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December 2015

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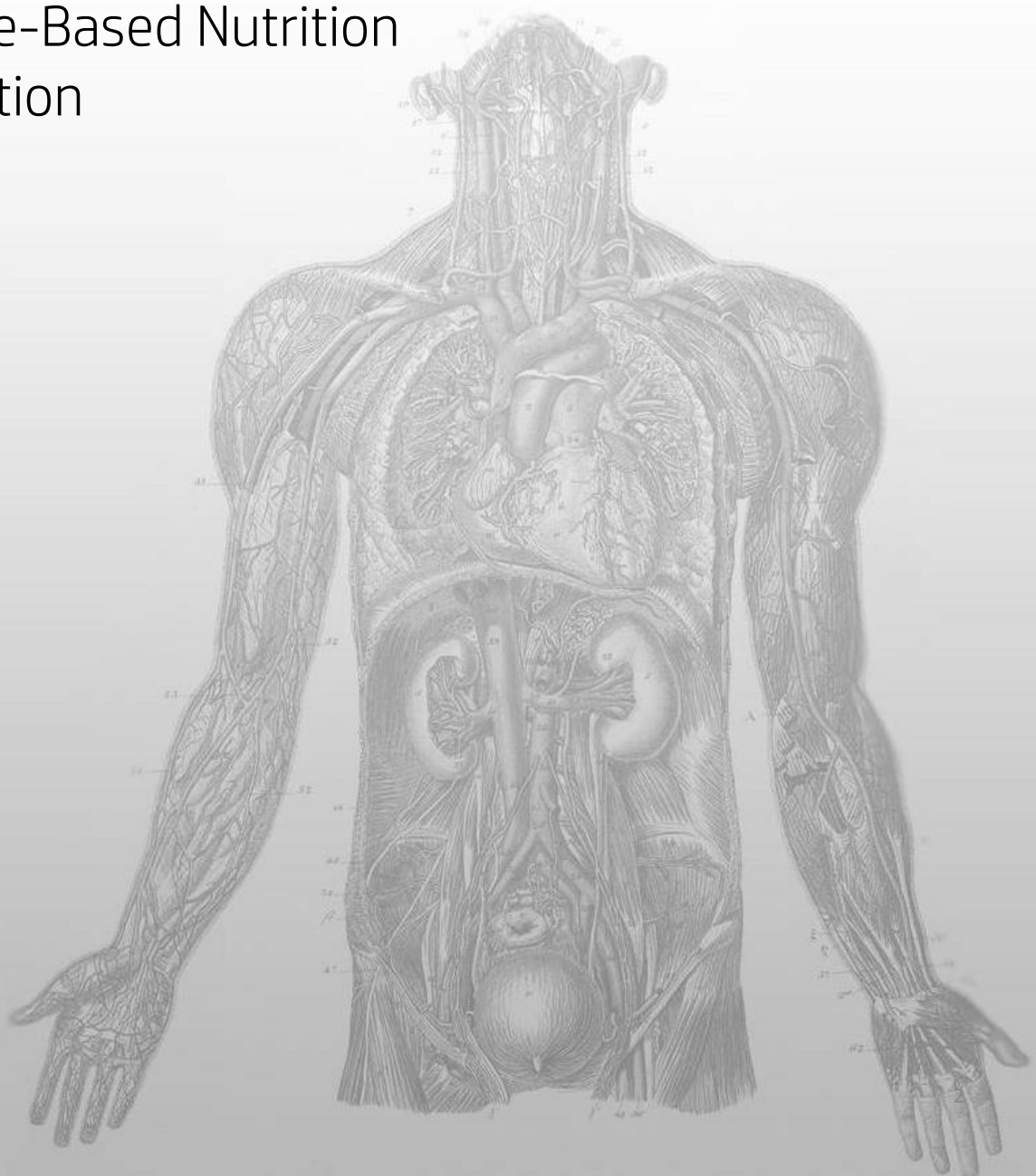

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Letter From the Editor

The success of the first edition is both a blessing and a curse. The bar was set high and now it must be raised. In order to do so we decided to tackle something big.

There may be no topic that is more highly debated, or raises more ire amongst people than the role of carbohydrates in the diet.

My personal views and opinions on the topic have gone the full spectrum over the past decade, swinging from ketogenic to mainlining dextrose. . . Ok, maybe not that extreme but my views have changed and developed overtime.

The role of carbohydrates in the diet is complex and highly nuanced. As a macronutrient it has implications in athletic performance, obesity, diabetes, cancer, mental disorders and a host of other prominent health conditions.

Unfortunately media often hyperbolizes research, using catchy titles to sell articles and ignoring the truth underlying the science.

In this issue of *Science Driven Nutrition* we took great care to assemble a lot of bright minds to be as objective as possible in their writing of the main articles as they convey their viewpoints on certain aspects of carbohydrates in the diet.

Joseph Agu masterfully covers the science behind carb-backloading and exactly how the timing of carbohydrates effects fat mass in the human body.

As an extension of some of Joseph's concepts I compiled several different lines of evidence to shed some light on the carbohydrate-insulin-obesity hypothesis. This article, while lengthy covers the concepts necessary to unravel the knot that researchers have used to hold that hypothesis together.

Sergio Fontinhas, uses his piece to present a paradigm of how carbohydrates are utilized in the context of your diet. The concepts conveyed in this article are robust and enlightening. They also build on the previous two article.

We are also fortunate to have Dr. Mike T Nelson provide an op-ed on how insulin resistance effects fat loss and tools to overcome some of hurdles that individuals in that position face.

We round out this issue with a research highlight by Dylan Dahlquist (aka The Jedi) on fat adaptation in athletes and how carbohydrates and fat effect performance.

-Brad Dieter, PhD, Executive Director

SCIENCE DRIVEN NUTRITION

Will Breakfast Make You Fat?

The Evidence About Carb Back-Loading

Carb Back-Loading: What Does the Evidence Say?

by Jose Antonio, PhD FNSCA FISSN CSCS

Carb Back-Loading (CBL) – a brainchild of John Kiefer - basically revolves around taking advantage of the supposed fluctuations in insulin sensitivity (IS) within the muscle and fat tissue throughout the day, as well as the non-insulin mediated uptake of glucose within the exercised muscles. For example, insulin sensitivity in both muscle and fat tissue is generally higher in the morning relative to the evening [1]. As such, Kiefer has suggested that eating carbs in the morning/earlier in the day (when overall IS may be higher) relative to the evening, will result in greater glucose uptake by the muscle (a good thing), but also in the fat tissue (a potentially bad thing).

Kiefer suggests that a way to get around this problem would be to train in the evenings as well as consuming almost all of your daily carbohydrate post-workout (PWO), whilst eating as little carbs as possible throughout the day. That way, you would supposedly take advantage of the reduced IS in fat tissue in the evenings, but also have the benefits of increased insulin sensitivity (more specifically, non-insulin mediated uptake of glucose) in the muscles PWO due to the evening training.

In addition, by avoiding carbs as much as possible during the day (when overall IS is high), fat gain via de novo lipogenesis (the creation of new fat tissue via carbohydrate), would apparently be minimised. Overall, this would hypothetically result in the potential for successful body recomposition (i.e. gain muscle whilst losing fat).

The following points briefly summarise how CBL works, in Kiefer's words:

1. Shift calories to later in the day, eating lighter in the morning and early afternoon, and feast at night. This may include skipping breakfast.

2. Keep carbs at an absolute minimum throughout the day until training.

3. Train in the afternoon, at around 5pm or so.

4. Start ingesting carbs after your training session, up to 30 minutes later.

5. Continue eating carbs throughout the night.

Does research support the idea of CBL?

There are correlational data signifying associations between shifting the intake of calories at different times of the day and adiposity (generally favouring eating earlier in the day). However, given the limitations of observational research, I'll solely focus on randomised control trials here.

The whole idea of shifting carbohydrate intake to later in the day is largely based on two studies, which are frequently cited throughout CBL. The first study by Keim et al [2] compared the effects of eating 70% of the day's calories in the morning (AM) vs. the evening (PM) on body mass and body composition during a six-week hypocaloric diet (60% CHO, 18% PRO & 22% FAT) in a group of 10 women. It was found that the ingestion of the larger AM meals resulted in greater weight loss compared with the larger PM intake, but this extra weight loss consisted of lean body mass (LBM). Therefore, the consumption of larger PM meals resulted in greater preservation of lean body mass (LBM) and resulted in a greater reduction of fat mass (see table below).

This study possessed several design strengths, the most notable of which was that it was conducted in a metabolic ward, meaning that food intake was strictly controlled.

The effect of large morning meals (AM pattern) versus large evening meals (PM pattern) on changes in weight and body composition during weight reduction[1]

	AM pattern	PM pattern
Weight, *kg/6 wk*	−3.90 ± 0.19	−3.27 ± 0.26[b]
Fat-free mass, *kg/6 wk*	−1.28 ± 0.14	−0.25 ± 0.16[a]
Fat percentage, *%/6 wk*	−1.83 ± 0.22	−2.52 ± 0.29[c]

[1] Values are means ± SEM, $n = 10$, and represent combined data for all subjects because statistical analysis showed no effect of order in presenting the meal patterns; PM pattern means significantly different from the AM pattern are indicated by superscript: [a] $P < 0.001$, [b] $P < 0.01$, [c] $P < 0.05$.

Furthermore, the 10 women underwent a structured exercise programme consisting of cardio and resistance training, making the results somewhat more applicable for those implementing CBL. However, two notable limitations of this study include the relatively small sample size (10) and method of assessing body composition (total body electrical conductivity, which is similar to BIA). BIA isn't the most accurate means of assessing body composition! [3]

The more recent trial used to support the evening carb intake of CBL is a 6-month study by Sofer et al [4], in which the authors compared the effects of carbs eaten mostly at dinner vs. eaten throughout the day, in diets consisting of 1300-1500 kcal (40-50% CHO, 20% PRO & 30-35% FAT) in a group of 78 Israeli police officers. It was found that reductions in weight, body fat and waist circumference were greater in the evening-carb experimental condition vs. the control condition. In addition, glucose control, inflammation, blood lipids and satiety were improved to a greater degree in the evening-carb group.

Moreover, leptin levels decreased to a lesser degree in the experimental condition and may partially explain the better maintenance in satiety within this group, as well as the greater observed weight loss. It is possible that the greater reductions in satiety in the control group led to a greater caloric intake in comparison to the evening-carb group, and thus explaining the more favorable body composition results seen in the experimental group.

Although this study looks extremely promising for CBL with respect to all the anthropometric, hormonal and biomarker data, the methodological limitations of the investigation are worth briefly discussing. Whilst a specified diet was prescribed, dietary intake was self-reported (unlike the shorter trial above). It is therefore possible that the participants' reported intakes were inaccurate, especially when considering the hectic work patterns on police officers. Similarly, caloric intake wasn't set according to the individual. Whilst still on the subject of food intake, an intake of 20% protein is the equivalent to roughly 65-75g per day. As the participants in this trial had an average body mass of 98.3kg, this would equate to a daily protein intake of 0.66-0.76g/kg, which is below the RDA of 0.8g/kg.

This intake is below that required [5] to spare muscle mass and promote satiety and is far below that typically consumed [6] by weight trainees looking to improve body composition. Therefore, the study's relevance to such populations is questionable (not to mention the lack of a structured exercise programme). Finally, when we look at the differences in weight loss between groups, the experimental group lost 2.54kg more than the control group over the 6-month trial, and was the only anthropometric measure to reach statistical significance. To put things into perspective, this equates to a greater weight loss of 14g per day, or 100g per week; this is hardly anything to write home about and surely isn't worth the hassle if it doesn't easily fit into your routine.

There are other controlled studies [7,8] similar to the two aforementioned, but no changes in body composition or weight loss were observed, probably due to short study durations (15 & 18 days, respectively) and other inherent limitations. Due to their neutral findings, these studies tend not to be mentioned by CBL advocates. Though, by the weight of the limited controlled evidence, it does seem that shifting caloric (and carbohydrate) intake to later in the day would provide a SLIGHT benefit with respect to body composition, hormonal changes and makers of health and disease.

Summary and Practical Recommendations

The general concept of CBL is supported by science. However, this science is limited to two studies with their fair share of limitations, rendering the topic inconclusive in the absence of compelling evidence. Moreover, if we consider shifting only carbohydrate as opposed to overall caloric intake, the data supporting the concept of CBL diminished to a single study. Nevertheless, if we consider the overall impact of calorie/carbohydrate placement on body composition from these two studies, though significant, in reality such differences are trivial.

In my opinion, the total macronutrients consumed by the end of the day will have the largest impact in terms of body composition changes; Kiefer even admits this (at least in regards to meal frequency), "the breakdown and distribution of calories and macro nutrients throughout the day matters far more than the number of meals".

At the end of the day, CBL will get some people results, but it will do so because of the caloric deficit and sufficient protein, not because of the intricate protocols.

As such, with respect to carb placement for body composition and performance, total intake is the primary consideration. A secondary consideration would be the positioning of these carbs in relation to training (around-workout nutrition) in order to optimise training performance. Once these factors are in place and consistently achieved, then, and only then, should someone have the option to experiment with hypothetical protocols.

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Do Carbs Make You Fat?

A Critical Analysis of the Carbohydrate-Insulin-Obesity Hypothesis

Carbs → Insulin → Obesity??

Carbohydrates have gotten a “bad rap” because they elicit an insulin response. And as everyone knows, insulin causes us to become fat (or does it?). In fact, the carbohydrate-insulin-obesity hypothesis has been blamed as the major cause of the obesity epidemic. Unfortunately, when we look closer at the science this idea doesn’t quite hold up

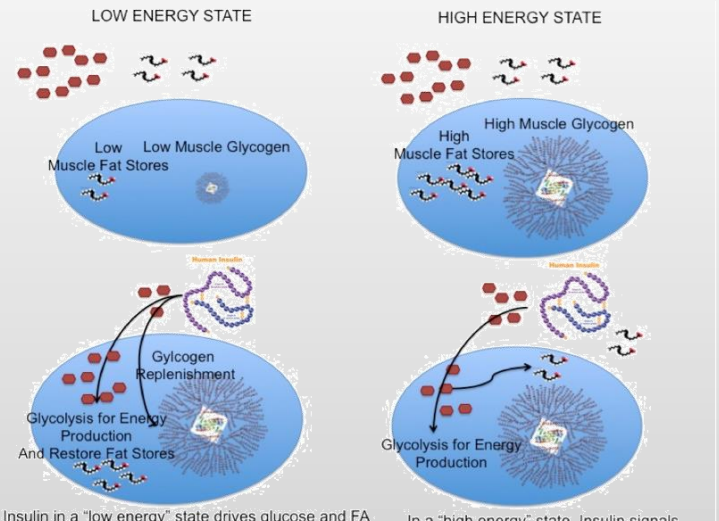
Does Insulin Increase Body Fatness?

Common reading literature such as Good Calories, Bad Calories* have put the hormonal regulation of body fat into limelight. In books such as GCBC, insulin is painted as the primary cause of common obesity. While it is true that hormones play a critical part in the regulation of body-fat, it is naïve and myopic to single out insulin as the sole contributor. Our endocrine system is extremely robust with almost countless signaling molecules. While insulin plays a critical part in substrate partitioning and metabolism (including the fate of fat and carbohydrates), it plays only one part in the signaling cascade. To ferret out whether insulin is in fact the main regulator in body fatness and if increased insulin levels due to carbohydrates cause obesity we need to look at the literature.

Insulin Action: Muscle, Liver, and Adipose Tissue

Insulin is an endocrine hormone, meaning it affects multiple tissues in the body. Specific to our focus is skeletal muscle, the liver, and our adipose tissue (fat).

When skeletal muscle is “exposed” to insulin several things happen within the muscle cell. First, glucose transporters (e.g. Glut4) move to the cell surface and begin transporting glucose into the muscle cell. Secondly, insulin signals the muscle cell to shift to carbohydrate-based metabolism. Now it is important to note that the energy status of the cell plays a role in exactly what happens in the muscle cell. If the muscle cell is low on glucose and/or muscle glycogen the insulin signal will instruct the cell to utilize the incoming glucose for fuel and to begin creating muscle glycogen from any spare glucose that is coming in.

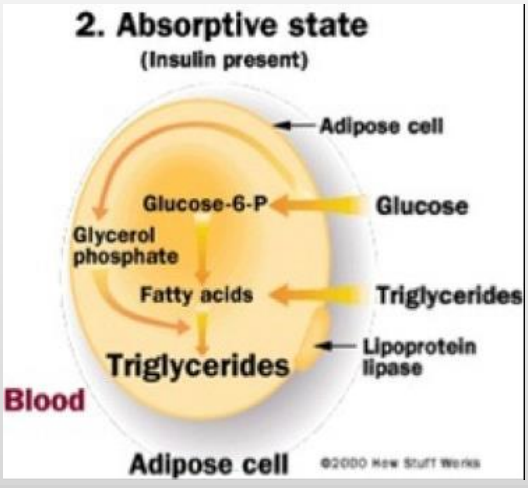


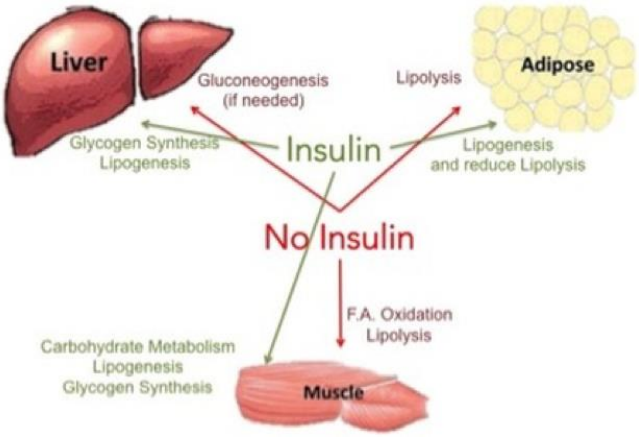
Insulin in a “low energy” state drives glucose and FA uptake while shifting metabolism to carbohydrate

In a “high energy” state, Insulin signals carbohydrate
Metabolism and fatty acid synthesis

Additionally, insulin will cause an uptake of fatty acids into muscle cells (Figure 1). If the muscle cell is already full of glucose and glycogen and topped off with intramuscular triglycerides then insulin signaling will do all of the things it does in a low energy state but will also turn excess glucose into fat through de novo lipogenesis (Figure 1).

What about the liver? The liver is one of the main sources of regulating blood sugar in the body. When our body sugar gets too low a hormone named glucagon signals the release of sugar from the liver (hepatic glucose output). Insulin, which is release in cases of elevated blood sugar due to carbohydrate (or protein) ingestion, signals the liver to stores its glucose, convert extra to liver glycogen, and then when that is full to synthesis fatty acids from extra glucose. Again, we do see that insulin can have a "lipogenic" effect. However, it is important to note the lipogenic effect of insulin on the liver is only seen in the case of saturated glucose and glycogen stores, which in essence is a state of excess energy.





Now to discuss the star of the "insulin-obesity" hypothesis, the role of insulin and our adipose tissue. Insulin does in fact decrease the rate of lipolysis in adipose tissue and does stimulate fatty acid and triacylglycerol synthesis (Figure 2). The science is crystal clear on this.

Now from this brief overview it would indeed appear that insulin promotes fat storage… well unfortunately that is the wrong wording for it. Insulin is "fat-sparing", not fat promoting. Yes, insulin does reduce fat oxidation and promote a lipogenic environment. However, for insulin to have a profound effect on body fatness and fat gain, we would have to have a constant high signal of insulin, something that is not typically seen. Our bodies default metabolic state is one of fat oxidation and insulin is simply the switch that turns it to carbohydrate metabolism (Figure 3). The net effect of insulin is simply a switch in substrate metabolism, it does not produce a large "obesegenic affect" due to dietary composition alone. FOR INSULIN TO HAVE A SIGNIFICANT OBESEGENIC EFFECT IN A HEALTHY INDIVIDUAL IT MUST OCCUR IN THE PRESENCE OF EXCESS ENERGY.

To examine this point scientifically, lets look a study that addresses the following question: do isocaloric diets that result in higher insulin levels increase body fatness?

One study had 8 participants consume a high-carbohydrate diet (60% CHO) for 7 days and a high-fat diet (60% fat) for 7 days and measured total energy expenditure and nutrient oxidation. The study showed that the composition of the diet did not effect total energy expenditure (a key component to long-term weight gain) but DID "rapidly shift substrate oxidation to closely reflect the composition of the diet" (1). Another study conducted by Dr. Kevin Hall showed that calorie for calorie, fat restriction leads to greater weight loss than carbohydrate restriction over short periods (2). This suggests that insulin signaling plays a major role in determining whether fat or carbohydrate is used as a primary fuel source, but not a major role in overall energy expenditure.

Additionally, if insulin were the driving factor in body fatness we would expect virtually all overweight or obese individuals to have elevated insulin levels to cause weight gain. Such is not always the case. In fact, there is a large portion of overweight individuals with normal, "healthy" insulin signaling.

We should also point to one critical piece of evidence: protein also elicits an insulin response. In fact, some high-protein foods elicit a greater insulin response than high-carbohydrate foods (has your brain exploded yet? I know mine did when I first read this article).

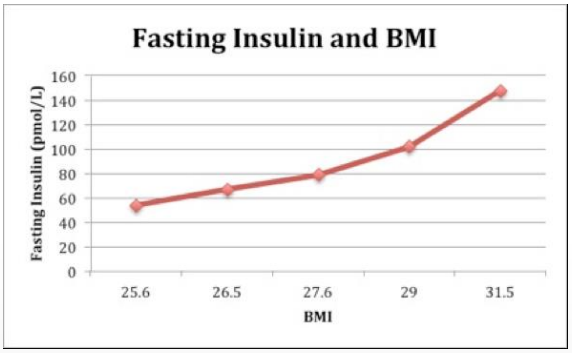
Why is this important? Well the most consistent finding from nutritional research is that diets higher in protein are effective in reducing body-fatness. So if insulin were the main culprit in body-fatness than consuming protein would also encourage fat accumulation and not fat loss.

When we take all this information together we can draw the following conclusion: insulin is a substrate regulator. It switches your body from burning mostly fat to mostly carbohydrate, promotes glycogen synthesis, and protein synthesis if necessary. In a metabolically healthy individual, the metabolism is flexible and robust enough to handle fluctuations in substrate and maintain leanness even in the presence of a high-carbohydrate intake that would, on the surface, seem to promote a "fat-accumulating" environment. Only in the presence of excess energy does insulin truly promote a fat-accumulating environment, even then our body has a nifty way of attempting to attenuate that environment.

Insulin and Obesity in Humans

I hate beating the dead horse (the poor thing is already a goner), but this is an important concept I want people to understand. It will be the basis for our approach to nutrition and fueling your performance in the gym. As a coach, I need us to be on the same page on the central idea of the last article and this one: carbohydrates are not the sole reason for body fat and in some cases increasing carbohydrate intake can be beneficial.

As we discussed earlier there is substantial common-reading literature that suggests that hyperinsulinemia is the driving cause of obesity in America. Specifically that increased sugar consumption leads to hyperinsulinemia which then leads to obesity. I would argue that increased sugar consumption has significantly contributed to the rise in obesity and metabolic diseases over the past few decades; however, from the data we will discuss in this post the increased insulin response leading to lipogenesis is not likely the mechanistic cause.*



Insulin Levels and Body Fat

Insulin levels are typically increased in obese individuals (figure 4); however it is not requisite. As mentioned last week, there are cases (more than you would think) where obese individuals do not show increased fasting insulin. For a reference, Hivert and colleauges reviewed the literature and showed the wide ranges of conflicting data on this topic back in 2007

This suggests that insulin is not the direct cause of common obesity. If it were, we would observe increased insulin in all cases of obesity (barring genetic disorders, extreme cases, or late stage T2DM where beta-cell dysfunction drops insulin levels).

For something to be a direct “cause” to “effect”, like stating that insulin causes obesity you would have to demonstrate (within measurable experimental error) that rises in insulin always precede obesity and are present in the obese state, which has not been shown.

Insulin Levels Rise and Fall with Body Weight

Ok, so now it may be plausible that increased fasting insulin is an effect of obesity, not a cause. Well, there is evidence to suggest this is likely the case. For example, track insulin as body weight moves up and down the scale; we also observe changes in fasting insulin with weight change. In studies where participants gain weight there is a concordant rise in insulin (Figure 5). Conversely, weight-loss leads to a decrease in insulin (Figure 6).

Separately it is difficult to tease out whether it is a cause or effect of weight gain but when taken together it is more likely that insulin levels are affect by weight, specifically adipose tissue.

What is going on?

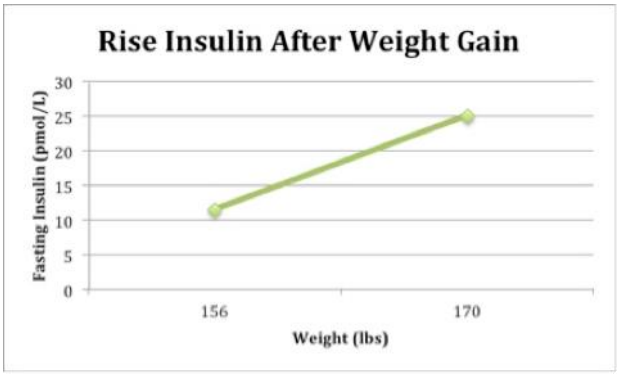
One of insulin's main roles is to maintain glucose homeostasis. Briefly, increased insulin promotes glucose uptake into tissues. Therefore, we would expect that increase levels present in obese individuals would result in lower blood glucose levels; however, what we observe is an increase in glucose (figure 7).

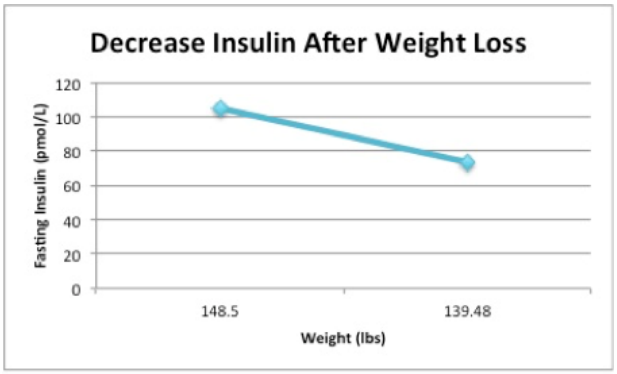
This suggests that while there is increased insulin, it is not functioning properly. While there is increased insulin in obese people (in most cases), it appears its action is reduced. This would explain the concurrent rise in both insulin and blood glucose

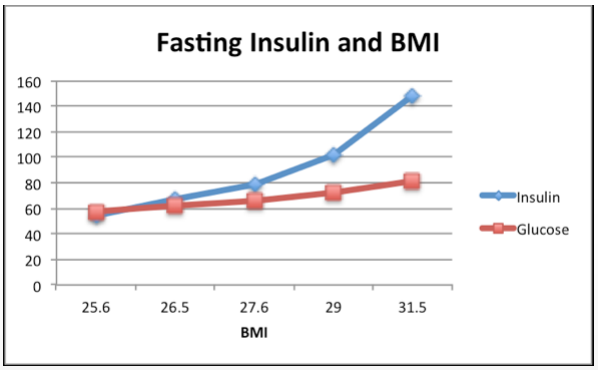
Additionally, in adipose tissue, insulin promotes lipogenesis and reduces lipolysis.

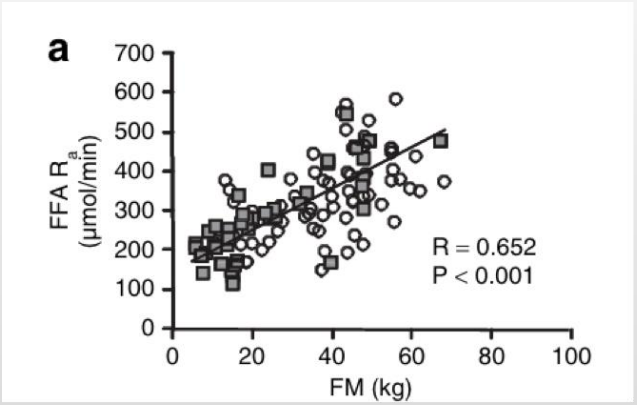
Therefore, we would observe decreased plasma fatty acids (FA) because insulin would promote fat uptake into the adipose tissue and suppress fat release.

However, we see the opposite; increased plasma FA in obese and diabetics, suggesting insulin action on adipose tissue is also decreased (figure 8) . If insulin action were increased in obese individuals due to the elevated insulin than we would observe decreased plasma FA as insulin action promotes lipogenesis and decreases lipolysis in adipose tissue; however we see the opposite in obese individuals.









This information suggests that in obese individuals, insulin is elevated, but the data suggests insulin action is actually decreased. This “jives” with the literature that obese individuals develop insulin resistance, which may eventually leads to type II diabetes. When we take all of this together it appears that elevated insulin is not the main cause of common obesity or fat gain.

Conversely, obesity is likely the cause of insulin resistance. **The main take home here is that insulin is not the culprit behind common obesity and substantial increases in adipose tissue.**

Losing Weight With Insulin Resistance

By: Mike T. Nelson, PhD

How Can I Lose Fat If I Am A Type 2 Diabetic?

Excellent question! If you have impaired glucose handling (as in the condition of type 2 diabetes), then you have increased insulin as the body tries to get glucose out of the blood. Remember that high levels of glucose in the blood is actually toxic.

Over time as the tissue becomes more insulin resistant, you need to put out more insulin in an attempt to get more glucose out, otherwise you will die.

The long term bugger is that as insulin goes up, it impairs fat oxidation. Oh crap! This is the opposite direction if you are looking to lose fat and get leaner.

Think of insulin as the "fuel selector switch" (1).

Low insulin = use fat. High insulin = use carbs.

Unfortunately, people with type 2 diabetes get short end of the stick. As the disease progresses they tend to have higher levels of glucose and fats in their blood

Low insulin = use fat. High insulin = use carbs.

Unfortunately, people with type 2 diabetes get short end of the stick. As the disease progresses they tend to have higher levels of glucose and fats in their blood (triglycerides), and as a result they are very metabolically inflexible (2). Their body is unable to effectively switch from using fat as a fuel to using carbs as a fuel due to the rising baseline levels of insulin. Remember, higher insulin makes it much harder for the body to burn fats.

Health Numbers For Fat Loss

If you are someone who is a type 2 diabetic and looking to lose fat, talk to your doctor about your blood work, especially your triglycerides (TGs), fasting glucose, and HbA1C levels (a marker of 3 month glucose levels).

This would give you a pretty darn good snapshot of metabolic health. Better metabolic health equates to being more metabolically flexible so your body can more effectively use fat as a fuel.

Dr Mike T Nelson

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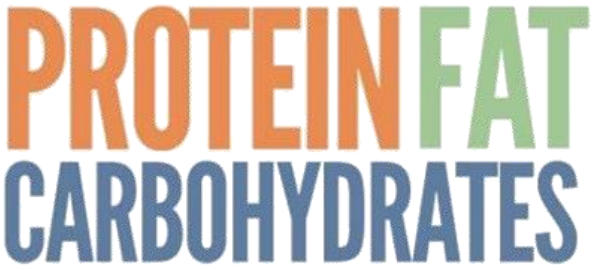


Carohydrate Disposal: A Hierarchy

By Sergio Fontinhas

The Abstract: AKA The TLDR Version

The oxidative hierarchy refers to the relative order in which fuels (alcohol, carbohydrates, protein and fat) are selected for oxidative disposal after ingestion. Giving the relative storage capacity for these different substrates, each fuel assumes different priorities within metabolic pathways. The hierarchy is dominated by alcohol, followed by carbohydrate and protein, with fat at the bottom. Alcohol, carbohydrate and protein elicit powerful auto-regulatory mechanisms - they promote their own oxidation – while fat does not. Glycogen stores can maximally accommodate 800-900g of carbohydrate and perhaps as much as 1000-1.100g in trained athletes.

When glycogen stores are saturated, massive intakes of carbohydrate are disposed of by high carbohydrate-oxidation rates. Along with a progressive carbohydrate oxidation, basal metabolic rate and total energy expenditure also increases. Another pathway to dispose of carbohydrates is de novo lipogenesis, however this process is quantitatively small in humans.

1.Fuel Storage Capacity

1.1 Alcohol

What is the fuel storage capacity for alcohol? The answer is virtually none, the body has no storage capacity for alcohol and therefore it must be “detoxified” and oxidized as soon as possible (1,3). For this reason, after ingestion, alcohol has a high oxidative reactivity and promotes rapid hepatic oxidation until alcohol is cleared, while at the same time suppressing the oxidation of other macronutrients when present (1,2,3).

1.2 Carbohydrate

After ingestion, dietary carbohydrates have four metabolic fates (4,5):

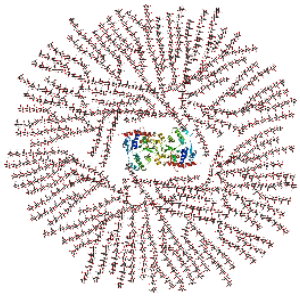
1. Used as fuel substrate and oxidized in several tissues of the body, mainly as glucose;

2. Storage as glycogen in skeletal muscle (80% stored) and liver;

3. Storage as triacylglycerol (TAG);

4. Conversion of glucose molecules into C3 precursors in the splanchnic area which pass on to the liver where they are used as substrates for gluconeogenesis

Glycogen is a homopolysaccharide composed of subunits of glucose. The synthesis of glycogen consists of adding individual glucose units to an existing glycogen chain, forming extensively linked branches every 8-12 residues on the glycogen molecule. The more extensively branched the molecule is, the greater the ability it has to rapidly supply energy.



The body's capacity for storing glycogen is limited. Most of dietary glucose is stored as glycogen in liver and skeletal muscles during the postprandial period and is subsequently released and oxidized (6). The capacity for storing large amounts of glycogen is relatively large (7,8,9,10,11).

Looking at the actual storage of glycogen in the body it was reported back in 1975 that a nonobese man weighing 70 kg stores about 350 g of muscle glycogen and 40-50 g of liver glycogen (12), equivalent to about 1600kcal of energy stored in the body in the form of glycogen.

Liver glycogen varies in relation to the patterns of eating and fasting (13). Liver glycogen concentrations vary in the range of 8-81g (50-500 mmol glycosyl residues/kg tissue in the post-absorptive state) with a mean of 44g (270 mmol glycosyl residues/kg liver) (14).

Skeletal muscle glycogen concentrations can also vary, concentrations depend on upon the muscle being measured (15). In biopsy samples from the quadriceps femoris muscle glycogen concentrations were found to be in the range of 60-l20 mmol glycosyl residues/kg with a mean of 85 mmol (14 g) glycosyl residues/kg tissue (15).

The body's glycogen reserves are usually maintained at between 250 and 500 g in a 70 kg adult man. More precisely, others reported that glycogen storage capacity in man is 15 g/kg body weight and can accommodate a gain of ~*500*g before net lipid synthesis contributes to increasing body fat mass (16). For a 70-kg man with ~40% of his weight as skeletal muscle and a liver weighing 1.8 kg, one can estimate that ~3 mol glycosyl residues or **almost 500 g of glycogen are stored in the body**.

However, higher values have been reported. For subjects with a mean body weight of 72kg, Hedman (17) calculated maximal values of 700 g glycogen. Others came to the same conclusion but suggested that a further 100g could be stored with 2 weeks of carbohydrate overfeeding or by using the glycogen-loading technique (18).

Back in 1967, the highest values ever reported were above 4/100g of muscle in three subjects (19), and liver glycogen content ranged from 14.3 g to 80.1 g per kg wet liver tissue, with a mean of 43.7 with 2.4 g water per g glycogen (20). If those highest values are extrapolated to the whole body, then up to 4.3 mol glycosyl residues or some 700 g of glycogen could be stored in the body.

Bergstrom et al (19) reported values in the **range 500-800 g** in some of their subjects who followed a glycogen-loading technique. **The maximum increase in stored glycogen ever observed was 1146g in one subject** (16). Data derived from this overfeeding study (16) suggest that the glycogen stores can **maximally accommodate 800-900 g of carbohydrate and perhaps as much as 1-1.1 kg** in trained athletes, which are among the highest glycogen storage values reported in the literature (16).

This glycogen serves as the primary supplier of energy during most forms of exercise (21). Glycogen is stored with two to four times its weight of water (22).

In addition, the extracellular fluid can accommodate less than an extra 10 g glucose in order to avoid glucosuria.

1.3 Protein

After digestion proteins are hydrolyzed into amino acids and reach the intracellular amino acid pool, a metabolic pool limited in size and not expandable (23, 24). From this pool amino acids can follow 3 major pathways (23):

1 .AAs can be used for the synthesis of new endogenous proteins and other biological substances;

2. AAs can be irremediably oxidized by the body, yielding urea (+ ammonia) and carbon dioxide (CO_2) as terminal end-products (see process of ureagenesis) and;

3. AAs can be converted into other compounds (gluconeogenesis).

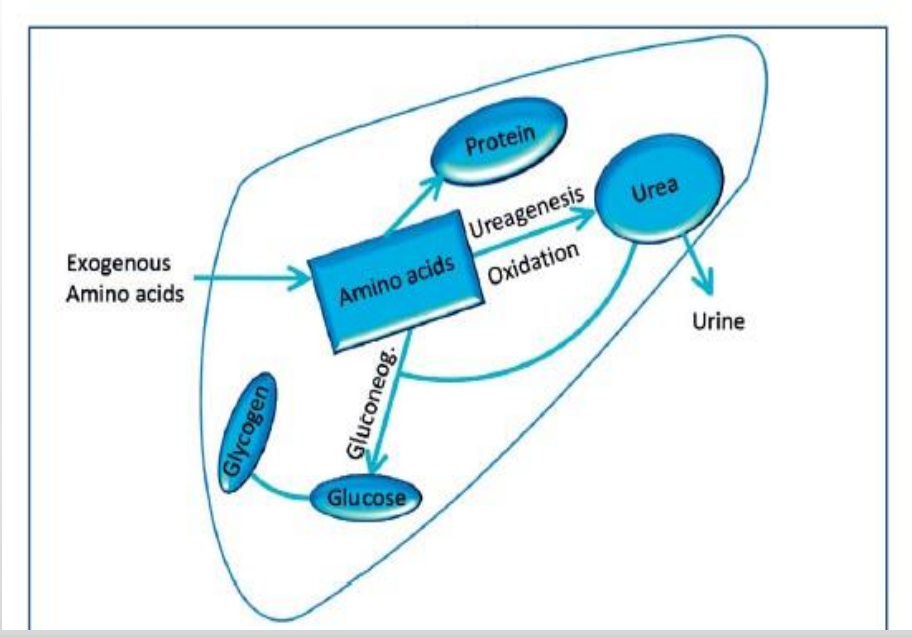
The free AA pool is maintained within tight limits (24), even under a variety of conditions of the free AA pool are very similar (25,26). The body tries to maintain body protein stores at constant levels (27). The concentration of each individual amino acid in the cell is precisely regulated (25).

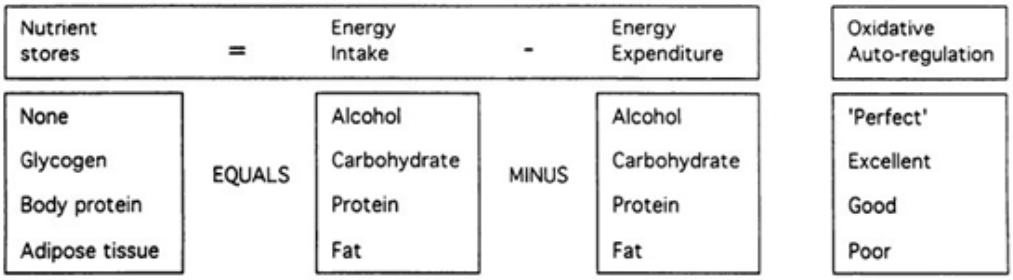
The free pool provides individual AAs for protein synthesis and oxidation, and it is replenished either by protein breakdown or AAs entering the body from the diet. For example, amino acids involved in charging of muscle tRNA apparently come from the intracellular pool (28).

Excluding taurine, the free pool has been estimated to contain only 100 grams of AAs, and including taurine the free pool increase up to 130 grams (29), with an additional 5 grams of free AAs circulating in the bloodstream (29). The free pool is approximately 1% of the size of the AA stored in tissue.

In an old study, 3g protein/kg only increased blood concentrations of most AAs by 30% above normal levels, with concentrations of BCAAs doubling over normal levels (30), which indicates that the pool is tightly regulated. The concentration of amino acids in the bloodstream are different from that seen within the muscle (31), changes in blood AA levels may have no impact on intramuscular AA concentrations.

Protein and amino acids ingested in excess of those needed for biosynthesis cannot be stored due to the limited size of the intracellular free amino acid pool, which cannot be much expanded (23). Although chronic strength exercises can increase the capacity for skeletal muscle protein storage there is eventually a limit.





It was once thought that the auto-regulatory control of protein oxidation was as efficient as that of carbohydrate (84). However, a 160% increase in protein intake (from 47 g/d during underfeeding to 122 g/d during overfeeding) only caused a 12% increase in protein oxidation (from 83 g/d during underfeeding to 93 g/d during overfeeding) (54). In contrast, a 550% increase in carbohydrate intake (from 83 g/d during underfeeding to 539 g/d during overfeeding) was almost matched by a 420% increase in oxidation (from 106 g/d during underfeeding to *551* g/d during overfeeding). **Carbohydrate was much more responsive**, and it certainly exerted a much greater influence on the reciprocal changes in fat utilization than did protein (54). **Protein takes a subordinate position to carbohydrate**.

1.4 Fat

Adipose tissue is highly plastic and can respond rapidly to changes in nutrient intake through fat-cell hypertrophy or hyperplasia.

Previously it was thought that the number of adipocytes remained constant, and that fat gain during adulthood was the result of adipocyte hypertrophy, not hyperplasia (32). However that supposition didn't quite fit well with the fact that fat-cell progenitors from different body-fat depots have distinct properties (33,34,35).

The ntion of fix adipocyte cell number was challenged by an overfeeding study and measurement of adipocyte size and number (36). This study reported a quick increase in femoral adipose tissue through formation of new fat cells, gain of only ~1.6 kg of lower-body fat (femoral fat) resulted in the creation of ~2.6 billion new adipocytes within 8 weeks (36).

Newly formed mature adipocytes arise from preadipocytes, resident in fat depots (37,38,39,40). In principle if adipocytes exceed an average lipid content of ~0.7–0.8 μg per cell new cells are created (16), in other words when adipocytes reach a critical volume or threshold they secrete factors that recruit new adipocytes (41,42,43). This response to overfeeding depends partially on sex and baseline adipocyte size.

The number of leg fat cells is greater in overweight than in normoweight persons (44), and obesity is associated with abdominal s.c. adipocyte hyperplasia (44,45,46).

This adipose tissue plasticity coupled with the fact that morbid obesity can be developed to a body mass index (BMI) higher than 40 and even 50 (47), illustrates the immense capacity for the body to store fat, unlike stores for carbohydrates and protein.

2.Substrate flux and hierarchy of fuel selection

The oxidative hierarchy follows the body's relative storage capacity for the different substrates and their role in ensuring survival.

1. Thus, the hierarchy is dominated by alcohol because the body has no storage capacity for it and must be eliminated by oxidative disposal through rapid hepatic oxidation (1,2,3).

2. Carbs come next in fuel selection. Due to the relatively small storage capacity for carbohydrates as glycogen (8,48,49,50) and the need to maintain glucose homeostasis within tight limits, ingestion of excess carbohydrates causes an acute autoregulatory increase in their oxidation (48,51,52,53,54). Lipogenesis is quantitatively unimportant (55,56).

- 3. Protein comes next. For quite some time there was some dispute as to which was dominant, carbs or protein (57), but there's clear evidence in favor of carbohydrate, at least under certain circumstances (54). Protein oxidation is related to protein ingestion (58).

- 4. Fat appears at the base of the hierarchy because there is virtually infinite capacity for fat storage. Thus, the rate of fat oxidation is regulated by the presence or absence of the other macronutrients. Futhermore, there is no autoregulatory mechanism between fat intake and fat oxidation, in other words, fat doesn't regulate its own oxidation after intake (49,59,63,64), and is further reduced under conditions of caloric excess (49,50). Fat oxidation is not increased after a single meal (59,60) and over an entire day excess fat intake does not increase fat oxidation (61). Increases in fat oxidation occur secondary to increases in the body fat mass (62). Not even in 50% excess calories from fat does fat promote its oxidation, oxidation rates remain practically equal to baseline before the overfeeding (64).

3.Carbohydrate disposal

So let's take a closer look at carbohydrate disposal. After glycogen depletion, glycogen stores takes up 4 days to saturate (11). Extreme carbohydrate manipulations have shown rapidly auto-regulatory adjustments in carbohydrate oxidation rates (49,62) over short periods of time, and the effect persists after normalization of the diet in response to the perturbed glycogen stores (49).

With the onset of carbohydrate overfeeding (after total glycogen depletion), there was a dramatic increase in carbohydrate oxidation from 74 ± 40 g/d (day 3) to 398 ± 87 g/d (day 4) (11). Thereafter carbohydrate utilization (ie, oxidation and that used for de novo lipid synthesis) increased progressively in response to the increase in carbohydrate ingestion, attaining 1010 ± 37 g/d on the last day of overfeeding (7 days) (11).

By the end of the second day of overfeeding, glycogen stores had increased by 500g. At this point carbohydrate oxidation and storage became insufficient to dispose of all of the ingested carbohydrate, and some had to be converted to fat, ie, de novo lipogenesis.

After 4 d of overfeeding glycogen stores became saturated at ~770g (occurred on day 5 for one subject). When the glycogen stores are saturated, massive intakes of carbohydrate are disposed of by high carbohydrate-oxidation rates (11).

In other metabolic ward study (12 days), carbohydrate intake of 540g and 83 g/d for overfeeding and underfeeding, respectively, exerted direct auto-regulatory feedback on carbohydrate oxidation (551 and 106 g/d at day 12 for overfeeding and underfeeding, respectively) (54).

With carbohydrate overfeeding there was a large increase in carbohydrate oxidation but also in glycogen storage (339 ± 82 g/d). Carbohydrate balance was achieved after the first few days and by day 12 carbohydrate oxidation was 551 g/d compared with an intake of 539 g/d. Carbohydrate oxidation was providing ~8.68 MJ/d, thus fat oxidation was suppressed. During overfeeding, BMR increased by 0.42 MJ (5.7%) and TEE increased by 0.75 MJ (6.2%).

This study also performed underfeeding. As expected over the first few days of underfeeding there was a sharp decrease in carbohydrate oxidation, reflecting a gradual but progressive decrease in muscle glycogen. After day 4 carbohydrate intake and oxidation were closely matched, with a small, persistent daily negative carbohydrate balance with intakes of 83 g/d compared with oxidation of 106 g/d (1.67 MJ/d) on day 12. In this case, to meet the body's energy requirement endogenous fat oxidation increased. BMR decreased by 0.82 MJ (8.3%) and TEE decreased by 1.20 MJ

(10.5%).

The contribution of protein to the fuel mixture during both interventions remained remarkably constant.

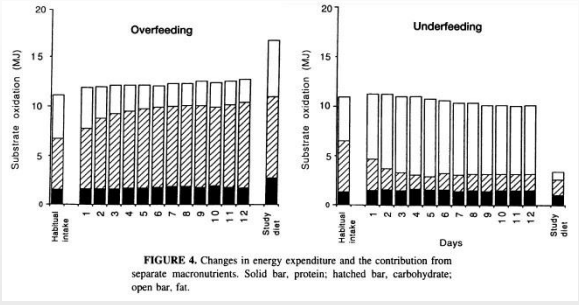


FIGURE 4. Changes in energy expenditure and the contribution from separate macronutrients. Solid bar, protein; hatched bar, carbohydrate; open bar, fat.

When protein intake surpasses the physiological needs of amino acids, **the excess amino acids are disposed of by** three major processes (23):

1**. Increased oxidation**, with terminal end products such as CO2 and ammonia
2. **Enhanced ureagenesis** i. e. synthesis of urea linked to protein oxidation eliminates the nitrogen radical
3. **Gluconeogenesis**, i. e. *de novo* synthesis of glucose. This is one of the mechanisms developed by the body to maintain blood glucose within a narrow range, (i. e. glucose homeostasis). Gluconeogenesis, uses non-glycogenic precursors; in particular certain specific amino acids (for example, alanine), as well as glycerol (derived from fat breakdown) and lactate (derived from muscles) to produce glucose

Progressive carbohydrate oxidation and total energy expenditure increases are seen with carbohydrate overfeeding (50% of excess energy intake) (65). Carbohydrate oxidation and total energy expenditure increases were evident in the first day of overfeeding and reached maximum by day 7 of overfeeding (14 days total). The increased energy expenditure was approximately double that which could be explained by the combination of increased TEF and increased body mass, meaning that more of the excess energy was oxidized and less stored in the body than was seen during fat overfeeding (65). Even on day 14 total energy expenditure was higher with carbohydrate overfeeding so that the total stored energy was less than with fat overfeeding.

This study also compared lean vs. obese subjects, and it was noted that obese subjects oxidized proportionally more carbohydrate and less fat than did lean subjects (65). The greatest reliance on carbohydrate oxidation during energy balance perturbations may be a risk factor for obesity (66,67,68). Subjects with the highest oxidative capacity of skeletal muscle have the lowest ratio of fat to lean mass in weight gain (69).

Another study showed a graded dose response in carbohydrate oxidation (70). Researchers used stable isotope-mass spectrometric methods with indirect calorimetry in normal subjects to quantify the metabolic response to six dietary phases (5 d each), ranging from 50% surplus CHO (+50% CHO) to 50% deficient CHO (-50% CHO), and 50% surplus fat (+50% fat). A dose response was observed in glucose production with increasing carbohydrate intake, which stimulated moderate hyperinsulinemia and decreased lipolysis and fatty acid availability. The net effect was to increase glycogen stores and deliver extracellular glucose, thus favoring increased carbohydrate oxidation and a reciprocal decrease in fat oxidation (70).

Carbohydrate disposal is not different between glucose, fructose, and sucrose, in lean and obese women (71). Different carbohydrates behave in an essentially identical manner. A 50% overfeeding with either glucose, fructose and sucrose resulted in no significant difference in fat balance, and there were no significant differences between lean and obese women in macronutrient oxidation or balances.

As expected, carbohydrate oxidation increased greatly in response to carbohydrate overfeeding (from 15.61 to 21.94, 21.64, and 21.97 MJ for fructose, glucose, and sucrose, respectively).

Of the excess carbohydrate ,74% was oxidized (compared to only 18% of the excess fat intake) and on average 12% of the excess energy was stored as glycogen and 88% as fat; there was no significant difference between overfeeding treatments.

As seen in other studies (54,72), almost all of the glycogen storage occurred on day 1, with minimal imbalance on subsequent days, which may suggest the need for glycogen stores to first be perturbed to generate feedback control.

The daily carbohydrate imbalance with sucrose overfeeding asymptotically approached zero as carbohydrate oxidation gradually increased until it exactly matched intake. This caused glycogen storage to plateau at a new level ~110 g above the initial value

Glycogen must be regulated within a relatively narrow window, and adipose tissue has evolved as the main energy storage compartment. Once any short-term changes in glycogen have resolved, additional energy excess or positive imbalances are buffered by fat stores.

4 .Lipogenesis

Net lipogenesis is quantitatively small in humans subjects following a typical diet (8,11,53,55,56). Net lipogenesis measurements using respiratory gas exchange (11,8) or from stable isotope tracer studies (55,56) indicate that under normal conditions the rates of lipogenesis from carbohydrates are small.

Hepatic lipogenesis is absent or very small in healthy subjects (55,73,74). Another study with 68% of complex carbs resulted in minimum hepatic lipogenesis (75). More studies confirmed the absence of a significant flux through hepatic lipogenesis under carbohydrate overfeeding conditions (76,77). Adipose tissue lipogenesis in both lean and obese subjects is equally small as hepatic lipogenesis (2-5g/d) under free living conditions (78).

Even with massive carbohydrate overfeeding (1000kcal) for 21 days there was only a conversion of 332 of glucose to fat from a total of 5250g (250g x 21 days), or only 6% (79). In another study with a mean energy surplus of 4.1 MJ/d (~1000kcal), carbohydrate oxidation predominated, but the RQ did not exceed 1 during any 24-h period, suggesting that despite this large energy surplus, there was no net lipogenesis (54).

Even though in the previous overfeeding study of 14 days discussed (65) de novo lipogenesis in tissues such as the liver could not be definitively determined, the calorimetry data indicated that net lipogenesis from carbohydrate did not occur.

This is no different in obese subjects with either glucose of sucrose overfeeding (50%), there is no different in lipogenesis rates in lean and obese subjects (80).

Some special conditions lead to an increase in hepatic lipogenesis, even in eucaloric conditions, such as in a diet with 10% or less energy from fat and 75% energy from carbohydrates (73,81). This contrasts with minimum lipogenesis in a diet comprised of 40% fat and 45% carbohydrates (73). Another conditions is when more than half of carbohydrates are consumed as simple sugars coupled with only 10% of fat (82).

But overall, the total increase rates are still small, lipogenesis is quantitatively small in humans, and the road less traveled (83).

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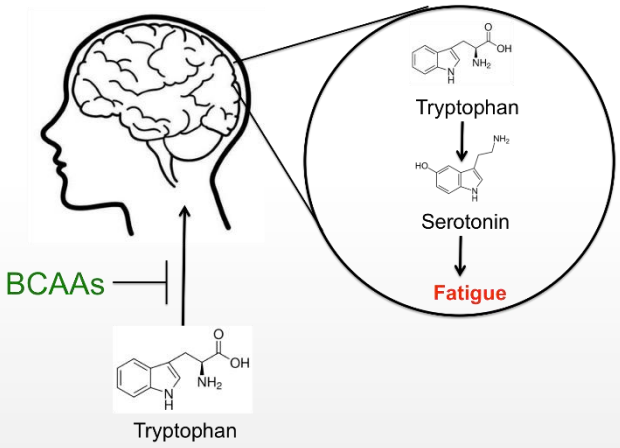
The Central Hypothesis of Fatigue: states that elevated levels of serotonin in the brain caused by increased levels of tryptophan (tryptophan is converted to serotonin) during exercise induces fatigue.

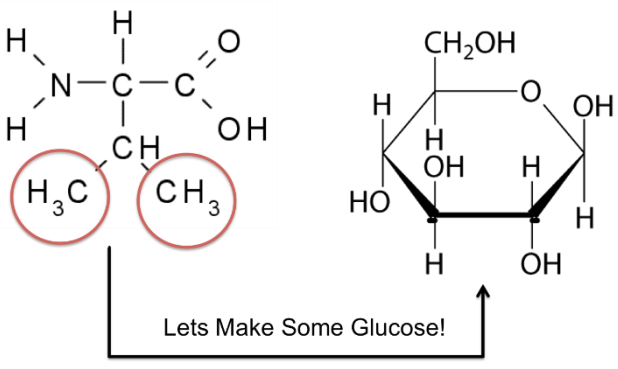
BCAAs are thought to prevent this because they compete for the same transporter into the brain. So the hypothesis is that if you increase your BCAAs in the blood by supplementation you prevent tryptophan uptake and thus reduce fatigue.



Now this sounds great as a biochemical and physiological theory. . . but unfortunately the research hasn't created any promising results and any anti-fatigue effects of BCAAs by reducing "Central Fatigue" appear to be minimal if there is any.

Valine: Glucose Creation

Valine is the least research and least well understood of the 3 BCAAs, and as such the currently known biological effects of it are minimal. Valine is a glucogenic amino acid, meaning it can create and/or be converted into glucose (1,2). The methyl carbons of valine can be utilized to produce glucose and ultimately glycogen.



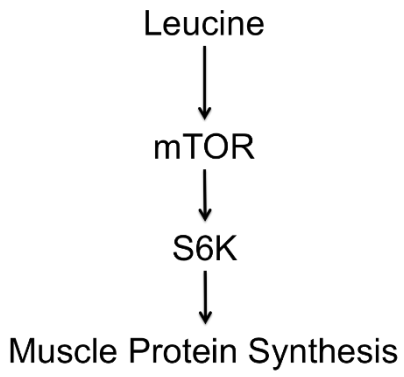
This process of valine oxidation for glucose is increased in skeletal muscle following injury, which suggests that consuming extra valine in times of muscle injury (i.e. heavy training) might be beneficial for muscle recovery. Unfortunately for valine, it is far less effective at this than Leucine (a theme that repeats itself).

Leucine: The King of BCAAs?

The main reason people use BCAAs is to optimize muscle building, and in the land of BCAAs leucine is the king of promoting muscle protein in synthesis.

When we think of the science behind muscle protein synthesis we think of two proteins, mTOR and S6K.

Interestingly, leucine is able to activate the mTOR pathway independently of other growth signals, like insulin.



A large amount of research has shown that protein supplementation can activate the mTOR pathway, and it is likely leucine is a major contributor to the anabolic capabilities of protein supplementation. For example, 5g of leucine elicits a greater muscle protein synthesis signal than 5g of a mixture of BCAAs.

There is even evidence that leucine provides the most potent growth signal when you compare leucine to other amino acids and even insulin!

Leucine for Fasted Training

Fasted training can be a valuable tool for increasing fat oxidation, and if done properly can increase GH responses. However, when done improperly cortisol levels can reach a level that shifts the balance towards catabolism. That being said, smart leucine supplementation may be able to help improve and even optimize an anabolic environment while still allowing the fat oxidation benefits of training fasted. Research studies have shown that consuming leucine pre-workout can reduce exercise induced cortisol and protein degradation. If you are training fasted, it appears that pre-workout leucine supplementation is probably a smart idea. Yes, I understand this means you are not truly training fasted, but who cares about semantics, you have muscle to build!

Leucine and Fat Oxidation

There is a saying amongst exercise enthusiasts that fat burns in a flame of carbohydrate. Well, that is like saying biceps curls are the only thing that build biceps. Just like rows and chins can build your guns, fat also burns in a flame of amino acids, not just carbohydrates. Without getting too technical, the carbon backbone of amino acids provides substrates to fuel the Citric Acid cycle (aka fat oxidation). Specifically, leucine is converted into Acetyl CoA, the major substrate that is “spins” the cycle. In fact, when you are low on carbohydrate your body will actually use a significant amount of amino acids to fuel fat oxidation.

Leucine supplementation provides the necessary ingredients to fuel the citric acid cycle and facilitate fat oxidation. Don't believe me? Read the research. Giving chronically active men 2.25g/day of leucine decreased their respiratory quotient when compared to placebo, indicating they were using more fat for fuel. Another study showed that leucine promotes energy partitioning from fat cells to muscle cells, decreasing energy storage and increasing fatty acid utilization in muscle.

Isoleucine

Isoleucine is an isomer (fancy word for meaning chemically identical but structurally different) of leucine. And like most little brothers, it is out shown by leucine in terms of its benefit. Now that being said, it does have some interesting properties we should discuss.

Isoleucine: the muscle glucose filler

Much like leucine, isoleucine can promote glucose uptake into skeletal muscle, indicating it can be beneficial for recovery. In fact, this appears to be a decent size of effect as well with some studies showing a 35%-70% increase in glucose uptake in muscle tissue. That's pretty darn interesting and worth investigating! Let us talk dosing real quick: max efficacy for increasing glucose uptake has been found to be at 72mg/kg which is about 5.7 grams for a 175 pound human.

Isoleucine: No love for glycogen synthesis

Sadly, the isoleucine induced increase in glucose uptake does not result in improved glycogen synthesis. So isoleucine pales in comparison to its brother leucine… but do not despair, because as we mentioned leucine can improved glycogen resynthesis so if you take leucine and isoleucine together you may get a synergistic effect!

BCAA Metabolites: Enter HMB

Remember way back up at the top were we mentioned that the breakdown products of BCAAs can inhibit the BDC complex and slow the breakdown of available BCAAs? Well some genius person decided it might be effective to just introduce those metabolites themselves into the system and stop the breakdown. This brings us to a rising star in the sport science world: β-Hydroxy β-Methylbutyrate (aka HMB). HMB is a metabolite of leucine and has actually been shown to prevent the breakdown of muscle tissue.

Without going too far down the research study rabbit hole, there is ample evidence to show that HMB can reduce markers of muscle injury, aid in recovery, and has anti-catabolic effects. Essentially, HMB is effective at reducing muscle-tissue breakdown at around 3g/day!

The Wrap Up

1.BCAAs have some interesting properties that support their use in athletes and in sedentary people alike with *most of the benefit coming from Leucine*.

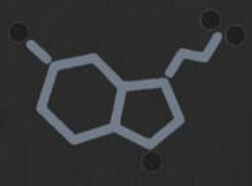
2.Supplementing with Leucine and HMB may convey some benefit to your recovery.

3.If you go with a BCAA try to get something with at least 5G of leucine in it*.

SCIENCE DRIVEN NUTRITION PRESENTS

FAT ADAPTATION AND ATHLETIC PERFORMANCE

PERIODS OF HIGH-FAT LOW-CARB MIGHT BE BENEFICIAL FOR CERTAIN PHYSIOLOGICAL ADAPTATIONS, BUT WILL LIKELY REMOVE TOP-END PERFORMANCE



Does Fat Loading Improve Performance?

By Dylan Dahlquist MS(c)

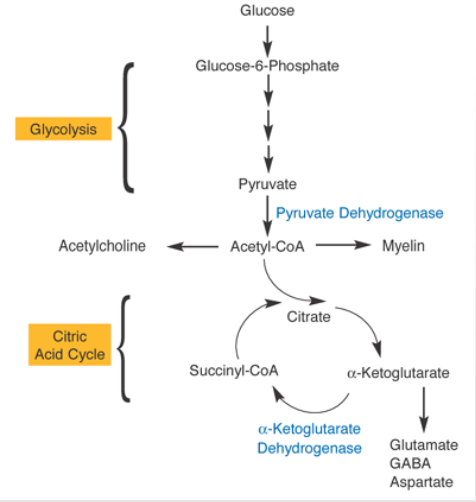
Fat for fuel in the athletic population has grown in popularity ever so more as various diets out there push the butter latent coffee, bacon and coconut oil. Various studies have begun to show that adapting to a high fat, low carbohydrate and moderate protein diet can augment AMPK activation (1), increase beta-oxidation and mitochondrial biogenesis (2).

During a period of fat adaptation, muscle and liver glycogen stores are depleted, thereafter, your ability to utilize fat as fuel is enhanced (3). So if the primary objective is to utilize fat more efficiently, fat-adaptation periods have merit.

However, when it comes to the sub-elite and professional athletic population, is a long-term fat adapted athlete actually beneficial? Or could this hinder an athlete performing high intensity exercise and/or competing in endurance based events at a very high threshold (think elite marathon runners running sub ~2 hrs)?

When you look at the amount of energy (or ATP) produced per gram of substrate, fat takes the cake, or this case bacon, over carbohydrates (CHO).

However, when you factor in intensity, as you further progress to higher and higher workloads, the rate at which one needs to produce energy accelerates at an exhilarating pace. In turn, CHO begins to trump fat as the preferred substrate for fuel. This is due in part that CHO can be oxidized into pyruvate and shuttled into the mitochondria of a cell more efficiently than that of fat. In order for this process to occur, a key enzyme is needed, pyruvate dehydrogenase (PDH).



If you decrease the activity of PDH you subsequently impair your ability to utilize both glycogen and glucose for fuel.

In order to demonstrate the above, we turn to a 2006 study that shows both the positive and negative effects of a fat adapted diet followed by the reintroduction of CHO in endurance trained individuals.

Decreased PDH activation and glyocogenolysis during exercise following fat adaptation with carbohydrate restoration

Stellingwerff and his team looked at the effects of a five day high fat, low CHO diet on performance, muscle tissue and blood biomarkers while continuously training during either a high fat, low CHO diet (HFLC) or a low fat, high CHO (LFHC) diet in trained participants.

The Subjects

7 endurance-trained male cyclists and triathletes were recruited for the study. Subjects were ~30 ± 0.7 years of age with a weight of 72.7 ± 2.9 kg and had a VO2peak of 60.7 ± 2.6 ml•kg-1•min-1.

The Dietary Intervention

Each subject went through two 7-day trials in a randomized, crossover fashion with a 2-week washout period between trials. Subjects were instructed to consume one of the two following diet protocols:

(1) 5-day fat adaptation diet followed up with a 1-day CHO refeed day all while performing supervised training sessions. On the 7th day, each participant went through a high-intensity cycling protocol after an overnight fast;

(2) Identical protocol to the above, just high CHO and low fat.

Note: Each diet was calorically matched.

HFLC diet consisted of 67% of total calories derived from fat, 18% consisting of CHO and 15% protein; where the LFHC diet consisted of 70% CHO, 15% fat and 15% protein. Fiber was matched at roughly 40g a day within a 5-10g margin

Furthermore, all the food the subjects consumed during the entire study was provided to them in prepacked containers and food diaries were checked daily to insure compliance.

Training

Training was individualized to the specific subject in order to match their training history and current training regime. Training logs were kept throughout the protocol and checked daily (similar to that of the food logs).

This is actually an awesome way to go about intervention towards training, and is highly applicable to real life scenarios because of the inter-individual variability between athletes.

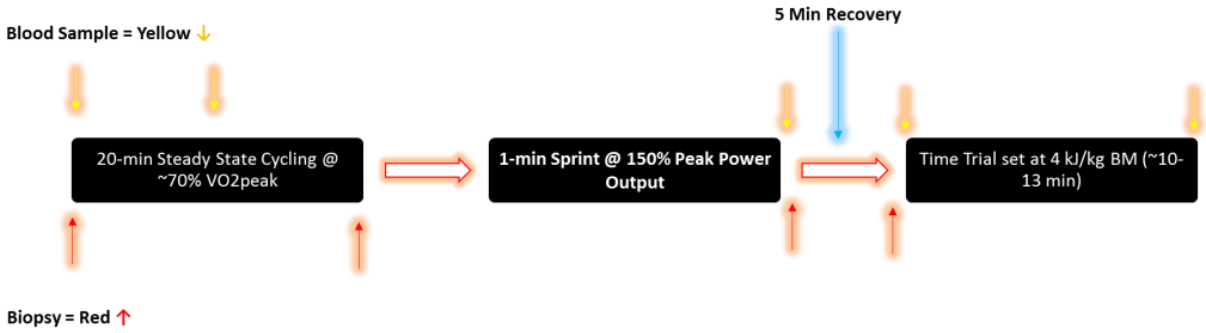


Figure Created from Stellingwerff et al (2006)

Steady State & Time Trial (TT) Performance

After a familiarization phase before the dietary interventions, subjects completed a 40-minute steady state cycling session at 70% VO2peak (assessed during the familiarization phase), rested for 1 min, and then commenced a 60 second all-out sprint at 150% peak power output followed by a 5 minute recovery and a final TT (4 kJ/kg BM) to be completed as fast as possible [see figure below for diagram of protocol.

Blood Biomarkers

Lactate, glucose, insulin, free fatty acids, epinephrine and norepinephrine were assessed at 5 different time periods throughout the experimental visit via a catheter inserted into the antecubital vein.

.

Muscle Metabolites

Four muscle biopsies of the vastus lateralis muscle for each experimental visit in order to assess the activity of PDHa, glycogen content of the muscle, HSL and various muscle metabolites (PCr, ATP, ADP, AMP, Pyruvate, G-6-P, Acetyl-CoA, and Acetylcarnitine).

Stats Baby!

The authors used a 2-way ANOVA (treatment x time) to determine significance of the finding between the treatments and the steady-state cycle. If significance was found, the authors assessed the finding further with a post-hoc. Paired sample t-tests were performed to look at the differences in muscle metabolites and blood biomarkers between dietary interventions

The Findings

First off, the authors did an impeccable job at controlling the subject's diet to the best of their abilities. The only way to further control/manipulate dietary adherence would have been throwing the subjects into a nutritional ward during the duration of the study (not very applicable). But since the authors provided prepacked meals throughout both interventions, it further increased the likeliness of adherence by the subjects.

Furthermore, dietary caloric value of both the HFLC and LFHC diets were matched calorie for calorie per day and cycling training load was identical between conditions (293.4 ± 19.9 km for HFLC and 292.6 ± 16.6 km for LFHC).

The CHO refeed day (day 6) was also matched between conditions and subjects were refrained from training on that day.

Upon CHO re-introduction, there was no difference in muscle glycogen storage between conditions prior to the exercise protocol on day 7.

During the experimental visit, the HFLC diet increased fat oxidation during the stead-state protocol by roughly ~45% and decreased CHO oxidation by ~30%, when compared to the LFHC diet. HSL activity increased by roughly 20% during this time frame as well (HSL is important in catalyzing stored triglycerides). This gives the notion that in 5 days, one can better utilize fat as fuel and spare muscle glycogen if CHO are restricted, and is supported by others (4 5).

Although there was no differences seen between the time to complete the TT, which lasted roughly ~13.18 ± 0.89 min HFLC and 13.26 ± 0.95 min LFHC, the interesting part of the study comes from the 50% reduction in PDH activity at rest even after the day prior of CHO reintroduction in the HFLC group.

Coupled with this was a reduction of pyruvate oxidation and glycogen utilization in the HFLC group. Meaning, individuals had an impaired ability to utilize glucose and glycogen for fuel even after the reintroduction of a high CHO diet the day prior.

But a question arises if this is actually a negative or a positive based on the performance measurements of the current study. If one were to only factor in the current study at hand, one might say yes. However, context becomes a virtue and if you pull up previous literature on the matter, you will find not so favorable outcomes on both high intensity sprint performances and prolonged 100-km time trials in endurance trained cyclists (6).

Furthermore, Dr. Stellingwerff himself made a great infographic showing the positive, negative and no differences seen of a low-CHO diet on athletic performance [Fig 2].

In addition to this, if we turn our attention to a few case-studies on both Olympic Level Marathon Runners (Canadians, Kenyans, and Ethiopians) and World Class Ultra-Endurance Runners (one of which won the Western States 100-mile foot race 2 times in a row from 2014 to 2015 and broke the course record), we will find a common trend regarding diet composition amongst all athletes.

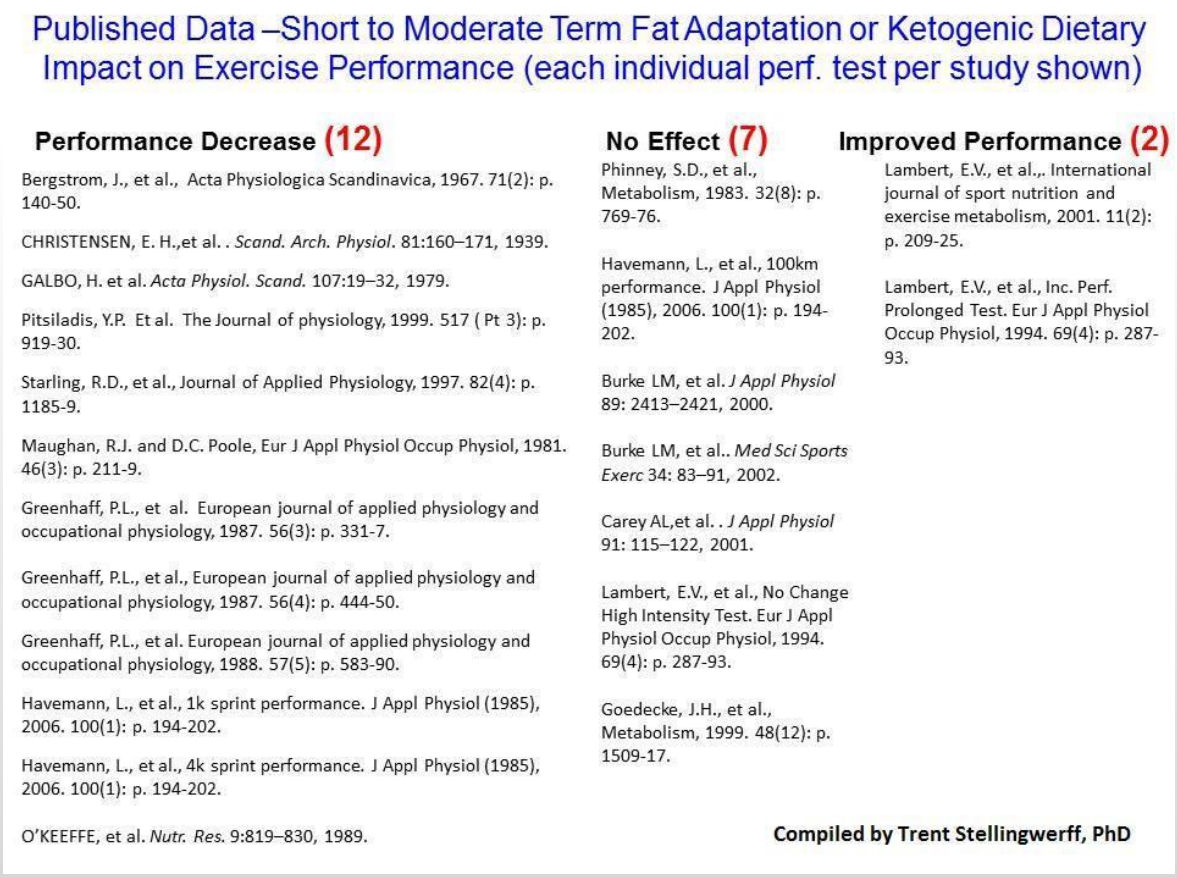
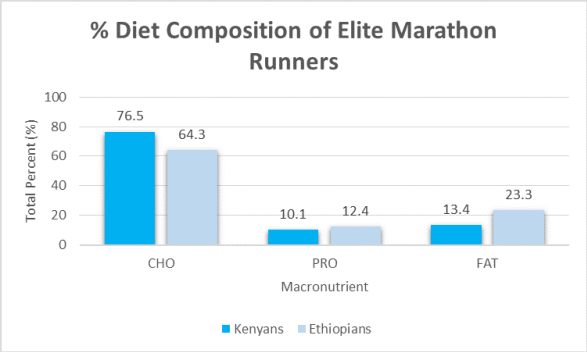
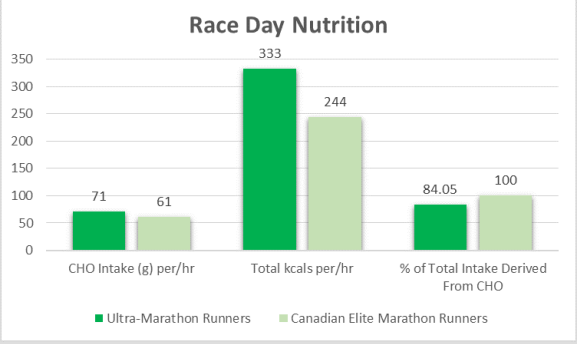


Fig 2 *Credit: Dr. Trent Stellingwerff via Twitter*

The first table below shows the dietary composition of elite Kenyan and Ethiopian Marathon runners, whom which hold the world record for the fastest marathon time recorded to date…and basically dominate this distance currently in The Olympic Games [Figure 3].



The second table will outline race day nutrition practices of both Canadian Olympic Marathon Runners and three professional Ultra-Marathon Runners [Figure 4].



The Wrap Up

In short, if you want to remove the top end of your race performance and go slower when trying to maintain intensities ≥75% [roughly] of your VO_2max, go low CHO *during* competition. However, if are trying to optimize your performance and be the best you can be, like a marathon runner trying to finish a race as fast as possible or a CrossFIT athlete trying to finish the WOD the fastest...ideally you should be consuming a good amount of CHO to fuel this both before and potentially during competition.

I'm not a LCHF zealot, and I do not believe a lot researchers out there that advocate high CHO consumption in order to improve athletic performance are either. Simply from a physiology standpoint, strategically planning a period of LCHF diet into ones training protocol for a short-duration could be extremely beneficial to further augment mitochondrial biogenesis and progress athletic prowess (**7**). I believe there is a good time for periods of low CHO availability, but based on the literature, it doesn't seem like it should be used both during or even days leading up to competition because of ones impaired ability to utilize CHO.

Author: Dylan Dahlquist

Dylan is from Ferndale, Washington US and is currently pursuing a Masters Degree at the University of British Columbia (Vancouver, BC Canada), working with Dr. Michael Koehle, Dr. Trent Stellingwerff & Dr. Don McKenzie, specializing in nutrient timing and hematology. He is a current intern for UBC Varsity Athletics, a Research Assistant for Lululemon Athletica's Innovation Team, and a Research Assistant for the Canadian Sport Institute – Pacific. When not locked in his dungeon [apartment] researching on various performance topics, he partakes in his other passions, weight-lifting, cycling (track and road), mountain running and watching an abundant amount of Dragon Ball Z.

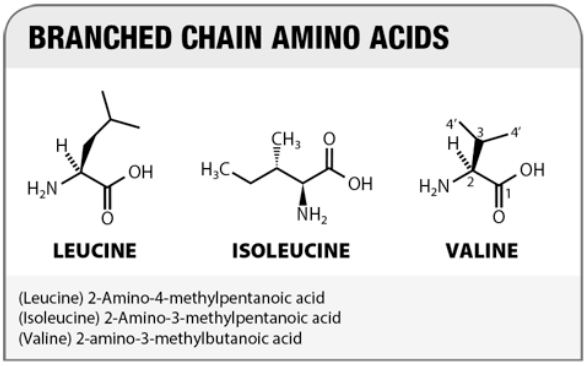


Image c/o Bodybuilding.com

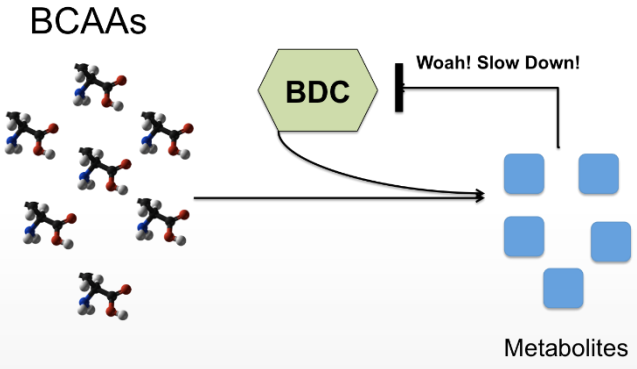
BCAAs:What are they?

Branched chain amino acids (BCAAs) are essential amino acids (meaning our body does not create them) that contain an aliphatic (branched) side-chain. These amino acids are key players in the regulation of muscle mass and must be consumed through your diet.

There are three BCAAs: valine, isoleucine, and leucine.

One really interesting aspect of BCAAs is their metabolism in the human body. The breakdown of BCAAs is regulated through an enzyme complex known as the BCAA dehydrogenase complex, which for our sake we will just call BDC.

Essentially, this means that when we have higher levels of BDC around, more BCAAs are broken down. Levels of BDC in the human body are increased when we exercise, indicating that exercise promotes breakdown of BCAAs. Another important aspect of this complex (which we will discuss later in this manifesto) is that when the metabolites of BCAAs are present (i.e. the products of the breakdown) they inhibit the BDC complex. Which means if you have a lot of BCAA breakdown products around you preserve the currently available amino acids.



The Anti-Fatigue Hypothesis: Battling Tryptophan

Before we dive into the individual BCAAs and their functions we need to cover one aspect of the collective group of BCAAs, their proposed ability to reduce fatigue.

There is a hypothesis about fatigue during training, it is called the central hypothesis of fatigue.