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CARBOHYDRATES

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➢Lecture [optional]

[Carbohydrates]

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Let's start with one of the most popular questions on carbohydrates, abbreviated to 'carbs' by all the cool kids, and along the way we'll cover the different types of carbs and their metabolic effects. Unless otherwise referenced, all the basic biology and biochemistry comes from the Advanced Nutrition and Human Metabolism textbook by Gropper et al.

Is a carb a carb when it comes to fat loss?

Are 50 grams of sugar more fattening than 50 grams of rice? Are whole grains always better than refined grains? Should you limit your fruit intake to avoid fructose overconsumption? Sure enough, not all carbohydrates are created equal. There are many methods in use to classify carbs and even terms for specific kinds of carbs.

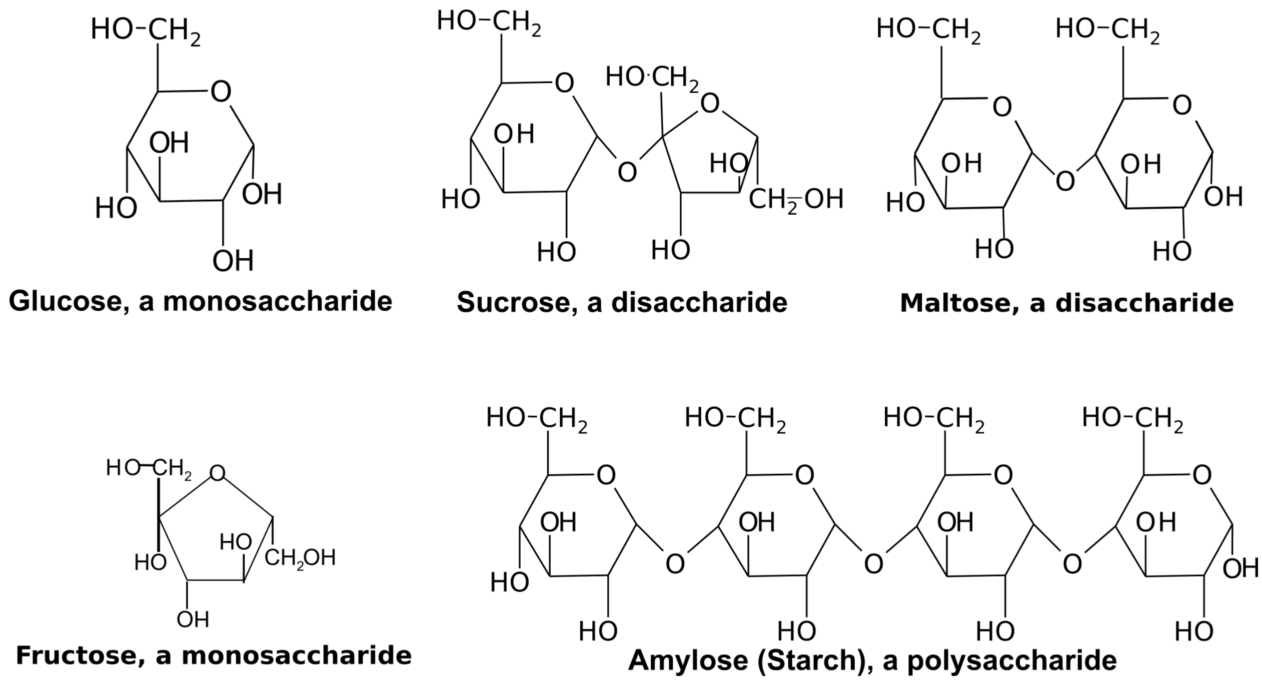
Take sugar, for example. Sugar is widely believed to be excessively fattening. That is, many people believe that calorie per calorie sugar is more fattening than other carbs. Anecdotally and even epidemiologically, it is true that people that eat more sugar tend to have more body fat. However, this relationship is confounded by energy intake. If you eat ad libitum – that is, until you're full – and you start adding sugar to your coffee or oatmeal, you are most likely going to increase your total energy intake. Sugar has very poor satiation, almost zero. It doesn't fill you up much relative to how much energy you consume. So if you add sugar to a meal, you won't eat much less of it. In fact, you may eat *more* of it because it's tastier (higher palatability). Adding sugar to your meals will thus generally increase your energy intake and we know consuming more energy than you burn can result in fat gain, especially without strength training.

What we're mostly interested in, is if different carbs are more fattening than others on a calorie- and protein-equated basis (isocaloric and isonitrogenous). Let's look at the different types of carbs and how they differ.

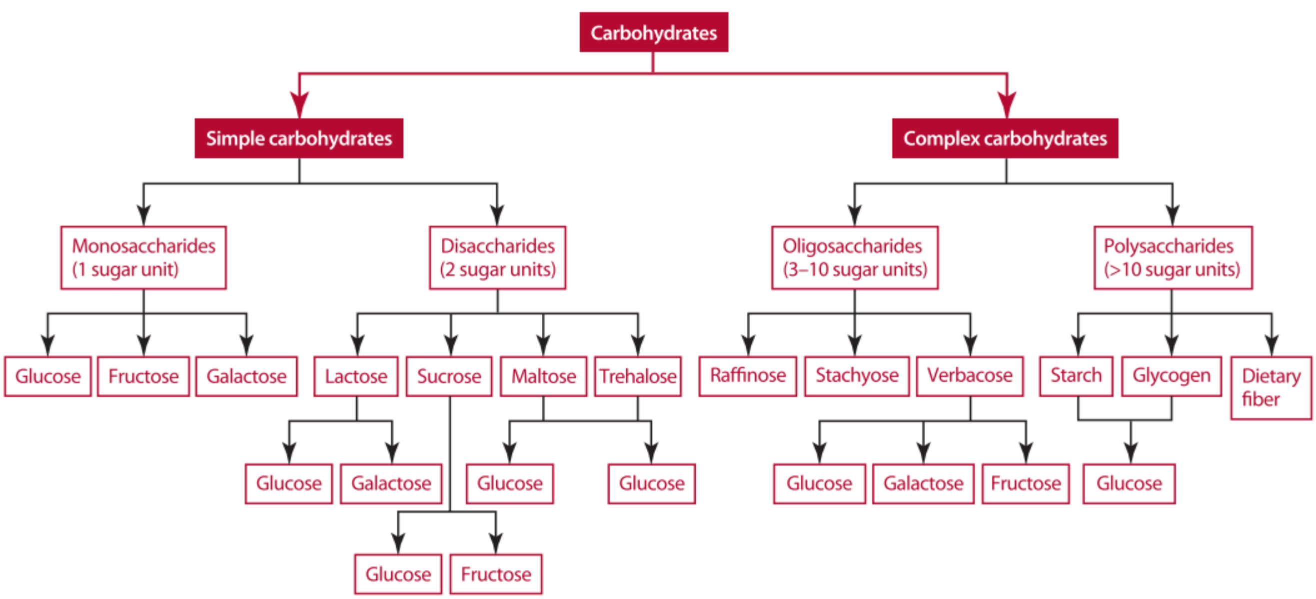
Simple vs. complex carbs

One way to distinguish carbs is by the simple/complex classification.

- Simple carbohydrates include monosaccharides and disaccharides: they have one (single/mono) or two (double/di) sugars (saccharides). Monosaccharides are structurally the simplest form of carbohydrate in that they cannot be reduced in size to smaller carbohydrate units by hydrolysis.
  Examples of simple carbs include:
  - glucose (dextrose)
  - fructose (fruit sugar)
  - lactose (milk sugar)
  - maltose (hydrolyzed starch found in beer and malt beverages)
  - sucrose (table sugar/cane sugar).
- Complex carbohydrates have three or more sugars and are called oligosaccharides (oligo meaning 'few') or polysaccharides (poly meaning 'many')."
  - Oligosaccharides are found in beans, peas, bran and whole grains. These products often make you fart more, because your digestive enzymes cannot hydrolyze all kinds of oligosaccharides: only the bacteria in your intestines can digest some of them.
  - Polysaccharides include starch, with its 2 forms amylose and amylopectin (both polymers of glucose). Starch is found in cereal grains, potatoes, legumes, and other vegetables. Amylose contributes ~20% and amylopectin ~80% of the total starch content of these foods. Glycogen, the body's stored form of glucose, is also a polysaccharide, as is cellulose.



The chemical structure of carbohydrates. More complex carbohydrates are generally just multiple simple carbohydrates linked together. Sucrose consists of glucose and fructose linked together, maltose consists of 2 glucose molecules linked together and amylose consists of multiple glucose molecules linked together.



The simple/complex carbohydrate classification.

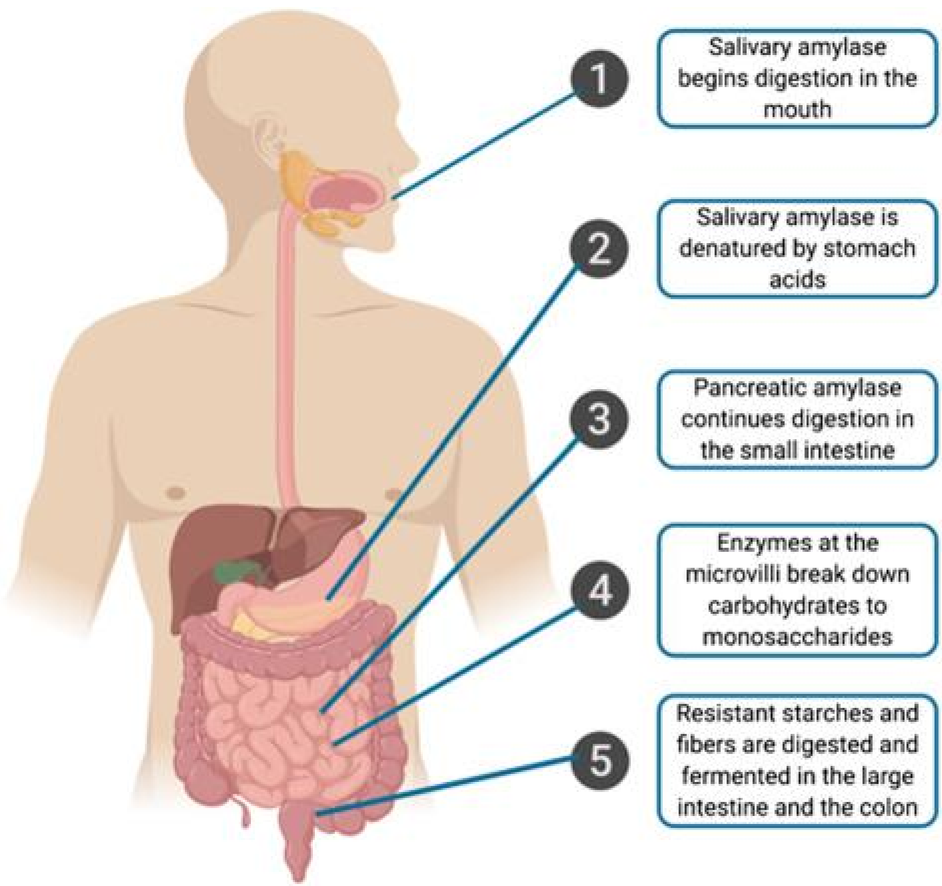
There are only 2 things that every diet expert in the world seems to agree on (and we know everyone is a diet expert these days). Vegetables are good and sugar is bad. But things aren't so black and white. While it is easy to classify simple carbs as *bad* and complex carbs as *good*, the distinction between simple and complex carbs is in fact completely arbitrary. It is merely a convention that we call carbohydrates with 3 or more sugars 'complex carbs' and we call carbohydrates with 1 or 2 sugars 'simple carbs'.

So, does it matter for a bodybuilder if a carb is simple or complex? In a 6-month study of 390 participants, one group ate a diet high in complex carbs and another group ate a diet high in simple carbs. Both diets contained the same number of calories and carbohydrates in total. There were no differences in fat loss or fat-free mass retention. The diets were also identical in their effects on blood lipids. Many other studies have confirmed that diets containing different amounts of sugar but the same number of calories result in the same body composition changes [2, 3]. Similarly, replacing part of a diet's complex carbs with simple carbs does not result in any body composition. A meta-analysis of the literature on the effects of fructose, 'fruit sugar', on body weight concluded that substituting fructose for other iso-caloric carbs does not cause weight gain. That's right: it's a myth that fructose is more fattening, calorie per calorie, than other carbs.

In conclusion, for fat loss it doesn't matter if the carbs in your diet come from simple or complex sources as long as the total carbohydrate intake is the same. To understand why simple and complex carbs have equal effects on your body composition, you have to understand the digestion of carbohydrates and their metabolic fate.

Digestion and absorption of carbohydrates

It doesn't matter if a carbohydrate is complex or simple for your physique, because your gut breaks down all polysaccharides to glucose before they reach your bloodstream (see illustration below for an overview of the digestive process). Simple carbs and oligosaccharides are all broken down to glucose or fructose. So different foods provide a different package of carbohydrates, but ultimately, they contain the same 1-2 types of fuel for your body.



The digestion of carbohydrates. *[Source]*

Lactose

If lactose ultimately all ends up as glucose just like complex carbs, why does it have such a bad reputation? Many people vilify dairy. The reason is probably indigestion. Lactase activity is high in infants, but in most mammals, including humans, it decreases a few years after weaning. This diminishing activity can lead to lactose malabsorption and intolerance. This comes with digestive discomfort, bloating and abdominal distension, which is easily mistaken for fat gain.

Lactose intolerance is particularly prevalent in African Americans, Jews, Arabs, Greeks and some Asians. Up to 75% of the world's population loses the ability to digest lactose at some point, while others have no problem with it. North-Western Europeans are rarely ever lactose intolerant, and in these cultures you correspondingly see little dairy phobia.

To ease the digestion, many types of dairy are available with added lactase, commonly but incorrectly marketed as 'lactose free'. Fermented dairy, including cheeses and yogurts, are also commonly much easier to digest for lactose intolerant individuals. We'll discuss lactose intolerance in greater detail in the course module on health sciences.

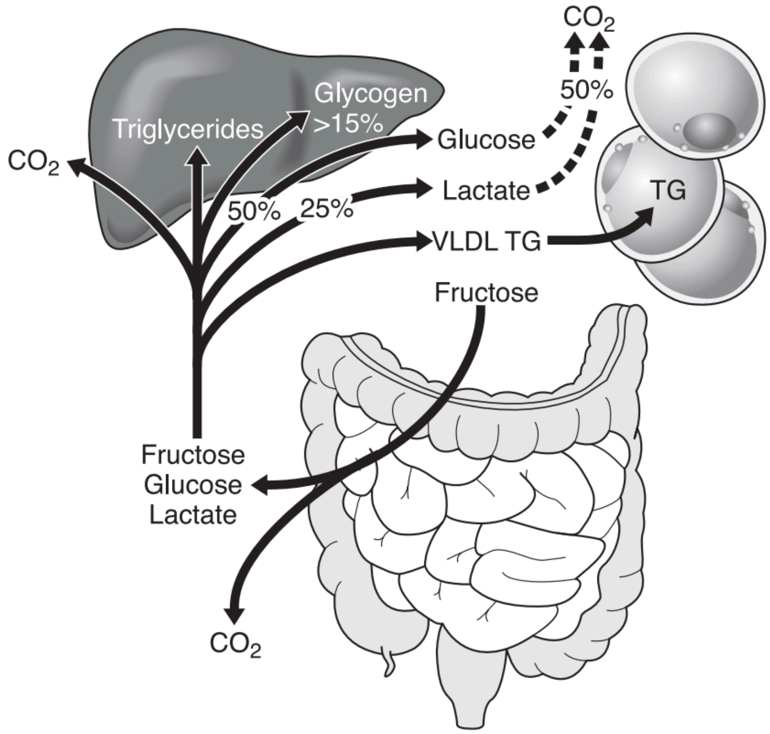
Fructose

Glucose and fructose are the 2 relevant metabolic products of the carbohydrates we eat, regardless of source. You learned what your body does with glucose: it's a preferred fuel source for many cells, notably muscle cells and the brain. What's the story of fructose?

Fructose in nature is found primarily in fruits, vegetables and honey. In modern societies, a lot of fructose consumption comes from table sugar, sucrose, which is a disaccharide consisting of glucose and fructose. Fructose is also indirectly consumed in the form of sorbitol, a sugar alcohol found in fruits and vegetables. Sorbitol is converted to fructose in the liver. Unbeknownst to many, the brain also produces some fructose itself from glucose.

Fructose is absorbed from your intestines via a specific glucose transporter protein called GLUT5 that isn't shared with glucose. The rate of uptake of fructose is much slower than that of both glucose and galactose but faster than sugar alcohols like sorbitol and xylitol, which are absorbed purely by passive diffusion. The absorption rate of fructose is increased in the presence of GLUT2, a more general monosaccharide transporter. Fructose's slow absorption is part of the reason it is unpopular with endurance athletes, who often desire rapid fuel replenishment after their workouts.

After absorption, all 3 monosaccharides (galactose, fructose and glucose) are transported into the liver by the transporter GLUT2. Some fructose is already metabolized to glucose in the small intestine. While glucose can pass through to the blood in significant amounts, most fructose is already metabolized in the liver, as many tissues cannot use fructose as an energy source efficiently. Some fructose is also metabolized by the kidneys. In the liver, fructose can have many metabolic fates. Most fructose is typically converted to glucose and subsequently stored as liver glycogen or released into general circulation. However, fructose can also be broken down to pyruvate and used as an energy source via the Krebs cycle or via fermentation to lactate. Fructose can also be indirectly converted to fatty acids (de novo lipogenesis).



The metabolism of fructose: in contrast to glucose, most fructose is already metabolized by the liver before it enters general circulation. The liver converts much of the fructose to glucose to be released into general circulation or to be stored as liver glycogen. Some fructose is converted into lactate in enterocytes (cells of the intestinal lining) and in the liver. A small portion of fructose is converted into fatty acids and can contribute to the formation of new triglycerides: de novo lipogenesis. [Source]

In contrast to what many textbooks still claim, your muscles have the GLUT5 transporter, which means [your muscles can also use fructose as an energy source]. The uptake of fructose is slow compared to glucose, however, and since it is not mediated by GLUT1 or GLUT4, it cannot be sped up by insulin or exercise. As such, glucose is the preferred fuel source for muscles. Some tissues cannot use fructose at all. [For example, red blood cells do not contain mitochondria and therefore rely on glucose to produce ATP].

Fructose toxicity

Despite the versatile metabolism of fructose, human fructose absorption is relatively poor. Around a third of people have fructose malabsorption and many people are unable to completely absorb doses of fructose over 20 to 50 g. Exceeding the small intestine's absorptive capacity of fructose results in water flowing into the intestines via osmosis. Large fructose and sugar alcohol intakes can lead to diarrhea, stomach cramps and bloating. Fructose malabsorption is not just a cosmetic or digestive problem. The accumulation of fructose and its intermediates can be toxic, causing a variety of health impairments including hypoglycemia and acidosis.

Another key health problem with large fructose intakes is de novo lipogenesis (DNL) in the liver. DNL results in an accumulation of triglyceride in the liver, which produces insulin resistance and subsequently fosters chronic inflammation. Fatty acids in the liver also cause production of very low density lipoprotein (VLDL). An accumulation of VLDL and triglyceride in the blood is a risk factor for cardiovascular disease (including clogged arteries).

Fructose's toxicity and potential for liver lipogenesis have given it a bad reputation to the point that many people fear eating lots of fruits or vegetables. This is misguided. The avoidance of fruits and vegetables poses many more health risks than fructose for strength trainees eating a whole food-based diet for several reasons.

First, co-ingesting glucose can aid passive fructose absorption via its accompanying water uptake. The relatively slow digestion of fruits and vegetables also gives the small intestine more time to metabolize fructose before it reaches the liver. As such, it's chiefly processed foods sweetened with fructose that pose a health risk. In fruits and vegetables, the ratio of fructose to glucose averages around 1:1, and it's mainly sweet plants that contain significant amounts of fructose in the first place.

Second, only daily fructose intakes over 100 grams in processed food form have a considerable potential for health complications, as they can exceed the metabolic capacity of the small intestine and liver. Some research still found no adverse health effects of diets with a whopping 150 g fructose per day for 8 weeks in healthy individuals and a 2021 meta-analysis concluded that overall, fructose is no more unhealthy than glucose, calorie per calorie.

Third, exercise provides significant protection against fructose toxicity [2]. The depletion of liver glycogen makes fructose more likely to be used to replete liver glycogen stores than be converted to lipid. Fructose can also be oxidized as fuel for muscles directly or after conversion to lactate or glucose.

In conclusion, it's completely misguided to avoid fruits and vegetables because they contain fructose. As we'll discuss in more detail in the course module on health, fruits and vegetables are generally very healthy and should be dietary staples. Fructose toxicity is generally only a concern for people that consume lots of processed foods with added fructose.

But what about blood sugar spikes and crashes?

Although practically all dietary carbohydrates end up as glucose in your blood, different carb sources can affect the rate at which glucose appears in your blood. Glucose is used by a wide variety of cell types that rely on its supply to form ATP and generate energy to keep the cellular processes active. So under normal conditions its concentration in the blood must be precisely controlled to keep all bodily processes functioning smoothly. Poor blood sugar control can lead to many health complications, in particular metabolic syndrome and type II diabetes.

However, simple carbs often don't actually spike our blood sugar levels that much more than complex carbs. The effect of food on your blood sugar is measured by the glycemic index (GI). Table sugar, due to its 50% fructose content, has a GI of ~68, which is a 'medium' effect on blood sugar. Table sugar actually has a lower GI than whole-wheat bread, which has a GI of ~71. Table sugar also has a lower insulin index than bread. (The exact values differ per publication, but the trend is consistent.)

The Glycemic Index (GI)

The glycemic index is another method of categorizing carbs that's become more popular than the simple/complex categorization of carbs. As the *GI Group* reported: "Terms such as complex carbohydrates and sugars, which commonly appear on food labels, are now recognized as having little nutritional or physiological significance. The *World Health Organization* and *Food and Agriculture Organization* recommend that these terms be removed and replaced with the total carbohydrate content of the food and its GI value. […]
The glycemic index (GI) is a ranking of carbohydrates on a scale from 0 to 100 according to the extent to which they raise blood sugar levels after eating. Foods with a high GI are those which are rapidly digested and absorbed and result in marked fluctuations in blood sugar levels. Low-GI foods, by virtue of their slow digestion and absorption, produce gradual rises in blood sugar and insulin levels."

A related quantitative measure, the glycemic load (GL), considers both the quantity and the quality of the carbohydrate in a food. The glycemic load equals the glycemic index times the grams of carbohydrate in a typical portion of the food.

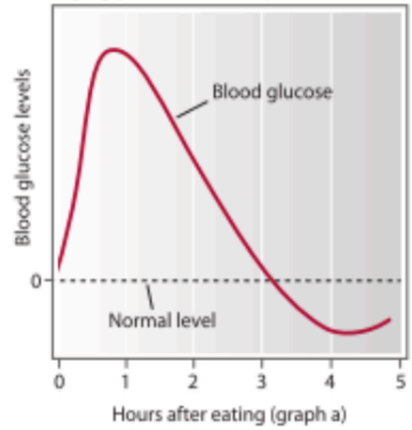
The illustrations below explain how the GI is calculated and what the GI values of various popular foods are. You'll find significant variation in these values due to differences in methodology, such as whether glucose or white bread is used as the reference value. Even the temperature of food can slightly affect its GI. Moreover, the data represent group averages that may not correspond to every individual. People's blood sugar responses are highly individual, even in the same individual at different times of the day.

Table 3.3 Glycemic Index of Common Foods with White Bread and Glucose Used as the Reference Food

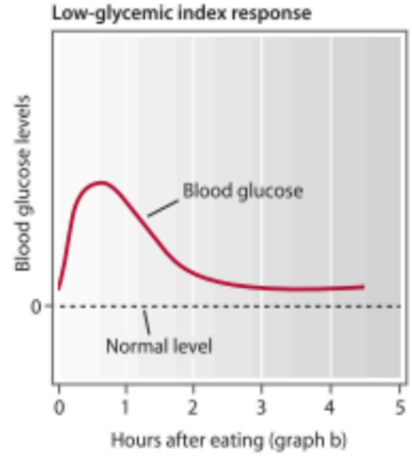
Food Tested	Glycemic Index White Bread = 100	Glycemic Index Glucose = 100
White bread[1]	100	71
Baked russet potato[1]	107.7	76.5
Instant mashed potatoes[1]	123.5	87.7
Boiled red potato (hot)[1]	125.9	89.4
Boiled red potato (cold)[1]	79.2	56.2
Bran muffin[2]	85	60
Coca Cola[2]	90	63
Apple juice, unsweetened[2]	57	40
Tomato juice[2]	54	38
Bagel[2]	103	72
Whole-meal rye bread[2]	89	62
Rye-kernel bread[2] (pumpernickel)	58	41
Whole-wheat bread[2]	74	52
All-Bran cereal[2]	54	38
Cheerios[2]	106	74
Corn Flakes[2]	116	81
Raisin Bran[2]	87	61
Sweet corn[2]	86	60
Couscous[2]	81	61
Rice[2]	73	51
Brown rice[2]	72	50
Ice cream[2]	89	62
Soy milk[2]	63	44
Raw apple[2]	57	40
Banana[2]	73	51
Orange[2]	69	48
Raw pineapple[2]	94	66
Baked beans[2]	57	40
Dried beans[2]	52	36
Kidney beans[2]	33	23
Lentils[2]	40	28
Spaghetti, durum wheat (boiled)[2]	91	64
Spaghetti, whole meal (boiled)[2]	32	46
Sucrose[2]	83	58

[1]Source for data: Fernandes G, Velangi A, Wolever TM. Glycemic index of potatoes commonly consumed in North America. J Am Diet Assoc. 2005; 105:557–62.

High-glycemic index response



Low-glycemic index response



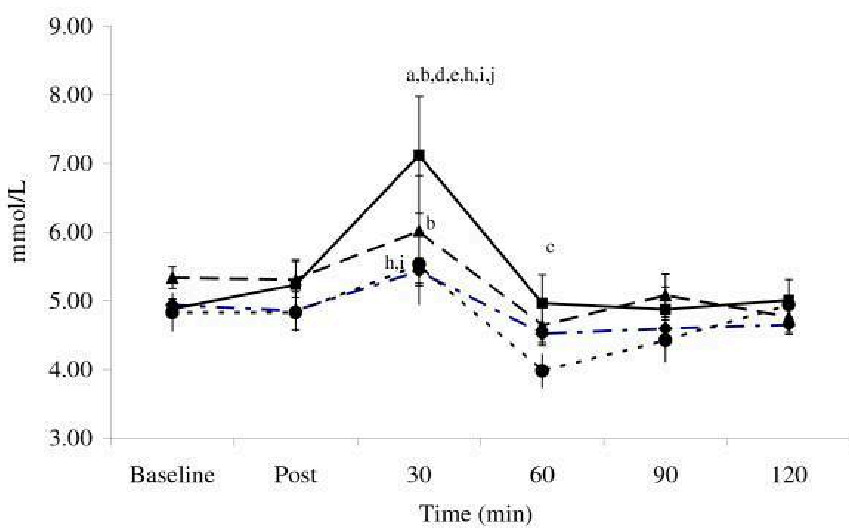
Calculation of Glycemic Index

1. The elevation in blood glucose level above the baseline following consumption of a high-glycemic index food or 50 g of glucose in a reference food (glucose or white bread). The glycemic index of the reference food is by definition equal to 100 (graph a).

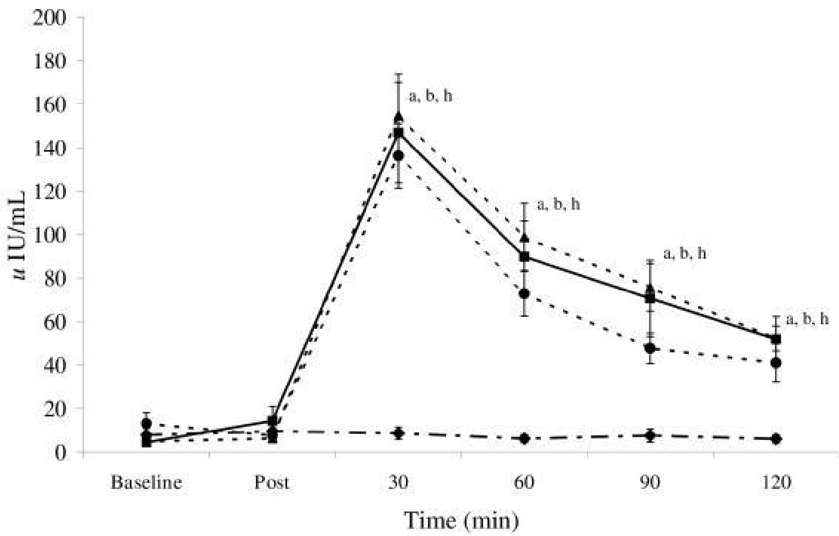
2. The elevation of blood glucose levels above the baseline following the intake of 50 g of glucose in a low-glycemic index food (graph b).

3. The glycemic index is calculated by dividing the area under the curve for the test food by the area under the curve for the reference food and multiplying the result by 100.

Another practical limitation of the GI is that the GI is based on each food being consumed in isolation, not within a meal. Other nutrients in a meal can greatly change the digestion and absorption rates. You may think the meal's GI index is just the weighted average of all its constituent foods, but there are interaction effects between the nutrients. In general, the weighted sum of a meal's GI overestimates the actual GI of meals by 22-50%. Other research has found that mixing macronutrients greatly reduces the variability in glucose and insulin responses of carbohydrate sources. For example, the differences in glucose and insulin responses between 120 g of sucrose (table sugar), honey and maltodextrin when combined with 40 g whey in a post-workout shake can be insignificant: see the figures below. So the idea that maltodextrin provides a massive glucose and insulin spike for 'rapid refueling' compared to table sugar is widely overblown in practical contexts.



Glucose excursions ('blood sugar spikes') after consuming glucose (diamond circle), sucrose (circle), honey (square) and maltodextrin (triangle). Most differences over time are statistically insignificant.



Insulin elevations after consuming sucrose (circle), honey (square) and maltodextrin (triangle) vs. a control group (bottom line). There are minimal differences between the sugars.

In fact, some research finds that different amounts of carbohydrate within the context of a balanced diet don't have any relation with blood sugar levels after eating or across the day as a whole, not even in type II diabetics. This finding would suggest a given number of calories results in similar increases in blood glucose levels in a person regardless of carbohydrate quantity, but this likely depends on what you replace the carbohydrates with, as other research like that on the GI does find the expected increase in average blood sugar levels on higher carbohydrate diets.

Theoretical limitations aside, let's look at the facts. Does the glycemic index of a diet determine its effects on your body composition?

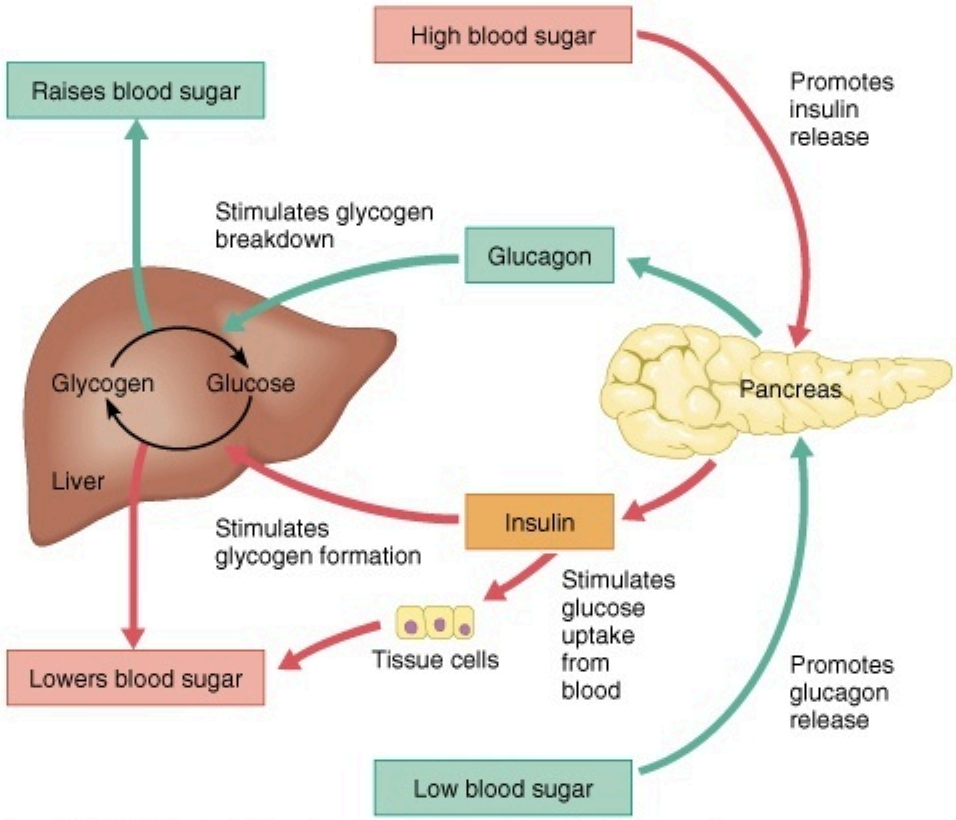
Nope. As Gaesser et al. (2021) summarized: "Results of 30 meta-analyses of RCTs from 8 publications demonstrated that low-GI diets were generally no better than high-GI diets for reducing body weight or body fat." Similar results are found in research on weight gain instead of loss. Given the same energy and protein intake, high and low glycemic index/load diets result in the same body composition change.

The blood sugar 'floods' and 'crashes' after you eat carbs are really more like gentle waves on the coastline of your blood vessels for a healthy individual. There are only around 4 total grams of glucose in your blood at any time point and blood glucose levels are maintained within a narrow range (homeostasis) by a balance among glucose absorption from the intestine, production by the liver and uptake and metabolism by the peripheral tissues.

These glucose homeostatic pathways are hormonally influenced, primarily by the antagonistic pancreatic hormones insulin (glucose storage) and glucagon (glucose release) and to a lesser extent by the glucocorticoid hormones of the adrenal cortex, like cortisol, which stimulates glucose production in the liver (hepatic gluconeogenesis).

Insulin plays a particularly central role in regulating the level of blood glucose during periods of feeding and fasting (blood sugar homeostasis). When blood glucose levels are elevated, insulin is released by the β-cells of the pancreas and release of its rival hormone glucagon is reduced. Insulin helps bring blood sugar levels back down via several mechanisms.

- Insulin inhibits the synthesis of glucose (gluconeogenesis) by the liver.
- Insulin binds with specific insulin receptors on cell membranes, particularly in muscle tissue and adipose tissue cells (fat), which causes the storage vesicles (GSV) to translocate to the cell membrane and the cells to take up glucose from the blood into their cells. Insulin basically opens a cell's floodgates to receive glucose.



Insulin and glucagon keep your blood glucose levels at a strict level.

In conclusion, healthy individuals don't need to worry about the glycemic index. If you're unhealthy, particularly if you're diabetic, the glycemic index still doesn't matter much for your body composition, but it may matter for your health. A meta-analysis found that the effects of the glycemic index on people's health markers are dependent on the health markers' initial values. Low glycemic load diets are good for your health if you're initially unhealthy, notably obese or diabetic, but in healthy populations there is no meaningful effect. This is an example of a ceiling effect. You can't fix what isn't broken, so if your body already keeps your blood glucose levels in check, there's no need to consume low-glycemic foods anymore to achieve this. There are many cultures in tropical climates thriving on diets of up to 90% carbohydrates [2, 3]. And we're not talking oatmeal and broccoli here. These cultures eat lots of sugary fruits. For example, honey is the favorite food of the Hadza from Tanzania. Evolution has made sure our bodies can deal with sugar, because it is found in many of the world's most nutritious foods: fruits. Fruit is in fact one of the foods humans have consumed for the longest period of our genetic existence. Fruit has been a staple in the human diet ever since we were still monkeys living in the jungle [2].

What about exercise performance?

Nope, not even endurance exercise performance is generally affected by the GI of your diet, including pre-workout meals [2, 3, 4]. Neither do beta-endorphin levels, rate of perceived exertion, heart rate, ventilation, lactate, respiratory quotient and substrate oxidation rate. For strength training, the GI of the carbs you eat normally makes absolutely zero difference in the gym. The only time when the GI of your carbohydrates may matter is in situations when extremely rapid glycogen resynthesis is necessary, such as during very long sports matches or competitions involving multiple bouts of exercise on the same day. At all other times, only the total amount of carbohydrate matters for performance, not its digestion speed. We'll discuss the effect of carbohydrate intake on exercise performance in more detail later in this module.

The Insulin Index

Since the glycemic index has proven to be rather useless for most purposes, researchers have created another index, the insulin index, to improve upon it. However, the insulin index is essentially equally useless as a measure of how fattening or unhealthy a food is. Many of the above studies implicitly also studied this. Replacing whole grain products like whole wheat bread with processed grains, like white bread, as was done in some of these studies, increases not just the glycemic index and load, but also the insulin index. In general, the glycemic and insulin index correlate strongly, with most differences being attributable to the fat and protein content of the foods instead of the carbohydrate content. As such, the above conclusions about the glycemic index also hold for the insulin index.

Plus, the idea that insulin elevations make us fat independent of calorie consumption makes no logical sense. Bosy-Westpahl et al. (2016) debunked the myth that insulin makes you fat as follows: "Diet-induced hyperinsulinemia may lead to a higher fat storage only at a positive energy balance. A shift in fuel partitioning towards fat storage requires improved or maintained insulin sensitivity in adipose tissue when compared with skeletal muscle." In other words, it's not as simple as 'more insulin means more fat storage'. Insulin is indeed a storage hormone, but it cannot store what isn't there and it stores nutrients in not just fat tissue but also muscle tissue. If insulin made you fat, high-protein diets should make you fat. Unbeknownst to many, it's not just carbs that spike insulin levels. Proteins do too. Insulin production after a meal is dose-response related to the leucine content of food, as leucine directly stimulates the pancreas to produce insulin. As a result, whey protein and dairy in general is highly insulinogenic, even though it has a relatively low glycemic index. And beef has an insulin index comparable to brown rice in some research. In spite of this, high-protein diets are well established to aid fat loss. Insulin cannot store what isn't there: energy balance still applies.

All of this may sound too good to be true, but sometimes you can literally have your cake and eat it too. You can get shredded without limiting yourself to rice as your only carb source. Eating sugar won't make your six-pack fade away into a tumorous gut if you watch your calories. And you certainly shouldn't avoid eating fruit or dairy because too much fructose or lactose will make you fat. That's exactly the kind of broscience that drives people into following obsessive and monotone diets that aren't healthy in psychological *or* nutritional terms.

Take-home messages

- For your body composition, it doesn't matter if a carb is simple or complex or if it has a high or low glycemic index or insulin index. Only the total amount of carbs in your diet matters and this only matters because carbs contain calories.
- For your health, the source of carbs is generally only relevant if you're unhealthy, such as diabetic. If you're metabolically healthy, the type of carbohydrate doesn't inherently matter.

Now before you go tell everyone it's ok to stuff yourself with candy, please remember that this section of the course only deals with carb sources at the level of macronutrients. Different carb sources contain not only different macros and different types of carbs, but lots of other substances as well, notably micronutrients. Fiber content also matters, as we'll discuss later. This cannot be emphasized enough. Calories from sugar may not inherently differ from calories from broccoli, but sugar still contains empty calories, whereas broccoli is packed with other stuff that's good for your health and it will satiate you much more. The sugar content and the insulin index of foods specifically are normally irrelevant for strength trainees. What matters is what else is in the food.

In sum, when it comes to fat loss, a carb is a carb.

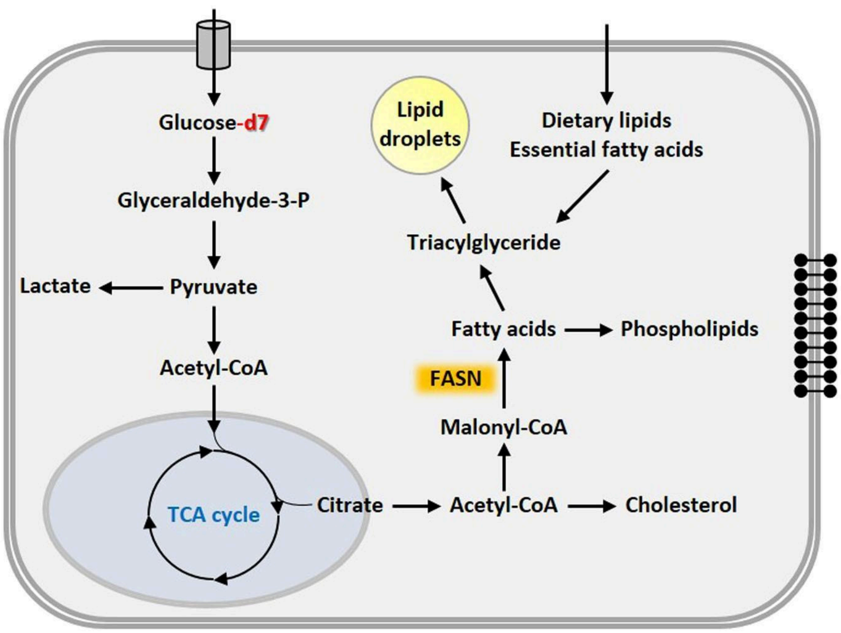
Does carbohydrate intake affect fat loss?

So far you learned that different types of carbohydrates have similar effects on fat loss. The glycemic and insulin index or whether a carb is simple or complex don't affect energy balance inherently. Only energy intake does. By that same logic, it follows that diets with higher or lower carb intakes result in similar fat loss, given the same energy deficit. Indeed, an abundance of research shows that fat loss is not affected by the ratio of carbohydrate to fat in the diet [2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12]: given equal protein and calorie intake, people lose the same amount of fat on higher- and lower-carbohydrate diets.

Interestingly, however, it appears that lower-carbohydrate diets do increase energy expenditure slightly. A 2020 meta-analysis by Ludwig et al. found that in longer-duration studies, low-carb diets resulted in slightly but consistently higher energy expenditure than high-carb diets. However, in a stricter reanalysis of the same data, Guyenet & Hall (2021) found the difference was only 70 kcal per day. Evidently, this difference is too small to significantly affect fat loss in long-term diet studies. There is also no established mechanism by which lower-carbohydrate diets increase energy expenditure. These studies carefully controlled for all energy and protein intake by providing all food to the participants. So *theoretically*, over the long run, low-carb diets should result in *a tiny bit* more fat loss than higher-carb diets with the same protein and energy intake.

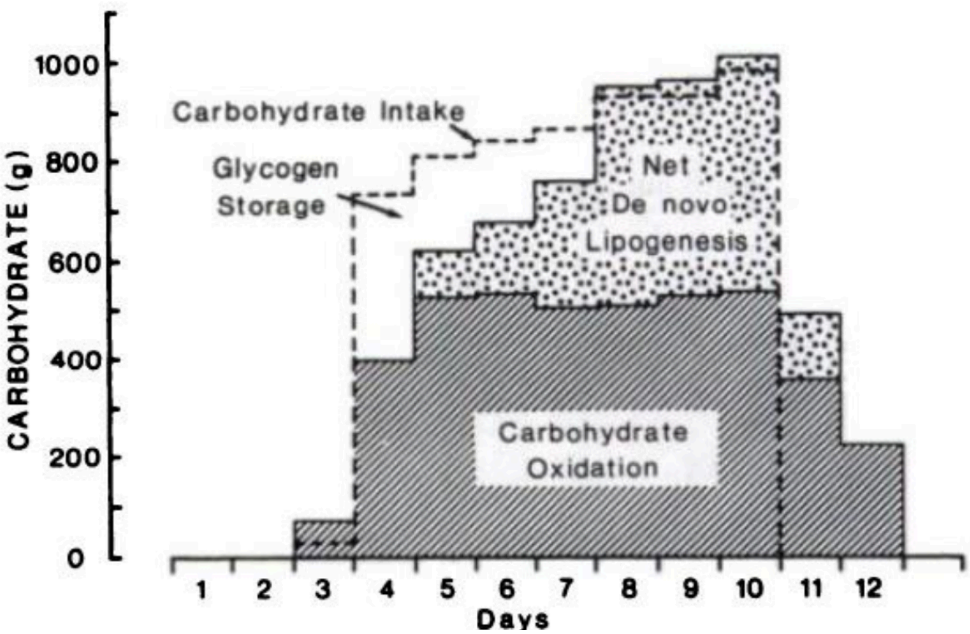
Overfeeding carbohydrates can't make you fat?

A common idea in bodybuilding circles is that overfeeding on a high-carb diet does not make you as fat as overfeeding on a high-fat diet. This is based on a misunderstanding of human metabolism. It's true that storing dietary fat as adipose tissue is a more straightforward process than converting glucose to fat and thereafter to adipose tissue, so the body normally stores fat as fat and burns carbs for energy. However, any carbs you consume and burn as energy free up more fats to be stored as fat, so carbohydrate intake contributes equally to fat gain as fat intake. When your body's fat storage rate exceeds what it can use from your dietary fat intake, it can convert the carbohydrate you consume to fat: this process is called *de novo lipogenesis* ('from anew lipid synthesis').



De novo lipogenesis inside a cell: the conversion of glucose to fatty acids (and thereafter to triglycerides or other lipids, which can be incorporated into adipose tissue, where the process also normally takes place). [Source]

Still, it's true that high-carbohydrate overfeeding results in less *acute* fat gain than high-fat overfeeding. What happens is that excess carbohydrates are first stored as glycogen before they spill over into fat storage, whereas excess fat is directly stored as fat. In the long term, however, this does not provide any advantage, since it's all stored energy. In energy surplus, a glycogen loaded individual will be more prone to fat gain than a depleted one, as they'll store all energy as fat that would go towards glycogen resynthesis in the depleted individual. In energy deficit, a glycogen loaded individual will lose less fat than a depleted one, as they'll first burn through their excess glycogen stores during exercise before they start losing more fat or will post-workout spend less energy on glycogen resynthesis. As such, overfeeding on carbohydrates results in just as much fat storage over time as overfeeding on fat when the total caloric intake is the same [2]. The image below illustrates how fat gain occurs after high-carbohydrate overfeeding.



Overeating on carbohydrates will cause increased glycogen storage, after which extra de novo lipogenesis *occurs to store the remaining carbohydrate excess. Regardless of* *whether you store glycogen or fat, it's all stored energy that must come off again later if you want to lose fat.*

If you're wondering if a huge carb refeed can be fully absorbed, the absorptive capacity of the human intestine has been estimated to amount to about 5,400 grams per day for glucose and 4,800 grams per day for fructose, so you'd have to consume well over 20k Calories before you get the 'benefit' of malabsorption. Digestion and absorption of carbohydrates are so efficient that nearly all monosaccharides are usually absorbed before the end of the jejunum in the small intestine.

Take-home message

The ratio of carbohydrate to fat in the diet normally does not directly affect long-term fat loss or fat gain: only total energy intake does.

Carbohydrate tolerance: the exception to the rule

Note: this section is highly advanced. If you're a lean strength trainee without diabetes, you can probably skip this section. If you have diabetes, PCOS or you're overweight, read on.

In the majority of individuals, at least in sedentary individuals, the ratio of carbohydrate to fat in the diet does not influence body composition change. Only total energy intake does. This is supported by a multitude of scientific studies. However, there are exceptions. What is true in the population on average is not necessarily true for each individual. Most people have normal carbohydrate tolerance: their blood sugar levels and insulin levels stay in the normal range after eating carbs and carbs don't affect their body composition independent of their calorie content. Yet some people are carb intolerant, in particular people with type II diabetes or metabolic syndrome. These people can experience various negative metabolic consequences of high carbohydrate diets. It can be hard to detect carb intolerance. If a study sample contains people with good and bad carb tolerance, on average a study may show no effect of the carb:fat ratio on weight loss success. It would take very high statistical power and a type of segmental/cluster analysis to find that there is individual variability. Since there is already marked interindividual variability in metabolic rate, diet compliance, activity level, etc. it is very difficult to find evidence of differences in carb tolerance in studies that are not specifically looking for this.

In such direct studies, there is indeed a rough correlation between insulin sensitivity and how well people do on high-carb diets. People with insulin resistance tend to lose more fat on low glycemic load diets [2, 3], adhere better to low carb diets and achieve better health outcomes on low carb diets, even when calories and protein are the same between groups (or at least self-reported to be), though these correlations are not always found.

Poor carb tolerance, specifically insulin resistance, might also impair nutrient partitioning, causing greater lean mass loss while dieting, at least in non-strength training individuals.

- Women with PCOS, which induces insulin resistance, tend to lose more fat and less muscle on a lower carb diet.
- Cross-sectional research by Wong et al. (2022) found insulin resistance in obese individuals was associated with significantly worse nutrient partitioning: increased lean body mass losses and reduced fat mass losses during diets, independent of diet composition.
- Bartholomew et al. (2024) found that individuals' insulin resistance at the start of a diet was associated with their percentage of lean body mass loss. Protein intake was supposedly controlled, but diet adherence was not reported.

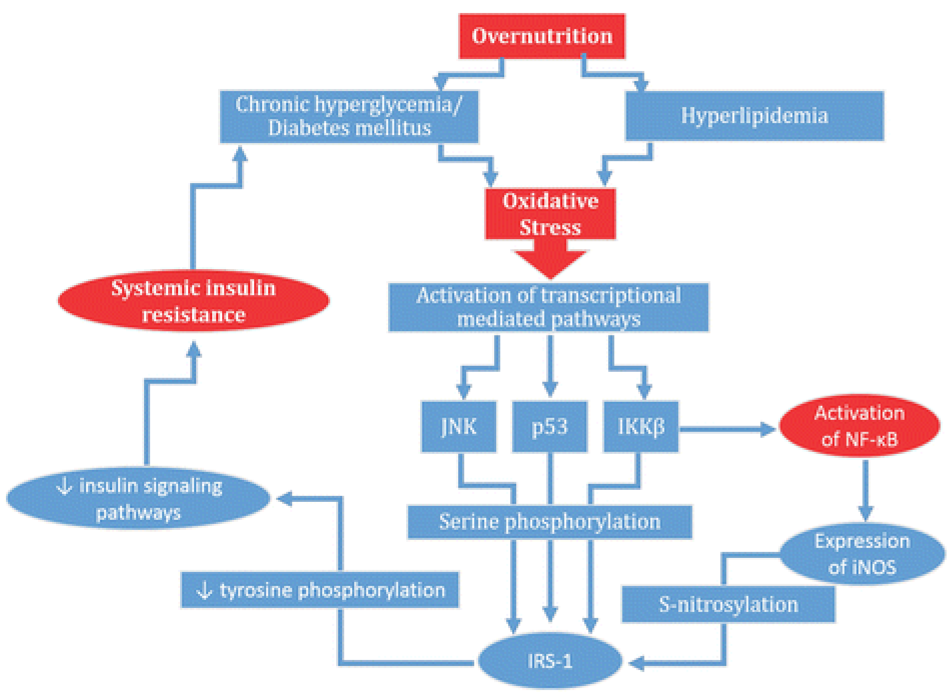
In exercising individuals, insulin resistance may also blunt training adaptations, but it seems to be mostly metabolic adaptations that are suppressed.

- Orsatti et al. (2022) found that insulin resistance predicted who would be non-responders in strength development during a strength training program in post-menopausal women.
- After aerobic exercise, insulin resistant and obese individuals show a blunted expression of genes responsible for endurance training adaptations, particularly those of mitochondrial adaptation [2, 3]. Stephens et al. (2015) found this blunted gene expression correlated with non-responsiveness to exercise during a 9-month intervention on both strength and endurance training, but unfortunately, responsiveness to exercise was defined as a decrease in body fat and insulin resistance. Muscle growth was not analyzed.

- A randomized controlled trial by Ekman et al. (2015) found that individuals with a family history of diabetes seem to have an impaired response to the training volume of endurance exercise. Exercise volume had a significant effect on $\dot{V}O_2$ peak, weight, and waist circumference in the control group, but not in the group with heredity of type 2 diabetes.
- Insulin resistance also correlates with mitochondrial dysfunction in general and Reiter et al. (2023) found that body fat percentage in healthy individuals was associated with reduced muscular energy production efficiency, which could impair work capacity.
- In contrast, Alvarez et al. (2017) found women with higher insulin resistance did not respond differently to a high intensity interval training (HIIT) program than women with lower insulin resistance in terms of fat loss or strength development. Muscle growth was unfortunately not measured.

A popular theory in traditional bodybuilding circles is that relative insulin resistance in muscle tissue may cause insulin to store more nutrients in fat than muscle. However, this would require selective insulin resistance of muscle in comparison to fat tissue, which is not normally how insulin resistance manifests. Muscle growth is unlikely to be affected as much as endurance training adaptations, because insulin sensitivity doesn't correlate with muscle growth in exercising individuals or with the ratio of muscle to fat gain during overfeeding trials (data unreported but shared with Menno by corresponding author Michael Jensen from the Mayo Clinic), including in identical twins, or with muscle protein synthesis [2]. Moreover, as you learned in the Energy module of the course, multiple studies have found that even older, overweight individuals generally don't show blunted muscle growth in response to strength training and those individuals often have some degree of insulin resistance.

Mechanistically, a high-carbohydrate/GI diet could have the following negative effects in someone with carbohydrate intolerance. The first issue is inflammation. High carbohydrate intakes, especially with a high GI, are associated with a high systemic inflammation level. Blood sugar is inherently inflammatory, because it causes oxidative stress. Oxidation forms reactive oxygen species (ROS), a type of free radical. Free radicals such as ROS destabilize other molecules by ‘stealing’ their electrons to stabilize themselves while destabilizing the other molecule. Excessively high levels of ROS without sufficient anti-oxidants to neutralize them can cause a variety of problems in the body, including inflammation. As a result, excessively elevated blood glucose levels could impair nutrient partitioning and muscular recovery by causing chronic inflammation and thereby interfering with the inflammatory signal for muscle repair



The relation between diet quality, inflammation and insulin resistance. High blood sugar and certain fatty acid levels stimulate reactive oxygen species (ROS) production, causing inflammation. Inflammation in turn causes insulin resistance by i.a. metabolic stress on the pancreas. Insulin resistance exacerbates the rise in blood sugar levels, which causes a reinforcing effect on the whole cycle. Source

Second, poor insulin sensitivity can reduce the thermic effect of food, decreasing energy expenditure. Individuals with poor carb tolerance often have a lower energy expenditure than healthy individuals with a similar body composition.

Third, since insulin has an appetite suppressing effect [2], fluctuations in insulin levels can cause similar fluctuations in hunger levels. Type II diabetics have been found to experience greater total daily fullness on low vs. high carb diets, though food choices are likely more important than total carbohydrate intake. Blood sugar fluctuations outside of the normal range can notably impair mental functioning, causing lethargy and fatigue when blood sugar levels are low. The blood sugar fluctuations can interfere with mental wellbeing and appetite control and thereby impair diet adherence, resulting in overeating.

Fourth, high-carb diets in carb-intolerant individuals could cause lethargy and reduce (activity) energy expenditure. Ebbeling et al. (2018) found that obese, insulin resistant individuals had a significantly lower energy expenditure and greater hunger, as estimated by increased ghrelin and lower leptin levels, when eating higher-carbohydrate diets with the same protein and total energy intake. The effects of high carb intakes were worse in individuals with greater insulin production in response to an oral glucose test. In the 33% of individuals with the lowest insulin secretion, there was no significant difference in energy expenditure between high- and low-carb diets, just like in most healthy individuals. In the medium insulin resistant individuals, higher carbohydrate intakes progressively reduced energy expenditure with a 264 kcal per day difference between the low- and high-carb diets. In the third most insulin resistant individuals, the difference between high- and low-carb diets was 478 kcal per day. The lower energy expenditure was partly the result of an unintentionally lower moderate-to-vigorous activity level. As we'll go into detail on later in the course, carbohydrates can stimulate both dopamine and serotonin production. These neurotransmitters are normally well regulated by the body, but if an individual has abnormal levels of either, carbohydrates can significantly affect mental performance and motivation. This can impact spontaneous physical activity and the amount of effort put into someone's training program. In support of this, large variation in the metabolic response to high- and low-carb diets has been found between individuals. Anecdotally, people with poor carb tolerance tend to get lethargic after eating lots of carbs.

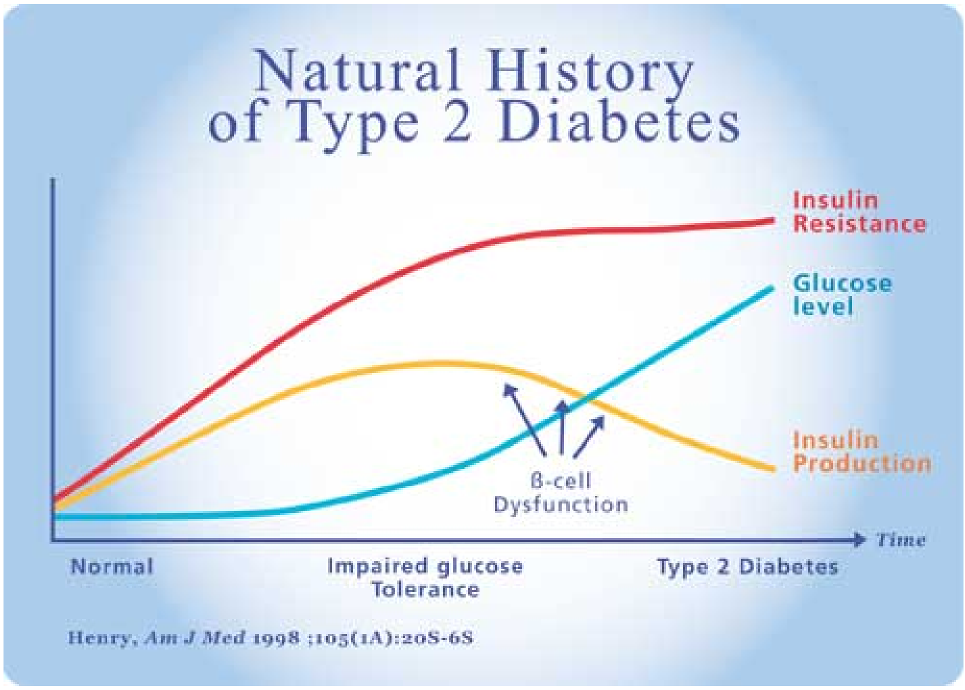
Measuring carb tolerance

Based on the above, we want to eat in line with our carb tolerance. But how do you find out if you're carb tolerant or not? Many people make this determination based on whether they prefer high- or low-carb diets, but this is not a good approach, because someone's subjective preference is not a reliable indicator for their objective carb tolerance. Allowing people to choose themselves if they should go on a high- or low-carbohydrate diet does not improve diet adherence, quality of life or weight loss success. In fact, giving people the choice of which diet to follow can reduce weight loss success. A 2018 meta-analysis looked at all studies in which participants were either put in a certain diet group by the researchers or they were allowed to choose which diet to follow. People that dieted based on their personal preference on average lost significantly **less** weight than people without a choice. Diet attrition rates were similar between groups. People's preferences are strongly colored by many other factors than their actual physiological responses, notably their beliefs and dietary preference. There are individuals with a paleo background that were indoctrinated that carbs were the poison of Satan, while in reality they had perfectly normal carb tolerance on all physical measures. Likewise, there are many fat-phobic bodybuilders that already get depressed by the idea of cutting out some carbs. They look in the mirror and see they're not as full, they give their workouts a half-assed try and immediately conclude: “I knew I couldn’t function on low carbs.” We’ll go into personal preference in more detail in the course module on psychology and compliance.

Rather than go by subjective measures, objective bloodwork is the most established test of carb tolerance. You can do a panel for type II diabetes to see if you’re likely carb intolerant. This test should ideally include the following.

1. An oral glucose tolerance test that measures fasting blood glucose and postprandial blood glucose after a high-carbohydrate test meal. This test is ideal, as it’s a very direct way to see how your body responds to carbs, but it’s not commonly done due to the extra time and effort involved, if you don’t already have signs there’s something wrong.
2. Fasting insulin and blood sugar levels.
3. A glycated hemoglobin (A1C) test, which is a measure of average blood glucose levels over the past months. Specifically, the A1C test measures the percentage of blood glucose attached to hemoglobin, the oxygen-carrying protein in red blood cells, to indicate past exposure to blood glucose.

Just measuring your insulin sensitivity is not always enough. In the 6-month CALERIE trial, insulin sensitivity did not predict weight loss in response to higher vs. lower carb diets in healthy men and women, but insulin production in response to an oral glucose challenge did. So, insulin resistant individuals didn’t necessarily lose more weight on a lower carb diet, but individuals with high insulin production did. By itself, insulin secretion is not related to fat gain though. Diets that vary wildly in their degree of insulin stimulation (insulinogenicity) can produce the same rate of fat loss, as you’ve learned. And insulin production in itself also doesn’t reliably identify if you’ll lose more fat on a low or high carb diet. It matters most if an individual’s insulin secretion is appropriate for the individual’s level of insulin sensitivity. This is what oral glucose tolerance tests measure.



The development of type II diabetes can have 2 stages of carb intolerance: at first there is insulin resistance with high insulin production but not too high blood glucose levels. Later, insulin production becomes 'exhausted' and blood glucose levels rise as a result.

It's even possible to be insulin resistant yet still do better on a highly insulinogenic diet if this makes you produce enough insulin. This scenario somewhat resembles a subclinical form of type 1 diabetes. This type of carb hyper-responsiveness is very rare, but some research found insulin resistant individuals lost more fat on a highly glycemic weight loss diet. Some other research has also found benefits of higher-carb diets in very insulin sensitive individuals. Insulin sensitive women have in a few cases been found to lose more fat on a higher-carb diet than a lower-carb diet [2]. These findings could well be explained by type I error or diet adherence issues, but there also seems to be a genetic component. Researchers have identified some of the genes responsible for carb hyper-responsiveness. The notorious 'fat mass and obesity–associated gene (FTO)' may also play a role in carb tolerance. There truly appear to be some special snowflakes among us that just don't respond to carbs the way most people do; however, it's crucial to remember these are the exceptions to the rule.

The interaction between insulin production and insulin sensitivity might also explain the anecdotal synergy between growth hormone and insulin usage reported by steroid users. The insulin compensates for the insulin resistance caused by growth hormone. Remember that insulin is a fundamentally anabolic hormone that can benefit muscle growth by increasing protein balance and the rate of glucose uptake in muscles, though supraphysiological dosages, i.e. insulin injections, are required to promote muscle growth.

Theory aside, in practice most people do not have access to readily available bloodwork. In such cases, you can use the following indicators of carb tolerance. Most of these predictors correlate reliably with insulin sensitivity. If someone has indications of poor carb tolerance, it's safest to err on the side of the higher end of the course's fat intake guidelines and thereby err on the side of a lower carbohydrate intake. However, don't compromise the intake of fibrous foods like vegetables or fibrous fruits to get more fats in. High-fiber foods are highly beneficial for all the potential issues of carb intolerance, including blood sugar control, appetite suppression and thermogenesis, so don't skimp on your veggies.

- The higher your body fat percentage, the lower your carb tolerance, generally. If you're overweight, it's unlikely you'll respond very well to a high carb diet. Body composition is generally by far the strongest predictor of carb tolerance and diabetes risk. Type II diabetes is exceptionally rare in lean individuals, whereas many obese individuals have indications of (pre)-metabolic syndrome.
- The closer your genetic ancestry is to Africa, the more likely it is that you have carb intolerance as a result of poor insulin sensitivity [1, 2]. This is probably related to the evolutionary adaptation period spent after the agricultural revolution. Asian Indians also seem to have a genetic predisposition for insulin resistance.

- Women are less likely to have problems with carb tolerance than men due to women's superior glucose and insulin metabolism.
    - Women with polycystic ovary syndrome are an exception to the rule and very often have very poor carb tolerance, so they typically fare better on low-carb diets. Anecdotally, this seems to apply to most women with medical conditions resulting in amenorrhea (loss of the menstrual cycle).
- Older individuals tend to be more prone to carb intolerance, though differences generally only become significant at a truly elderly age (65+), depending on how healthy, lean and active they are. (See the course module on the effects of age.)
- Highly trained individuals are unlikely to be carb intolerant, since muscle mass increases insulin sensitivity. (See course module on health sciences.)
- Individuals exercising daily are unlikely to suffer from carb intolerance, since exercise greatly improves insulin sensitivity acutely.
- People on androgenic-anabolic steroids (AAS) are very unlikely to be carb intolerant, as AAS improve insulin sensitivity and nutrient partitioning enormously.

Carbohydrates as fuel for exercise performance

To understand the effects of carbohydrates on exercise performance, it is important to understand how exactly dietary carbohydrates provide energy to your muscles. As you've learned, most of the starch you eat, regardless of which food it came from, ends up as glucose before it reaches the blood or any organ beyond the liver. Glucose is a key fuel source that can be used to produce energy.

Energy production from glucose

Glycolytic reactions convert glucose to pyruvate. From pyruvate, either an aerobic course (complete oxidation in the TCA cycle) or an anaerobic course (to lactate) can be followed. Aerobic means it requires oxygen. Anaerobic means it doesn't require oxygen.

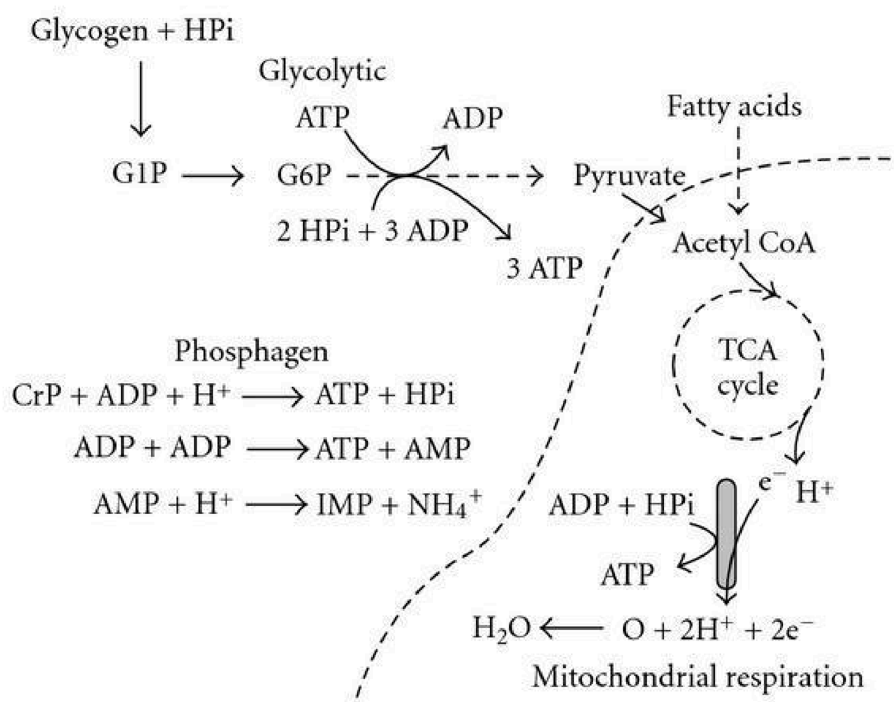
Nearly all the energy formed by the oxidation of carbohydrates to CO_2 and H_2O is released via the TCA cycle. On complete oxidation, approximately 40% of this energy is retained in the high-energy phosphate bonds of ATP. The remaining energy supplies heat to the body.

Ultimately, ATP provides the energy for all the work output of your body (specifically the terminal phosphate group). However, glucose is not the only substrate the body can use to produce ATP.

Energy production pathways

Your body has 3 pathways by which it can obtain ATP.

1. The aerobic system (aerobic glycolysis, TCA cycle, and β-oxidation of fatty acids) relying on mitochondrial respiration.
2. The ATP-CP (creatine phosphate) system.
3. The lactate system relying on anaerobic glycolysis.

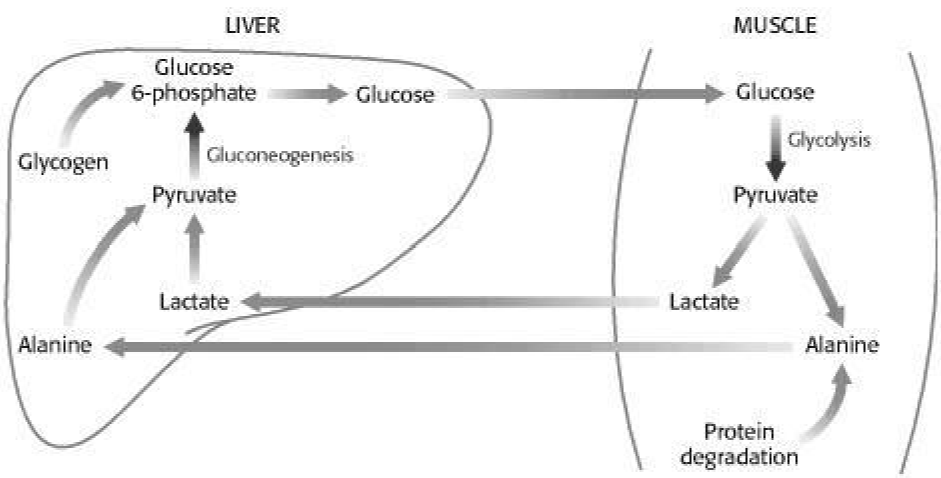


The 3 energy production pathways of ATP (Source)

The aerobic system

The aerobic system involves the TCA cycle, through which carbohydrates, fats, and some amino acids are completely oxidized to CO_2 and H_2O. During low-effort daily life activities, fats are the primary substrate, providing 80-90% of energy with only 5-18% from carbs and 2-5% from amino acids.

The oxidation of amino acids contributes only minimally to the total amount of ATP used by your muscles, because your body preferentially uses the protein you consume for protein synthesis. Significant breakdown of amino acids normally occurs only toward the end of long endurance exercise, when carbohydrate supplies are getting depleted. Amino acids can then be transaminated to form alanine from pyruvate. The alanine is transported to the liver and is a primary substrate for gluconeogenesis. This process is termed the glucose-alanine cycle or Cahill cycle. The carbon skeleton of some amino acids can be oxidized directly in the muscle.



The glucose-alanine cycle AKA the Cahill cycle.

We can estimate the ratio of carbohydrate-to-fat use with the respiratory quotient (RQ) or respiratory exchange ratio (RER). RQ is the ratio of the volume of carbon dioxide (CO_2) production to oxygen (O_2) consumption:

$$RQ = VCO_2 / VO_2.$$

The RQ varies per type of substrate used by the body as energy.

- Carbohydrate: 1.00
- Fat: 0.70. Fatty acid oxidation relies more on oxygen than glucose oxidation.

- Protein: 0.82.

The RQ tells us which macronutrients the body is using as fuel. The RQ for an ordinary mixed diet consisting of the three energy-producing nutrients is around 0.85. An RQ of 0.82 approximately represents the metabolism of a mixture of 40% carbohydrate and 60% fat. The higher the RQ, the more carbs are being burned and the lower the RQ, the more fat's being burned, assuming no significant protein oxidation. In clinical practice, an RQ below 0.8 suggests that a patient may be underfed, an RQ below 0.7 suggests starvation or ingestion of a low-carbohydrate or high-alcohol diet, and an RQ of 1.0 suggests that fat synthesis is occurring. Overfeeding can increase the RQ, because de novo lipogenesis has a RQ well above 1.0, while underfeeding, alcohol consumption and ketosis can lower the RQ due to the oxidation of body fat stores, alcohol or ketones, which increase the ratio of oxygen consumption to carbon dioxide production. Certain pathological metabolic states, such as hyperventilation and metabolic alkalosis, can skew the RQ so that it no longer accurately reflects substrate oxidation. The RQ is not of much practical relevance for strength trainees, but you'll see the measure often in research.

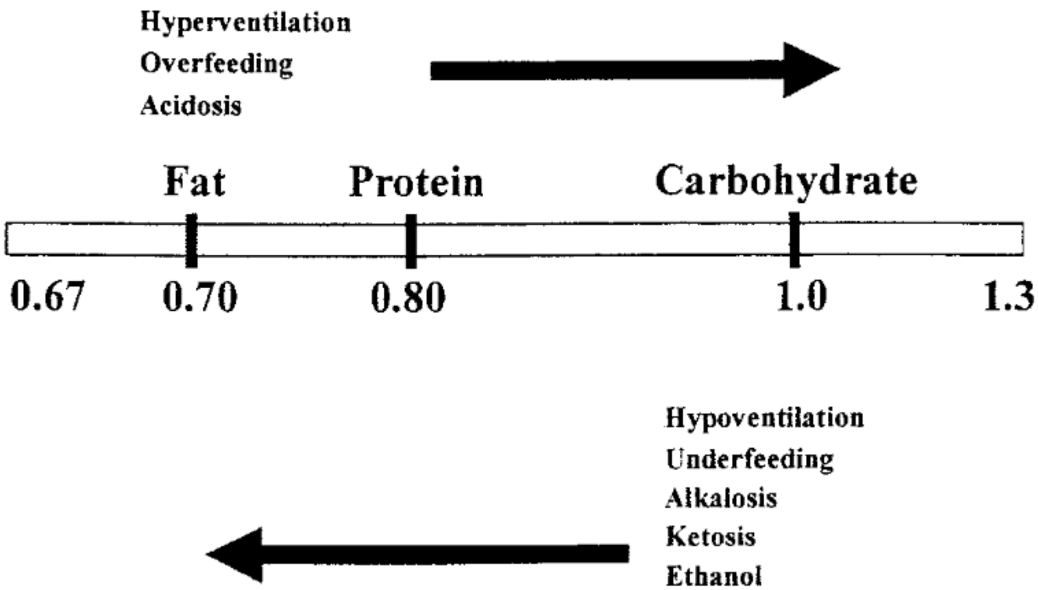


FIG. 1. Physiologic range for the overall measured respiratory quotient.

Source

The aerobic system is highly efficient in terms of how much ATP it produces per unit of substrate and is therefore the body's default energy production pathway. It has a crucial limitation, however: as the name indicates, the aerobic system relies on oxygen to function. As work increases in intensity, coming closer to maximal force output, the volume of oxygen taken up by the body also increases. VO_2 max is defined as the point at which a further increase in the intensity of the exercise no longer results in an increase in the volume of oxygen uptake. Aerobic, oxygen reliant, energy production has maxed out at this point. The intensity level of an endurance workload is most commonly expressed in terms of the percentage of the VO_2 max that it induces. For strength trainees, training intensity is better defined differently (discussed in the course topic on optimal program design), but your aerobic system is still limited by your VO_2 max.

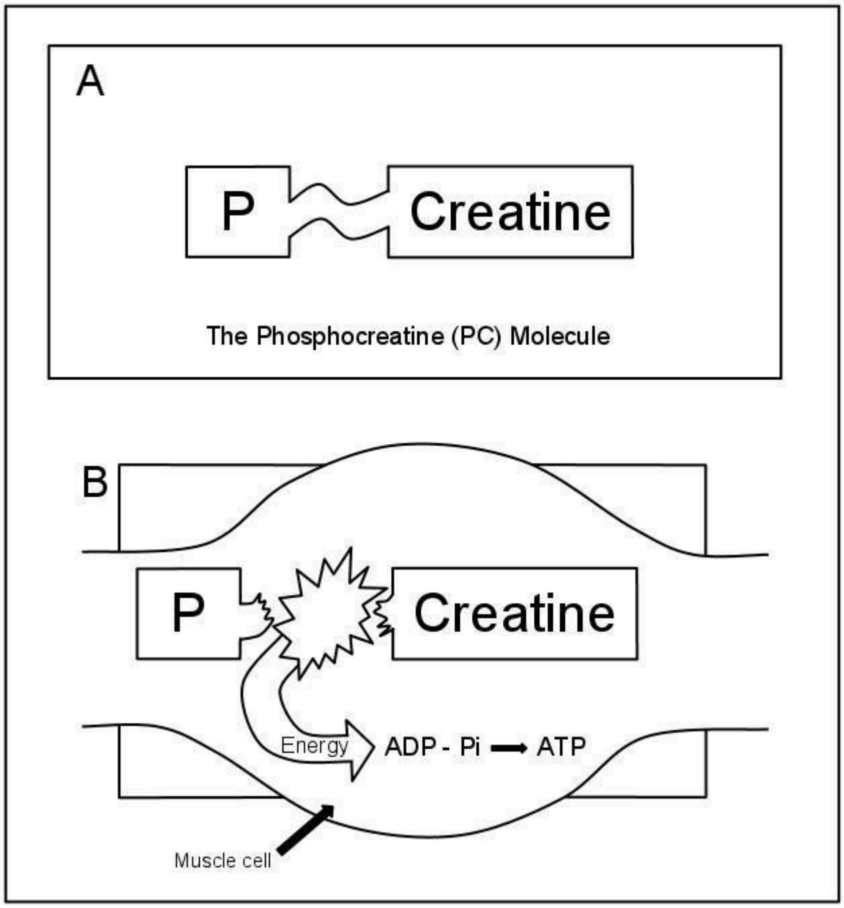
Your VO_2 max depends on your cardiovascular system's ability to deliver blood (which carries the oxygen, glucose, and fatty acids) to exercising muscle, pulmonary ventilation, oxygenation of hemoglobin, and the release of oxygen from hemoglobin to the muscle. As such, VO_2 max is a useful general measure of how endurance trained someone is.

Fatty acids have only two oxygen molecules, compared to carbohydrates' equal number of oxygen and carbon molecules. So fatty acid catabolism depends on the cardiovascular system to deliver extra oxygen. Within the exertion range of 60-75% VO_2 max, fat cannot be oxidized fast enough to provide needed energy, and therefore nearly half of the required energy must be furnished by carbohydrate oxidation. Fatty acids are the favored substrates for intensities of up to about 50% VO_2 max. As exercise intensity increases to 85% VO_2 max, the relative contribution of carbohydrate oxidation to total metabolism increases sharply. Above 85% VO_2 max, carbohydrates supply the vast majority of energy.

When your body cannot supply oxygen fast enough for the aerobic system to produce enough energy to fuel your exercise, your body must use its other 2 systems to keep up with the demand for energy production. One of these systems consists of effectively dipping into the muscle's supply of stored ATP: the ATP creatine phosphate system.

The ATP-CP (creatine phosphate) system

Our muscles have a small supply of ATP that can be used to provide a very rapid boost of energy production without any need for oxygen. More ATP can be generated rapidly by breaking down creatine phosphate (CP), as there is a lot of energy in the phosphate bonds.



The creatine phosphate system produces energy by breaking down high-energy phosphate bonds.

The creatine phosphate system is continuously active but at a very low pace, because our muscles' ATP stores are very limited. CP stores are 4-5 times greater than those of ATP, but still, most energy from this system is depleted after a mere 10-25 seconds of strenuous exercise. The ATP-CP system is thus primarily used as a 'kickstart' during high-intensity exercise before the other systems can pick up the slack.

The ATP-CP system recovers quickly afterwards, within a matter of minutes. Full recovery of phosphocreatine stores in between sets of weight training is possible within 3 minutes.

During exercise when the ATP-CP system is exhausted and the aerobic system cannot keep up with energy production demands, anaerobic glycolysis picks up to produce more ATP to sustain exercise.

The glycolytic system

This system involves the glycolytic pathway in skeletal muscle: ATP is produced by the incomplete, anaerobic breakdown of glucose through phosphorylation into lactate. This is the system in which carbs are the dominant fuel source, as this system relies on glucose for ATP production.

The glycolytic system is engaged to provide a rapid source of energy: not as rapidly as the ATP-CP system, but it has more staying power. When an inadequate supply of oxygen prevents the aerobic system from furnishing enough ATP to meet the demands of exercise, the glycolytic system will continue to function for up to several more minutes of high intensity exercise, resulting in what is called oxygen debt.

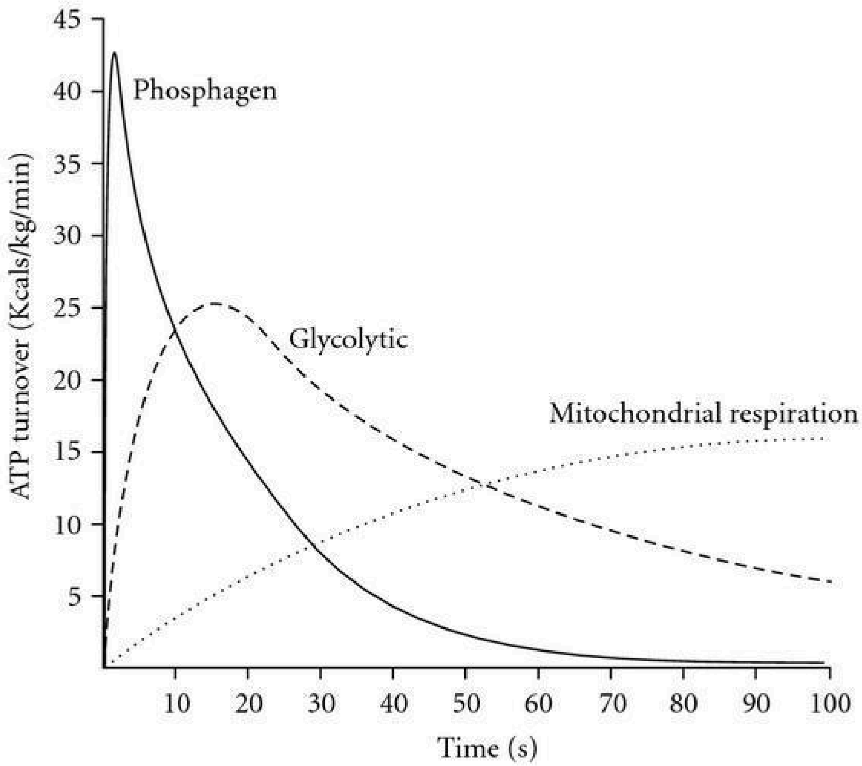
Although the lactate system becomes active as soon as strenuous exercise begins, it becomes the primary supplier of energy only after CP stores in the muscle are depleted, which occurs after 15-25 seconds of maximal energy output. As a backup to the ATP-CP system, the lactate system becomes important in high-intensity anaerobic power events that last from about 20 to 75 seconds, such as sprints of up to 800 m and swimming events of 100 or 200 m. During these events the anaerobic system (lactate system) and the aerobic system each supply about 50% of the energy at maximal energy output.

The lactate produced by this system rapidly crosses the muscle cell membrane into the bloodstream, from which it can be cleared by other tissues (including the liver) for aerobic production of ATP or gluconeogenesis. If the rate of production of lactate exceeds its rate of clearance, blood lactate accumulates. The quantity of lactate released at the start of exercise is low, but when it accumulates, it lowers the pH of the blood and this causes fatigue, eventually causing your muscles to be unable to produce more force.

Activity of the glycolytic system is strongly associated with recruitment of fast-twitch muscle fibers, compared to the recruitment of more slow-twitch muscle fibers when only the aerobic system is active. We'll get to muscle fiber type differences in detail in the training modules of the course.

Energy system interactions

The following figure illustrates the relative contributions of the 3 energy production systems as a function of exercise duration. The primary supplier of energy for activity is the ATP-CP system during the first seconds of a strenuous activity. The glycolytic pathway takes over quickly thereafter as the primary energy production pathway during high intensity exercise. The aerobic system is always as active as possible but limited by oxygen supply, meaning its contribution decreases with exercise intensity. The exact values vary per individual, particularly the individual's VO_2 max.



The contribution of energy production pathways over time (from Baker et al. (2010)).

The following data show the actual contributions from a Wingate power test, commonly used as a test of extremely anaerobic performance. It's essentially a maximal sprint on a bike ergometer. Even in this case the aerobic system still contributes about 20% of total energy demands and during the first seconds, almost all energy is produced by the CP system.

Energy sources for the Wingate test: J. C. Smith and D. W. Hill

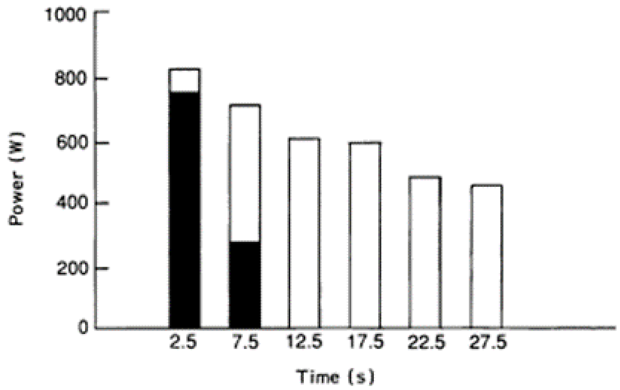


Figure 1. ATP-PC contribution (W) at each 5-s interval ATP-PC contributions are superimposed on total power output in each 5-s interval

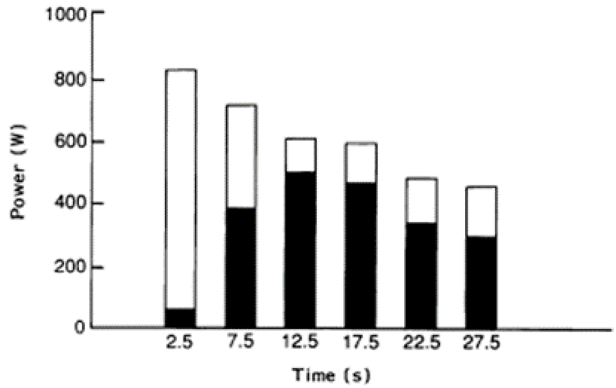


Figure 2. Glycolytic contribution (W) at each 5-s interval. Glycolytic contributions are superimposed on total power output in each 5-s interval

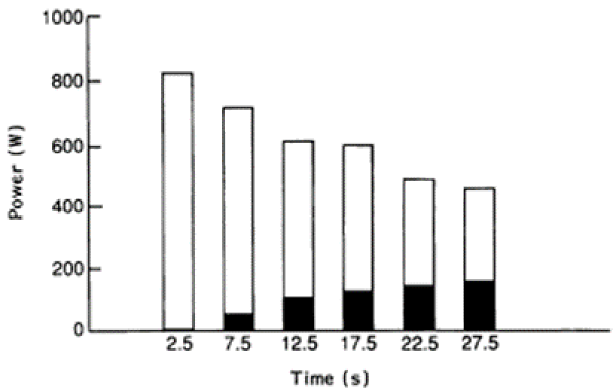


Figure 3. Aerobic contribution (W) at each 5-s interval. Aerobic contributions are superimposed on total power output in each 5-s interval

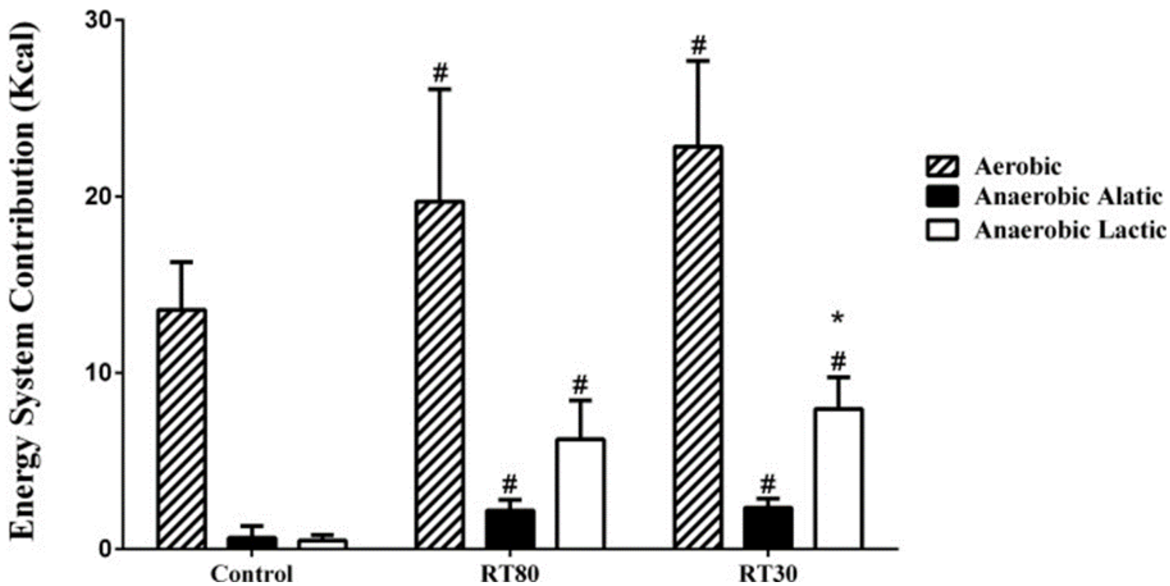
Table 1. Total power produced (i.e. 'peak power' or 'peak anaerobic power'), and peak power of the various energy systems

Energy system	*W*	*W kg⁻¹*	*Time (s)*
Peak power	819(16)	11.1(0.6)	First 5
ATP-PC power	750(14)	10.2(0.6)	First 5
Glycolytic power	497(11)	6.7(0.7)	10–15
Aerobic power	157(5)	2.1(0.3)	25–30

Values are means (s.e.m.)

Br J Sp Med 1991; **25**(4) **197**

Strength training is far more aerobic than a bicycle ergometer sprint due to the non-continuous nature of exercise and the dynamic muscle contractions, which are less energy-intensive than concentric muscle contractions (more on this later in the module on exercise selection). During strength training at 80% of 1RM, the aerobic energy contributes at least ~20-40% of energy and according to some research, much more. The data below from Brunelli et al. (2019) show the energy system contributions of 3 sets of leg extensions performed at 30% or 80% to failure in untrained individuals. Interestingly, 70% of energy was produced aerobically during this workout with the glycolytic system a distant second and the creatine phosphate system contributing the least energy but still a significant proportion.



Overall, glucose is often the primary fuel substrate for high-intensity exercise, but the ATP-CP system and the aerobic system also contribute very significantly to total energy expenditure. Your body can use the glucose you consumed via carbohydrates, but it can also create glucose from glucogenic amino acids or the glycerol backbone molecule of triglycerides.

In contrast to what you may intuitively think, when your muscles burn glucose, this is generally not the glucose that entered your bloodstream after the digestion and absorption of the carbohydrates you ate. Rather, your muscles obtain their glucose by breaking down their glycogen stores.

Glycogen vs. glucose

There are only around 4 grams of glucose in the blood, so the body has a way to store glucose for later use in the form of glycogen. The conversion of glucose into glycogen is called glycogenesis. Without glycogen, the body would be extremely reliant on having consumed carbohydrates before any kind of prolonged high-intensity activity. Glycogen is the major form of stored carbohydrate in animal tissues, located primarily in liver (~80-120 g) and skeletal muscle (~350-500 g in strength trainees).

The glucose residues within glycogen serve as a readily available source of glucose. When energy is needed, glucose residues are sequentially removed enzymatically from the glycogen chains. The breakdown of glycogen into individual glucose units is called glycogenolysis, not to be confused with glycolysis, the breakdown of glucose. **Glycogen**olysis is catalyzed by the enzyme phosphorylase.

The figure below illustrates the relative contributions of plasma glucose, muscle glycogen and fats during (endurance) exercise. Stored glycogen, not blood glucose, is the primary fuel substrate during exercise.

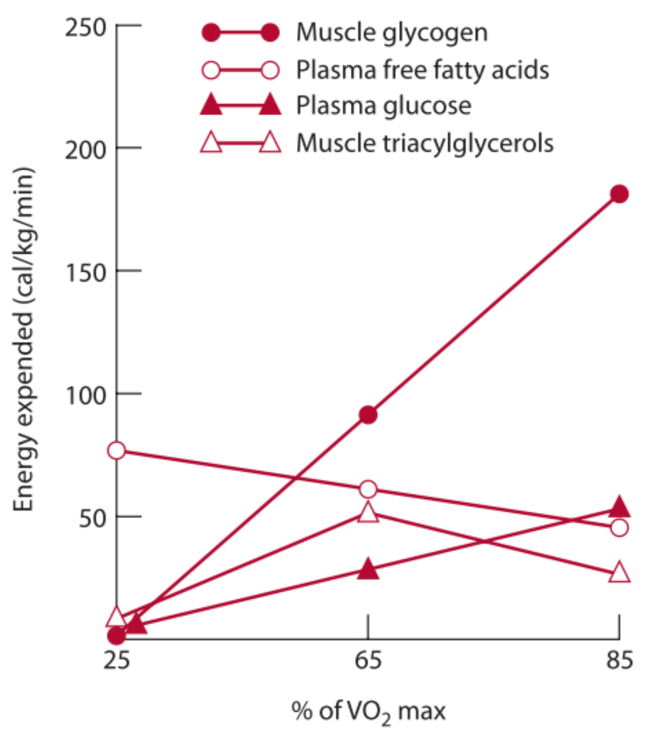


Figure 7.15 Contribution of the four major substrates to energy expenditure afte 30 minutes of exercise at 25%, 65%, and 85% VO_2 max.

Liver vs. muscle glycogen

Liver and muscle glycogen stores have a different purpose. In human skeletal muscle, glycogen generally accounts for only 1-2% of the weight of the tissue. Although the concentration of glycogen in the liver is greater at ~10%, muscle stores account for most of the body's glycogen (~75%) because muscle makes up a much greater portion of the body's weight than the liver does. The glycogen stores in muscle are an energy source within that muscle fiber and cannot directly contribute to blood glucose levels, because muscle lacks the enzyme glucose-6-phosphatase, which converts the phosphorylated glucose back to free glucose. So only the muscle itself can use its local glycogen store as fuel.

The liver does have the enzyme glucose-6-phosphatase, so liver glycogen can be broken down to glucose and re-enter the bloodstream. Therefore, it plays an important role in maintaining blood glucose homeostasis: the liver can both produce glucose as well as reduce the blood glucose level when it becomes high. Moreover, the liver is not dependent upon insulin for glucose transport into the cell, though glucokinase (*enzyme that phosphorylates glucose)* is inducible by insulin. As such, the liver is a key regulator of glycogen synthesis and storage.

Acute effect of carbohydrate intake on strength performance

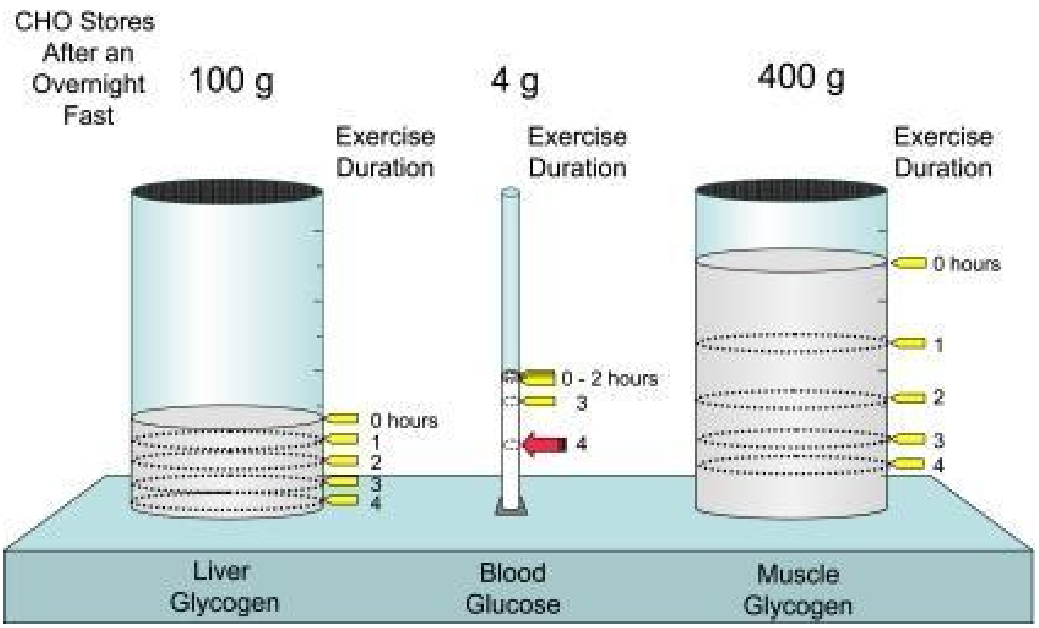
As carbohydrate is an efficient fuel source for the body, traditional sports nutrition recommendations have emphasized the need to consume carbohydrates before your workouts. While this is well supported to be beneficial for long-duration, high-intensity interval sports like soccer, traditional carbohydrate intake recommendations are most likely excessive for pure strength trainees.

Based on what you learned so far, you can estimate how many grams of carbohydrates it would take to fuel a typical weight training workout. Even assuming the lowest reported contributions of the aerobic and creatine phosphate systems you learned above, 20% and 16%, respectively, thereby assuming a 64% glycolytic contribution, and assuming a hypothetical but realistic strength training session energy expenditure of 500 kcal, this would require 500 × 0.64 / 4 = 80 g carbohydrate to fuel. Assuming 500 g glycogen storage in a typical athlete, this would theoretically amount to only 80 / 500 = 16% glycogen depletion.

Indeed, as you can see in the literature overview below, glycogen depletion levels after strength training workouts range from negligible levels to 39%. No published study has found greater glycogen depletion levels than 39% after traditional weight training. For example, Essén-Gustavsson & Tesch (1990) found only 28% quadriceps glycogen depletion in bodybuilders after 5 sets each of front squats, back squats, leg presses and leg extensions to failure at ~12RM (20 sets total).

➢ Literature overview

Glycogen depletion from strength training



General glucose and glycogen levels in the liver, blood and skeletal muscle in relation to exercise duration after an overnight fast. Source

In conclusion, muscle glycogen stores are generally not a limiting factor for energy production during strength training. Should you ever fully deplete your glycogen stores, you'll know it, it feels like you're literally unable to move. Endurance athletes call this 'hitting the wall'.

Since our muscles rely chiefly on stored glycogen, not the glucose coming into our blood from our last meal, and most strength training workouts don't deplete glycogen stores enough to impair muscular functioning, carbohydrate intake generally shouldn't affect strength training performance acutely. Indeed, a 2022 systematic review of the literature by Henselmans et al. found the majority of studies do not find any effect of carbohydrate intake on strength training performance. A 2022 meta-analysis by King et al. confirmed that there was no effect of pre-workout carbohydrate intake on strength training workout performance in the literature with control groups fasting up to 8 hours and training with set volumes up to approximately 10 sets per muscle group. For example, Raposo et al. (2011) had strength-trained women perform 5 sets of bench presses at 75% of 1RM and 5 sets of leg presses at 85% of 1RM. On one occasion, they performed the workout after an overnight fast. On another occasion, they performed the workout after consuming 81 grams of carbs (randomized cross-over). There was no significant difference in how many reps they could do on either exercise or their total training volume loads. As another example, Smith et al. (2017) had strength-trained men perform 10 sets of presses and 10 sets of rows at 65% of 1RM once after an overnight fast and once after consuming 36 g carbs. There was no significant difference between conditions in repetitions to failure for either exercise.

Moreover, our paper and King et al. (2022, 2024) found no dose-response effect of carbohydrate intake. There was no trend of higher carbohydrate intakes to result in more positive findings, not in the literature as a whole or in any study measuring 3+ different carbohydrate intakes. For example, Krings et al. (2016) studied the effect of 15 grams per hour, 30 g/h and 60 g/h of carbohydrates vs. placebo on strength training, running and jumping performance. The highest total repetition numbers were achieved by the *lowest* carbohydrate intake group (15 g/h). However, all carb groups showed some evidence of outperforming placebo, so combined with the other studies showing fasted training isn't optimal, we recommend consuming at least 15 grams of carbohydrate within 3 hours of your training sessions. As per the protein module, you should also consume at least 0.3 g/kg protein.

➢ Literature overview

Acute effects of carbohydrate intake on strength performance

There are multiple studies that do find significant effects of carbohydrate intake on strength training performance and they generally favor the higher-carbohydrate conditions, but they all had a crucial limitation: none of them had isocaloric control groups. Thus, they only show consuming something before your workouts is better than nothing. This is very useful to know, because it means you shouldn't train fasted. However, outside of fasted training, the practical question for most people is if you need to fill your macros specifically with carbs and if you need to consume carbs pre-workout. If we look at the limited isocaloric research on the effect of acute carbohydrate intake, there is no effect. For example, Lynch (2013) had strength-trained men perform a ~17-minute high-intensity weight training workout. Post-workout, they consumed either a high-carbohydrate shake (68 g carbs) or an isocaloric high-protein shake. Two hours later, they performed a test workout of agility T-tests, push-ups to failure and 40-m sprinting. The high-carbohydrate drink resulted in *worse* performance than the high-protein drink on aggregated test performance. In another study by Welikonich (2010), a high-carb shake with 89 g carbs improved leg press performance identically compared to a shake with 79 g carbs and 21 g protein.

Some research even suggests the performance-enhancing (ergogenic) effect of consuming something rather than nothing is psychological and it has nothing to do with macronutrient intakes. Mears et al. (2018) found that a high-carbohydrate breakfast improved high-intensity endurance exercise performance compared to water. However, a texture-matched placebo without any carbs increased performance just as well as the carbs. Thinking they had consumed a meal rather than actually having consumed a meal improved performance, indicating that it was merely the perception of having consumed energy rather than an actual effect of carbohydrate on energy production that increased performance. Naharudin et al. (2020) replicated the placebo effect of breakfast on performance in strength trainees. A calorie-free but texture-matched placebo breakfast improved squatting performance (sets of squats totaling around 40 reps) just as well as a high-carb breakfast. Both increased performance compared to having consumed only water, indicating the perception of having consumed energy induced a significant placebo effect on performance. In the latter study, both groups also experienced a similar reduction in hunger, so it's possible that the negative effects of training fasted are partly due to hunger: feeling hungry and weak may reduce training efforts, even though there is no physical shortage of energy in the muscles. In support of hunger being more important than energy intake, a follow-up study from Naharudin et al. (2021) compared two isocaloric breakfast meals, a semi-solid one vs. a liquid one. The semi-solid meal reduced hunger more and improved back squat repetition performance more than the liquid meal, suggesting hunger suppression can have a positive effect on resistance training performance.

So is carbohydrate intake completely irrelevant for strength trainees? No, there are 2 scenarios in which carbohydrate intake may be important [2]:

1. When performing over 10 sets per muscle group.
2. When training twice on the same day with a high training volume.

In these scenarios, glycogen depletion may reach critical levels in some muscle fibers. Hokken et al. (2020) found that a whole-muscle glycogen depletion level of 38% after 12 sets of quad training was associated with approximately 50% subcellular depletion specifically within type II muscle fibers and almost complete depletion in half of the type II fibers. While this still wasn't complete depletion, glycogen depletion can start to impair neuromuscular functioning when levels are reduced to approximately 250-300 mmol/kg dry weight, which is roughly 50% depletion for typical strength trainees. High-volume resistance training could thus deplete some type II muscle fibers sufficiently to impair performance even though most muscle fibers still have ample glycogen. However, Koopman et al. (2006) found fiber-specific glycogen depletion was limited to 40% in the IIa and 44% in the IIx fibers after 16 sets quadriceps resistance training at 75% of 1RM, although glycogen content was estimated rather than directly measured. Also, the participants in Hokken et al. were weightlifters and powerlifters with a pre-exercise glycogen storage level of 92 mmol/kg wet weight, which is relatively low for strength trainees in a fed state. Strength trainees generally have resting glycogen stores around 120 mmol/kg. It's possible the powerlifters and weightlifters were unaccustomed to high-volume 'bodybuilding style' workouts. Moreover, it remains to be determined if pre-workout carbohydrate intake can in fact prevent glycogen depletion from reaching critical levels during very high-volume training. In Hokken et al.'s study, the participants consumed a mixed meal with 63 g carbohydrate and 560 kcal within 90 minutes of the workout and glycogen levels still reached critical levels in some of the type II muscle fibers. In Koopman et al.'s study, the participants did not reach the same level of depletion despite training after an overnight fast. Other factors, notably pre-exercise glycogen storage levels, may be more important than acute carbohydrate intake to prevent performance-limiting levels of glycogen depletion.

If training a muscle with 16 sets in a single workout can indeed induce critically low glycogen levels in some muscle fibers, a high carbohydrate intake before the workout may be needed to prevent this. Indeed, if we specifically look at studies measuring performance in workouts with more than 10 sets per muscle group, the majority finds at least trends in favor of higher carb intakes.

- Haff et al. (2001) found greater total work output on a carb intake of 143 grams compared to a zero-calorie placebo during 16 sets of isokinetic leg extensions, although there was no effect for leg curls.
- Lambert et al. (1991) investigated how many sets of 7 reps of leg extensions at a supposed 80% of 1RM strength trainees could do on a carb intake of 125 g compared to a zero-calorie placebo. They could do 17 sets with the carbs vs. 14 without them, which was almost statistically significant (p = 0.06).
- Welikonich (2010) investigated how many sets of 8 reps of leg presses strength trainees could do on a carb intake of ~60 g vs. a non-caloric placebo. Total repetition volume was greater on 89 g carbs than placebo with 135 reps vs. 95

reps. However, a group consuming 79 g carbs and 21 g protein achieved identical results to the higher-carb group.

In contrast, Dalton et al. (1999) found strength trainees could perform just as many repetitions on 15 sets of quad training and 5 sets of bench presses after an overnight fast as after a shake with 1 g/kg carbs. The workout consisted of 5 sets each of bench presses, squats, leg presses and leg extensions at 60-80% of 1RM. Similarly, Mitchell et al. (1997) found strength trainees could perform just as many reps while they were on a high-carb diet with 643 grams per day as while they were on an isocaloric low-carb diet with 32 grams per day during a workout of 15 sets of quadriceps strength training (5 sets each of squats, leg extensions and leg presses) at 15RM to repetition failure, even though they had performed an exhaustive bicycling glycogen depletion workout 48 hours earlier.

If you train twice on the same day, it's also plausible your performance in the second workout is going to suffer on a low carb intake, as resynthesis won't be complete in time after the first workout. Two studies show consuming carbohydrates in between bidaily workouts can improve performance.

1. Haff et al. (1999) had strength-trained men train twice on the same day. The morning session consisted of 15 sets of high-rep, submaximal squats to deplete glycogen stores. Four hours later, they performed as many sets of 10 squats with 55% of 1RM as they could either fasted on one occasion (both groups did have a meal 2.5 hours before the first workout) or while basically continuously drinking carbs (~3.2 g/kg carbs in total) at another occasion. On the massive carbohydrate intake, they could perform 19 sets compared to 11 in the fasted state. Of course, it's questionable if it was the carbohydrate intake per se that really mattered here or just the massive difference in energy intake to fuel

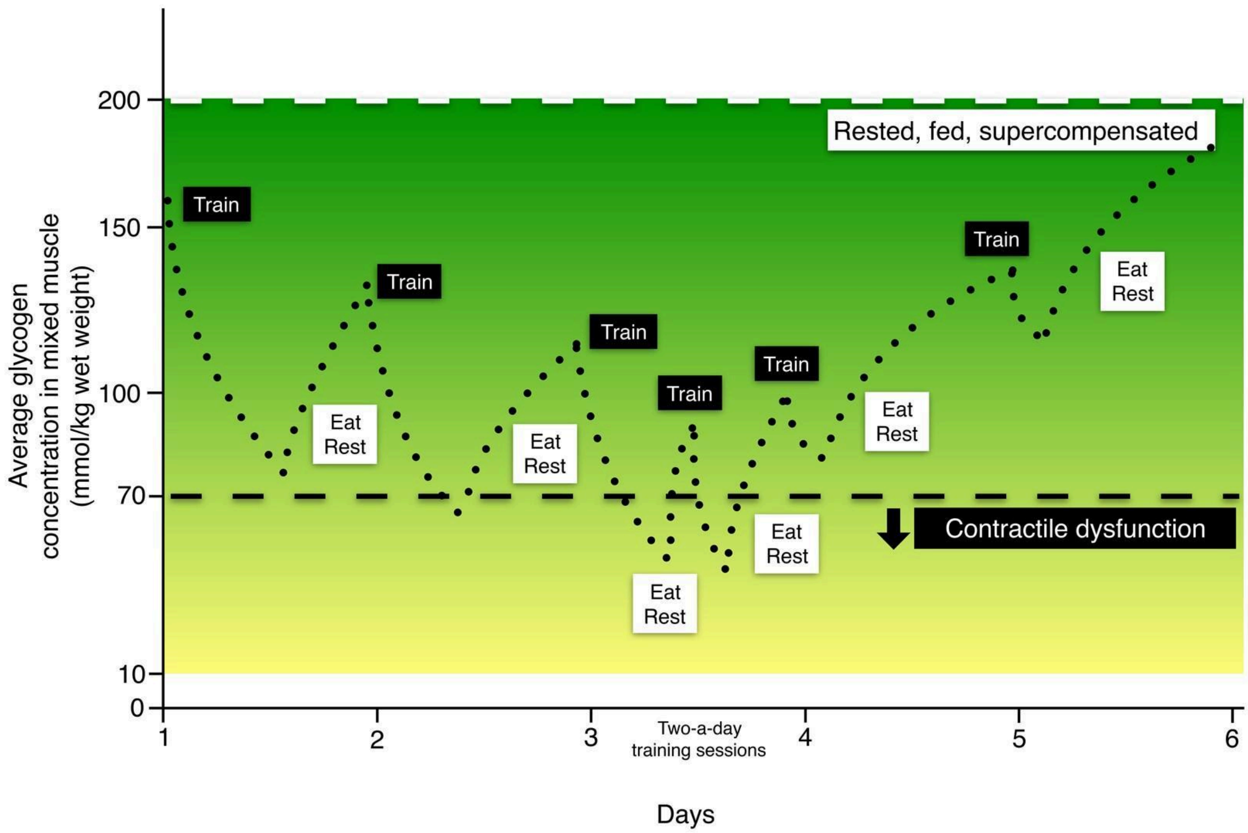
recovery in between the 2 workouts. It's also worth noting that the difference in total work output did not strictly reach statistical significance (p = 0.07).

2. Oliver et al. (2016) had strength-trained men perform 5 sets of 10 squats as explosively as possible 2 hours after an exhaustive glycogen depletion bicycling workout, either completely fasted overnight on one occasion or with 106 g carbs after the depletion workout on another occasion. Average power outputs were higher after the high-carb meal, although differences in average force output were 'trivial'.

In sum, carbohydrate intake per se generally does not acutely impact strength training performance via physiological mechanisms in a fed state. Our muscles rely primarily on previously stored glycogen rather than incoming blood glucose to fuel performance and glycogen stores are generally not depleted below critical levels during strength training workouts. Still, to be on the safe side, it's advisable to consume at least 15 g net carbs and 0.3 g/kg protein within 3 hours of your workouts. If you perform workouts with well over 10 sets per muscle group or bidaily workouts, you may deplete glycogen stores to performance-limiting levels in some muscle fibers. Thus, you may want to consume more carbs beforehand, but as we'll discuss later, this programming structure is generally not advisable anyway.

Effect of long-term carbohydrate intake on strength performance

Carbohydrates are rarely acutely needed to sustain energy production during strength training because of the modest glycogen depletion. However, repeated exercise can deplete glycogen stores over time if there is not enough resynthesis in between workouts, as illustrated below.



Repeated glycogen depletion over time may cause levels to fall to performance-limiting levels when there is insufficient glycogen resynthesis in between workouts. Data on endurance training. Source

To maximize glycogen resynthesis, muscles need glucose and insulin. Before glucose can be taken up by a cell, it has to cross the plasma membrane of the cell via a transport system. The family of protein carriers involved in the transport of glucose are called glucose transporters, abbreviated GLUT. Different tissues use different GLUTs. GLUT4 transports glucose into muscle and adipose tissue. GLUT4 is stimulated by insulin: insulin translocates the preformed GLUT4 from intracellular vesicles to the cell membrane.

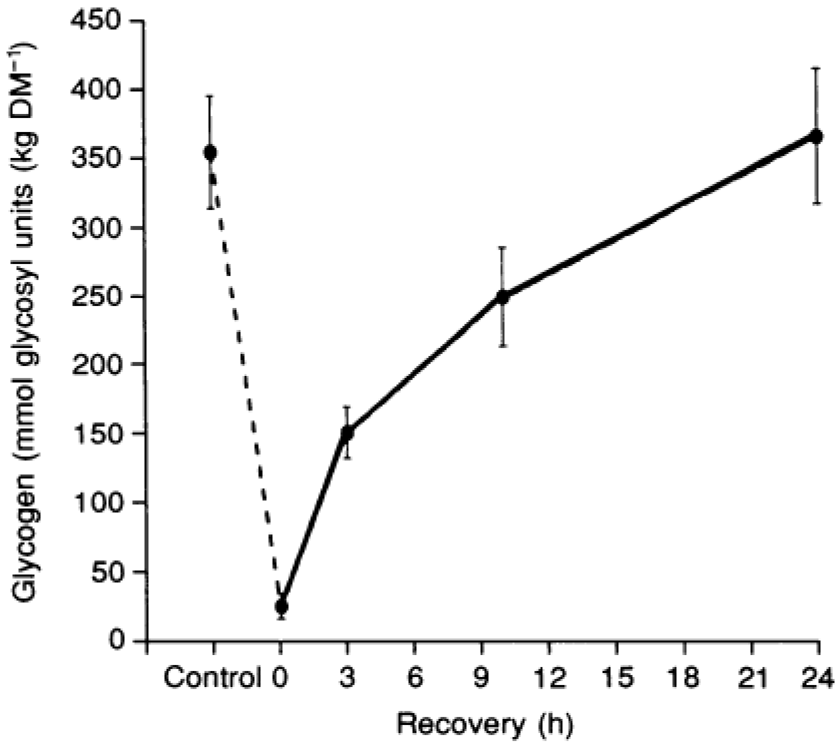
Based on the above theoretical knowledge, traditional sports nutrition recommendations have generally emphasized a high-carbohydrate diet for strength trainees. However, the supporting literature for this is commonly in endurance trainees and the theoretical rationale does not consider the body's high flexibility in how it can produce energy. A 2022 systematic review of the literature by Henselmans et al. found no beneficial effects of higher carbohydrate intakes on strength training performance in all 8 short-term studies and 16 out of 17 long-term studies. Every study with isocaloric control groups found carbohydrate intake influenced neither resistance training performance nor strength development. There was only a single study favoring higher carbohydrate intakes, Vargas-Molina et al. (2020), and in this study the ketogenic diet group consumed significantly fewer calories than the high-carbohydrate diet group, resulting in fat loss in the keto group compared to more muscle and strength development in the high-carb group. In contrast, two studies actually showed evidence in favor of the lower carbohydrate intake groups. A subsequent 2024 meta-analysis confirmed that 1RM strength gains in the squat and bench press didn't significantly differ between ketogenic and non-ketogenic diets in strength-trained individuals.

➢ Literature overview

Long-term effects of carbohydrate intake on strength training performance

Glycogen resynthesis

The primary reason for the null effect of carbohydrate intake on performance over the course of a strength training program is that full glycogen resynthesis practically always occurs before the next workout of a given muscle group. On high carbohydrate intakes, you can resynthesize glycogen extremely rapidly at around 10 mmol/kg/h after strength training. Glycogen resynthesis rates are maximized by a carbohydrate intake of 1—1.2 g/kg per hour, up to 10 g/kg per day. Such rates enable full glycogen resynthesis after even major depletion within 24 hours. For example, see Casey et al. (1995)'s measured time-course of glycogen resynthesis in untrained individuals after cycling exercise to repeated exhaustion with practically maximal glycogen depletion. Even in this scenario, glycogen resynthesis was complete within a day of high carbohydrate dieting.



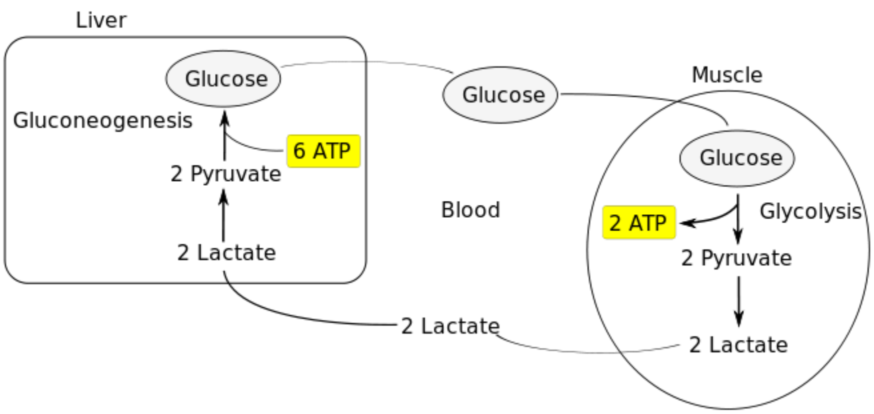
Glycogen resynthesis in the day following practically maximal glycogen depletion in untrained individuals on a high-carbohydrate diet. [Source]

However, strength training generally results in only modest depletion. [At maximum glycogen resynthesis rates, modest depletion levels (~40 mmol/kg) can recover within 4 hours]. Since you generally only train a muscle again at least 24 hours after a given workout, there's no need for such fast glycogen recovery and it can be enough to rely on carbohydrate-independent glycogen resynthesis pathways. The body has 3 mechanisms by which it can produce glucose without using any carbohydrates (gluconeogenesis).

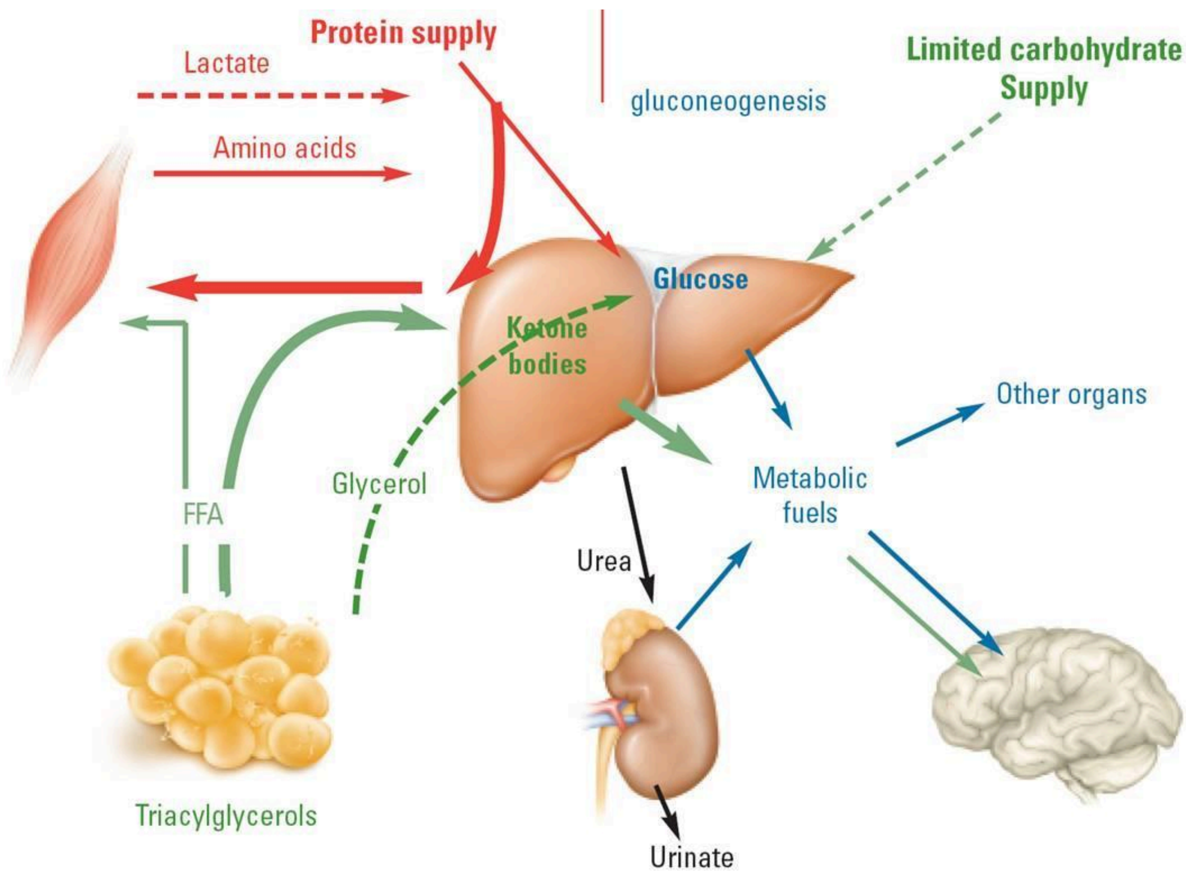
1. Protein: All but 2 of the natural amino acids (lysine and leucine are the exceptions) are glucogenic, meaning they can be converted to glucose. Of course, this would only be desirable for excessive protein intakes so that it doesn't reduce muscle protein synthesis levels.

2. Fats: The glycerol backbone of triglycerides from your diet or adipose tissue can be converted to glucose by the liver. Though the contribution of glycerol to glucose production is normally modest, the body is capable of deriving a significant percentage of its glucose needs from glycerol, up to full daily requirements during starvation. Given that a strength trainee on a low-carb, high-fat diet has a high glucose requirement and an abundant supply of dietary triglyceride, major conversion of glycerol to glucose is plausible.

3. Lactate: The Cori cycle effectively 'recycles' lactate produced during anaerobic exercise to glucose. The energy for this process can be derived from fatty acid oxidation (i.e. burning fat). The capacity of this system is limited though: only a minority of glycogen resynthesis at any time can occur via the Cori cycle.



The Cori cycle.



Ways the body can create glucose.

Moreover, muscle glycogen stores are partly autoregulated. The more you deplete glycogen, the faster the glycogen resynthesis, as glycogen content is inversely related to glycogen synthase I activity, thereby allowing faster glycogen resynthesis after greater depletion, at least if substrate is available. Exercise also causes insulin release resulting from the elevation in blood glucose levels, as well as translocation of GLUT4 from the GSVs to the cell membrane in your muscles to promote glucose uptake. The higher the intensity of exercise, the more lactate is produced and the faster the resynthesis.

As a result, even fasted after a strength training workout, glycogen resynthesis can occur at a rate of approximately 1.9 mmol/kg/h [2]. Phielix et al. (2019) estimated the glycogen synthesis rate of sedentary and endurance-trained individuals in an overnight fasted state (with saline infusion) at approximately 2.0 mmol/kg/h and 3.7 mmol/kg/h, respectively. Even the lowest of these fasted rates is generally high enough for complete glycogen resynthesis after strength training within a day. For example, if we take the glycogen depletion amount of the study with the highest reported depletion in the literature after resistance training, 39% or 46.6 mmol/kg, this would require an average glycogen resynthesis rate over 24 hours of 1.9 mmol/kg/h for full recovery. That's possible even while fasting.

The carbohydrate-independent glycogen resynthesis or glycogen sparing pathways can further improve in efficiency over time on low-carb diets, although most data are in endurance trainees. Volek et al. (2016) compared the glycogen depletion and recovery of ultra-endurance athletes on a habitual 59% carbohydrate diet to those on a habitual 10% carbohydrate diet after a 3-hour run. Ninety minutes pre-workout and immediately post-workout, the participants consumed an isocaloric (5 kcal/kg) and isonitrogenous (14% protein) shake with either 5% carbohydrate for the low-carbohydrate dieters or 50% carbohydrate for the high-carbohydrate dieters. Despite the large difference in carbohydrate intakes, resting glycogen concentrations were similar between groups and they decreased similarly immediately post-workout (62-66%) and recovered similarly over two hours back to 34-38% depletion. Other research [2, 3] finds habituated exercise in carbohydrate-restricted conditions may decrease reliance on glycogen, although again all research so far is on endurance rather than strength training. For example, Phinney et al. (1980) found that during a ketogenic, protein-sparing modified fast with endurance training, muscle glycogen levels stabilized at 69% of baseline after six weeks on the diet, compared to 57% of baseline after the first week. Moreover, there was a decrease in the amount of glycogen depletion of their treadmill endurance test over time: 16% at baseline, 13% in week one and unmeasurable levels in week six, while the time to exhaustion increased by 55%.

In conclusion, scientific research is quite conclusive that low-carbohydrate diets can be equally effective for strength training performance as higher-carbohydrate diets over the long run. Carbohydrate intake generally does not impact long-term strength training performance. The body has several pathways by which it can generally recover all glycogen lost with a typical strength training workout within 24 hours without needing to consume any dietary carbohydrate.

What about sports and cardio?

If you only train with weights, your carbohydrate intake isn't that important, but what about other athletes for more mainstream sports, like tennis, soccer or football? These types of sports burn far more glycogen than strength training, as you can see in the literature overview below, for 3 reasons.

1. Rest periods in between exercise bouts are typically much lower, resulting in more continuous exercise and often simply more total time spent exercising.

2. More exercise is spent in the most glycolytic exercise intensity range: repeat sprints of more than 10 seconds in length are notorious for burning a lot of glycogen.

3. A greater reliance on concentric (shortening) muscle actions, in which the actin-myosin cross-bridges for muscle contractions have to be formed chemically. This costs a lot of energy compared to the mechanical cleaving that occurs during eccentric contractions. (More on this in the module on understanding muscle growth.)

➢ Literature overview (not comprehensive)

Glycogen depletion from endurance training and sports

If you engage in highly glycolytic exercise like soccer or bicycling, you may need to increase your carbohydrate intake, especially pre-workout and post-workout. Based on a 2016 meta-analysis, competitive bicyclists significantly benefit from pre-workout carbohydrate consumption when bicycling for over 90 minutes. For shorter duration exercise, there was also a positive trend, but it did not reach statistical significance.

The following table lists the number of calories you burn per unit of time per bodyweight for various activities. Note that this is total energy expenditure. When you factor in how much to increase your calories, you have to calculate above basal energy expenditure: you have to subtract the amount of resting energy expenditure of that time period. After that, you can generally add all those calories in the form of carbohydrate, though it's not a disaster if you eat some fat or especially some protein as part of the additional calories, since this can largely be converted to glucose. It's best to consume the additional carbohydrates either before the endurance training or after the *previous* workout before the endurance training to maximize glycogen storage. You either want to store the carbs as glycogen or use the glucose directly.

Second, note that this is the energy expenditure for the actual activity performed non-stop or at least at a competitive/intense pace. For strength training in particular, you want to use the amount of time in which the workout *could* be completed with the rest intervals set so that you're basically only catching your breath in between sets. Powerlifters and Olympic weightlifters, I'm afraid these 3-hour workouts where you only perform 50 total reps don't burn 2000 Calories. More like 400.

As an example of how to calculate carbohydrate requirements, Mr. Orange is a 100 kg bodybuilder that decides to take up gardening. We're talking hardcore gardening here (surely it exists?), not your 2-by-2-yard gardening, so he spends an hour and a half in the yard now several days a week where he previously just sat in front of his computer.

Let's say 60 of these 90 minutes are actual activity with the rest is finding the tools, taking a break, etc. As per the table, he then burns 0.099 * 60 * 100 = 594 kcal. In total. Let's say his daily resting energy expenditure was 3000 kcal. That means we must subtract 3000 / 24 = 125 kcal and his energy expenditure only increases by 594 - 125 = 469 kcal. On these days, you can thus advise Mr. Orange to add 469 / 4 = 117 g carbohydrate to his diet. These carbohydrates are best consumed before the gardening or after the last workout *before* the gardening (if this can be planned) to maximize glycogen storage.

For cardio specifically, we'll get to more precise energy expenditure estimates in the course module on cardio.

Activity	kcal/lb/min	kcal/kg/min
Aerobic dance (vigorous)	.062	.136
Basketball (vigorous, full court)	.097	.213
Bicycling		
13 mph	.045	.099
15 mph	.049	.108
17 mph	.057	.125
19 mph	.076	.167
21 mph	.090	.198
23 mph	.109	.240
25 mph	.139	.306
Canoeing, flat water, moderate pace	.045	.099
Cross-country skiing		
8 mph	.104	.229
Gardening	.045	.099
Golf (carrying clubs)	.045	.099
Handball	.078	.172
Horseback riding (trot)	.052	.114
Rowing (vigorous)	.097	.213
Running		
5 mph	.061	.134
6 mph	.074	.163
7.5 mph	.094	.207
9 mph	.103	.227
10 mph	.114	.251
11 mph	.131	.288
Soccer (vigorous)	.097	.213
Studying	.011	.024
Swimming		
20 yd/min	.032	.070
45 yd/min	.058	.128
50 yd/min	.070	.154
Table tennis (skilled)	.045	.099
Tennis (beginner)	.032	.070
Vacuuming and other household tasks	.030	.066
Walking (brisk pace)		
3.5 mph	.035	.077
4.5 mph	.048	.106
Weight lifting		
light-to-moderate effort	.024	.053
vigorous effort	.048	.106
Wheelchair basketball	.084	.185
Wheeling self in wheelchair	.030	.066
Wii games		
bowling	.021	.046
boxing	.021	.047
tennis	.022	.048

Source: Rolfes, Pinna, Whitney, Understanding Normal & Clinical Nutrition, 9/e. © Cengage Learning.

Muscle growth

Other than the need for carbohydrates to affect your performance in the gym, what about the need for carbs to fuel muscle growth?

Old-school dieticians often cite the textbook science that liver glycogen levels need to be maintained to prevent amino acids being converted to glucose. For endurance training, low starting glycogen levels can indeed result in muscle catabolism [2]. However, as you learned, for strength trainees glycogen stores are generally not a limiting factor. Accordingly, even very depleted pre-exercise glycogen levels do not significantly impair post-workout anabolic signaling or muscle protein synthesis in strength trainees [2*, 3, 4]. The anabolic signaling for endurance training adaptations can even be enhanced by training in a glycogen-depleted state.

> **: The second study found a non-significant trend for one of the 3 signaling pathways, but it did not include mTOR and this study was conducted in cyclists performing only 3 submaximal sets of leg extensions (3x10 at 70% of 1RM) in a fasted state after 2 days of intensive glycogen depletion consisting of a very low-carbohydrate diet and over 3 hours of intensive endurance exercise, including 6 maximal sprints, so it's actually miraculous there was no negative effect, as you'd expect a negative effect from the muscle damage or interference effect alone.*

Carbohydrate intake can still influence protein balance via insulin. Insulin is an anabolic hormone, so it's sometimes theorized that it can increase muscle protein synthesis. However, the role of insulin in promoting protein synthesis is greatly overrated and you need to inject insulin with supraphysiological bodybuilding dosages to cause meaningful further increases in protein synthesis. Carbohydrates also cannot stimulate muscle protein synthesis (MPS), as they are obviously not protein. However, while neither carbs nor insulin normally stimulate MPS in natural trainees, consuming carbohydrates in isolation can have an anti-catabolic effect by increasing insulin production. Insulin does suppress muscle protein breakdown (MPB). As such, a post-workout meal with just 100 g carbohydrates does not stimulate MPS but does decrease MPB, thereby improving protein balance slightly. Emphasis on 'slightly', as protein balance was still negative. The contribution of MPB to protein balance is relatively minor: most changes in protein balance occur due to changes in MPS, which can vary severalfold. The body is efficient: it doesn't break down proteins unless needed and rather changes the rate of synthesis based on its needs and dietary protein intake.

Unbeknownst to many people, carbohydrates are not the only insulinogenic macronutrient. Protein is too and maximal inhibition of protein breakdown already occurs at an insulin concentration of 11, 15 or 30 mU/l, depending on which study you look at. Good protein sources are highly insulinogenic, just like carbs, so just ~20 g protein will have you exceed the insulin threshold for maximal MPB suppression [2, 3]. As such, protein consumption makes carbohydrate consumption redundant for protein balance [2]. Carbs may reduce protein breakdown a little when you barely consume any protein, but if you already consume enough protein, carbs have no additive effect.

It's worth noting there are 3 studies (Miller et al. 2003; Bird et al. 2006; Bird et al. 2006 II) that did find beneficial effects of adding carbohydrates to protein to decrease muscle protein breakdown. These studies are often cited in support of post-workout nutrition by supplement manufacturers. Their key limitation is that only 6 g of protein was consumed in all 3 studies. That's not enough to maximally stimulate insulin production to prevent muscle catabolism. 6 grams of essential amino acids aren't even enough to stimulate muscle protein synthesis in some research. Koopman et al. (2007) used a much more practically relevant study design. They examined the difference in protein balance in groups consuming either 0, 0.15, or 0.6 g of carbs per kg of body weight when also consuming ~25 g protein after resistance training. The results: “Whole body protein breakdown, synthesis, and oxidation rates, as well as whole body protein balance, did not differ between experiments. […] In conclusion, co-ingestion of carbohydrate during recovery does not further stimulate post-exercise muscle protein synthesis when ample protein is ingested.” Staples et al. (2011) wanted to replicate the above finding to end the discussion once and for all. After a weight training session, they gave their subjects either 25 g of whey or both 25 g of whey in combination with 50 g of maltodextrin. They found that consuming 50 g of maltodextrin along with 25 g of whey did not stimulate muscle protein synthesis or inhibit protein breakdown more than 25 g of whey alone.

Thus, you generally don’t need to consume any carbohydrates post-workout to maximize muscle growth. Just make sure you consume enough protein, at the very least 0.4 g/kg. You don’t have to actively avoid carbs either, but adding sugar to your post-workout shake is, well, just like adding sugar to any other meal. So beware of supplement companies trying to sell you sugar for the price of steak because it’s now a ‘special post-workout supplement’.

Another purported benefit of pre-workout carbohydrate intakes is suppressing cortisol. The idea is that cortisol is catabolic and therefore bad for our gains, so suppressing it is beneficial. However, a 2022 meta-analysis found carbohydrate intake does not affect long-term, resting cortisol levels. Low-carbohydrate diets may result in a temporary rise in cortisol levels, at least in endurance trainees, but afterwards the levels normalize back to baseline. This adaptation may relate to a change in fuel substrate use when we change our diet. Cortisol’s primary role during exercise is to mobilize fuel substrates for energy production [2], not to meaninglessly break down our muscles. As such, cortisol release is normal and in fact beneficial during exercise, especially high-intensity exercise, so it's questionable if we even want to suppress it in the first place.

Overall, most research shows that the ratio of carbohydrate to fat in the diet does not affect protein balance [2, 3] and consuming carbohydrates on top of protein does not increase protein synthesis. Some tenuous research even finds that nitrogen balance is greater on high-**fat** diets than on low-fat diets. The high fat group also lost significantly less lean body mass than the high carb group. Other research also finds that carbohydrates mainly increase protein balance in your organs due to the nutrient partitioning effect of insulin, whereas fat actually causes more protein to reach peripheral tissue like muscle.

In line with the above theory, most research does not find any effect of carbohydrate intake on muscle growth over the course of a strength training program. Even ketogenic diets result in similar muscle growth as higher-carb diets, provided energy intake is high enough. A 2022 meta-analysis by Vargas-Molina et al. found no significant difference in muscle hypertrophy in strength trainees between ketogenic and control diets. Another 2022 meta-analysis on ketogenic vs. non-ketogenic diets for concurrent athletes found that the diets did not differentially affect body fat percentage or lean body mass. Fifteen of the studies from Henselmans et al.'s 2022 systematic literature review on the effect of carbohydrate intake on strength training performance also estimated or measured fat-free-mass (FFM) or muscle thickness (MT). Ten of these studies found no significant between-group differences in these measures and thereby presumably muscle growth, in line with the lack of differences in strength development. Five studies favored higher carbohydrate intakes, but they were clearly confounded by energy intake. People on lower carbohydrate intakes often unintentionally reduce their energy intake, resulting in better fat loss but less muscle growth.

- A considerable discrepancy in energy intake was evident in self-reported energy intakes in the studies by Vargas-Molina et al. and Rozenek et al.
- LaFountain et al.'s study was probably similarly confounded, as it had *ad libitum* diets without instructions regarding energy intake, resulting in significant fat loss in the ketogenic diet group (-5.9 kg) but not the control diet (-0.6 kg).
- Greene et al.'s study was isocaloric based on self-reported food logs and the authors noted that the loss of FFM in this study may have largely been water and glycogen, combined with a previously reported overestimation of FFM loss in carbohydrate-restricted athletes. Lack of significant loss of contractile muscle tissue would explain why neither basal metabolic rate (BMR) nor performance measures significantly differed between groups. FFM is the strongest predictor of BMR, so significant FFM loss should decrease BMR.
- Similarly, in Paoli et al.'s study, the participants did not report significantly different energy intakes between groups; however, again only the ketogenic diet group lost a significant amount of fat (-1.4 kg) and strength performance and resting metabolic rate did not significantly differ between groups, so the greater FFM gain in the Western diet group may have been attributable largely to water and glycogen losses in the ketogenic group.

Glycogen levels and associated water retention can significantly influence FFM measurements. Notably, Wilson et al. (2017) demonstrated that one week of carbohydrate reintroduction increased FFM by 4.8% after a 10-week ketogenic diet, while no change was observed in the traditional high-carbohydrate group. Over the course of the total 11-week isocaloric and isonitrogenous diets, the change in muscle thickness and lean body mass was significantly positive in the ketogenic diet group but not the higher-carbohydrate diet group.

Another study by Wrzosek et al. (2021) that did control for protein and calorie intake in trained men found no body recomposition differences after 12 weeks on 20% carb vs. 40% carb diets. A subsequent study by Ribeiro et al. (2023) found identical lean mass gains (1.4 kg) in high- vs. low-carb diets in strength-trained men after 8 weeks of training. We also have 3 unpublished studies by Werner et al. (thesis), Rinehart (1988) (in book) and Zajac et al. (7th ICST conference abstract) that all found no differences in the development of body composition, nitrogen balance or strength between the higher- and lower-carbohydrate groups.

Overall, carbohydrate intake generally does not have a significant effect on muscular development independent of energy intake.

➢ Research overview

The effect of carbohydrate intake on muscle growth

Note: The potential anabolic function of fats is discussed in more detail in the course module on dietary fat.

Fiber

Carbohydrates are commonly recommended for their alleged performance enhancing effects, which is generally unfounded as we've discussed so far, yet a special type of carbohydrate does come with many benefits for fat loss and health: fiber.

Fiber is carbohydrate that is indigestible in the human small intestine and ferments in the large intestine. Fibers can be natural (dietary fiber) or synthetic (functional fiber) to mimic the health benefits of dietary fiber.

Dietary Fibers	Functional Fibers
Cellulose	Cellulose
Hemicellulose	Pectin
Pectin	Lignin*
Lignin	Gums
Gums	β-glucans
β-glucans	Fructans*
Fructans	Chitin and chitosan*
Resistant starches	Polydextrose and polyols*
	Psyllium
	Resistant dextrins*
	Resistant starches

*Data showing positive physiological effects in humans are needed.

For health and physique benefits, we are mostly interested in dietary fiber, as the food matrix changes its effects in the human body. We'll revisit supplemental fibers in the supplements module.

Soluble vs. insoluble fiber

Nutritionists commonly classify dietary fiber as soluble and insoluble fiber, depending on whether the fiber dissolves in water, as this significantly changes its effects in the body. Cellulose is the most common dietary form of insoluble fiber. Soluble fibers include pectin, psyllium and gums. Soluble fibers generally provide more health benefits than insoluble fibers.

Insoluble fiber mainly has mechanical effects while it passes through your gut and is therefore commonly said to prevent constipation. However, the digestive effects of fiber are not merely a result of the fiber's solubility but also its fermentability, viscosity and hydration capacity. Fiber normally has laxative effects, making your bowel movements more regular and voluminous, but many people have some degree of intolerance to certain types of fiber, which makes these fibers cause constipation. FODMAP intolerances are further discussed in the module on health science.

Soluble fiber binds to water and certain nutrients, turning it into a thick, viscous gel, which delays gastric emptying and food transit times. As a result, most dietary soluble fibers make you feel full and satiated. By slowing down the digestion and absorption of glucose into the blood, (soluble) dietary fiber also improves blood sugar control and insulin sensitivity, thereby reducing the risk of metabolic syndrome and type II diabetes.

By binding to bile, digestive enzymes and fatty acids, (soluble) dietary fiber reduces the digestibility of fats and proteins from your diet. Reduced protein digestibility is generally only a problem for vegetarians, as discussed in the course module on protein. Reduced digestibility of fat is beneficial for most people, as it theoretically allows you to eat more at the same level of energy balance. However, the effect is trivial with the possible exception of very high fat, high-protein diets. The Atwater factors already correct for the metabolizable energy in food with fiber, so you generally don't have to take the digestibility effect of fiber into account with your macros. Still, it's possible that a high fiber intake on a high protein, high fat diet will allow you to increase your energy intake a few percent without storing more energy.

Dietary fiber is also good for your cardiovascular health. A high dietary fiber intake lowers LDL cholesterol levels and blood pressure via several mechanisms. Fiber, particularly soluble fiber, can bind to bile and cholesterol, causing reduced cholesterol absorption, lower liver cholesterol levels and subsequently greater removal of LDL cholesterol from the blood. Cholesterol metabolism will be discussed in greater detail in the course module on fats.

While you commonly see foods categorized as being either rich in soluble or insoluble fiber, almost all fruits, vegetables and grains contain both types of fiber, just in somewhat different ratios, so you don't have to pay much attention to which food contains which type of fiber. As long as you consume enough total fiber from a variety of whole-food sources, you should get enough of both types of fiber.

Fermentation

Since fiber is indigestible for the human small intestine, it reaches the large intestine, your colon, intact. There, colonic microflora ferment the fiber. Soluble fibers generally ferment to a greater degree than insoluble fibers. Cellulose barely ferments, for example.

When fiber ferments, it provides energy for your microflora. This is called a prebiotic effect, which helps form probiotics that aid your digestive health.

The fermentation reaction forms short-chain fatty acids (SCFA), primarily acetic, butyric, and propionic acids. Other products of fiber fermentation are hydrogen, carbon dioxide, and methane gases that are excreted as flatus (yes, those are your farts) or expired by the lungs. Different fibers are fermented to different short-chain fatty acids in different amounts by different bacteria. SCFAs are rapidly absorbed, causing water and sodium absorption in the colon and growth of mucosal cells from the colon. During their metabolism, they increase blood flow to the whole splanchnic area (the organs in the abdominal cavity such as the liver, spleen, and intestines) and promote immune system activity.

Fermentation also forms lactic acid, lowering the colon's pH. This acidic environment increases the absorption of bile and fatty acids, which is thought to prevent colon cancer. In addition, the lower colonic pH favors the growth of beneficial lactobacilli and bifidobacteria and inhibits the growth of certain pathogenic bacteria.

Effect on energy harvest

SCFAs provide energy to the body, so dietary fiber is not devoid of energy. However, dietary fibers have fewer than 2 kcal per gram on average and due to their anti-nutritive effects, some fibers even have negative calories. Food labels should already take the energy value of fiber into account in their estimates of metabolizable energy content. However, a very large increase in dietary fiber can decrease your energy balance by a few percent due to fecal energy losses. Corbin et al. (2023) compared a low- and a high-fiber 'microbiome feeding' diet in a carefully controlled metabolic ward. Both diets had the same macronutrient and thus energy intakes, but the fiber intake in the low-fiber diet was 6.4 g/1000 kcal, around 14 g total per day, compared to 26 g/1000 kcal with 10 g/1000 g resistant starch, around 55 g total, in the high-fiber diet. The high-fiber diet resulted in 5% lower energy balance due to fecal energy loss: they literally pooped out 5% of their energy intake. This fecal energy loss should slightly improve fat loss or allow you to eat 5% more kcal for the same fat loss. However, 5% 'free calories' without a change in energy expenditure or macros is probably as high a microbiome effect as you'll get in practice, given the major difference between the diets. Going from a healthy to a healthi*er* diet will probably only lower energy yield by a few percent at most.

Dietary fibers can also bind to toxins. For example, insoluble fibers may adsorb hydrophobic carcinogens. This can lower your risk of cancer. A high fiber diet is a better detox than the majority of pseudoscience detox nonsense you commonly see advertised in mainstream media.

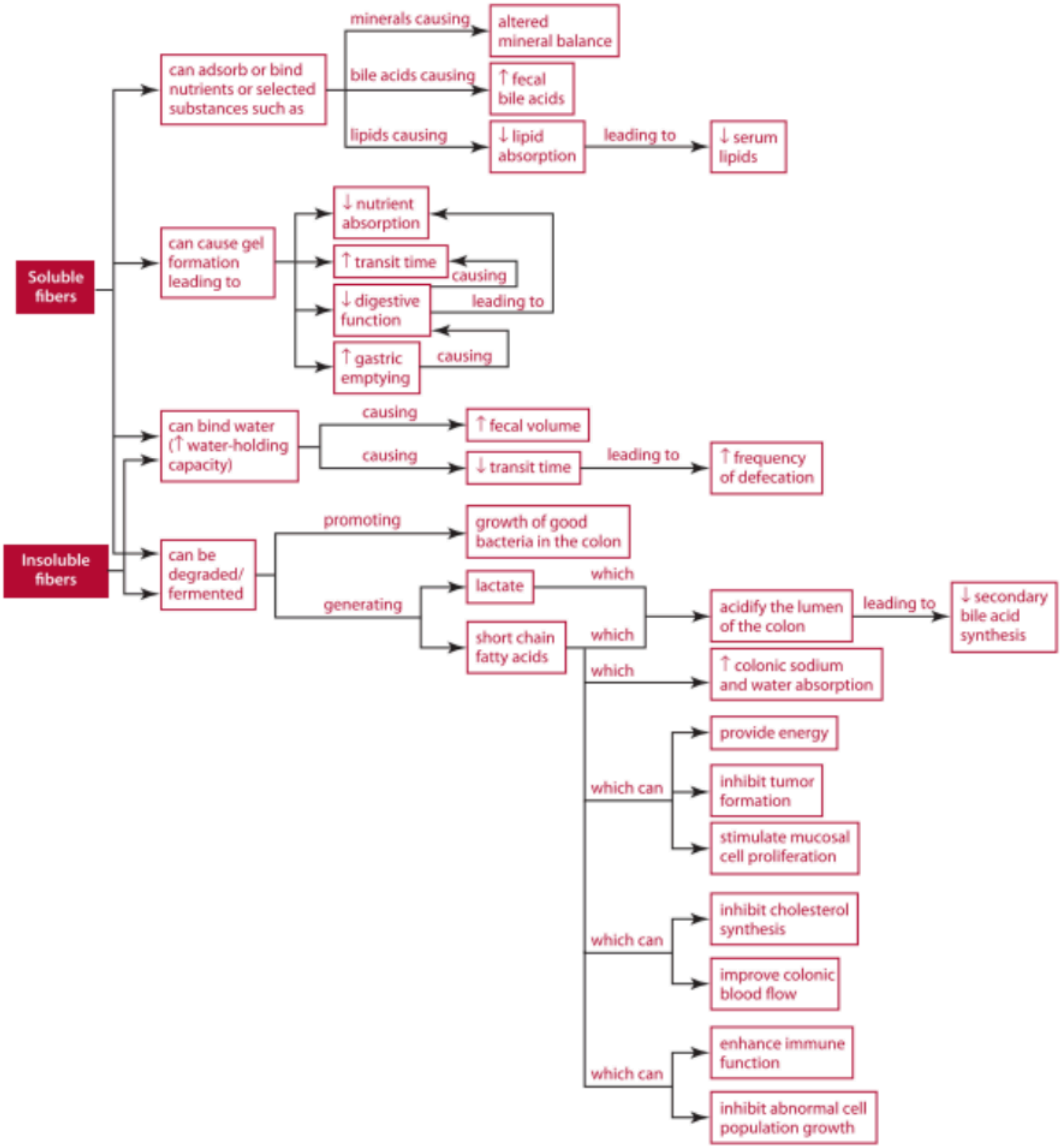
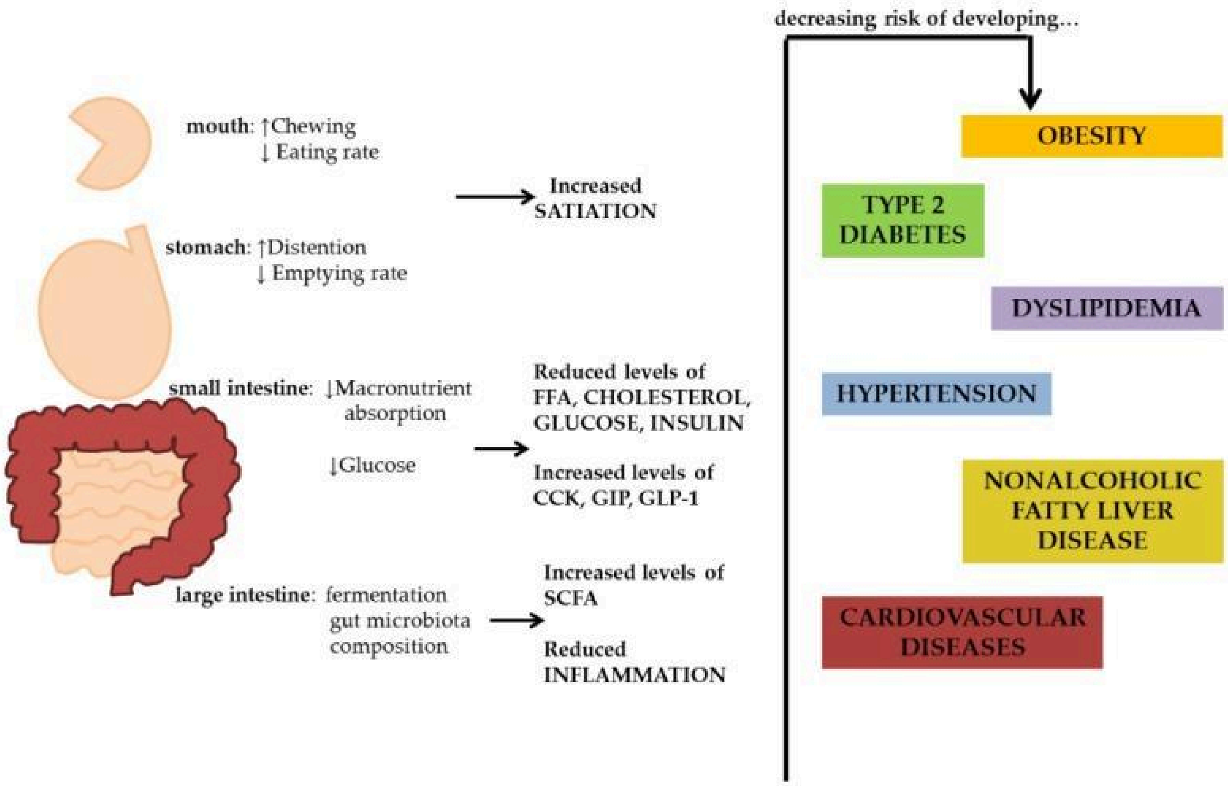


Figure 4.4 Gastrointestinal response to soluble and insoluble fibers.



Health effects of dietary fiber (Source)

Conclusion

In short, high fiber diets are great for your health [2, 3, 4, 5]. They improve your digestion, glucose control and immune system and they lower the risk of cardiovascular diseases by decreasing your blood pressure and cholesterol level, ultimately amounting to lower mortality and morbidity and an increased lifespan. Higher fiber intakes have been found to decrease all-cause mortality by up to 30%.

High fiber intakes also aid fat loss by increasing satiety, making dieting easier. They may also slightly increase energy expenditure and reduce energy harvesting. High fiber intakes are associated with greater gut microbiota diversity and less weight gain over time. Some studies find that even on an isocaloric basis (self-reported/planned at least), large intakes of brown rice lead to more fat loss than white rice [1, 2]. Since we know different carbs typically do not have different effects on our body composition, these effects are most likely attributable to the extra fiber content of brown rice.

Based on the latest series of systematic reviews and meta-analyses on dietary fiber intake, the health benefits of fiber increase up to intakes of at least 35 grams per day: see the dose-response meta-analysis below.

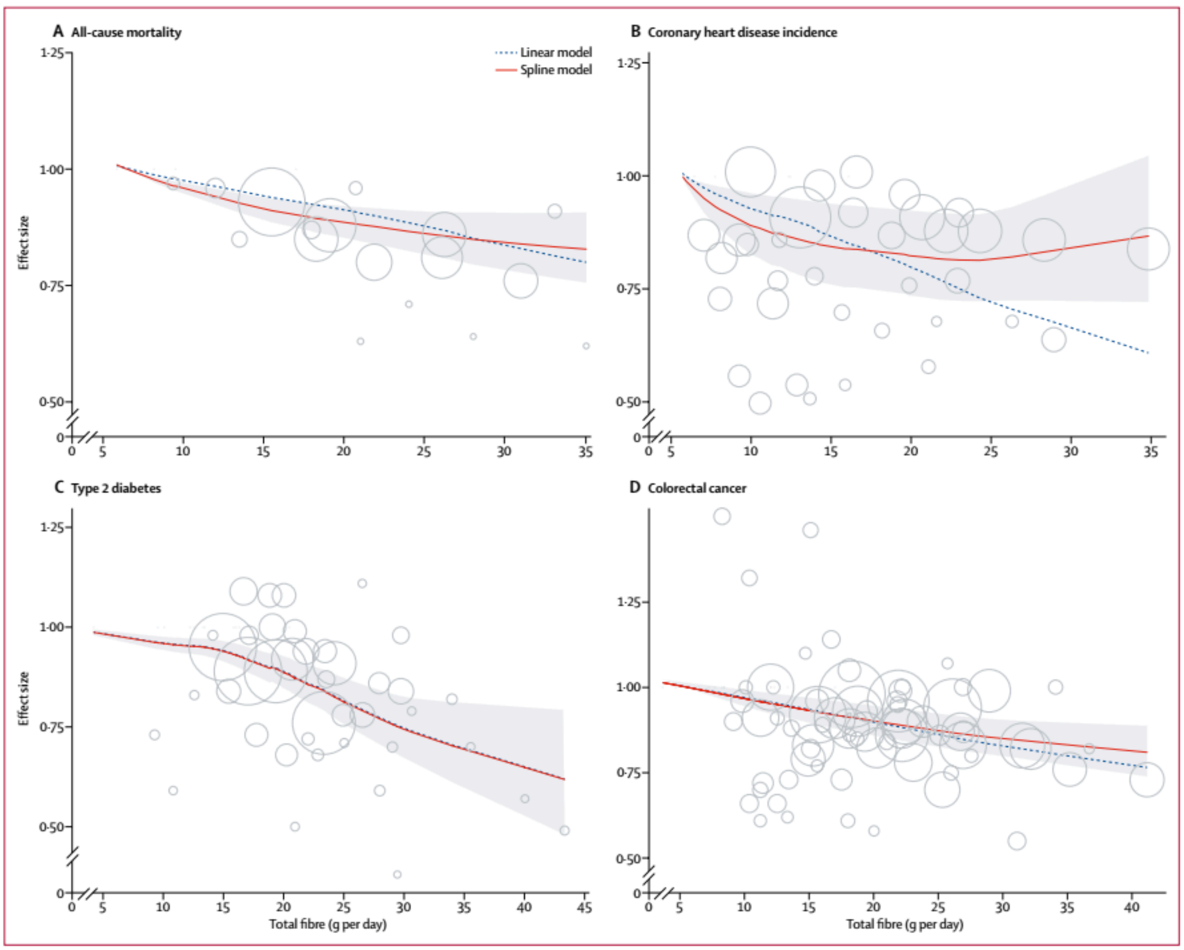


Figure 1: **Dose-response relationships between total dietary fibre and critical clinical outcomes based on data from prospective studies**
(A) Total fibre and all-cause mortality. 68 183 deaths over 11·3 million person-years. Assuming linearity a risk ratio of 0·93 (95% CI 0·90–0·95) was observed for every 8 g more fibre consumed per day. (B) Total fibre and incidence of coronary heart disease. 6449 deaths over 2·5 million person-years. Assuming linearity a risk ratio of 0·81 (0·73–0·90) was observed for every 8 g more fibre consumed per day.(C) Total fibre and incidence of type 2 diabetes. 22 450 cases over 3·2 million person-years. Assuming linearity a risk ratio of 0·85 (0·82–0·89) was observed for every 8 g more fibre consumed per day. (D) Total fibre and incidence of colorectal cancer. 20 009 cases over 20·9 million person-years. Assuming linearity a risk ratio of 0·92 (0·89–0·95) was observed for every 8 g more fibre consumed per day.

Several research organizations recommend daily dietary fiber intakes of 25 grams for women and 38 grams for men. These recommendations are for non-strength training individuals, so for strength trainees, you should see them as an absolute minimum for optimal health, assuming no digestive pathologies are present.

If this seems like a lot of fiber compared to the abysmally unhealthy modern Western diet, consider that many hunter-gatherer cultures habitually consumed fiber intakes upwards of 100 grams with estimates of average daily fiber intakes of 46 g [2], 86 g and 104 g. This is in line with estimates of 40-80 g/d fiber in Australian Aboriginals and a whopping 150-225 g/d fiber in a hunter-forager group in the northern Chihuahuan Desert. “Analysis of vegetable foods consumed by foragers in this century and evaluation of archaic native American coproliths suggest that ancestral human fiber intake exceeded 100 g/d (Eaton 1990). Rural Chinese consume up to 77 g/d (Campbell and Chen 1994), rural Africans up to 120 g/d (Burkitt 1983)”. (Source)

The following table lists the highest fiber foods per 100 Calories of the food based on the USDA database. As you can see, most starches commonly recommended as fiber sources are in fact poor sources of fiber. Vegetables and fruits, especially berries, are far richer in fiber.

Food	Fiber per 100 kcal (g)
Endive	18.2
Beet greens	16.8
Yambean (jicama)	12.8
Raspberry	12.5
Blackberry	12.3
Eggplant	12
Kale	11.7
Artichokes, (globe or french)	11.5

Celery	11.4
Passion-fruit, (granadilla), purple	10.7
Asparagus	10.5
Cabbage	10
Radish	10
Spinach raw	9.6
Carambola, (starfruit)	9
Brussels sprouts	8.9
Chayote, fruit	8.9
Lettuce, green leaf	8.7
Beans, snap, green	8.7
Chard, swiss	8.4
Peppers, sweet, red	8.1
Bamboo shoots	8.1
Cauliflower	8
Guavas, common	7.9
Broccoli	7.7
Broad beans, mature seeds	7.3
Green peas	7
Carrots	6.9
Beets	6.5
Turnips	6.4
Oat bran	6.3
Strawberries	6.2
Zucchini, includes skin	5.9
Mushrooms, portabella	5.9
Pear	5.4

Oranges, all commercial varieties	5.1
Seeds, flaxseed	5.1
Kiwifruit, green	4.9
Tomatoes, green	4.8
Pomegranate fruit	4.8
Apples, with skin	4.6
Mushrooms, white	4.5
Beans, black, mature seeds	4.5
Barley, pearled	4.4
Onions	4.2
Avocados, all commercial varieties	4.2
Blueberries	4.2
Apricots	4.2
Papayas	3.9
Peaches, yellow	3.9
Figs	3.9
Yellow nance	3.8
Sweet potato	3.7
Cucumber, with peel	3.4
Grapefruit, pink and red and white, all areas	3.4
Tangerines, (mandarin oranges)	3.4
Whole grain pasta	3.3
Chickpeas, mature seeds	3.2
Plums	3.1
Leeks, (bulb and lower leaf-portion)	3
Lentils	3

Whole grain wheat cereal	3
Banana	2.9
Buckwheat	2.9
Soybeans, green	2.9
Pineapple, all varieties	2.8
Mangos	2.7
Edamame, unprepared	2.7
Steel cut oats	2.7
Potatoes, flesh and skin	2.7
Melons, cantaloupe	2.6
Custard-apple, (bullock's-heart)	2.4
Corn, sweet, yellow	2.3
Millet	2.3
Whole grain bread	2
Quinoa, uncooked	1.9
Wild rice	1.7
Garlic	1.4
Grapes, red or green	1.3
Cassava	1.1
Soybeans, mature seeds, sprouted	0.9
Brown rice	0.7

More fiber does not always convey digestive health benefits: the type of fiber and the specific problem determine to what extent dietary fiber can improve health outcomes. Some individuals may even feel worse when increasing their fiber intake. This is commonly related to FODMAPs, which we'll discuss in the module on health science.

Practical applications

As with the other macronutrients, there's a lot of theory a serious lifter should know about carbohydrates. However, the practical implications are not difficult.

- The type of carbohydrate you eat matters preciously little: what matters is what else is in the food, notably its fiber and micronutritional content.
- A high protein intake generally makes carbohydrates redundant for muscle growth and performance. Just make sure you're consuming some carbohydrates in the meal before and after each workout and you don't train fasted.
- Carbohydrates generally offer no long-term advantage over dietary fat for fat loss or the prevention of fat gain.
- Carbohydrate requirements are effectively zero for bodybuilders engaging in strength training or low intensity cardio as their only modalities of activity. For most sports and high intensity interval training, carbohydrate requirements rapidly increase.
- The only type of carbohydrate with distinct benefits for satiety and overall health is dietary fiber. The recommended minimum fiber intake is 25 g for women and 38 g for men.