degrees of dietary carbohydrate and protein restriction. This response range is called 'nutritional ketosis', and is associated with metabolic adaptations allowing your body to maintain a stable state of inter-organ fuel homeostasis. This process is dependent on an adequate, albeit minimal, ability of the pancreas to produce insulin in response to dietary protein and serum ketones, thus maintaining serum B-OHB in the range where it replaces much of your body's (and your brain's) need for glucose without distorting whole-body acid-base balance.

Nutritional ketosis is by definition a benign metabolic state that gives human metabolism the flexibility to deal with famine or major shifts in available dietary fuels. By contrast, 'diabetic ketoacidosis' is an unstable and dangerous condition that occurs when there is inadequate pancreatic insulin response to regulate serum B-OHB. This occurs only in type-1 diabetics or in late stage type-2 diabetes with advanced pancreatic burnout. In this setting of deficient insulin, when exogenous insulin is withheld, serum B-OHB levels reach the 15-25 mM range – 5-to-10-fold higher than the levels characteristic of nutritional ketosis.

Unfortunately, among the general public and even many health care professionals as well, these two distinct metabolic states tend to be confused one for another. Understanding how different they are is key to being able to capture the many benefits of nutritional ketosis while avoiding the risks in that very small minority of the population subject to developing diabetic ketoacidosis. To this end, a full chapter later in this book is devoted to the clinical use of carbohydrate restriction in diabetes.

Utility and Sustainability of Carbohydrate Restriction

Up until 150 years ago, the apparent motivation for humans to eat a low carbohydrate diet was because that was what their regional environment provided. For example, absent wild orchards and fields of waving grain, the Inuit had little choice other than meat and fat from the arctic tundra and the sea. However, some cultures with long experience and apparent choice attempted to actively defend their low carbohydrate lifestyle. Examples of this included the Bison People of the North American Great Plains, who maintained their nomadic existence until the bison were virtually exterminated, and the Masai of East Africa who still avoided vegetable foods (against the vigorous advice of the British) into the 1930s. Manifestly, for these cultures, not only were their low carbohydrate dietary practices sustainable – allowing them to survive and reproduce for hundreds of generations under difficult environmental conditions – they regarded their diet of animal products as preferable to an agricultural lifestyle, despite the latter having been available to them.

In the 1920s, carbohydrate restriction was employed in mainstream medical practice in the management of diabetes and in the treatment of seizures. In both of these clinical situations, as there was no other effective treatment, these dietary interventions were sustained by individual patients for years. With the advent of insulin for diabetes and anti-seizure drugs like diphenyl-hydantoin (Dilantin), these dietary interventions began to fall out of favor. However now that the practical limitations and side effects of modern pharmaceutical therapy are becoming recognized, the wheel may be again turning.

One of the perceived limitations of modern low carbohydrate diets is that they have become stigmatized as extreme and thus necessarily limited to temporary use. Adding to this sense of transience, most popular diet books promoting carbohydrate restriction have effectively only described an initial energy restricted phase to promote weight loss. So what does the reader do after 3 months eating 1400 kcal/day with a 30 lb weight loss? No healthy adult over 5 feet tall achieves energy balance on 1400 kcal per day, so the transition from weight loss to long-term weight main-

tenance necessarily means adding back food. But how much, and from what foods? Carbohydrates? Protein? Fat?

It is a primary hypothesis (if not principle) of this book that a low carbohydrate diet that is sustainable in the long term (e.g., for the management of type-2 diabetes, seizures, or severe obesity) necessarily contains an appropriate fat content in its weight maintenance phase. Thus, if a book promoting a low carb diet does not contain practical instruction and recipes promoting the inclusion of fat in its maintenance diet, it is not likely to result in much long-term success among its readers. To this end, later chapters in this book will describe the physiology of fuel partitioning, clinical use of low carbohydrate and ketogenic diets, plus two full chapters and a week's worth of menus addressing the practicalities of preparing and consuming a maintenance diet that is rich in fat.

Recent and Future Research

The last decade has seen a dramatic increase in the volume of research publication on the topic of carbohydrate restriction. Multiple randomized, controlled trials (RCTs) have been performed comparing a variety of other diets to carbohydrate restriction. Many of these have demonstrated clear advantages in favor of low carbohydrate and ketogenic diets. However, the interpretation of these studies is sometimes clouded by reticence of authors or editors to give them due credit or by pitfalls in research methodology. The pros and cons of this recent body of research are addressed throughout this book. In addition, multiple contributing authors provide their insights on topics pertinent to the range of effects and benefits of carbohydrate restriction.