# Carbohydrate Intolerance

# Its Implications in Health and Fitness

Perhaps the most common cause of low quality of life, accelerated aging and chronic disease is the trio of increased body fat, chronic inflammation and insulin resistance. This white paper discusses the practical rationale for referring to the related dysfunctions associated with poor carbohydrate metabolism under the umbrella term Carbohydrate Intolerance.

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#### Introduction

Many diseases begin to develop early in life. This happens long before most people, including healthcare professionals, would even consider the progressive nature of poor health in apparently young and healthy bodies.

In the 1920s, endocrinologist Hans Selye (who coined the term "stress") observed that the body has a progressive response to physical, biochemical and mental-emotional stress. He called this the general adaptation syndrome, which from today's clinical perspective can be presented in three stages:

- Functional (relatively minor but important) signs and symptoms.
- Increasingly measurable dysfunction with elevated disease risk factors.
- Chronic diseases.

Also during the early 1920s, it was discovered that a series of ailments and risk factors clustered together: hypertension, Type 2 diabetes and cardiovascular disease. By the 1980s, other related conditions, including increased abdominal fat, obesity and high blood triglycerides, had been tied together in the same common thread by a condition called *insulin resistance*.<sup>2</sup> Recognition that insulin resistance is a spectrum disorder was first proposed in 1979.3 This is a serious problem seen as the earliest manifestation of what became known in the 1990s as metabolic syndrome,4 and which today incorporates all the above conditions.

These conditions, along with numerous others described here, follow a progression very similar to Selve's model of stress. The beginning of the process appears to be some combination of the trio of insulin resistance, chronic inflammation and increased body fat. In fact, stress (in all its physical, biochemical and mental forms), can further worsen, and even trigger, this trio.

Since around 1980, the author has referred to this complex array of dysfunctions and illnesses — the full spectrum of associated conditions — under the umbrella term Carbohydrate Intolerance (CI). This term emerged from the acknowledgment that the most common ailment shared by people suffering from these conditions is an inability to properly metabolize carbohydrates.5

While these problems tend to occur in people who are overweight or obese (referred to as overfat), up to 40 percent of normal-weight people have metabolic dysfunction like that of an overfat person.<sup>6</sup> Most of these individuals are also considered to be carbohydrate-intolerant.

As a chronic condition, CI continues to worsen worldwide: of all age groups, obesity is growing most rapidly in children. They are also developing conditions that at one time were only seen in adults: hypertension, high blood fats and other components of the metabolic syndrome.4

Unfortunately, the early signs and symptoms of CI, while well-recognized by some researchers and clinicians, are ignored by too many health practitioners, patients and other individuals. Traditional medicine considers these as separate, isolated conditions, rather than part of a spectrum of metabolic impairment. It is therefore typical to find that CI is not treated until it has progressed to later stages, when risk factors such as hypertension or disease states like Type 2 diabetes are diagnosed (and even then is treated symptomatically rather than by addressing the cause).

#### **Impaired Physiology**

While the medical mainstream often highlights signs and symptoms (fatigue, pain, memory loss, etc.) in people with CI, the cause of these common complaints reflects underlying physiological dysfunction. As shown by the following examples, the development of CI can be quite extensive and complex:

- Mitochondrial dysfunction:9 Energy production (fatigue) and muscle function (physical injuries and pain) is impaired.
- Accelerated aging:10 Becoming physiologically older than one's chronological age due to oxidative and immune stress (increasing disease risk).
- Neurodegeneration:11 Brain injury (including memory loss, depression and other mental-emotional impairment).

A central goal of this paper is to argue the benefits of using the term carbohydrate intolerance to refer to the full spectrum of this common metabolic problem, from the earliest subtle impairments to end-stage diseases. Our secondary goal is to discuss the etiological and therapeutic role of regulating dietary carbohydrates, as many conditions are reversible through simple adjustments in food choices. (Fewer than 5 percent of our modern diseases can primarily be ascribed to genetic factors.)7

By referring to this condition as CI, an opportunity emerges: Key underlying problems (insulin resistance, chronic inflammation, increased body fat) and the full spectrum of their associated conditions, and signs and symptoms, can be presented as part of one integrated model. The benefits of this terminology include:

- · Simplifying the full spectrum of CI, in all its complexities.
- Aiding in the process of assessment and treatment.
- · Preventing lower quality of life.
- Avoiding chronic end-stage diseases.
- Defining CI's impact on exercise and the overtraining syndrome.
- Patient education.
- · Self-management of health.

## Carbohydrate Intolerance: Its Spectrum and Stages

The progression of CI closely follows Selye's three-stage model of the general adaptation syndrome. Individuals with CI can manifest different signs and symptoms at various points throughout their lives. While one person may progress through all three stages quite quickly, and develop a serious condition in childhood, another may go through the first two stages more slowly, and be diagnosed with a disease later in life.

Most importantly, the earliest indications of CI can begin early in life or even before birth. For example, certain factors present at birth or in childhood are predictive of Type 2 diabetes, including higher or lower birth weight, and children who are taller than average for their age.8



#### Stages of CI

In Stage 1 of CI, the earliest signs and symptoms of dysfunction may begin to appear. These may be very subtle, but can be uncovered through a good history-taking process. By mainstream medical standards, however, most people in this stage are viewed as "otherwise normal." Due to this, early recognition of CI is often overlooked by health practitioners despite patient complaints. The need for preventive care is overlooked, and the opportunity to tackle the problem at its roots is missed. Individuals with these problems often begin self-medicating to control symptoms.

In Stage 2 of CI there are often more numerous and well-defined signs and symptoms.<sup>13, 14, 15, 16</sup> This allows the condition to be more easily assessed from history, physical evaluations and laboratory tests. Dietary and other lifestyle interventions at this stage can play a key role in reversing CI, but are also often overlooked. (A commonly ineffective strategy is the application of low-fat, high-carbohydrate diets, ostensibly to promote weight loss.)

In Stage 3 of CI, clear disease conditions can develop. These include heart disease, cancer, stroke, Alzheimer's and Type 2 diabetes. According to the Centers for Disease Control and Prevention, these are five of the most common causes of death in the U.S., while at the same time are considered preventable.

The main feature of CI is the trio of insulin resistance, chronic inflammation and increased body fat. At various points along the spectrum different patterns associated with this trio can exist.

#### For example:

- In the early stages, insulin resistance may be associated with the overproduction of insulin (hyperinsulinemia). Later in life, insulin levels may fall significantly, leading to a diagnosis of Type 2 diabetes.
- Chronic inflammation is difficult to recognize early in life (and often referred to as "lowgrade"). It becomes more obvious later, especially when inflammatory-related conditions appear. These include pain, arthritis, and even cardiovascular disease and cancer.
- Body fat stores may begin increasing at various points in life. While this typically manifests as an increase in waist size, many "normal-weight" people can have increased body fat and CI.

#### **Insulin Action**

In a healthy body, the hormone insulin is responsible for enabling glucose (the simplest form of carbohydrates) to be carried across the cell's insulin-dependent membrane into muscle fibers and other cells.

If the action of insulin is hindered (sometimes due to a fault in the cell's insulin receptors), the body becomes incapable of properly metabolizing carbohydrates, often leading to increased insulin production (hyperinsulinemia) and eventually to diminished insulin sensitivity (insulin resistance). This can lead to more carbohydrate foods being converted to stored fat. (It should be noted that insulinindependent glucose receptors also exist in human muscle.<sup>17</sup>)

#### Table 1.

Depression

#### The Full Spectrum of CI

Some signs and symptoms are associated with the three stages of CI. Many can continue or worsen beyond the stage in which they initially appeared. For example, Stage 1 symptoms such as fatigue, hormone imbalance, and sleep problems continue to be common complaints in all Stage 3 diseases. Accelerating aging may be common to all stages.

Stage 1	Stage 2	Stage 3
Birth weight $< 5^{1}/_{2}$ lbs or $> 9$ lbs	Chronic pain	Type 2 diabetes
Children taller than average for	High LDL cholesterol	Heart disease
their age	Low HDL cholesterol	Stroke
Sleepiness after meals	High triglycerides	Alzheimer's/dementia
Loss of focus after meals	Liver dysfunction	Cancer
Intestinal bloating after meals	Arthritis	
Increased abdominal fat/waist size	Pre-hypertension/hypertension Gout	
Overfat		
Fatigue	Sleep apnea/disorders	
Hormone imbalance	Kidney stones  Gall stones	
Family history of CI conditions	Obesity	
Excess hunger	Pre-diabetes	
Increased craving for sweets		
Oxidative stress/poor immunity	Aerobic deficiency Osteoporosis	
(associated with aging)	Hormone imbalance	
Hormone imbalance	Menstrual irregularities	
Impaired glucose regulation		
Sleep problems	<ul><li>Polycystic ovaries</li><li>Infertility</li></ul>	
Brain dysfunction	Neurodegeneration	
<ul><li>Poor learning</li><li>Attention disorders</li><li>Hyperactivity</li><li>Aggression, anger</li></ul>	<ul><li>Poor memory, cognition</li><li>Depression</li><li>Anxiety</li><li>Poor learning</li></ul>	
Aggression, anger	1 oor learning	



#### **The Stress Connection**

The brain and body respond to physical, biochemical and mental-emotional stressors through a mechanism called the Hypothalamic-Pituitary-Adrenal (HPA) axis. Examples of stress include over-exercise, a drop in blood sugar, or anxiety. The adrenal glands, which help mediate the stress response through the production of the hormone cortisol, can become impaired with too much stress. This is known as adrenal dysfunction.<sup>18, 19</sup> The result can be a wide range of other adrenal hormone imbalances that affect muscle repair, exercise recovery, water regulation, sodium and other electrolyte balance, control of inflammation, energy needs, sexual activity and many others. The accumulation of stress can trigger or worsen Cl.

Dysregulation of the HPA axis can also disturb one's mental-emotional state. Clow et al. write that this "regulation of physiological function across the day (e.g., the immune system) and its sensitivity to psychosocial variables make it a prime candidate as an intermediary linking mind to health."20

A common and perhaps primary trigger of CI is an excess consumption of moderate- and highglycemic, processed and refined carbohydrates (junk food). See Figure 1.

Figure 1

# **MODEL OF CARBOHYDRATE INTOLERANCE**

High refined-carbohydrate diet



Insulin resistance, chronic inflammation, increased body fat





More insulin resistance, chronic inflammation and increased body fat



Accelerated aging & bodywide dysfunction



Increased disease risk/ subclinical illness



Chronic disease

#### **Mental-Emotional Symptoms and CI**

Various aspects of CI have been shown to have significant adverse impacts on brain function.<sup>21</sup> For example, it has been demonstrated that in neurologically intact people with metabolic syndrome, the brain's response to a cognitive challenge is blunted. This suggests that metabolic disturbances are associated with early brain impairment.<sup>22</sup>

Because the brain relies on stable blood sugar for proper function, CI can significantly impact a person's mental and emotional state. An early symptom of CI can include depression.<sup>23</sup> Holden writes, "The fundamental problem does not lie with the neurotransmitter per se, but rather with uncontrolled fluctuations of brain glycaemic levels acting in conjunction with insulin resistance."23

Hyperactivity, aggression and anger in children can also be associated with CI.<sup>24</sup> High insulin levels have particularly been correlated with attention deficit disorder (ADD), violence and early alcohol abuse.<sup>24</sup> Holden's contention is that "mental illness, in its many guises, is a general manifestation of a diabetic brain state which has been termed 'cerebral diabetes.''23

- In tests, children and adolescents with CI scored significantly worse on attention/ concentration, visual-spatial tasks, mathematics, executive function, memory and attention.<sup>26, 27, 28</sup>
- In adults, CI-related conditions have been linked to deficits in memory, visuospatial abilities, executive function, processing speed and overall cognitive function.<sup>29, 30, 31, 32, 33</sup>

#### CI and Exercise

An understanding of CI is of paramount importance to all those who exercise, from those who work out to lose weight to competitive athletes. A critical component of the body — the *aerobic system* — constitutes the key connection between CI and its effects on athletic performance. The function of this system is to provide a continuous and reliable supply of energy to the body by oxidizing (burning) fats. A well-functioning aerobic system improves circulation, helps the immune system, supports anaerobic muscles, protects joints, ligaments, tendons and bones, and aids other functions.34

Carbohydrate intolerance impairs metabolism, reducing fat-burning. Insulin inhibits the action of leptin, the hormone that enables the body to break down fat and use it for fuel,<sup>35</sup> and also helps convert larger amounts of dietary carbohydrate to fat for storage.<sup>36</sup>

Impaired fat-burning and increased fat storage prevents the aerobic system from providing the body with reliable and nearly unlimited energy over the long-term. Increased body fat often means increased weight, which can reduce running economy, along with endurance and overall performance. This can also raise exercise heart rate at the same power output, increasing the risk for overtraining.

#### CI and the Overtraining Syndrome

The overtraining syndrome (OTS) in athletes can be tightly related to CI and aerobic dysfunction. In fact, many of the signs and symptoms of CI are shared in the OTS.37 In particular, pain and inflammation are frequent conditions associated with CI, with concomitant high injury rates in those who exercise.



High-intensity training — a hallmark of OTS - can trigger a high production of growth hormone.<sup>38</sup> Since increases in growth hormone are accompanied by insulin resistance,39 highintensity workouts can worsen both CI and OTS.

### Addressing the **Full Spectrum of CI**

Addressing CI at any point along the spectrum may best be accomplished with primary dietary intervention. Reducing physical, biochemical and mental-emotional stressors is also important. Even young, healthy adults can overproduce insulin when consuming highly refined carbohydrate meals.<sup>40</sup> Lowering dietary carbohydrate intake while raising healthy fats is a successful therapy for various components of CI, even for serious, chronic conditions such as:

- Reversal of metabolic syndrome in children (hypertension, high triglycerides and LDL-cholesterol, hyperinsulinemia) and improvements in glucose tolerance in just 10 days.41
- Improvements in fertility and reproductive function by reducing insulin resistance,42 and carbohydrate intake.43
- Reduction and elimination of exogenous insulin requirements in Type 2 diabetes.<sup>44</sup>
- Successful management of Type 1 diabetes, including the reduction and sometimes elimination of exogenous insulin.45
- Among the most promising cancer therapies are diets extremely low in carbohydrate and high in healthy fats.<sup>46, 47</sup>

No dietary intervention is better than carbohydrate restriction for weight loss, consistently outperforming low-fat diets in studies for whatever time period they are compared, and frequently show dramatically better outcomes.44 Low-carbohydrate, highfat eating plans can also successfully be implemented in endurance athletes to improve performance.48,49,50

While low-fat diets still remain popular, this approach has been a failure.<sup>51, 52</sup>

The ideal percentage of dietary carbohydrates consumed is based strictly on an individual's need, which may change throughout life. Generally, as one moves further along the CI spectrum, becoming more insulin resistant, lower levels of carbohydrate foods are best tolerated. One important recommendation for everyone with CI is the elimination of all refined carbohydrates including processed flour and sugar. As these foods are reduced, the intake of healthy dietary fats must be increased to maintain caloric balance.

The Two-Week Test developed by the author in the early 1980s (and described elsewhere) is a useful tool to help people find dietary balance. This test is a dietary challenge that can help individuals determine their optimal carbohydrate (hence, fat and protein) intake based on specific signs and symptoms.

#### Conclusion

The term "carbohydrate intolerance" is exceedingly useful for bringing into focus an array of interrelated functional problems and their associated end-stage diseases. Most are induced or influenced by diet, and respond well to some degree of dietary carbohydrate restriction. The notion of CI also helps individuals in all walks of life better understand how food immediately impacts their physical, biochemical and mentalemotional health, and to become more proactive in their own program of preventive care.

#### References

- Szabo S, Tache Y, Somogyi A. The legacy of Hans Selye and the origins of stress research: a retrospective 75 years after his landmark brief "letter" to the editor of the journal Nature. Stress. 2012; 15(5):472-8.
- Samaras K, McElduff A, Twigg SM et al. Insulin levels in insulin resistance: phantom of the metabolic opera? Med J Aust. 2006; 185(3):159-61.
- Skyler JS. The Spectrum of Insulin Resistance. Diabetes Care. 1979; 2 (3): 319-322.
- Ram Weiss, Andrew A Bremer, Robert H Lustig. What is metabolic syndrome, and why are children getting it? Ann N Y Acad Sci. 2013; 1281(1): 123–140.
- Maffetone P. Complementary Sports Medicine: Balancing traditional and nontraditional treatments. Human Kinetics Publishers: 1999.
- Voulgari C, Tentolouris N, Dilaveris P, et al. Increased heart failure risk in normal-weight people with metabolic syndrome compared with metabolically healthy obese individuals. J Am Coll Cardiol. 2011; 58(13):1343-50.
- Ruiz-Núñez B, Pruimboom L, Dijck-Brouwer J, Muskiet F. J Nutritional Biochemistry. 2013; 24:1183-120.
- Diego Gomez-Arbelaez, Laura Alvarado-Jurado, Miguel Ayala-Castillo, et al. Evaluation of the Finnish Diabetes Risk Score to predict type 2 diabetes mellitus in a Colombian population: A longitudinal observational study. World J Diabetes. 2015; 6(17): 1337-1344.
- Min Hi Park, Dae Hyun Kim, Eun Kyeong Lee, et al. Age-related inflammation and insulin resistance: a review of their intricate interdependency. Arch Pharm Res. 2014; 37(12): 1507-1514.
- Mokdad AH, Bowman BA, Ford ES, et al. The continuing epidemics of obesity and diabetes in the United States. JAMA. 2001; 286(10):1195-200.
- Ma L, Wang J, Li Y. Insulin resistance and cognitive dysfunction. Clin Chim Acta. 2015; 444:18-23.
- Nestler JE. Assessment of insulin resistance. Scientific American Science & Medicine. September/October, 1994: 58-67.
- Corbould AM, Judd SJ, Rodgers RJ. Expression of types 1, 2, and 3 17 beta-hydroxysteroid dehydrogenase in subcutaneous abdominal and intra-abdominal adipose tissue of women. J Clin Endocrinol Metab. 1998; 83(1): 187-194.
- Hollmann M, Runnebaum B, Gerhard I. Impact of waist-hip-ratio and body-mass-index on hormonal and metabolic parameters in young, obese women. Int J Obes Relat Metab Disord. 1997; 21(6): 476-483.
- Schiavon R, Altamirano-Bustamante N, Jimenez C, et. al. Fasting and postprandial serum insulin in Mexican adolescents with menstrual disorders. Rev Invest Clin. 1996; 48(5): 335-342.
- Franks S, Robinson S, Willis DS. Nutrition, insulin and polycystic ovary syndrome. Rev. Reprod. 1996; 1(1): 47-53.
- Pertti Ebeling, Heikki A Koistinen, Veikko A Koivisto. Insulinindependent glucose transport regulates insulin sensitivity. FEBS Letters. 1998: 436 (3): 301-303.
- Keltikangas-Jarvinen L, Ravaja N, Raikkonen K, Lyytinen H. Insulin resistance syndrome and autonomically mediated physiological responses to experimentally induced mental stress in adolescent boys. Metabolism. 1996; 45(5): 614-621.
- Lembo G, Vecchione C, laccarino G, Trimarco B. The crosstalk between insulin and the sympathetic nervous system: possible implications in the pathogenesis of essential hypertension. Blood Press Suppl. 1996; 1: 38-42.
- Clow A, Thorn L, Evans P, Hucklebridge F. The awakening cortisol response: methodological issues and significance. Stress. 2004: 7:29–37.

- Yates KF, Sweat V, Yau PL, et al. ATVB in focus metabolic Syndrome and Insulin Resistance: Mechanisms and Consequences. Impact of Metabolic Syndrome on Cognition and Brain. Arterioscl Throm Vas. 2012; 32: 2060-2067.
- Hoth KF, Gonzales MM, Tarumi T, Miles SC, Tanaka H, Haley AP.
   Functional MR imaging evidence of altered functional activation in metabolic syndrome. AJNR Am J Neuroradiol. 2011; 32:541–547.
- Holden RJ. Schizophrenia, suicide and the serotonin story. Med Hypotheses. 1995; 44(5): 379-391.
- Ravaja N, Keltikangas-Jarvinen L. Temperament and metabolic syndrome precursors in children: a three-year follow-up. Prev Med. 1995; 24(5): 518-527.
- Virkkunen M. Reactive hypoglycemic tendency among habitually violent offenders. Nutr Rev. 1986; 44 (Suppl): 94-103.
- Lande MB, Kaczorowski JM, Auinger P, Schwartz GJ, Weitzman M. Elevated blood pressure and decreased cognitive function among school-age children and adolescents in the United States. J Pediatr. 2003; 143:720-724
- Logroscino G, Kang JH, Grodstein F. Prospective study of type 2 diabetes and cognitive decline in women aged 70-81 years. BMJ. 2004; 328:548-551
- Yau PL, Javier DC, Ryan CM, et al. Preliminary evidence for brain complications in obese adolescents with type 2 diabetes mellitus. Diabetologia. 2010; 53:2298-2306
- Bokura H, Nagai A, Oguro H, Kobayashi S, Yamaguchi S. The association of metabolic syndrome with executive dysfunction independent of subclinical ischemic brain lesions in Japanese adults. Dement Geriatr Cogn Disord. 2010; 30:479–485.
- Cavalieri M, Ropele S, Petrovic K, et al. Metabolic syndrome, brain magnetic resonance imaging, and cognition. Diabetes Care. 2010; 33:2489-2495.
- Hassenstab JJ, Sweat V, Bruehl H, Convit A. Metabolic syndrome is associated with learning and recall impairment in middle age. Dement Geriatr Cogn Disord. 2010 29:356–362.
- Komulainen P, Lakka TA, Kivipelto M, et al. Metabolic syndrome and cognitive function: a population-based follow-up study in elderly women. Dement Geriatr Cogn Disord. 2007; 23:29–34.
- Schuur M, Henneman P, van Swieten JC, et al. Insulin-resistance and metabolic syndrome are related to executive function in women in a large family-based study. Eur J Epidemiol. 2010; 25:561–568.
- 34. MAF HR WHITE PAPER
- 35. Gonzalez-Yanes C, Sanchez-Margalet V. Signalling mechanisms regulating lipolysis. Cell Signal. 2006; 18:401-408
- Postic C, Girard J. Contribution of de novo fatty acid synthesis to hepatic steatosis and insulin resistance: lessons from genetically engineered mice. J Clin invest. 2008; 118(3):829-38.
- Kreher JB and Schwartz JB. Overtraining Syndrome. A Practical Guide. Sports Health. 2012 Mar; 4(2): 128–138.
- Fry AC, Kraemer WJ. Resistance exercise overtraining and overreaching. Sports Med. 1997; 23(2): 106-129.
- Lindgren F Dahlquist G, Efendic S, Persson B, Skottner A. Insulin sensitivity and glucose-induced insulin response changes during adolescence. Acta Paediatr Scand. 1990; 79: 431-436.
- Coulston AM, Liu GC, Reaven GM. Plasma glucose, insulin and lipid responses to high-carbohydrate low-fat diets in normal humans. Metabolism. 1983; 32(1): 52-6.
- Lustig RH, Mulligan K, Noworolski SM, et al. Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. Obesity. 2015; 24(2):453-60.



- Velazquez EM, Mendoza S, Hamer T, Sosa F, Glueck CJ. Metformin therapy in polycystic ovary syndrome reduces hyperinsulinemia, insulin resistance, hyperandrogenemia, and systolic blood pressure, while facilitating normal menses and pregnancy. Metabolism. 1994; 43(5): 647-654.
- 43. Chavarro J, Willett W, Skerrett P. The Fertility Diet: Groundbreaking research reveals natural ways to boost ovulation and improve your chances of getting pregnant. McGraw Hill Professional; 2009 Apr 27.
- 44. Feinman RD, Pogozelski WK, Astrup A et al. Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base. Nutrition. 2015; (31):1-13.
- 45. Tóth C, Clemens Z. Type 1 diabetes mellitus successfully managed with the paleolithic ketogenic diet. Int J Case Rep Images. 2014; 5(10):699-703.
- 46. Seyfried TN, Flores R, Poff AM, D'Agostino DP, Mukherjee P. Metabolic therapy: a new paradigm for managing malignant brain cancer. Cancer Lett. 2015; 356(2 Pt A):289-300.
- 47. Seyfried TN, Flores RE, Poff AM, D'Agostino DP. Cancer as a metabolic disease: implications for novel the rapeutics. Carcinogenesis. 2014 Mar; 35(3):515-27.
- 48. Volek JS, Freidenreich DJ, Saenz C, et al. Metabolic characteristics of keto-adapted ultra-endurance runners. Metabolism. 2015: 65(3):100-10.
- 49. Noakes T, Volek JS, Phinney SD. Low-carbohydrate diets for athletes: what evidence? Br J Sports Med. 2014; 48(14):1077-8.
- 50. Maffetone P. Season-Long Progress of an Elite Endurance Athlete on a Low-Carbohydrate/High-Fat Ketogenic Eating Plan. (Tampa, FL, USA), Nutritional Ketosis and Metabolic Therapeutics 1st Annual Conference. January 28-30, 2016.
- Howard BV, Manson JE, Stefanick ML, et al. Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. JAMA. 2006; 295(1):39-49.
- 52. Dansinger ML, Schaefer EJ. Low-fat diets and weight change. JAMA. 2006; 295:94-5.

