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EVIDENCE-BASED KETO

YOUR NO-HYPE GUIDE TO THE KETOGENIC DIET



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Reviewed by Kamal Patel and the Examine.com team

How to Use This Guide

A quick Google search will bring up countless results on ketogenic diets — about 147 million hits at the time of writing.

Not only are blog posts (and magazines, and celebrities ...) into keto, but the past few years have seen a sharp increase in the number of scientific articles investigating the diet. All this attention has brought along with it a lot of hype and confusion about just what the keto diet can do.

Figure 1: Scientific articles on keto published per year

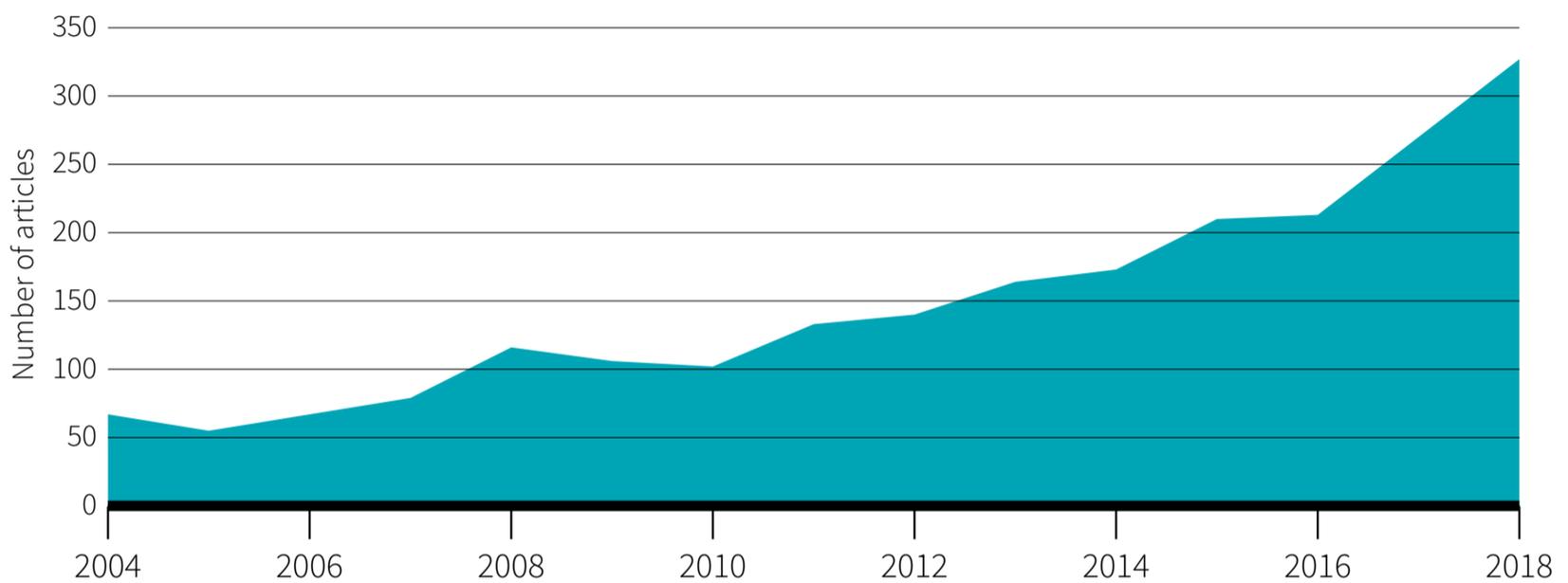
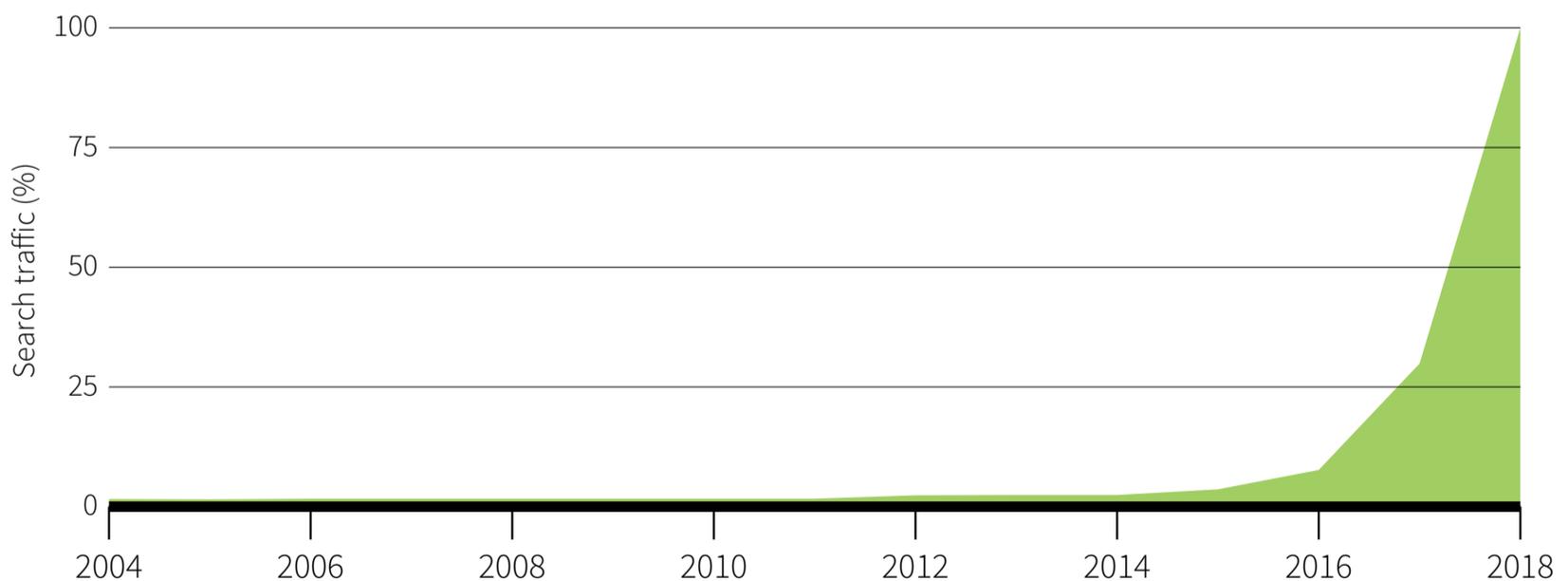


Figure 2: Google search trend for *keto diet*



That's why we've written this guide. What does the latest high-quality evidence say about keto? Is it safe? What are its benefits and downsides? What (and whom) is it best for? How well does it work in the long term? What are the results experienced by endurance athletes, strength athletes, regular gym goers, couch potatoes, and many other categories? That's what you're about to discover.

Choose your own adventure

Give me what I need to get started

If you're a cut-to-the-chase kind of person, someone who just wants to know the "what" of keto, then [Keto 101: The Practice of Keto](#) is where you should start. You will find:

- An overview of the keto diet (basic information and core concepts)
- A guide to getting your diet started
- Advice to help you stick to your diet in a world filled with carbs
- Tips to help you deal with unwanted side effects that may arise on a keto diet
- Answers to common questions about keto's interactions with specific health conditions

Give me all the facts

If the basic facts aren't enough for you, if you want to know the underlying "why" behind each "what", then head over to [Keto 201: The Science of Keto](#), where each chapter provides a deep dive into the scientific literature. Is keto better for fat loss than any other diet? Does it hurt muscle growth? How does it affect your blood sugar levels? We've got you covered.

Additionally, every chapter is enriched with explainer boxes:

- Boxes that **teach** important scientific concepts
- Boxes that **analyze** essential studies
- Boxes that **introduce** emerging research
- Boxes with **warnings** and **tips** to keep in mind

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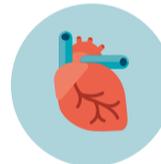
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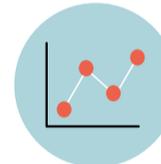
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Medical Disclaimer

This guide is a general-health document for adults 18 or over. Its aim is strictly educational. It does not constitute medical advice. Please consult a medical or health professional before you begin any exercise-, nutrition-, or supplementation-related program, or if you have questions about your health.

This guide is based on scientific studies, but individual results do vary. If you engage in any activity or take any product mentioned herein, you do so of your own free will, and you knowingly and voluntarily accept the risks.

Preface

Being the nutrition nerds that we are, many of us at Examine.com have tried a ketogenic diet at one point or another in our lives. Below, we recount our experiences.

Kamal

The Atkins diet was popular when I was in college, in the late 1990s, but the science didn't seem to make sense. What was the deal with all those diet phases? Eventually, I started reading about clinically investigated ketogenic diets, until finally I decided to try each of the main flavors in turn: a standard ketogenic diet (SKD), the targeted variety (TKD), and the cyclical approach (CKD).

My aim was narcissistic to the max: not only did I want to be the most muscular and ripped Kamal I could be, but I also wanted to be more muscular than the loud Indian frat bros who had previously dwarfed me (I hadn't lifted a single dumbbell before age 19). CKD worked the best for me, when combined with glycogen supercompensation and a strict training regimen. Since then, I've used ketogenic diets periodically, first for body composition, then later for its potential therapeutic benefits. It doesn't seem to be a silver bullet for my joint pain, but does prod me to avoid several types of junk foods I'm apt to overindulge (junk foods can worsen joint problems).



Kamal Patel, Co-founder
MBA, MPH

A co-founder and the director of Examine.com, Kamal holds two master's degrees from the Johns Hopkins University, in business and in public health, and is on hiatus from a Ph.D. in nutrition for which he's investigated the link between diet and chronic pain. He's published peer-reviewed articles on vitamin D and calcium, as well as on a variety of clinical research topics. He's also been involved in research on fructose and liver health, on nutrition in low-income areas, and on mindfulness meditation.

Pierre-Alexandre

I got sick. After a few months, I got better. But among the symptoms that never went away was a constant brain fog. Sometimes it is lighter and sometimes denser, but it never truly dissipates. And among the many things I tried to get rid of it was a keto diet.

As Wyatt will tell you, there are anecdotal reports of keto benefits for about every health issue under the sun — including brain fog. Since ketones act a little differently in the brain than does glucose, there's actually a rationale for why a keto diet may have an effect. Moreover, there's preliminary evidence that keto helps with Alzheimer's (maybe through the same mechanisms that make it useful to control epilepsy).

So, I tried keto. I cut carbs out from my diet nearly entirely, usually staying closer to 30 than 50 grams per day. Fortunately, I really like eggs, because for one month they were my staple food. After the first few days, I managed to stay in ketosis, according to the urine strips I used (though I now regret not buying a blood ketone meter).

In the end, did a ketogenic diet turn me into a ketogenius? Alas, no. It had no discernible effect, positive or negative, on my thinking or mood. My digestion was better than ever, though, and I could eat breakfast then nothing until dinner without the slightest pang of hunger. Over my keto month, I lost 6 kg (13 lb), and since I was very lean to begin with, now you could have played the xylophone on my ribs. This musical perk wasn't the keto benefit I was hoping for, however, so I terminated the keto experiment.

In my first week post-keto, I regained 4 kg. The next week, I lost 1 kg again. Both were probably glycogen and water, so over my keto month I may have lost 3 kg (6.6 lb) of mixed fat and muscle. The fact that I was never hungry *and* was barred from eating my favorite treats is enough, I surmise, to explain this weight loss.



Pierre-Alexandre Sicart,
AA in English, PhD in
French Literature

Pierre-Alexandre Sicart holds graduate degrees from New York University, the University of Toulouse II, and the University of St Andrews. At NYU, he was MVP then captain of the Taekwondo Club, president of the Karate Club, and founder of the Martial Arts Club. After graduation, he wrote a grammar book, then found himself working as an assistant professor of French in Taiwan. After some years enjoying the best foods in Asia, he moved back to France to freelance as a writer, translator, and copy editor. He's Examine.com's resident copy editor and has been overseeing our [Supplement Guides](#) since 2016.

Michael

I took a dive into the keto diet right as I started my undergraduate degree in exercise and nutrition. It has been very interesting for me to look back on that period, now that I have a better understanding of what my body was going through at the time. When I started keto, I did so gradually — I did not suddenly drop my carbs down to under 50 grams a day (which helped me avoid the dreaded “keto flu”). Over the first month, I cut out alcohol, added sugars, grains, legumes, and dairy. So I was certainly on a low-carb diet, but not yet full-on ketogenic.

Over the second month, I got down to eating only meat, seafood, nuts, seeds, and non-starchy vegetables. That was it! My blood ketone levels hovered around 2.0 mmol/L (four times higher than required for ketosis), my body fat dropped by 2.2%, and my vitamin D levels went up. But my exercise performance absolutely *tanked*. I was doing a fair amount

of high-intensity training at the time, and my workout capability didn't fully recover until I started eating more carbs. While I haven't gone back to keto, the experience ultimately made me more knowledgeable about some of the challenges people face when following this diet.



Michael Hull, MSc in Nutrition,
CISSN, PN-L1

Michael holds a bachelor's degree in exercise science and sports nutrition from George Mason University and completed his master's degree in human nutrition at McGill University, where he published a thesis on lifestyle correlates of vitamin D status. As a full-time senior researcher at Examine.com, he primarily writes and updates the [Supplement Guides](#), maintains their database of supplement studies, and [blogs about various health topics](#). In his free time, he enjoys finding ways of using technology to remove barriers to science communication.

Alex

My interest in the ketogenic diet came about from seeing the many different camps that exist on the Internet. You have those who promote a therapeutic ketogenic diet regardless of one's goals; you have those who believe ketogenic diets are simply another tool in the toolbox, with ketosis being largely irrelevant; and you have those who believe that a ketogenic diet is an abomination for human health. Maybe all of them have a point, depending on the context.

I've never eaten what many would consider a true ketogenic diet. My "keto" experience comes from running cycles of a protein-sparing modified fast with weekly carb binges — a cyclical ketogenic diet by some standards, but being in ketosis was simply a side effect of eating nothing but protein and doing glycogen-depletion workouts; it wasn't the goal. The goal was to get as lean as possible while retaining most all of my muscles. Although it worked, it wasn't sustainable for me in the long run. Today, I enjoy eating a diet high in both protein and carbohydrate (and moderate in fat); it has helped me build much more muscle while staying relatively lean.



Alex Leaf, MS in Nutrition,
CISSN

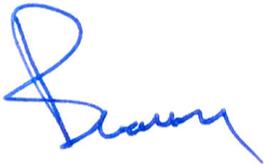
A certified sports nutritionist and personal trainer, Alex Leaf holds a master's degree in nutrition from Bastyr University. He's a full-time senior researcher at Examine.com, involved in updating the supplement database, editing [ERD](#) articles, and [blogging about nutrition](#). Alex also teaches young minds about nutrition and functional medicine at the University of Western States. He enjoys blending the scientific aspects of nutrition with the pragmatic realities of life to help others achieve their goals.

Wyatt

There was a period of about two years, around the early 2010s, when I was fascinated by the Paleo diet. I was looking for ways to deal with a rather painful condition, and many people had stories about the Paleo diet helping with *their* painful conditions. So, I tried it out and kept on reading and discussing.

My initial Paleo diet wasn't ketogenic, but it was naturally lower in carbohydrates than I was used to. The Paleo sphere also overlaps considerably with the low-carb and keto spheres, so I heard a lot about the keto diet and became interested in giving it a try. While I didn't have diabetes, didn't need to lose weight, and certainly didn't have epilepsy, anecdotal reports of keto benefits extend to probably all other health conditions out there, so I rolled the dice to see what it could do for me ... sort of.

I ate about 40 grams of carbohydrates daily for at most two weeks and didn't really notice any effects on pain or mood. While that's probably an insufficient amount of time to test a diet, I was never very sold on keto in the first place, and there certainly wasn't any scientific research on it for my condition or even a good rationale. I also really love fruit, so ketosis was quite a drag to maintain.



Wyatt Brown, World's most interesting man

Wyatt enjoys searching for ways to improve his health and, frequently confused by the conflicting messages from publications and popular authors, dove headfirst into the scientific research and became fascinated by its logic and methods. Contributing to his most respected website has only intensified his interest and motivated him to pursue an education in nutrition.

KETO 101:

THE
PRACTICE
OF KETO

Chapter 1: The Basics of Keto

In this chapter, you will learn [what a keto diet is](#), [what ketones and ketosis are](#), and [how you can measure your ketone levels](#) to make sure you really are in ketosis.

What is a keto diet?

Your body breaks down the carbs you eat into glucose, which it uses as fuel. If you don't eat carbs, your body uses fat to create another kind of fuel: [ketones](#). **A ketogenic diet is, literally, a ketone-producing diet.** It is low in carbs (less than 50 grams per day) and high in fat (usually more than 60% of daily calories).

As a rule, the less carbohydrate (and, to a lesser extent, the less [protein](#)) you consume, the more ketones your produce. Getting a higher proportion of your fat from *medium-chain triglycerides* ([MCTs](#)) will also increase ketone levels.

Is “keto” the same as a “low-carb”?

A keto diet is an extreme type of low-carb diet. In practice, whether your diet is called “low-carb” or “keto” depends on how little carbohydrate you consume each day:

- Between 50 and 150 grams, your diet is **low-carb**.
- Under 50 grams, your diet is **ketogenic**.

Those numbers, proposed by a group of prominent low-carb scientists,¹ have been echoed in other scientific publications.²

In studies, a low-carb-but-not-keto diet is usually called a *low-carbohydrate diet* (LCD), whereas a keto diet is sometimes called a *low-carbohydrate ketogenic diet* (LCKD) or *very-low-carbohydrate ketogenic diet* (VLCKD). There is no difference between LCKD and VLCKD.

Table 1: Carbohydrate intake for different diets

DIET				AVERAGE US INTAKE
Ketogenic	Low-carb	Moderate-carb	High-carb	
<50 g/day	50–150 g/day	40–65%	>65%	49%

% = percentage of total daily calories

References: Wylie-Rosett et al. *Curr Diab Rep*. 2013. PMID:[23266565](#) • Ford and Dietz. *Am J Clin Nutr*. 2013. PMID:[23426032](#) • Westman et al. *Am J Clin Nutr*. 2007. PMID:[17684196](#)

Note that “under 50 grams to reach keto” is only a rule of thumb. Depending on the individual, the “carb threshold” below which ketone levels rise could be as low as 30 grams or as high as 80 grams.

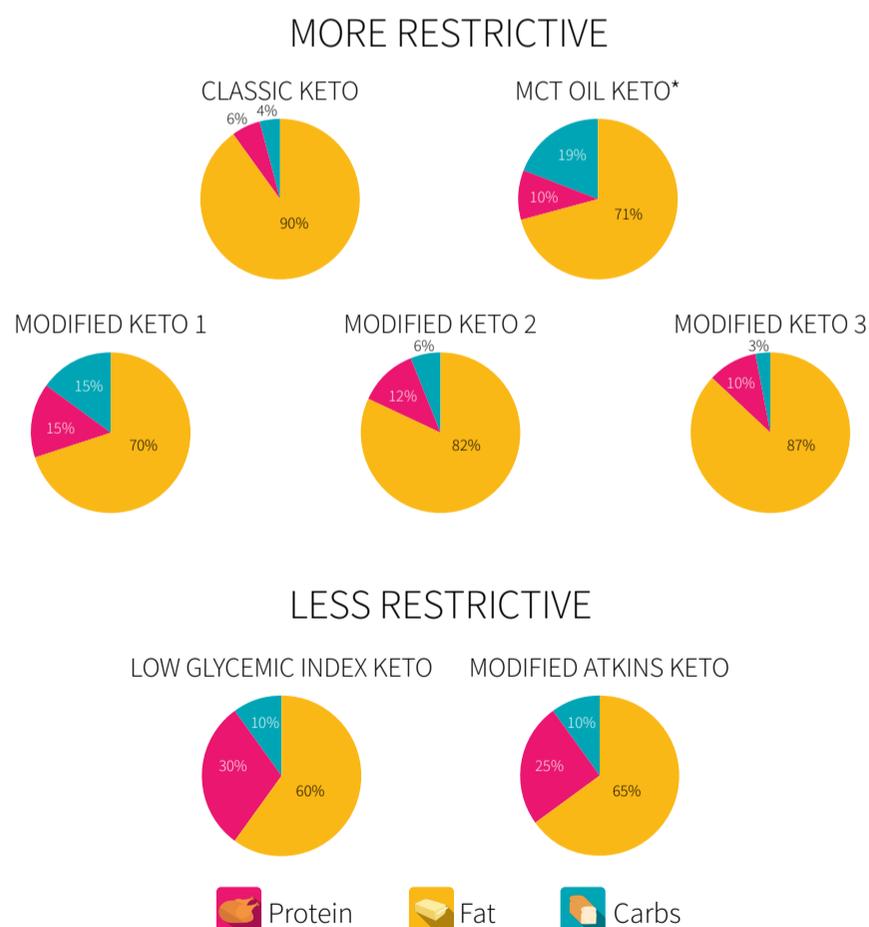
Is there only one type of keto diet?

A keto diet is, in essence, pretty simple: **eat less than 50 grams of carbs per day**. And yet, there isn't one or two but several types of keto diet, which differ either by their macronutrient ratios and sometimes food types (let's call those types **variants**) or by their carb-intake timing (let's call those types **approaches**).

Variants of the keto diet

Variants of the keto diet differ by their macronutrient ratios and sometimes food types (Low Glycemic Index Treatment, for instance, restricts you to low-GI carbs). The figure below depicts the seven most common variants.³ As a rule, people follow one of the “more restrictive” diets only if it was prescribed to them to treat a specific condition, such as drug-resistant [epilepsy](#). Most people will follow one of the “less restrictive” variants, or simply make sure to eat less than 50 grams of carbs per day.

Figure 1: Variants of the ketogenic diet



* Fat is 50% *medium-chain triglycerides* (MCTs) and 21% *long-chain triglycerides* (LCTs).

Reference: Payne et al. *Epilepsia*. 2011. PMID:[22004525](#)

Approaches to keto dieting

Approaches to keto dieting differ in how strictly you follow the “<50 g/day” rule. Here are the three main approaches:

Standard Ketogenic Diet (SKD)

An SKD is maintained all of the time — indefinitely. It can be initiated **quickly**, through fasting or immediate carbohydrate restriction, or **gradually**, by eating less and less carbohydrate every day until the keto threshold (<50 g/day) is reached. The quick options will produce ketosis faster, but the gradual option may be more tolerable, and once you’re on a keto diet, [how you got there](#) won’t matter.

Cyclical Ketogenic Diet (CKD)

A CKD cycles between days when you eat keto and days when you don’t. Weekly cycles are the most common: 5 or 6 keto days, then 1 or 2 non-keto days each week (people who have 2 non-keto days a week usually have them in succession). Depleting your carb stores may take several days, as [we’ll see](#), so if your successive keto days are too few or if you eat too much carb on your non-keto days, you may not reach the degree of ketosis you want (i.e., the blood levels of ketones you want), or not for as long as you’d like.

Targeted Ketogenic Diet (TKD)

A TKD allows for a small amount of carbs just before a workout. The idea is that you should get just enough carbs to fuel your workout without getting kicked out of ketosis. TKD has [three potential advantages](#): it may improve exercise performance, it can help refill your glycogen stores, and it provides you with a time when you can get away with a “carb treat” (which may also help motivate you to go to the gym).

Which keto diet is best?

Which keto diet is best *for you* depends on your lifestyle and medical considerations. In general, the best [variant](#) and [approach](#) are simply the ones you can stick with.

Is keto the best diet for fat loss?

As a fat-loss diet, [the keto diet isn’t inherently superior](#). Different diets work differently for different people, but **your best fat-loss diet** will have at least two qualities: it’ll be **hypocaloric** (it’ll make you eat less than you burn) and **sustainable** (it’ll fit *your* food preferences and lifestyle well enough that you can stick to it).

Despite the constraints of keto, some people find hypocaloric keto more sustainable than other hypocaloric diets. Some people simply have a preference for [the foods allowed on a keto diet](#). Others may find keto especially sustainable because it reduces their appetite.

Will a keto diet suppress your appetite?

One of the more common anecdotes you hear from people who have tried keto is how well this diet suppresses their appetite, and studies tend to concur: participants on *ad libitum* keto diets (i.e., people who were put on a keto diet but allowed to eat as much or as little as they pleased) do report a decrease in feelings of hunger and desire to eat.⁴

Let's mention three possible reasons:

- People who start eating less carbohydrate usually start eating more protein ([the most satiating](#) of the three macronutrients⁵).
- Ketones may also have an appetite-reducing effect, although to what degree is still an open question.⁶
- Some people find high-fat foods more satiating than high-carb foods; this is very subjective.

Unfortunately, the appetite-suppressing effect may decrease with time. Compared to other diets, low-carb diets (including keto) tend to promote greater weight loss during the first months, but not after one year.⁷

How much fat will you lose?

If you want to lose weight, you need to eat fewer calories than you burn. How much weight you'll lose depends on the extent and duration of your caloric deficit. Moreover, if you want most of your weight loss to be in the form of fat, not muscle, you also need to [get enough protein](#) and preferably to exercise.



Tip: Calculating your caloric needs

Your height, weight, age, and level of physical activity all contribute to your caloric needs. There are many calorie calculators out there, but one does stand above the rest:



This calculator has been tested and validated against real-world data.⁸ It can estimate the number of calories *you* need to reach then maintain a specific weight. Click on the image above to get going!

Note that ketone levels and fat loss are not correlated. In other words, **higher ketone levels DO NOT mean greater fat loss.**

What are ketones and ketosis?

As we saw at the start of this chapter, a ketogenic diet is, literally, a ketone-producing diet. Your immediate purpose when you follow a keto diet is to produce enough **ketones** to enter a state of **ketosis**.

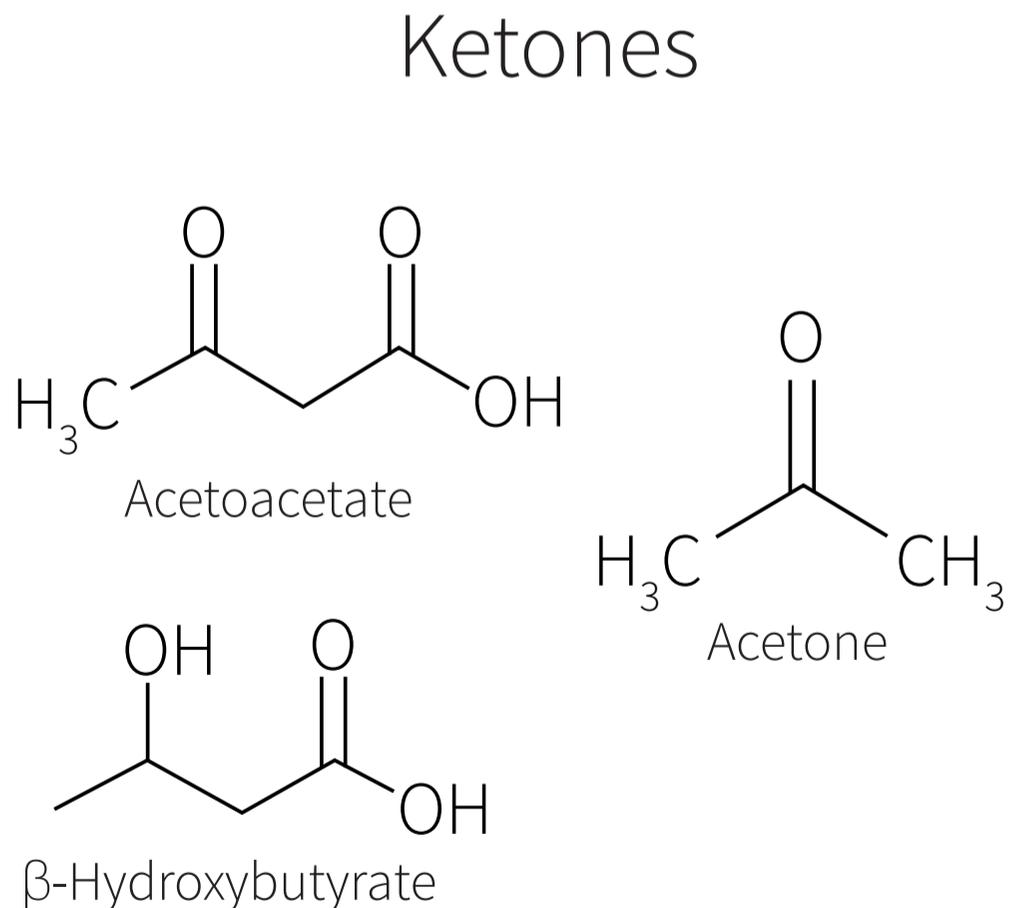
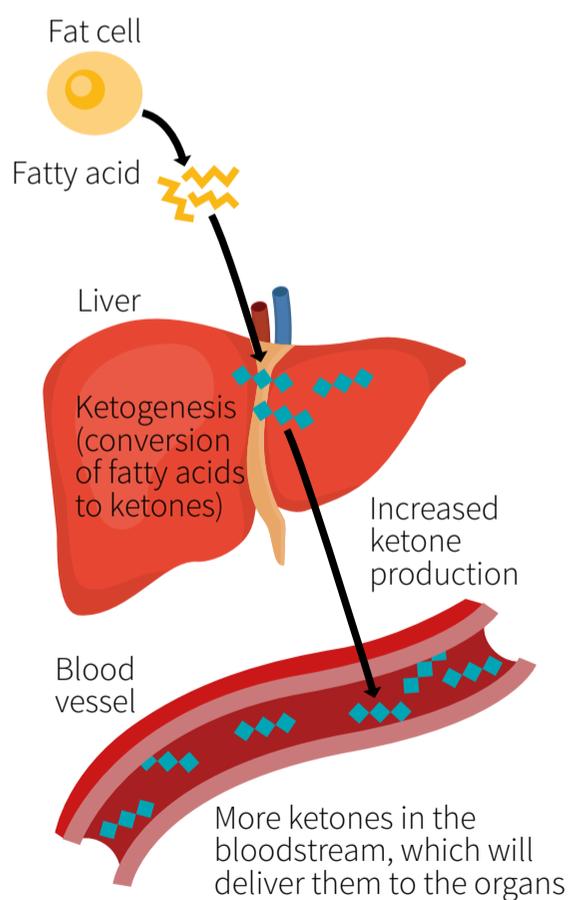
What are ketones?

Through a process called ketogenesis, your body can break down fat (primarily in the liver) to produce three types of ketones:

- Acetone
- Acetoacetate
- β -hydroxybutyrate (β HB)

Ketones are water-soluble, so your liver can easily release them into your bloodstream, which can then deliver them wherever they're needed. When glucose is scarce, ketones replace it as fuel for your organs and tissues (notably your brain), so it makes sense that ketone availability should increase when glucose availability decreases, as happens when you drastically reduce your carb intake.

Figure 2: Ketone production



What is ketosis?

You are in ketosis (also known medically as *hyperketonemia*) when your blood levels of ketones are high. How high?

Under normal, mixed-diet conditions, you usually have 0.1 millimoles of ketones per liter of blood, or less (≤ 0.1 mmol/L). A state of ketosis is generally defined as >0.2 mmol/L,⁹ but most studies use ≥ 0.5 mmol/L as their practical threshold. (Unless otherwise specified, when this guide mentions ketone levels, it mentions [blood](#) ketone levels.)

Your ketone levels can vary even if your diet doesn't, because they depend on your production (ketogenesis) *and* utilization (ketone uptake by your tissues). Higher levels could be due to increased production or decreased utilization, and likewise, lower levels could be due to decreased production or increased utilization.

Note that, when we speak of ketosis in this guide, we speak of **nutritional ketosis** — a kind of “low level” ketosis anyone can reach through carb restriction. This kind of ketosis shouldn't be confused with **ketoacidosis**, a life-threatening condition where blood ketone levels exceed 12–15 mmol/L. You cannot reach such high levels just by excluding all carbs from your diet, fortunately; you need to suffer from a [medical condition](#).

How do you know if you're in ketosis?

Subjectively, some people have reported being able to “feel” when they're in ketosis. While this is plausible, the reliability of this feeling is likely to vary greatly from person to person.

Somewhat objectively, you can ask someone if you have the “[keto breath](#)”. When the ketone acetone is excreted through your breath, it produces an odor that has been described both as “nail polish remover” and “slightly fruity”.

Objectively, the only way to make sure that you're in ketosis is to measure your ketone levels via a [blood](#), [breath](#), or [urine](#) test.

Do you need to measure your ketone levels?

No, but doing so can give you some level of assurance that you really are in ketosis. This is useful for two reasons:

- The “under 50 grams to reach keto” is only a rule of thumb. Depending on the individual, the “carb threshold” below which ketone levels rise could be as low as 30 grams or as high as 80 grams. To know your personal threshold could be useful.

- Assessing your macronutrient intake can be tricky,¹⁰ especially since nature itself doesn't use a precise scale. You may know how much carbohydrate there is in the [average carrot](#), but the specific one you eat may have a little less or a little more, and small discrepancies can accumulate.

Measuring your ketone levels may be a way for you to obsess less about your by-the-scale food intake. But don't obsess about your ketone levels instead! If anything, your focus should be on the general **composition** and **quality** of your diet.

How do you measure your ketone levels?

You can measure your ketone levels through a **blood**, **breath**, or **urine** test.

Blood ketones

β -hydroxybutyrate (β HB) is the predominant ketone in your blood. Most blood ketone meters only test for β HB, so most often "blood ketone levels" actually means "blood β HB levels".

Practically speaking, blood β HB levels of ≥ 0.5 mmol/L indicate a state of ketosis. Unless you are on a medically prescribed keto diet, your ketone levels don't really need to be higher than this. Note that individual results will vary: some people eating less than 50 grams of carbs per day might even see levels barely over 0.2 mmol/L (in which case you still are in ketosis, but a milder ketosis).

Pros

- A blood test can be taken at any time (unlike a urine test).
- A blood test is more precise, accurate, and reliable than a breath or urine test.
- A blood test reflects your current ketone production (whereas a urine test reflects your *past* ketone production).

Cons

- A blood test requires a finger prick.
- The blood ketone meter, testing strips, and lancets can be expensive.

Considerations

- Samples can be taken at any time of day, but readings taken during or after exercise can be misleading. When you exercise, ketone production increases (leading to higher blood levels), but so does ketone utilization (leading to lower blood levels). As a result, your blood levels could be higher or lower than your daily normal, and change very quickly. Be sure to take your readings at rest, either before or at least one hour after your workout.

- A caloric deficit can increase your ketone levels, which may then take about a week to normalize.

Tip: How to use a blood ketone meter

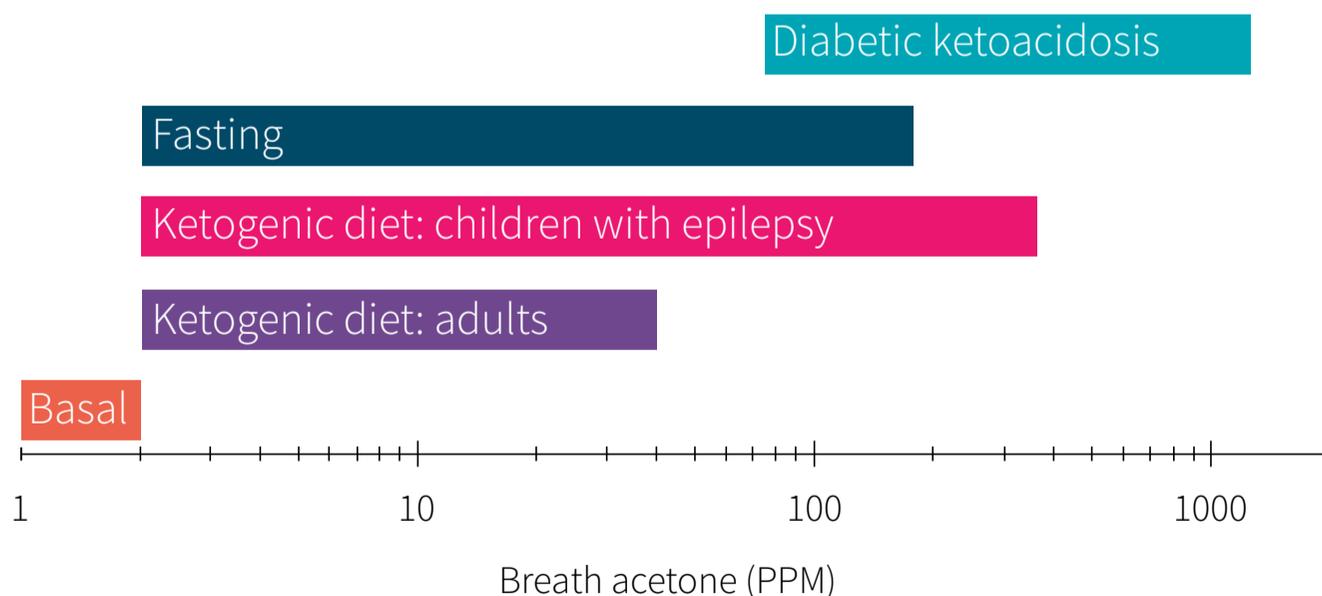
There are a few procedures to follow when measuring blood ketone levels.

- First, lay out everything you will need: ketone meter, lancet (used to draw blood), alcohol swab, ketone blood strip, and a tissue.
- Sterilize your fingertip with the alcohol swab.
- Turn on the meter and insert the strip.
- Place the lancet at the tip of one of your fingers and press the button to depress the needle tip.
- After the lancet has pricked your finger, a drop of blood should appear. If it doesn't, or if it is too small, try massaging your finger, but be careful not to touch your blood, or you risk contaminating the sample.
- Place the correct end of the strip right on the edge of the blood drop. The strip should easily suck up enough blood from the drop.
- Use the tissue to clean your finger while you wait for the machine to process the blood sample. Your reading should appear fairly quickly.
- If no reading appears, you may not have drawn out enough blood. Repeat the steps above with a new strip on another fingertip.

Breath ketones

Breath ketone meters measure the acetone in your breath. Breath tests aren't as accurate as blood tests but correlate fairly closely.¹¹ Blood β HB levels of 0.5–3.0 mmol/L correspond to a breath acetone levels of 4–30 ppm (parts per million).

Figure 3: The breath acetone spectrum



Adapted from Anderson. *Obesity (Silver Spring)*. 2015. PMID:[26524104](https://pubmed.ncbi.nlm.nih.gov/26524104/).

Pros

- A breath test can be taken at any time (unlike a urine test).
- A breath test is non-invasive (unlike a blood test).
- A breath test can be taken easily on the go (unlike a blood or urine test).
- A breath test reflects your current ketone production (whereas a urine test reflects your *past* ketone production).

Cons

- A breath ketone meter can be expensive.
- A breath test isn't quite as accurate as a blood test. Readings can vary depending on many factors, such as weight loss, a sudden change in carb intake, or simply how much breath you exhale.

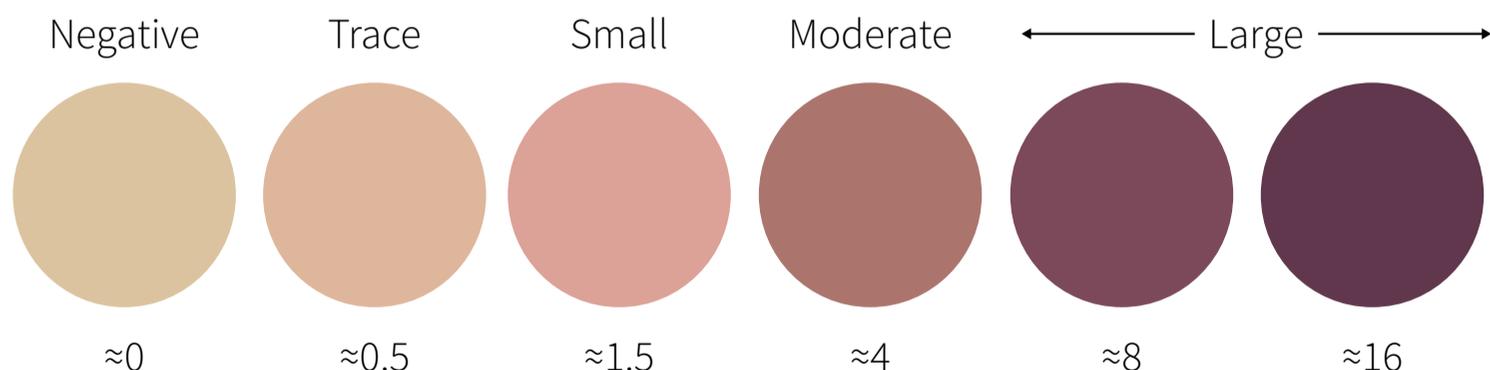
Considerations

- A sudden increase in carb intake — as little as 20 grams — can throw off your readings for 5 hours or more.¹¹ So if you practice a Cyclical Ketogenic Diet (CKD) or Targeted Ketogenic Diet (TKD), where carb intakes vary constantly, a breath test may not be ideal.
- Exercise can elevate breath acetone levels for a few hours post-workout.¹¹ Be sure to take your readings at rest before your workout.
- A caloric deficit can increase your ketone levels, which may then take about a week to normalize.

Urine ketones

A urine ketone test requires that you expose a chemically coated paper strip to your urine, wait for the strip to change color (for 15 seconds, usually, but read your product's instructions), then compare the strip's color to a color chart typically printed on the bottle the strips came in. You need to compare quickly, because the strip's color can keep changing.

Figure 4: Color chart results for urine ketone levels (mmol/L)



Pros

- The test strips are very small and portable.
- The test strips are usually very inexpensive.

Cons

- The test strips can become defective if exposed to moisture or damp environments.
- A urine ketone test cannot be taken anywhere (you need access to toilets) or at any time (you need to feel like urinating).
- A urine ketone test doesn't reflect what your ketone levels *are*, but rather what they *were* an hour or so ago.
- A urine ketone test reports only *approximate* ketone levels.
- The results of a keto test vary with fluid intake.¹² The less hydrated you are, the less diluted the ketones, the higher the reported levels. In general, it means that if your urine is especially dark, your ketone levels will be skewed up, whereas if it is especially clear, your ketone levels will be skewed down.
- Excess fluid can dilute urine ketones, thus lowering your levels, whereas dehydration can elevate them.

Considerations

- The best time of day to test urine ketone levels is usually in the morning, before breakfast, or late in the evening, at least two hours after your last big meal.¹³
- Readings taken after exercise can be thrown off. Be sure to take your readings at rest or at least two hours after exercise.
- A caloric deficit can increase your ketone levels, which may then take about a week to normalize.
- If a strip doesn't change color, it may have become defective due to previous exposure to moisture.¹² Keep the strips in a dry environment.

Chapter 2: Eating Keto

On a keto diet, you should eat very little [carbohydrate](#), a lot of [fat](#), and enough [protein](#). We'll talk about all three macronutrients, then consider if a [vegan or vegetarian keto diet](#) is realistic. Finally, we'll have a closer look at [five specific foods](#) people interested in keto often ask about: alcoholic drinks, artificial sweeteners, coconut oil, eggs, and red meat.

Carbohydrate

In a guide dedicated to a type of diet that consists of eliminating nearly all carbs, you may be surprised to hear this, but **carbs are not *inherently* bad**.

Cutting down on your carb intake (especially on overly processed carbs, which tend to be high in calories and poor in macronutrients) is always a good idea, but if a keto diet makes you eat worse or feel worse or if you really can't stick with it, [you should consider other options](#).

How much carbohydrate can you eat?

“Less than 50 grams per day” is only a rule of thumb, as the degree of carbohydrate restriction required to elevate ketone levels vary between individuals. Some people cannot eat more than 30 g/day, while others can eat over 80 g/day. *If you want to know your personal carb threshold, you'll need to both [measure your ketone levels](#) and, of course, monitor your daily carb intake.*

More precisely, you'll need to monitor your daily *net-carb* intake.

Warning: Net carbs

When we talk of carbs in this guide, we actually talk of [net carbs](#).

The net-carb content of a food is its carb content minus its fiber content. In other words, **fiber doesn't count**. So, for instance, don't shun avocados: they may have 8.6 grams of carbs per 100 grams of fruit (yes, avocados are fruits), but that number includes 6.8 grams of fiber.¹

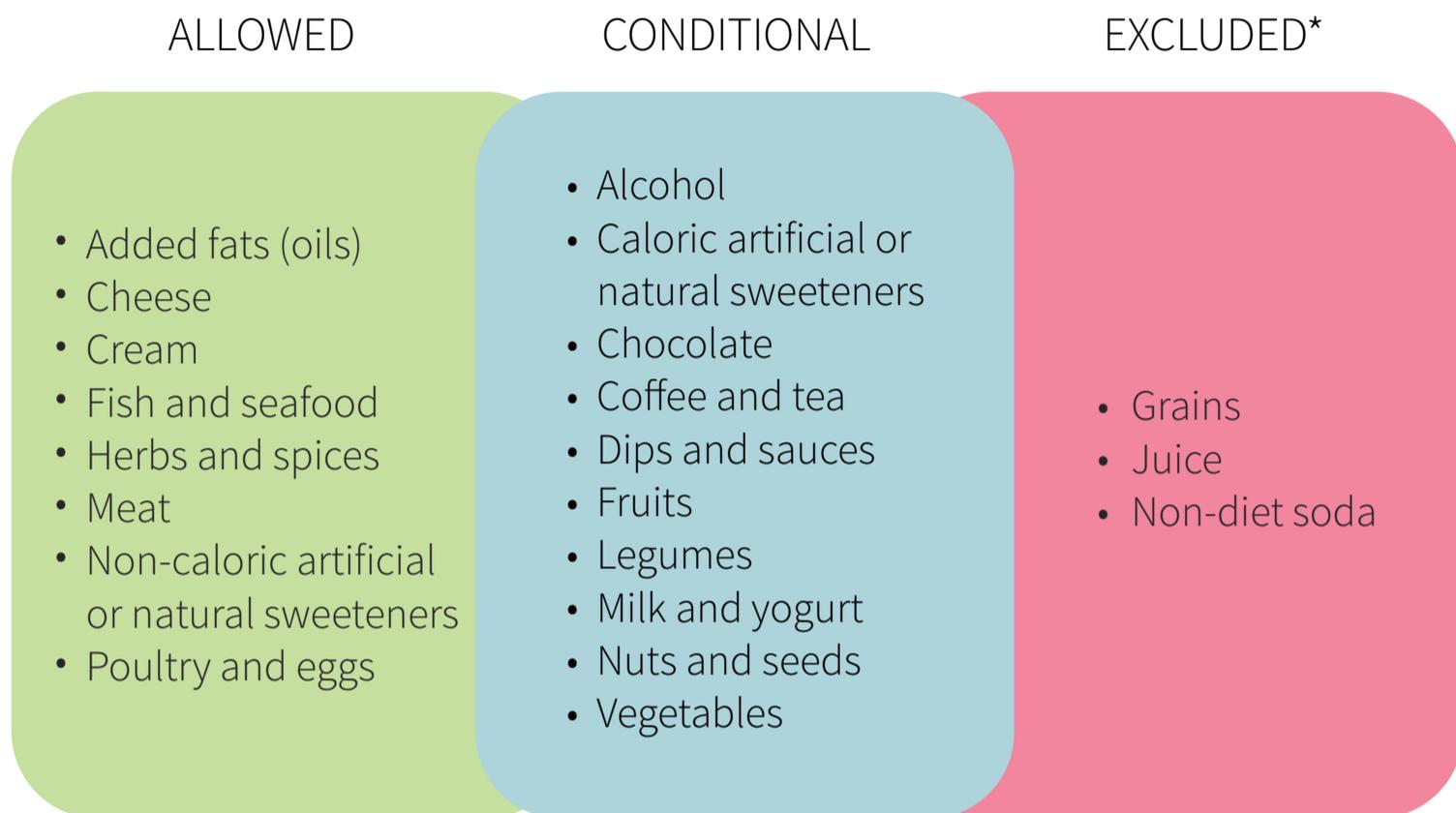
[Fibers belong in a healthy diet](#), so don't avoid them because they're carbs. For the purpose of a keto diet, they aren't.

Foods low and high in carbs

A keto diet requires that you limit your carbs. Foods high in carbs are **excluded**, foods very low in carbs are **allowed**, and other foods can be consumed in moderation. Beware that some food groups combine high-carb and low-carb foods.

If you can eat a certain food depends on its carb content, your daily carb limit, the amount of carbs you've already consumed that day, and your plans for the rest of the day. For instance, if you eat 45 grams of carbs at breakfast and your daily limit is 50 grams, you'll find your food choices for the rest of the day to be very, very limited!

Figure 1: Food choices permissions on a keto diet



* It is possible to include small amounts of these in a keto diet, though not very practical.

Vegetables are a prime example of **conditional** food. Your keto diet should be rich in low-carb veggies (leafy greens, peppers ...) but poor in tubers (potatoes, parsnips ...). Another example is chocolate: most chocolate on the market is loaded with sugar, but dark chocolate can have very little sugar, and you'll find chocolate made with non-caloric sweeteners.



Tip: Low-carb friendly foods

Knowing which foods may be low in carbs can take a little bit of learning. To make this easier, we've included tables of low-carb foods (in [Appendix A](#)) and tables of low-carb foods rich in nutrients commonly underconsumed on a keto diet (in [Appendix B](#)). If you don't know where to start, have a look there to get some ideas.

Fat

Like carbs, fats are not universally bad. Even the much maligned [saturated fat isn't inherently harmful](#), though too much of it may have a detrimental effect on your cholesterol profile. In fact, [trans fat](#) is the only fat that has been shown to be categorically

detrimental to health — a little won't kill you ([CLA](#), a naturally occurring trans fat, has even been investigated for its potential health benefits), but it will do harm.²

Saturated fat

Many people see their *low-density lipoprotein* (LDL, the “bad cholesterol”) increase when they go keto,^{3,4} maybe because they start eating more saturated fat.

Saturated fat doesn't affect everyone's LDL levels the same, though, and LDL itself is a more complicated matter than was once assumed: some forms of LDL have a greater potential for harm than others, and your levels of *high-density lipoprotein* (HDL, the “good cholesterol”) also matter. (See the “LDL and HDL” section in [this article](#).)

Still, since the full implications of elevated LDL levels on a keto diet aren't wholly understood, it would be prudent to try to keep your LDL levels from rising too high.

Warning: Keto and cholesterol

If you currently have an unfavorable lipid panel, you should take extra caution before going keto; but even if you've never had any problem with your cholesterol, you'd be prudent to ask your physician for a baseline lipid panel. Then, after a month on keto, get another lipid panel done to see what has changed.

We address some strategies to handle elevated LDL [in the “Warnings” chapter](#).

Trans fat

Naturally occurring trans fat and industrially produced trans fat seem to have a similar effect on blood lipids,⁵ but you don't need to worry about the minute amounts of trans fat naturally occurring in whole foods.⁶ The trans fat you need to shun is a byproduct of partially hydrogenated oils: this type of trans fat was once a common ingredient of processed foods — so common that trans fat consumption was linked to more than half a million *coronary heart disease* (CHD) deaths worldwide ... just in 2010.^{7,8}

Industrially produced trans fat was banned in the US in 2015, and all products were supposed to be phased out by June 2018, but manufacturers received an extension until July 2019.⁹ That means that a lot of products with this type of trans fat are still on the shelves today.

And you might not even know it by looking at food labels, because the FDA used to allow for a product to be labeled as having 0 grams of trans fat as long as a *serving* of the product had less than 0.5 grams. However, even today, the manufacturer usually gets to decide what a “serving” is, which means that, while a 5-gram serving (maybe a small treat the size of your thumbnail) may have officially 0 grams of trans fat, 100 grams of the product may have 8 grams (if 5 grams of the product contains in fact 0.4 grams of trans fat).

Considering that keto diets are high in fat, trans fat is a greater risk for keto dieters than for the general population. The solution? To **focus on whole foods**.

Is your keto diet full of expertly designed, overly-processed, keto junk food? Are your meals drowning in added fats? Or, are you making sure your fat is coming from mostly whole-food sources like fish, meat, nuts, seeds, and fruits like avocado and olives? The **composition** and **quality** of your keto diet may have a bigger effect on your health than the carb restriction itself!

Protein

From a keto point of view, you can't get too little carb or too much fat. Protein, however, is another matter: you *need* some in your diet, to provide you with the *essential amino acids* (EAAs) your body needs not just to build muscle but also to synthesize hormones, enzymes, and neurotransmitters; but at the same time, since you removed so much carb from your diet, isn't it possible that you're now getting *too much* protein?

How much protein should you eat?

Does your being on a keto diet play a role in determining [your protein intake](#)? It's hard to say; there simply isn't a lot of data on the topic. A safe assumption is that you need to consume *at least* 1.2 g/kg (0.54 g/lb) a day^{10,11,12,13} — if you are not in a caloric deficit.

However, many people seem to spontaneously eat less (i.e., decrease their caloric intake) when they switch to a keto diet, thus ending in a caloric deficit. If that's your case, you'll probably need more than 1.2 g/kg to increase or at least preserve your muscle mass.^{14,15,16,17}



Tip: Use our Protein Intake Calculator

Your protein needs hinge on many factors — notably your weight, health goals, and level of physical activity. Based on our research and the data you input, we can calculate your optimal daily protein intake. Click on the image below to get started!

YOUR OPTIMAL PROTEIN
INTAKE:

???

Can you get *too much* protein?

If you remove all carbs from your diet, you'll likely replace some of the carb-calories thus lost with not just fat-calories but also protein-calories. In other words, keto diets tend to be naturally high in protein. But then, isn't there a risk that "all this protein" will kick you out of **ketosis**, or [hurt your bones or kidneys](#)?

Protein and ketosis

Since medically prescribed ketogenic diets require that your ketone levels stay high at all times, they require that you restrict your intake not just of carbs but also of protein. But whereas high protein intakes can *reduce* ketogenesis (the generation of ketones) within the liver,¹⁸ they don't seem to *prevent* it.

In keto dieters (not on a medical keto diet), higher protein intakes may reduce ketogenesis, but to what extent is very uncertain and highly variable.^{19,20,21,22,23,24,25,26,27,28,29,30,31,32,33,34,35,36} The evidence is all over the place, ranging as it does from one study correlating a protein intake of 1.3 g/kg with ketone levels of 0.33 mmol/L³⁰ to a study correlating a protein intake of 2.1 g/kg with ketone levels of 1.80 mmol/L.²¹ (As a reminder, a state of ketosis is generally defined as >0.2 mmol/L,³⁷ but most studies use ≥ 0.5 mmol/L as their practical threshold.)

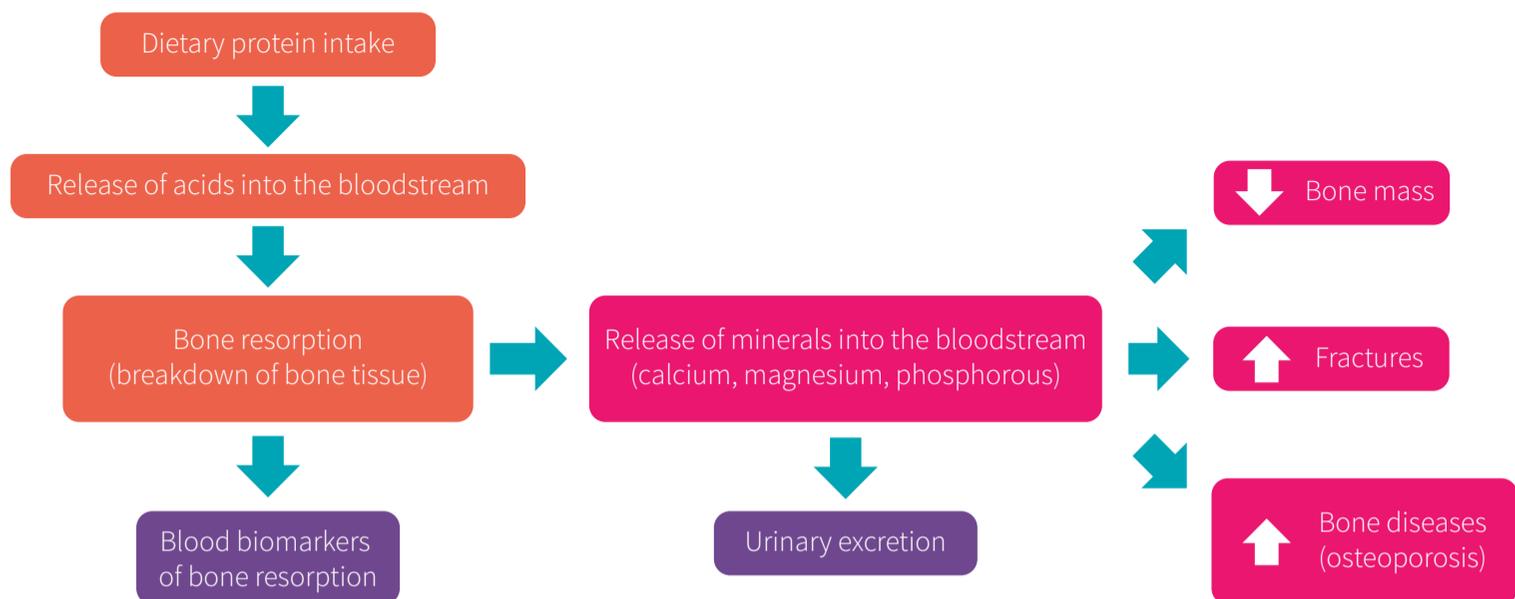
If you want to evaluate the effect of your protein intake on your ketogenesis, start at 1.2 g/kg, [measure your ketone levels](#) for at least a week (starting from no earlier than the third week after you switched to a keto diet, to ensure that your body has had time to adapt), then increase your protein intake little by little each week, and see how your ketone levels are affected.

Protein and bone health

Your bones are composed of minerals (mainly calcium, magnesium, and phosphorus) attached to a structural matrix made of collagen, a protein. For that reason, dietary protein should benefit bone health.

However, protein metabolism increases the amount of acid in the blood.³⁸ Your bones can neutralize this acid, but at the cost of releasing some of their minerals, which get excreted in the urine.³⁹ It would explain why more protein in the diet has been linked to more calcium in the urine.⁴⁰

Figure 2: Mechanism through which dietary protein might hurt bone health



Adapted from Carnauba et al. *Nutrients*. 2017. PMID:28587067.

Except that ... most studies that looked at protein intake and calcium excretion list dairy products as a protein source,⁴¹ and milk is rich in calcium. Therefore, more calcium in the urine could simply be the consequence of more calcium in the diet (i.e., more calcium in, more calcium out).

Therefore, looking only at calcium *excretion* wasn't enough. Subsequent studies have shown that dietary protein promotes dietary-calcium absorption⁴² and that a diet high in protein “promotes bone growth and retards bone loss[, whereas a] low-protein diet is associated with higher risk of hip fractures”^{43,44}

All in all, the current body of evidence suggests that **protein actually has a neutral or even protective effect on bones.**^{43,44,45}

Protein and kidney health

Because high-protein diets increase *glomerular filtration rate* (GFR), a marker for waste filtration in the kidneys,⁴⁶ they were hypothesized to put undue stress on the kidneys.⁴⁷ Later research, however, has shown that **high-protein diets do no harm to healthy kidneys.**⁴⁸ If your kidneys are damaged, however, dietary protein may accelerate their decline in function.^{49,50,51}

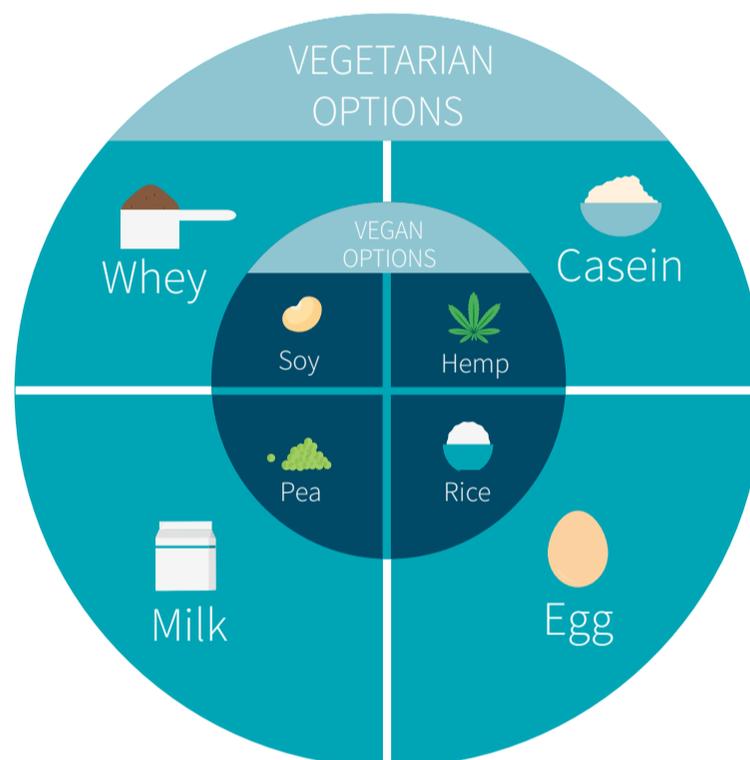
Can you be vegan or vegetarian *and* keto?

Low-carb diets tend to rely heavily on animal products for carb-free fat and protein, but one group of researchers set out to modify the popular Atkins Diet into a vegan alternative, using gluten, nuts, soy products, and vegetable oils for much of the protein and fat.⁵²

In their study, they compared their “Eco-Atkins” diet (25% carbs, 31% protein, 43% fat) to a high-carb lacto-ovo-vegetarian diet (58% carbohydrates, 16% protein, 25% fat). For the first month, they fed the participants; for the next six months, they let the participants select their own diet-appropriate foods. At the end of the study, the Eco-Atkins dieters had lost slightly more weight (1.1 kg, so 2.4 lb) and had greater lipid improvements, including a greater decrease in LDL (the “bad cholesterol”).

Obviously, “Eco-Atkins” isn’t low-carb enough to be keto, but it is the closest thing to a vegan keto diet to have ever been studied. It suggests that a vegan keto diet is at least possible with ample fat from avocados, olives, coconuts, nuts, and seeds, and their associated oils. Vegan protein sources are often high in carbs, however, so you may have to supplement whole foods with plant-based protein powders, such as pea protein isolates and soy protein isolates.

Figure 3: Vegetarian- and vegan-friendly sources of protein



A vegetarian keto diet will be less challenging than a vegan keto diet, of course, but still, keep in mind that keto by itself is already very restrictive. By cumulating food restrictions, you increase the risk not only of making the diet unbearable but also of underconsuming nutrients critical for your health.

Specific foods

The five foods people interested in keto most often ask about are **alcoholic drinks**, **artificial sweeteners**, **coconut oil**, **eggs**, and **red meat**.

Alcoholic drinks

Will alcohol (ethanol) kick you out of ketosis? No. But many alcoholic drinks will.

- Mixed drinks often include a high-carb ingredient — soda or fruit juice.
- Non-distilled fermented drinks (e.g., beer, cider, wine) all contain carbs, though the actual content varies greatly.
- Distilled fermented drinks (e.g., brandy, gin, mezcal, rum, tequila, vodka, whisky) are virtually carb-free and can actually increase ketogenesis, as your body strives to convert the ethanol to ketones. While this happens, however, your body is burning less fat (remember, alcohol contains 7 kilocalories per gram).

As always, keep your alcohol intake moderate.

Artificial sweeteners

Will artificial sweeteners kick you out of ketosis? No, and neither will natural nonnutritive sweeteners, such as the steviol glycosides extracted from [Stevia rebaudiana](#) or the mogrosides extracted from *Siraitia grosvenorii* (a.k.a. monk fruit and *luo han guo*).

Coconut oil

[Coconut oil](#) is one of those foods the [media](#) (and even the occasional [Harvard professor](#)) loves to sensationalize. So, [is coconut oil a superfood or a poison?](#)

Coconut oil may have gotten its original “superfood” status from its [medium-chain triglycerides](#) (MCTs), a subtype of [saturated fat](#) that can promote [ketogenesis](#) and might promote [satiety](#) and [weight loss](#). But although rich in MCTs *compared to other oils*, [coconut oil is less than 13% MCTs](#).

- Coconut oil is 0.5% **caproic acid** (C6:0).
- Coconut oil is 6.8% **caprylic acid** (C8:0).
- Coconut oil is 5.4% **capric acid** (C10:0).
- Coconut oil is 41.8% **lauric acid** (C12:0).

MCTs have 6–10 carbons.⁵³ Lauric acid, having 12 carbons, is a [long-chain triglyceride](#) (LCT). You may have heard that coconut oil is half MCTs, but that’s because lauric acid is sometimes called an MCT, even in scientific papers.⁵⁴ However, although lauric acid does

display properties intermediate between MCTs and LCTs,⁵⁵ it's still more of an LCT,^{56,57} and it hasn't been shown to affect ketogenesis or weight loss as do true MCTs.

Human studies on MCTs that reported benefits for ketogenesis or weight loss used 10–42 grams a day of purified MCT oil.^{58,59} To get that amount from coconut oil, you'd need to ingest 79–331 grams of coconut oil, worth 705–2,953 kcal.

Even the lower end of this range is way too high, and not just because there is preliminary evidence that high intakes of coconut oil may cause an increase in your LDL (your “bad cholesterol”), which in turn can cause an increase in your risk of heart disease.⁶⁰ Remember that coconut oil is a refined product and lacks many of the components present in the flesh of this fruit (such as polyphenols, fiber, and vitamin E). Coconut oil isn't a poison, but neither is it the superfood the media often made it to be; you can incorporate it into your keto diet, but don't go crazy! There is very preliminary evidence that it might support weight loss,⁵⁵ but only if it *replaces* other fats — not if it increases your daily caloric intake.

Eggs

[Eggs can increase cholesterol levels](#), but not in everyone.⁶¹ The risk of an increase is higher in people with specific pre-existing conditions such as [diabetes](#), hyperglycemia, or familial hypercholesterolemia.⁶²

Most people can eat up to 4 eggs a day without worry. Still, [as we saw](#), many people see their *low-density lipoprotein* (LDL, the “bad cholesterol”) increase when they go keto,^{3,4} before you do, ask your physician for a baseline lipid panel. Then, after a month on keto, get another lipid panel done to see what has changed. We address some strategies to handle elevated LDL [in the “Warnings” chapter](#).

Red meat

Claims that red meat is unhealthy are based on observational studies that found a relationship between red meat consumption and both cardiovascular disease⁶³ and death from cardiovascular disease.⁶⁴ However, some researchers argue that this relationship is the product of faulty data collection — the participants hadn't been asked to keep a food log, so they could only rely on often vague memories to fill the form given them, and furthermore, this form didn't differentiate between processed and unprocessed red meats.^{65,66}

Regardless, observational evidence cannot serve to establish cause and effect. But then, of course, randomized controlled trials that evaluate long-term health outcomes (such as dying from a heart attack) are terribly difficult and expensive to conduct. For that reason, we can never be sure that any food is entirely safe, but that doesn't mean we should stop eating altogether, nor does it mean we shouldn't avoid some foods if they seem especially risky.

So, in the end, should you ban red meat from your diet, just to be safe?

Going that far shouldn't be necessary. Current evidence suggests that processed red meats, particularly those that are more charred during cooking, *can* increase the cancer risk of people with poor diets and lifestyles,^{67,68} but if you moderate your red meat intake, exercise regularly, eat your fruits and veggies, consume adequate fiber, don't smoke, and drink only in moderation, the effect of red meat on your health isn't something worth worrying about.

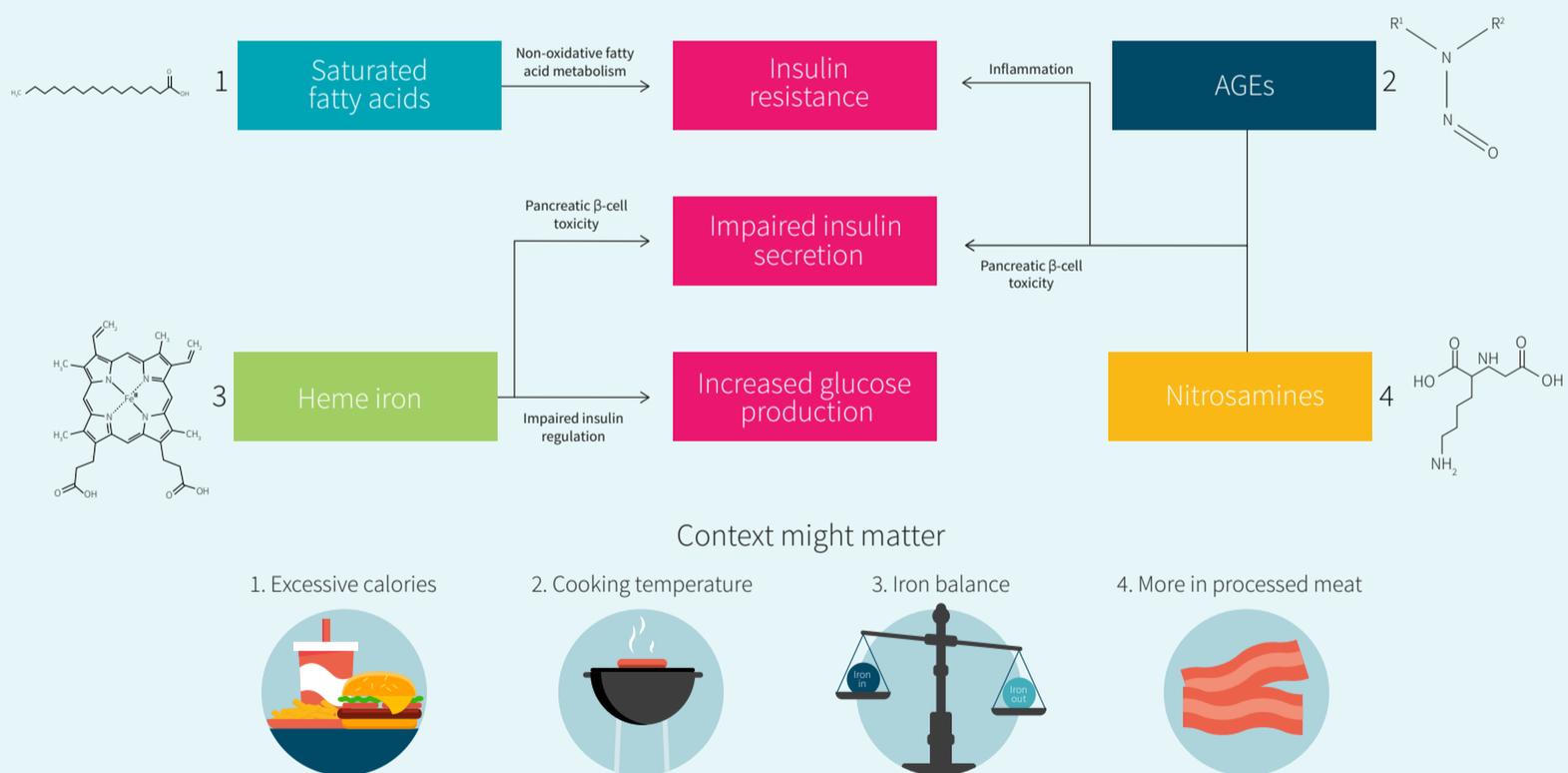
In short, the healthfulness of red meat depends not only on the needs of the individual (notably their iron requirements⁶⁹) but also on the type of red meat, the way it is prepared, and the overall diet it is a part of.

🔍 Digging Deeper: Making red meat healthier

There is some evidence that red meat may exacerbate metabolic diseases, such as diabetes. From a mechanistic standpoint (illustrated below),⁷⁰ eating red meat has the potential to contribute to insulin resistance by increasing your intake of nitrosamines, heme iron, *advanced glycation end-products* (AGEs), *heterocyclic amines* (HCAs), and *polycyclic aromatic hydrocarbons* (PAHs).

If that sounds a bit scary, know that merely changing the way you cook can reduce insulin resistance and other markers of inflammation and oxidative stress, possibly by reducing your exposure to AGEs, HCAs, and PAHs.⁷¹ Notably, you could opt to cook red meat "gently" (through boiling, microwaving, poaching, steaming, or stewing) more often than "harshly" (through baking, frying, grilling, or roasting).⁷²

Figure 4: Possible roles of red meat in type II diabetes



Reference: Kim et al. *Metabolism*. 2015. PMID:25838035

Chapter 3: Starting Keto

First of all, you should [make sure keto isn't contraindicated for you](#). If you have any doubt, talk to your physician. (Actually, even if you don't have any doubt, you should talk to your physician before you go keto, if only to get another opinion and [request a baseline lipid panel](#).)

In this chapter, we'll review [four methods to go keto](#) and estimate [how long it takes](#) to, first, reach ketosis and, second, become keto-adapted.

How should you go keto?

Of the three main methods to go keto — **start high**, **start low**, and **jump to 50** — the first is considered the easiest and safest.

Keep in mind that “under 50 grams of carbs per day” is only a rule of thumb. Depending on the individual, the “carb threshold” below which ketone levels rise could be as low as 30 grams or as high as 80 grams.

Start high, decrease gradually

Start [low-carb](#), then gradually go (down to) keto.

First, drop your daily carb intake to 100–150 grams. Then, over the next week or weeks, remove 20 grams each day (or every other day) until you eat less than 50 grams of carbs per day.

This approach is slow but has two advantages:

- It reduces the risk of suffering from side effects (such as the [“keto flu”](#)).
- It gives you the opportunity to adjust your diet gradually, rather than all at once (which can be overwhelming!).

Start low, increase gradually

This is essentially the **modified Atkins diet** approach (the “modified Atkins diet” is a ketogenic version of the Atkins diet which was created at the Johns Hopkins Hospital¹).

First, drop your daily carb intake to 20 grams. Then, over the next week, add 5–10 grams each day until you eat a little less than 50 grams of carbs per day.

Jump straight to <50

If you're feeling confident, you can cut your carb intake right down to <50 g/day. To succeed, you will need to have a clear idea of what your meals should look like, so as not to consume more carbs than you realize.

Fast for a day

If you want to reach ketosis quickly, a one-day fast can help. Have a meal plan ready to transition from the fast to a keto diet. Planning is everything!

During your fast, remember to stay hydrated. Adding a little salt (i.e., sodium) to your water or zero-calorie beverage may help alleviate some negative effects of the fast, such as fatigue. Note that a keto diet also requires that you keep an eye on your intake of [fluids](#) and [electrolytes](#).

How long does it take to go keto?

It really depends on what we mean by “going keto”. If we mean **adopting a keto diet**, then, as we saw in the previous section, it could take anything from a day to a few weeks (the latter only if you cut carbs down very, very slowly). If we mean **reaching ketosis**, then it should take less than a week. And if we mean **being keto-adapted** (i.e., having a body geared toward burning fat rather than carbs), then it should take less than two weeks.

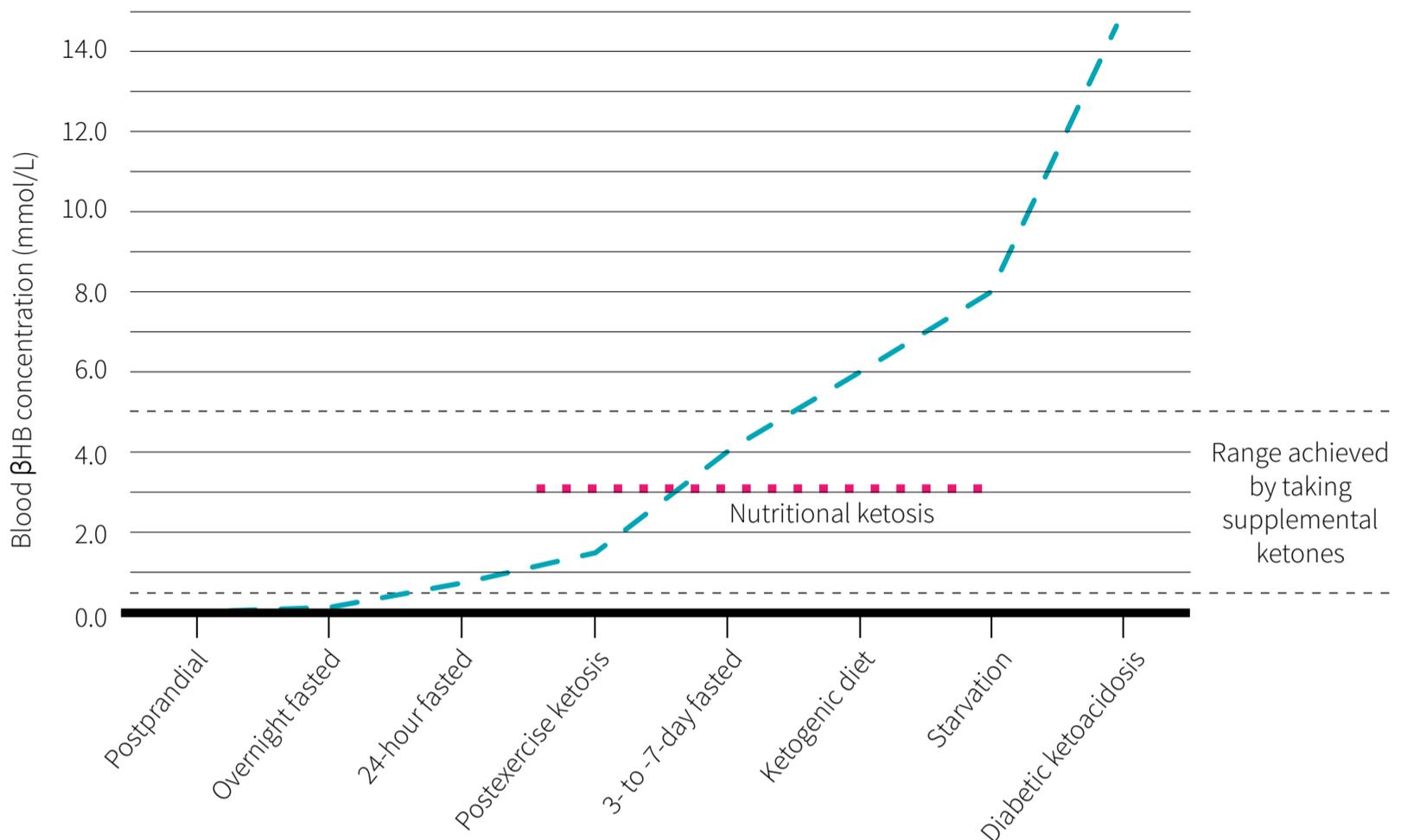
How long does it take to reach ketosis?

How quickly you'll reach [ketosis](#) depends on a number of factors:

- **Carbohydrate restriction.** The lower your carb intake, the sooner [your carb stores will get depleted](#), the quicker you'll reach ketosis.
- **Caloric restriction.** The less you eat, the more your body will need to use its stored carbs to fuel your activities, the sooner your carb stores will get depleted, the quicker you'll reach ketosis. That's why some people kickstart their keto diet with [a one-day fast](#).
- **Physical activity.** The more active you are, the faster you'll empty your carb stores, the quicker you'll reach ketosis.

There is a fair amount of **individual variability**, too, but most people who immediately drop their carb intake down to <50 g/day will achieve ketosis in less than 7 days.

Figure 1: Changes in β -hydroxybutyrate (β HB) levels under various physiological states



Adapted from Evans et al. *J Physiol.* 2017. PMID:[27861911](https://pubmed.ncbi.nlm.nih.gov/27861911/)

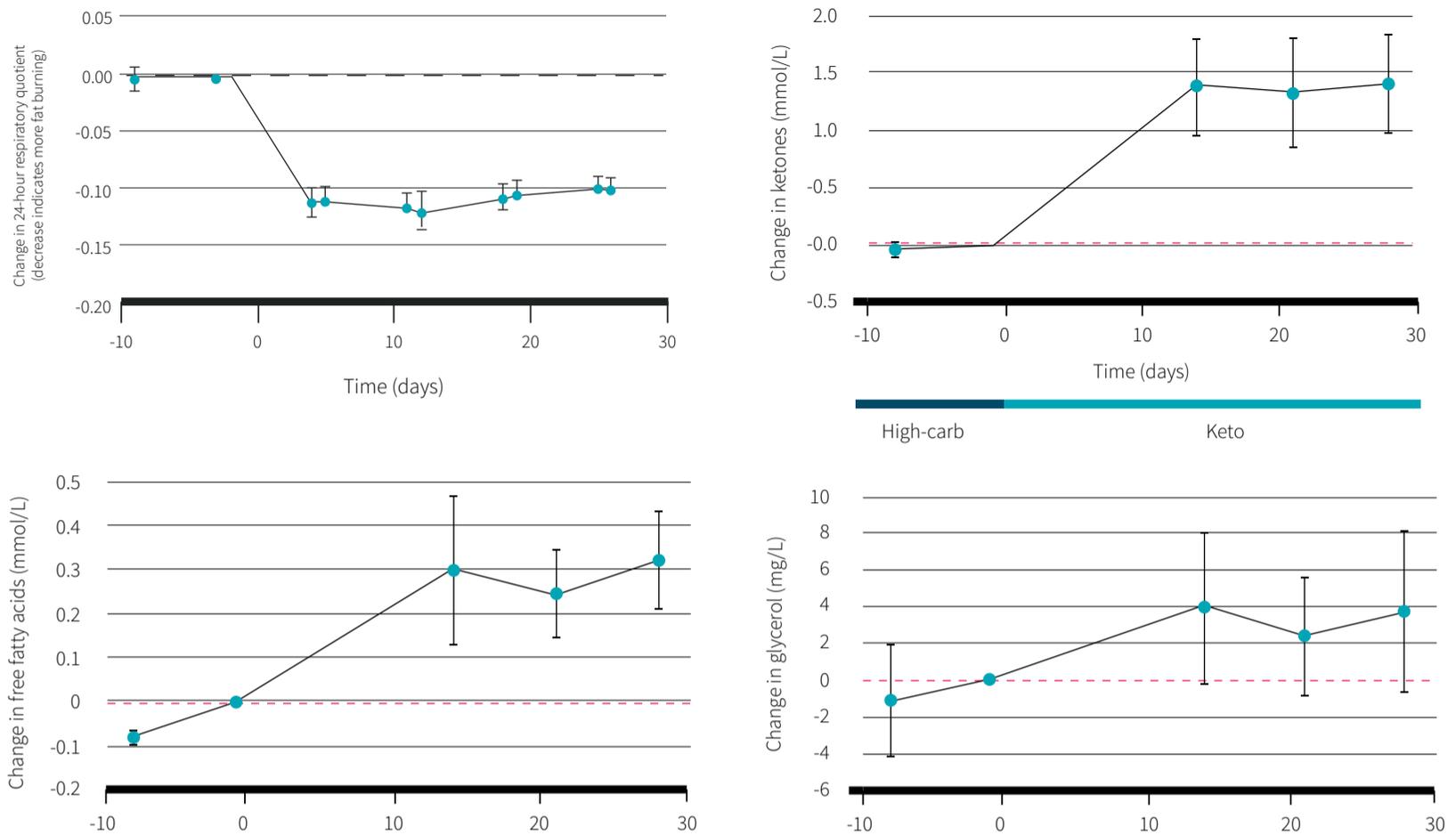
How long does it take to become keto-adapted?

Your body is [keto-adapted](#) when it's optimized for fat-burning rather than carb-burning.

To see how long it takes for your body to switch from carbs to fat as its favored energy source, we can consider three metrics indicative of body-fat breakdown: **fat oxidation**, blood levels of **ketones**, and blood levels of **fat metabolites** (glycerol and free fatty acids).

In a [metabolic-ward study](#) whose participants switched from a high-carb diet to a keto diet, **fat oxidation** maxed out at around day 5, then plateaued, whereas **ketones** and **fat metabolites** maxed out at around day 12 (or earlier, but we don't have earlier measures to confirm this), then plateaued.²

Figure 2: How various measures of fat-adaptation behave once a keto diet is initiated



Adapted from Hall et al. *Am J Clin Nutr.* 2016. PMID:[27385608](https://pubmed.ncbi.nlm.nih.gov/27385608/)

You may have heard that it takes up to one month to become properly keto-adapted. This belief likely stems from the “keto flu” lasting 1–4 weeks, but while it *may* take you that long to feel normal again after switching to a keto diet, your energy systems actually make the transition from preferentially burning carbs to preferentially burning fat rather quickly.

Other biomarkers (e.g., hormones, enzymes) might take longer to fully stabilize after you switch to a keto diet, but further research is needed to ascertain this.

Chapter 4: Maintaining Keto

Is the keto diet especially hard to maintain? That wouldn't be surprising, since it's so restrictive, but we'll see what the studies have to say before we consider what you can do if you want to eat out or if you find a keto diet too hard to maintain.

Is a keto diet hard to maintain?

Diet adherence hinges on many factors, notably these three:

Diet adherence hinges on many factors. In addition to “high internal motivation”, let's mention the POSE factors:

- **Preferences.** If you like the foods your diet allows more than the foods it disallows, you'll find adherence easier. To speak in broader terms: the better a diet fits your lifestyle and food preferences, the more likely you are to stick to it.
- **Organization.** If you prepare diet-appropriate meals and snacks in advance, you'll find adherence easier. (Studies in which meals are provided by the researchers see greater diet adherence.) Relatedly, following any diet usually means spending a little more time planning your grocery shopping. Finally, if you intend to eat out, you'll want to do some research to find restaurants with keto-friendly items on their menu.
- **Support.** If your family is supportive (if only by not leaving in plain sight the foods your diet disallows — see below), you'll find adherence easier. Your friends and colleagues might need to be supportive too (especially if you want to eat out — see above). Having access to a support system (dietician, dedicated online group ...) will also help.
- **Environment.** If you live alone, consider removing all disallowed foods from your place. If you don't live alone and the people you live with don't share your diet, you should at least make sure that disallowed foods don't lie in plain sight and aren't easily accessible, so they won't constantly tempt you. Another risk factor is your TV: anytime you watch it, you're likely to be assaulted with commercials for high-carb foods.

You'll notice we didn't mention hunger. That's because not all diets are hypocaloric, or need to be. Hypocaloric diets (diets that have you eat less than you burn) are useful only if you wish to lose weight. Many diets (Mediterranean, paleo, keto ...) are not intrinsically hypocaloric. Still, it is worth noting that people tend to be less hungry on a keto diet: in studies that didn't limit food intake, people on a keto diet tended to naturally eat less (i.e., fewer calories) than people on higher-carb diets.¹

But in the end, hypocaloric or not, are keto diets easier to adhere to than other diets? To answer this question, we can look at studies whose **participants were randomly assigned to a keto diet** and studies whose **participants chose a keto diet** (and are thus more likely to adhere to it, in theory).

Study participants assigned to keto

An unpublished meta-analysis of 30 keto studies (1,307 participants total) found the average dropout rate to be similar for hypocaloric keto diets and hypocaloric non-keto diets: 24%.²

This number, however, includes only the participants who either declared they dropped out or stopped responding to the researchers; it doesn't include the participants who decided to stick to their diet but failed to do so. In the longer-term studies, we see that blood ketone levels decrease progressively, while reported carb intakes increase; this shows that many participants in the keto groups struggled more and more to eat less than 50 grams of carbs per day. In other words, some of the keto participants who didn't drop out weren't in ketosis at the end of the study.

We also witness this creeping increase in carb intake in the DIETFITS randomized controlled trial.³ This trial was not strictly a keto study, but the 300 participants randomly assigned to the low-carb group were instructed to consume no more than 20 grams of carbs daily for the first 2 months of this 12-month trial, after when they were allowed to stay at these low levels or increase their carb intake to the minimum they felt was sustainable.

Likewise, the 300 participants randomly assigned to the low-fat group were instructed to consume no more than 20 grams of fat daily for the first 2 months of this 12-month trial, after when they were allowed to stay at these low levels or increase their fat intake to the minimum they felt was sustainable.

Neither group was able to stick to the very low starting intakes: by month 3, the low-fat group was already consuming an average of 42 grams of fat (378 kcal) per day, whereas the low-carb group was consuming an average of 97 grams (388 kcal) of carbs per day.

A very small minority of the low-carb participants did choose to stick to a keto-level carb intake and reported consuming less than 50 grams per day by the end of the study, but they tended to report unrealistically low caloric intakes, which suggests they may also have underreported their carb intake (since it wasn't a keto study, blood ketone levels weren't measured).

The [DIETFITS study](#) found the average dropout rates to be similar in the high- and low-carb groups (21%), just as the meta-analysis discussed above found the average dropout rate to be similar in the keto and control groups (24%). The average dropout rates were also similar between the **three groups** of the Framingham State Food Study (which wasn't a keto trial):⁴

- 29.6% in the **high-carb group** (60% of calories as carbs)
- 26.4% in the **moderate-carb group** (40% of calories as carbs)
- 24.6% in the **low-carb group** (20% of calories as carbs)

Importantly, in the Framingham State Food Study, the DIETFITS study, and each of the 30 keto studies in the meta-analysis discussed above, the “competing” diets were equally hypocaloric. On the other hand, the researchers didn't restrict calories in any of the 12 studies of adults with epilepsy (270 participants total) covered by a 2015 meta-analysis. This meta-analysis found that, across the [different types of keto diets](#), the dropout rate was about 55%.⁵ This number is exceptionally high, which may seem strange for two reasons:

- Unlike the diets in the other studies discussed in this section, these keto diets weren't hypocaloric, so hunger wasn't (or shouldn't have been) an issue.
- [The prospect of experiencing fewer seizures](#) should have been a strong motivator for *most* of these participants (some epileptics do not experience fewer seizures on a keto diet; for them, the motivation to stick to keto would be very low).

It isn't surprising, on the other hand, to see lower dropout rates for the modified Atkins diet (44%) than for the classical keto diet (62%). Importantly, it is probable that the modified Atkins diet saw better adherence not just because it is slightly higher in carbs (10% vs. 4%) but also because it is much higher in protein (25% vs. 6%).

Study participants who chose keto

In theory, people who choose to go keto should show better diet adherence than people who were assigned to the diet, but in practice, it may not be the case.

For the [Virta Health open-label trial](#), 349 type II diabetics chose which group they wanted to be in: 262 chose the keto intervention (and received intensive support and monitoring), whereas 87 chose the usual-care intervention.⁶

At the two-year mark, the dropout rates were 35% for the keto group and 28% for the usual-care group (“usual care” means that the patients were given instructions by their usual physicians; the actual instructions differed between physicians; presumably, since the patients were either overweight or obese, caloric restriction was one of the instructions).

As in the trials where participants were randomly assigned to keto, blood ketone levels decreased over time, indicating a decrease in adherence to the “under 50 grams of carb” daily limit. Still, it should be noted that the keto group lost weight (14 kg, so 31 lb, on average) while the usual-care group *gained* weight (5 kg, so 11 lb, on average).

Bottom line

In both types of studies (those whose participants were randomly assigned to a keto diet and those whose participants chose a keto diet), we see a progressive increase in carb intake and a concomitant decrease in ketone levels. In the long term, few participants were able to maintain a keto diet (<50 grams of carbs per day), but many more were able to maintain a low-carb diet (50–150 g/day). The adherence rate for hypocaloric low-carb diets is close to 75%, so no better or worse than the adherence rate for any other type of hypocaloric diet (including the simplest type: the diet with no other restriction than its being hypocaloric).

Note that “few participants” doesn’t mean “no participants”. In nearly all diet trials, as well as in the wider world, there are [individuals](#) who stick to their diet — including keto. The only way to know if keto works for you is for you to try it out.

How can you eat out and stay keto?

When you feel like eating out, you have three main options:

- Just go to the restaurant and hope that something on the menu is keto enough for your needs.
- Just go to the restaurant and treat yourself to a non-keto meal. This approach is valid within the context of a [Cyclical Ketogenic Diet](#) (CKD).
- Do some advance research to find a restaurant that fits your keto needs. If you intend to go out with friends, you may want to prepare a list of several keto-friendly restaurants, to give your friends a choice.

What should you do if you can’t stick to keto?

If you have repeatedly tried and repeatedly failed to keep your daily carb intake under 50 grams, then it may be time to consider a new approach:

Try a *Cyclical Ketogenic Diet* (CKD)

If you've been carb-cycling by accident, why not make it intentional? CKD consists in taking 1 or 2 (usually successive) non-keto days a week, as [we saw](#) in the “Basics of Keto” chapter.

Transition to a low-carb diet

If you struggle with keto (less than 50 grams of carbs per day), consider switching to low-carb (50–150 g/day). Instead of randomly increasing your carb intake, though, increase your daily limit by 5–20 grams, then reassess the situation over a few days. Do you feel that this new carb level is sustainable? If you do, stop there. If you don't, increase your daily limit by 5–20 grams again, and reassess again. Repeat until you hit a level you feel you can sustain indefinitely.

Reassess

You may give it a serious go and still discover that keto — or even just low-carb — simply isn't working out for you. **Don't feel bad about this!** Your efforts weren't wasted. Take stock of what worked well and what didn't:

- **Note what you disliked most** about this specific diet, then try to find a diet pattern that doesn't include those most hated elements!

Note what you enjoyed about this specific diet, then try to incorporate these elements into whatever diet pattern you choose to explore next.

Chapter 5: Troubleshooting Keto

What can you do about the “keto flu”?

In the first 1–4 weeks of trying the keto diet, you may experience fatigue, nausea, bad breath, intestinal discomfort, headaches, brain fog, or other various ailments. This collection of symptoms is commonly referred to as the “low-carb flu” or “keto flu”. Luckily, they are often temporary and we walk you through what steps to take to help prevent or alleviate some of these unwanted side effects.

The keto flu will manifest in different ways to different people, if at all. [As we saw](#), we go into more depth on how to avoid or manage adverse reactions and side effects you may experience on the keto diet. Very briefly, here are some general strategies you may try.

- Increase your [fiber](#) intake, which can help to alleviate constipation.
 - [Psyllium](#) can be a good option due to its high viscosity and low degree of fermentation, making it less likely to produce unwanted side effects. Take 10–15 g/day in divided doses across multiple meals.
- Increase your [fluid](#) intake, which may help to generally alleviate symptoms.
 - Add at least 710 mL (24 oz) of a carb-free beverage to your diet.
- Increase your [electrolyte](#) intake, as many people see intakes drop on keto
 - For **sodium**, try increasing your daily intake to 3–5 g/day. Remember, this is a *total* of 3–5 g/day from foods plus added sodium, not just from added sodium. If you have been prescribed a low-sodium diet, speak with your physician before upping your intake.
 - For **magnesium**, start with 200 mg of *elemental magnesium* once a day. If you don’t notice symptom improvement, you can increase your dose to 350 mg/day.
 - For **potassium**, supplements will likely be insufficient for improving overall potassium intake, as most contain <100 mg (compare that with an RDA of 3,400 mg for adult males and 2,600 mg for adult females).¹ Incorporating more potassium-rich foods into your diet can help maintain adequate levels during your transition to the keto diet. Remember, too much potassium at once on an empty stomach can lead to [hyperkalemia](#) (very high levels of potassium in the blood).

- For **calcium**, supplementation is best considered only after a dietary evaluation, as overconsumption may have negative effects on cardiovascular health.^{2,3} Track what you eat for a week and compare the calcium content of your diet with the RDA for your gender and age. If, on average, you are getting less than 80% of your RDA, supplementation becomes a possibility, but you should first consider tweaking your diet.
- Get sufficient and quality **sleep**. Can never go wrong with getting good sleep.

Table 1: Recommended hours of sleep, by age

AGE	RECOMMENDED	MAY BE APPROPRIATE	NOT RECOMMENDED
0–3 months	14–17	11–19	<11 or >19
4–11 months	12–15	10–18	<10 or >18
1–2 years	11–14	9–16	<9 or >16
3–5 years	10–13	8–14	<8 or >14
6–13 years	9–11	7–12	<7 or >12
14–17 years	8–10	7–11	<7 or >11
18–25 years	7–9	6–11	<6 or >11
26–64 years	7–9	6–10	<6 or >10
≥65 years	7–8	5–9	<5 or >9

Adapted from Hirshkowitz. *Sleep Health*. 2015. PMID:[29073412](https://pubmed.ncbi.nlm.nih.gov/29073412/)

What can you do about the “keto breath”?

The ketone body acetone can be excreted via your breath, giving it a “nail polish remover” or “slightly fruity” smell. This smell is commonly referred to as “keto breath”. If you don’t like it, here are some potential solutions:

- Wait it out. Sometimes this can just go away on its own once your body has better adapted to the keto diet.
- Drink more fluids. Acetone can also be excreted through your urine. Theoretically, drinking more fluids may help decrease the amount that is excreted through your breath.
- Decrease ketone levels. If your breath is really bothering you, you can try to increase your daily carb intake level to help decrease the formation of ketones. Try adding 10–20 grams of carbs back into your diet and see what results this produces. Individual results will vary, so some trial and error testing will be needed.

- Mask the smell. Keep some sugar-free or low-carb mints, gums, mouthwashes, strips, or sprays on hand. Use as needed to keep “keto breath” at bay.

How do you avoid constipation?

If you experience constipation, try taking the following actions.

- Increase your soluble fiber intake. [Psyllium](#) may be a preferential fiber to start with due to its high viscosity and low degree of fermentation, thus being less likely to produce unwanted side effects. To supplement with psyllium, take 10–15 g/day in divided doses across multiple meals.
- Hydrate. Increase fluid intake throughout the day so that you’re adding at least 710 mL (24 oz) to your diet. The infamous cup of coffee may help here too.
- Strategically use oils. Adding 1–2 tablespoons (15–30 grams) of [MCT oil](#) or mineral oil to your diet per day may help.
- If you need more immediate relief, the use of the over-the-counter drug [polyethylene glycol 3350](#) (MiraLAX®) can also be used.

How do you avoid muscle cramps?

It is possible this is occurring due to being under-hydrated and not consuming enough electrolytes.

Should you increase your fluid intake?

A keto diet can have a diuretic effect on your body, and maintaining adequate hydration can be helpful for fending off symptoms of the “keto flu” and constipation. Your body can adjust to this after a few weeks, but if you’re feeling a little dehydrated be sure to increase your daily fluid intake. A good starting point may be to take in at least 710 mL (24 oz) extra fluids per day.

Should you increase your electrolyte intake?

On a keto diet, a drop in electrolyte intake may be responsible for some of the symptoms of the “keto flu” (nausea, fatigue, brain fog, etc). To alleviate some of these symptoms, you can increase your intake of magnesium, sodium, and potassium in the first few weeks of a keto diet.

- For **sodium**, try increasing your daily intake to 3–5 g/day. If you have been prescribed a low-sodium diet, speak with your physician before doing so.
- For **magnesium**, start with 200 mg of *elemental magnesium* once a day. Increase to 350 mg as needed if no symptom improvements are seen.
- For **potassium**, supplements will likely be insufficient for improving overall potassium intake, as most contain <100 mg (compare that to an RDA of 3,400 mg adult males and 2600 mg for adult females).¹ The incorporation of more potassium-rich foods into your diet can help maintain adequate levels during your transition onto the keto diet. A list of low-carb foods rich in potassium is available in appendix B.
- For **calcium**, supplementation with calcium is best considered only after a dietary evaluation, as overconsumption may have negative effects on cardiovascular health.^{2,3} Track what you eat for a week and compare the calcium content of your diet with the RDA for your gender and age. If, on average, you are getting less than 80% of your RDA, supplementation becomes a possibility, but you should first consider tweaking your diet.

Are there supplements you should take?

There are certain micronutrients that may be underconsumed on a keto diet.⁴ Where practical, supplements may be used to help fill these nutrient gaps. Here are the nutrients where you may experience low intakes.

- [Calcium](#)
- [Fiber](#)
- [Iodine](#)
- [Iron](#)
- [Magnesium](#)
- [Potassium](#)
- Sodium
- [Vitamin A](#)
- [Vitamin B1](#) (thiamine)
- [Vitamin B9](#) (folate)
- [Vitamin C](#)
- [Vitamin D](#)

We cover each of these nutrients in the [Warnings](#) chapter and show you how to modify your diet with foods and/or supplements to ensure you are receiving adequate levels.

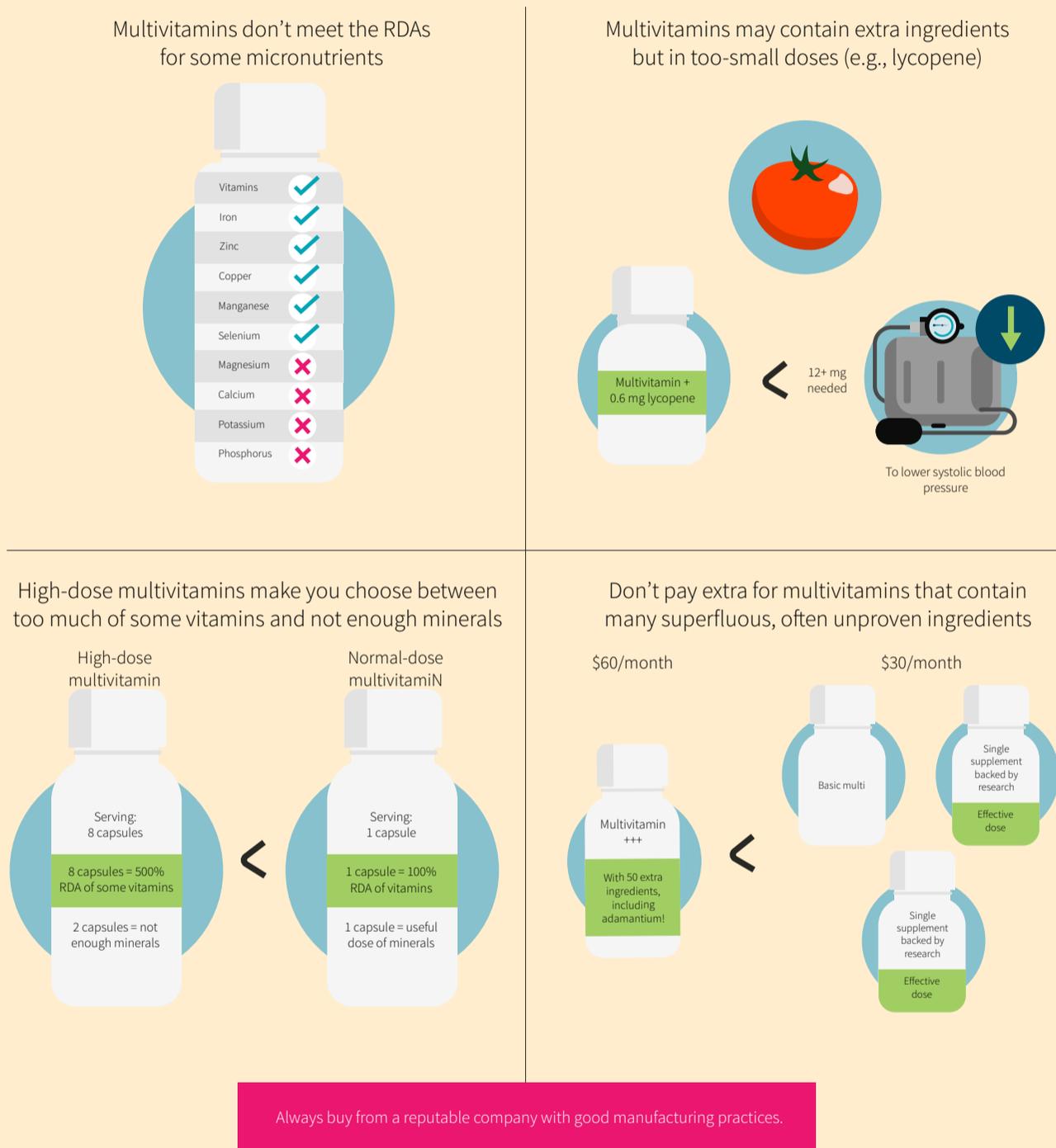
Additionally, if you are looking to increase your exercise performance, the addition of creatine monohydrate may help. The standard dose is 5 g/day. People with more muscle mass may benefit from as much as 10 g/day, but this claim is not fully supported by the evidence. To supplement 10 g/day, take 5 grams twice a day.

Tip: Much ado about multivitamins

You may be inclined to take a multivitamin while you are on a keto diet as a sort of insurance policy against nutrient deficits. A multivitamin isn't strictly necessary but could make things easier **if it is well formulated**.

When buying a multivitamin, check on the label the content of each serving, the number of pills per serving, and the number of servings per day; don't pay more for dubious bells and whistles; and stick to a company with a reputation for good manufacturing.

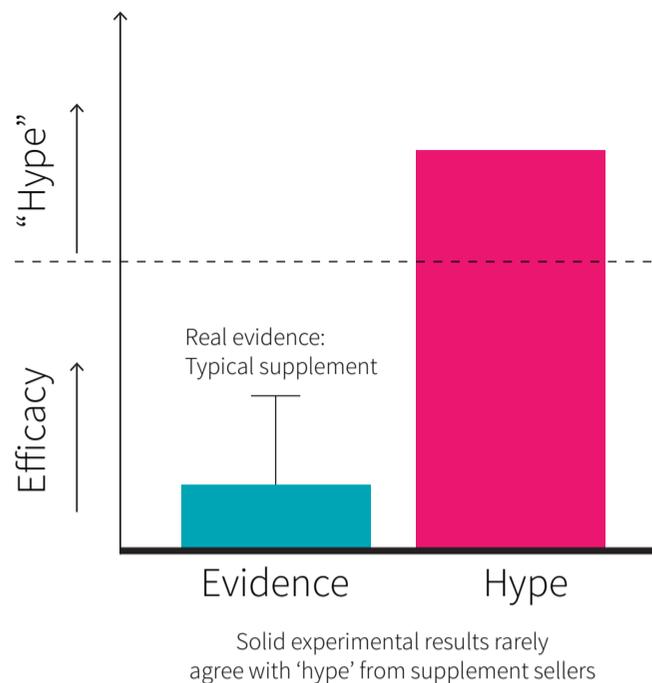
Figure 1: Factors to consider when buying a multivitamin



Should you take ketone supplements?

Ketones taken as a supplement (aka exogenous ketones) are very trendy in the keto diet space. As with most new supplements, there's a lot of hype and precious little research. Let's review the data to get a clear picture of what the evidence currently says.

Figure 2: Hype versus reality



Ketone supplements for performance

Of the eight placebo-controlled studies investigated how ketone supplements affect endurance performance, only one has shown a possible benefit. Even here, it was only a combination of pre-workout carbs and ketones that led to a 2% improvement over the carb-only group (and the study was only in eight athletes).⁵ Studies in recreational athletes did not show any apparent benefit from exogenous ketones, with or without carbohydrates. Many more trials with larger samples will be needed before any concrete conclusions can be made.

Ketone supplements for other health endpoints

There have been a wide variety of studies looking at exogenous ketones for brain disorders, epilepsy, glioblastomas, cognitive decline, and psychiatric conditions. However, there are not enough studies looking at any one endpoint to draw firm conclusions at the moment. Overall, research at this point is more speculative than practically useful.

Should you supplement with MCT oil?

[Medium-chain triglycerides](#) (MCTs) have been shown to increase blood ketone levels relative to diets high in *long-chain triglycerides* (LCT). But does this effect confer any benefits?

A number of studies have suggested that MCTs can lead to [greater satiety](#) and produce a [very small increase in fat loss](#) over LCTs. Yet most of these studies are short, and it's unclear what the implications for weight loss are in the long term. Before you go adding a bunch of MCT oil to your diet, keep in mind it is largely devoid of micronutrients and low nutrient intake are a concern in a keto diet.

Chapter 6: Health Concerns

Is the keto diet healthy and safe?

As we will see, keto is contraindicated if you have certain medical conditions. Does that mean that keto is healthy and safe if you *don't* have any specific health condition?

When asking about the safety or healthfulness of any diet it's important to ask this question:

Compared to what?

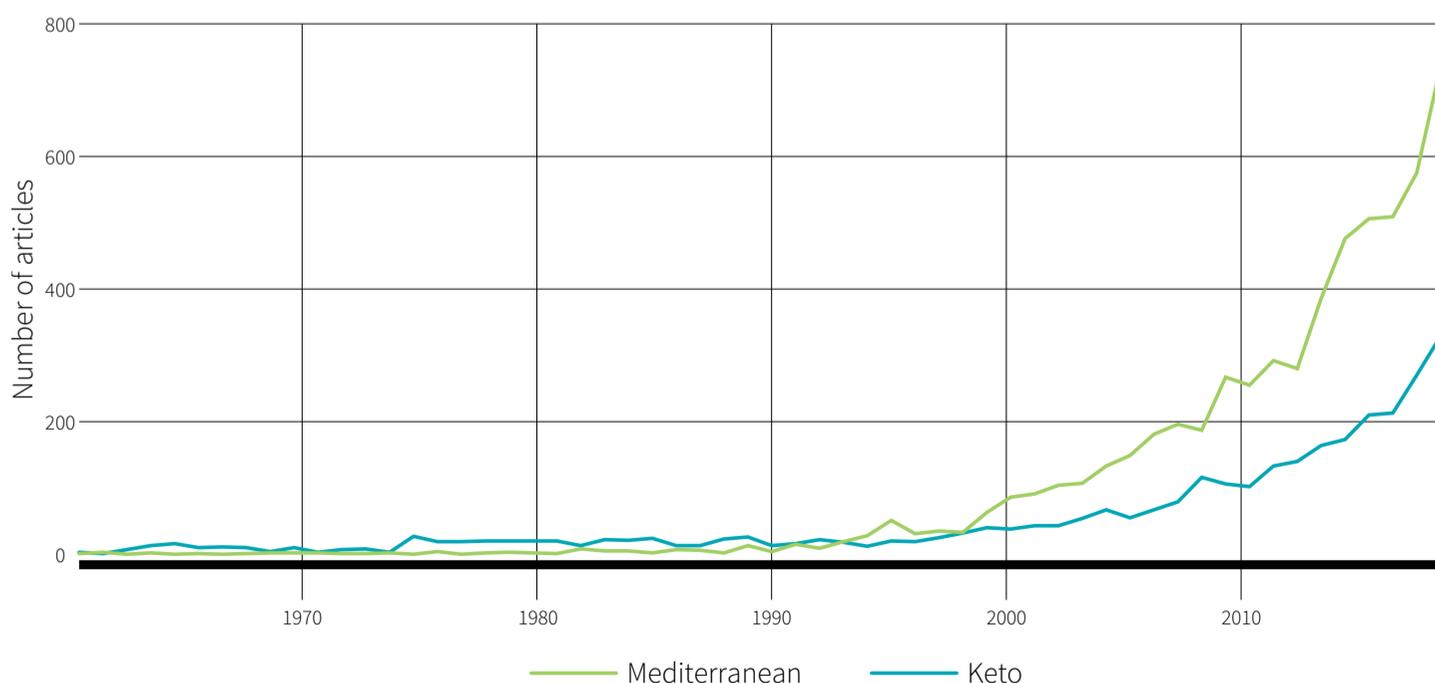
Is a well-formulated ketogenic diet better than a diet of 100% deep-fried butter? Absolutely.

Is a well-formulated ketogenic diet better than a Mediterranean diet? Well, this is where it starts to get nuanced.

We have a good amount of short-term research (<1 year) that tell us many will experience side effects and adverse events on a keto diet. Yet, these are typically temporary and manageable. However, there are very few long-term keto studies — especially when compared to the Mediterranean diet. As such, the long-term effects of keto are not as well established, be they good, bad, or neutral. So depending on how risk-averse you are, a keto diet might strike you as not sufficiently safe.

Broadly speaking, we have more information about the risks and benefits of a Mediterranean diet pattern than we do a ketogenic diet pattern. Thus, we have more information with which to make an informed choice about the Mediterranean diet.

Figure 1: Articles published on the ketogenic or Mediterranean diets



Does that mean you should pick Mediterranean over keto? Not necessarily. In fact, you could pick both and do a ketogenic Mediterranean diet!¹ There is a spectrum of potentially healthy diets but a diet pattern that works wonders for one person may be an unmitigated disaster for another. Thus, a keto diet can be safe and healthy for some while being unsafe and unhealthy for others (see [When is keto contraindicated?](#)). This depends on a few factors:

- How the diet is executed (mostly nutrient-dense whole foods, or mostly junk food?)
- How the diet intersects with personal health history and considerations (including mental health!)
- What that person is *not* doing because they are doing keto (i.e., the opportunity cost, are you missing out on something that could work better for your circumstances?)

In short, the keto diet *can* be safe and healthy, but that doesn't mean it *will* be all the time.

Different diets are tolerated differently from person to person. There is no One Ideal Diet. The best diet is the healthy whole-foods based diet you can stick to. A keto diet may be that diet for some.



Tip: A ketogenic Mediterranean diet

Traditionally, a Mediterranean diet is defined by a high intake of grains, fruits, vegetables, herbs, olive oil, fish, and moderate amounts of red wine. So how do you adapt this carb-heavy diet to be keto-friendly? Try to focus on incorporating the following.

- Vegetables. Incorporate as many as you can without exceeding your carb limit.
- Olives. Consuming olives and olive oil is encouraged. Try and make it your go-to fat source.
- Fish. Try to incorporate fish into your meals at least 2 times per week. More is certainly encouraged.

When is keto contraindicated?

A keto diet is not universally safe; in people with certain medical conditions, it could be inappropriate or cause harm.^{2,3} The table below lists two kinds of contraindications:

- **Absolute contraindications.** If you have any of these conditions (many of which are identified in early childhood), then a keto diet — of any type — just isn't for you.
- **Possible contraindications.** If you have any of these conditions, then a keto diet may be contraindicated, but you might still be able to start one — under medical supervision. Talk to your physician first.

ABSOLUTE CONTRAINDICATIONS	POSSIBLE CONTRAINDICATIONS
Carnitine palmitoyltransferase I or II deficiency	Cancer (a keto diet may alter treatment efficacy or cause possibly undesirable weight loss)
Carnitine translocase deficiency	Concurrent use of propofol (a keto diet may increase the risk of propofol infusion syndrome)
Fatty acid beta-oxidation defects	Diabetes (medication adjustment/monitoring may be required; see below the table for more information)
Long-chain 3-hydroxyacyl-CoA deficiency	Dysphagia (difficulty swallowing)
Long-chain acyl-dehydrogenase deficiency	Eating disorders
Medium-chain 3-hydroxyacyl-CoA deficiency	Familial hyperlipidemia (a genetic disorder that increases blood lipids)
Medium-chain acyl-dehydrogenase deficiency	Gallbladder disease or no gallbladder (because of issues with digesting fat)
Organic acidurias	Gastroesophageal reflux disease (GERD)
Porphyria	Gout
Primary carnitine deficiency	History of kidney stones
Pyruvate carboxylase deficiency	Hypertension (high blood pressure; medication adjustment/monitoring may be required)
Short-chain acyl-dehydrogenase deficiency	If 18 years old or younger (or still growing, as a general rule)
	Kidney disease or failure
	Liver disease or failure
	Multiple food allergies
	Pregnancy or breastfeeding
	Received bariatric surgery (because of possible issues with digesting fat)

Can you go keto if you're diabetic?

A keto diet can treat [diabetes](#). However, if you have diabetes or prediabetes and are considering a keto diet, first broach the topic with your physician, a registered dietitian, or both. Because a keto diet drastically decreases carb intake, medications may need to be adjusted and monitored to make sure they do not cause any adverse events. Plans may also need to be put into place so you are ready to deal with hypoglycemic events, should they occur.

Should you worry about ketoacidosis?

Ketosis (a.k.a. nutritional ketosis) should not be confused with [ketoacidosis](#), a life-threatening condition where blood ketone levels exceed 12–15 mmol/L (remember that in nutritional ketosis, levels typically range from 0.2–3.0 mmol/L). Under normal circumstances, ketogenesis operates under a negative feedback loop — a “fail-safe” system that decreases ketone production when it senses concentrations are getting too high.

Ketoacidosis is a potential risk only when these feedback mechanisms do not function, which can be the case in those with diabetes or with chronic alcoholism. If you do not have these conditions you need not worry about ketoacidosis.

Keto and exercise

Will keto hurt your exercise performance?

It might, but not always. Let's take a look at two components of exercise: strength and endurance.

Strength performance on keto

The effect of keto diets on strength hasn't been as well-studied as much as their effects on endurance, and the data we have on the effects of carb-restriction on muscle gains are limited. What studies we do have indicate that keto diets neither hinder nor enhance strength performance or gains, for two reasons.

1. Tests of strength commonly last only a few seconds, during which your muscles use creatine phosphate as fuel, rather than glucose. Thus, you won't actually be dipping into your glycogen stores as much during a typical resistance training session.
2. When you don't eat carbs, your body still produces enough glucose to refill your glycogen stores between workouts, thus ensuring that you can train just as hard as someone on a high-carb diet.

Endurance performance on keto

Since your body has a limited storage capacity for carbs, and a near endless storage capacity for fat, wouldn't a diet that turns you into an efficient fat-burning machine also make you better at long, endurance activities? The evidence here is a little mixed but ultimately suggests that a keto diet has either a neutral or negative effect on endurance exercise performance. This conclusion seems to hold true for both elite and recreational athletes.

Why the disconnect? At the exercise intensities seen in endurance events, what matters is how efficiently you burn glucose, not fat. By making you more reliant on fat, a keto diet actually *impairs* your ability to burn glucose, which hurts your performance.

But what about non-competitive endurance events such as long walks, hikes, or a casual jog? Could a keto diet help you in these scenarios? It's possible, but the truth is we don't yet know as there are no studies looking at this. If this changes, we'll keep you updated!

Can you gain muscle on keto?

Yes, you absolutely can (and quite a bit of muscle to boot). Is it the 100% Most Optimal Muscle Building Diet Of All Time? No, but this is unlikely to matter to the vast majority of people (aka those not competing at an über elite level).

If you're looking to optimize muscle building on a keto diet, these dietary strategies may help.

- Time your carb intake to before a workout (aka a Targeted Ketogenic Diet). Consume 10–50 grams of carbs during your workout or up to 30 minutes before. Finding how much extra carbohydrate your body can tolerate without getting kicked out of ketosis will be a trial-and-error endeavor requiring that you measure your ketone levels (preferably via blood testing, given the low accuracy of urine testing).
Ensure your daily protein intake is at an optimal level. You should be consuming *at least* 1.2 g/kg (0.54 g/lb) a day if you are not in a caloric deficit. However, many people seem to spontaneously decrease their caloric intake when they switch to a keto diet. If that's your case, you'll probably need more than 1.2 g/kg to increase or at least preserve your muscle mass. You can use our Protein Intake Calculator to find your optimal protein needs [here](#).
- Take creatine monohydrate with fluid. The standard dose is 5 g/day. People with more muscle mass may benefit from as much as 10 g/day, but this claim is not fully supported by the evidence. To supplement 10 g/day, take 5 grams twice a day.
- Ensure your sodium and magnesium intake is sufficient.
 - For **sodium**, try increasing your daily intake to 3–5 g/day. If you have been prescribed a low-sodium diet, speak with your physician before doing so.
 - For **magnesium**, start with 200 mg of *elemental magnesium* once a day. Increase to 350 mg as needed if no symptom improvements are seen.

Chapter 7: Can Keto Treat ... ?

We've seen that keto is [contraindicated](#) if you have certain medical conditions. But can it, conversely, help if you have certain other medical conditions? According to the Internet, the answer is yes. According to the Internet, keto can potentially treat — even cure! — everything.

In the following pages, however, we'll have a quick look at what the *scientific evidence* has to say on keto's effects on [Alzheimer's](#), [autism](#), [cancer](#), [diabetes](#), [epilepsy](#), [Lou Gehrig's](#), [migraines](#), and [Parkinson's](#).

Can keto treat Alzheimer's?

Alzheimer's disease, a common form of dementia, is a brain disorder that impairs a person's memory and thus their ability to function and conduct routine daily activities (i.e., showering, eating, driving). Of the five clinical studies conducted to date, four used an MCT oil supplement as the primary intervention while one actually testing a keto diet.¹ Most of these trials have been small and were primarily designed to test safety and feasibility, with changes in cognitive functioning commonly being secondary outcomes.¹

None of these trials have been long-term, so little can be said of a keto diet's effect on the progression of Alzheimer's. It is important to acknowledge that patients with dementia are often at increased risk of malnutrition, as eating may become challenging as the disease advances. A keto diet may cause a reduction in appetite and can increase the risk for certain nutrient deficiencies. These effects can complicate the implementation of a keto diet in a patient with dementia.

Can keto treat autism?

Autism spectrum disorder (ASD) is a developmental disorder that usually manifests in childhood. Only one clinical trial has tested a keto diet alone on children with diagnosed ASD.² Of the 30 children initially enrolled, only 18 tolerated the diet and followed through for the entire six-month trial — a 40% dropout rate. Those with more mild symptoms saw greater improvement over those with more severe symptoms.

It should be noted that nutrient intake issues can be especially tricky in children with ASD. One study found that boys with ASD had lower bone mineral density and vitamin D intake from food as well as measured in serum.³ These issues can be compounded by a keto diet, where ensuring sufficient intake of nutrients can take some diligent planning.

Can keto treat cancer?

It is safe to say that a keto diet is not a cancer cure-all, but it might have some applications in select types of cancer. The preclinical evidence (i.e., cancer cells and animal models) for the uses of a keto diet as a complementary addition to standard treatments is quite mixed, in part due to different study designs and typically small sample sizes.^{4,5} Furthermore, most of the mice studies have been focused on examining the effects of a keto diet on brain cancers (the results of which have hinted at potentially favorable effects).⁴

Moving onto human trials the evidence is currently sparse and low quality, with much of the data we have coming from limited case studies or small open-label trials. Even here, most of these trials are primarily testing the viability and safety of the diet — not its efficacy in treating cancer.

- A systematic review examined six case studies including 39 patients with gliomas (a rare form of brain and spinal cancer).⁶
 - Adherence was variable, and some studies lacked good reporting on this endpoint.
 - Some adverse events were reported, such as inadvertent weight loss, increased cholesterol, and negative impact on quality of life.
 - There was insufficient evidence to determine a keto had any effect on cancer or survival time.
 - The review found a lack of high-quality evidence available.
- A systematic review of eleven studies (3 early-phase single-arm trials, 3 prospective cohort studies, 1 retrospective review, and 4 case reports) looked at 102 participants with either brain, rectal, or mixed cancer sites.⁷
 - Adherence was very low, as only 49% of patients were able to complete the diet. As a result of this, many studies could not perform the necessary statistical tests needed to compare the keto to the non-keto group.
 - Adverse events were reported in 50 patients, and the most common were fatigue, constipation, diarrhea, hyperuricemia, and vomiting.
 - Of the analyses that were performed on changes in cancerous tumors or survival, results were highly mixed. Confident conclusions could not be drawn.
 - The review deemed the overall quality of evidence to be very low.

- A systematic review of fifteen studies (8 prospective cohort studies, 2 retrospective studies, and 5 case reports) looked at 330 participants on isocaloric keto diets (i.e., they tried to keep the patient's weight-stable).⁸ Cancer sites were variable, including lung, stomach, ovarian, breast, prostate, rectal, head, and neck.
- Adherence was low, with only 53% of patients being able to follow the diet at any one point in time. Only 20% were able to adhere very well.
- Adverse events included fatigue, dehydration, gastrointestinal upset, high cholesterol, and vitamin, mineral, and enzyme deficiencies.
- Most studies were designed to test adherence, quality of life, and feasibility of the KD — not the effects of keto on the cancerous tumors or survival.
- The analyses that were performed on tumor progression and survival were, again, highly mixed.
- No study included was found to be of rigorous methodological design.

At present, there is no reliable manner in which to assess which cancer types or cancer patients might respond favorably to a keto diet. Longer-term and higher-quality randomized trials will be needed in the future to gain greater insight into keto-cancer interactions.

Can keto treat diabetes?

It can treat diabetes, but not necessarily cure you of it. A true cure would mean you could go back to eating carbs without any blood sugar or insulin issues (note that cure does not apply to type I diabetes, as there is no cure for this type). At present, a keto diet has not been shown to cure diabetes by this definition. Yet, a keto diet (particularly when paired with weight loss) can certainly help improve your blood sugar control, HbA1c, insulin sensitivity, and production, and may help you reduce the number or the amount of diabetes-related medications you may be taking.

One active area of unanswered research is if there are any inherent diabetes benefits to a keto diet. We have a good idea of the improvements keto + weight loss can confer for those with diabetes, but what about just keto itself? Losing weight can greatly benefit health in diabetics, so researchers are now trying to figure out if there is anything special about going keto, when no weight is lost, that could provide additional advantages. Stay tuned!

Can keto treat epilepsy?

Epilepsy is a neurological disorder where the affected are prone to seizures. Fasting as a treatment for what fits the description of epilepsy has existed for thousands of years, and it was a modern medical treatment beginning in 1911.⁹ However, extended fasting is unsustainable so in the 1920s a ketogenic diet gained popularity under the hypothesis that it could mimic the effects of fasting (high ketone levels are a common feature of both). Today, the ketogenic diet can be used to treat refractory epilepsy — epilepsy that hasn't been well-controlled by medication.¹⁰

A Cochrane review from 2018 looked at randomized trials on refractory epilepsy and included studies on classical ketogenic diets, medium-chain triglyceride ketogenic diets, and the modified Atkins diet. They included 11 studies but were unable to perform any statistical analysis due to large differences between study designs, which make performing such analyses unhelpful.¹¹

Overall, a ketogenic diet for childhood epilepsy was deemed to be promising, but the quality of evidence was consistently rated as low or very low for both complete seizure freedom or >50% reduction in seizure frequency. More trials are needed to assess the average expected reduction.

It's quite plausible that those who are able to stick to a ketogenic diet saw better results, though some dropouts were due to ineffectiveness, and at the same time, tolerability was a concern; with vomiting and constipation being the main adverse effects (but not extremely common).

Can keto treat Lou Gehrig's disease?

Lou Gehrig's disease, also known as amyotrophic lateral sclerosis (ALS), is a neurodegenerative disorder that causes nerve cells to be degraded. The disease is currently lethal, with lifespan from time of symptom onset being 3–5 years.

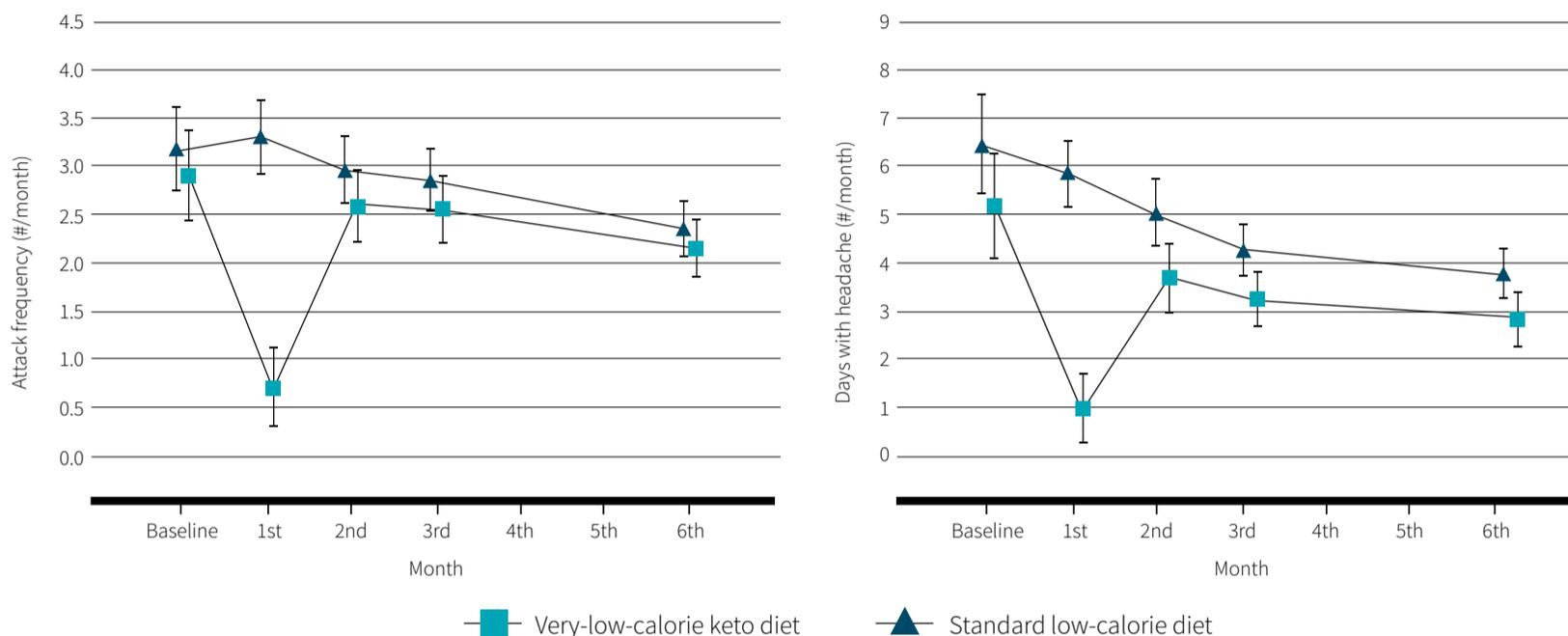
At present, there have been no clinical trials studying keto for ALS. One phase 3 trial was initially planned but later terminated for unknown reasons.¹² Studies in rodent models of ALS suggests that a keto diet might be helpful for decreasing symptoms or disease progression, but controlled human trials are needed to confirm these findings.¹³

Can keto treat migraines?

A migraine headache is a recurring headache that often causes moderate or severe pain. Two clinical studies have looked at keto diets as a treatment for migraines.^{14,15}

The smaller trial of 18 subjects saw reductions in migraine frequency with a keto diet after one month.¹⁵ The larger of the two enrolled 96 females who were overweight and randomly assigned them to either receive a standard low-calorie diet for six months or a very-low-calorie keto diet for one month followed by five months of the low-calorie standard diet.¹⁴ Though the keto group experienced an improvement in symptoms after the first month compared to the standard diet group, these improvements greatly diminished as the keto group began consuming the standard non-keto diet.

Figure 1: Headache occurrence over six months on a keto or non-keto diet



Adapted from Di Lorenzo et al. *Eur J Neurol*. 2015. PMID:[25156013](https://pubmed.ncbi.nlm.nih.gov/25156013/)

Can keto treat Parkinson's disease?

Parkinson's is a progressive disorder of the nervous system that gradually reduces a person's ability to control their movements. There are currently only two studies examining the effects of keto on this disease.

The first study involved five patients that were observed for a month while consuming a keto diet at home.¹⁶ Some improvements were seen but it's hard to know what to make of these as there was no control group. One caveat here is that the diet they consumed was very low in protein, accounting for only 8% of their daily calories.

The second study compared low-fat to a keto diet over eight weeks in 47 patients (only 38 completed the trial, a 19% dropout rate).¹⁷ At the end of the study, both groups had seen significant improvements in symptoms but the keto group saw a bit better results in nonmotor symptoms (i.e., changes in mood, cognition).

Given the very limited data we have, further studies are necessary before any conclusions can be drawn.

KETO 201:

THE
SCIENCE
OF KETO

Chapter 8: Body Composition

Summary

As a weight-loss diet, low-carb (including keto) isn't inherently superior to low-fat, and vice versa. Different diets work differently for different people, but **your best diet** will have at least two qualities: it'll be **hypocaloric** (it'll make you eat less than your burn) and **sustainable** (it'll fit *your* tastes and lifestyle well enough that you can stick to it).

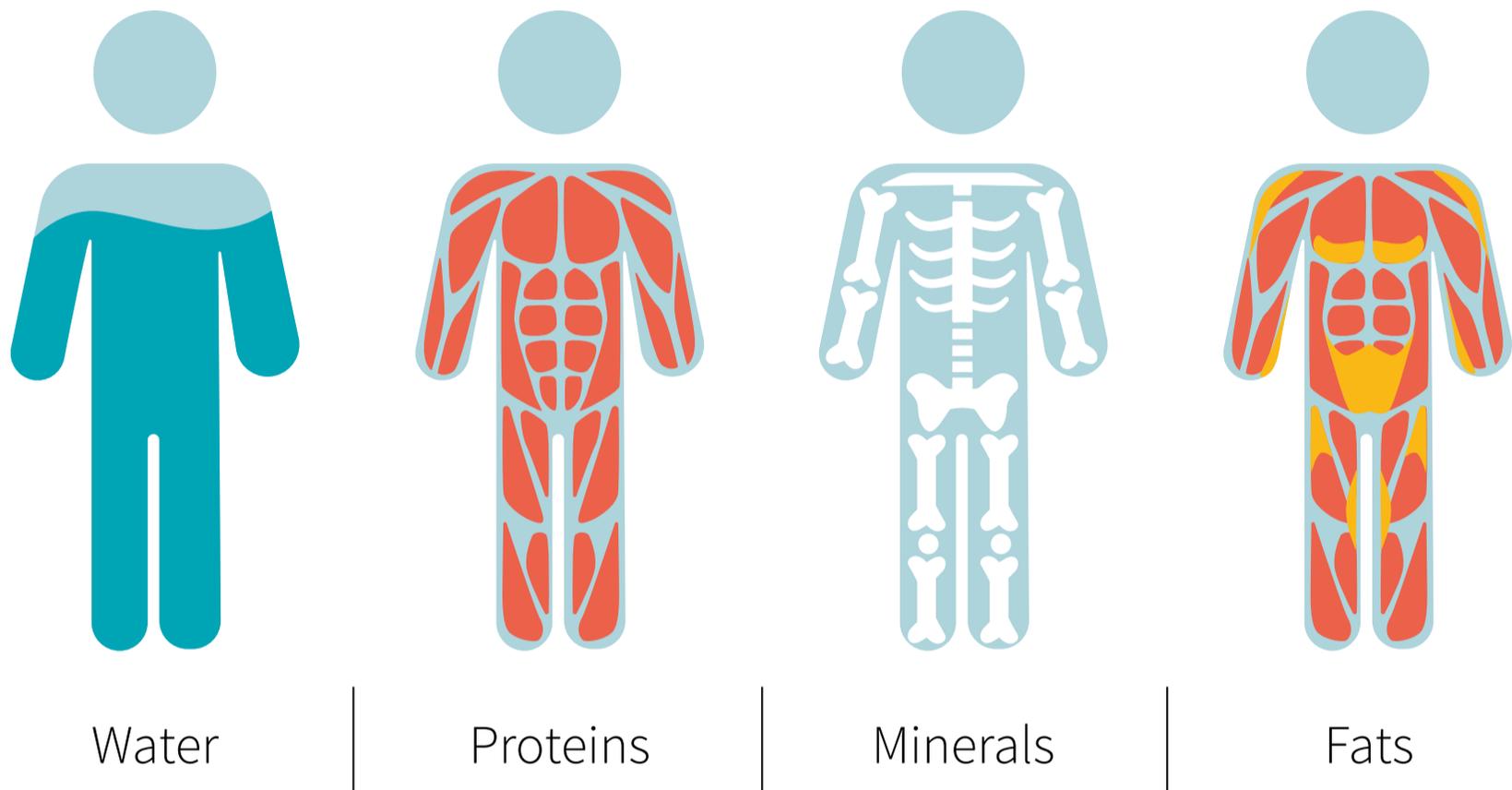
- Body weight = fat mass + lean mass. (In this guide, “weight” and “mass” are interchangeable.) Your lean mass is everything that isn't your fat; it includes notably your muscle, water, and glycogen (i.e., your carb stores).
- At the onset of a keto diet, people can lose a lot of water and glycogen, totaling 2 kg (4.4 lb) on average. Because, in most studies, this loss is recorded as a loss of lean mass, it can easily be misinterpreted as a loss of muscle mass.
- To improve your body composition, you must increase your lean-to-fat ratio by losing fat or building muscle (or both).
- When you lose weight, you want it to be fat, not muscle. To build or preserve muscle, you need **resistance training** and enough **protein**.
- **High protein** intakes might reduce your blood ketone levels, but to what extent is uncertain and highly variable. Note that, unless you follow a **medical** keto diet, ketone levels of 0.5 mmol/L should suffice (your primary fuel source will have switched from carbs to fat).
- **Training** considerations are mostly the same on keto as on any other diet, but keto dieters may need to consume more salt and fluids, and may benefit from taking creatine more than other people. It is also possible that, by taking some extra carbs around your workout, you could improve the quality of this workout without kicking yourself out of keto.

What is body composition?

Some of the most intense discussions around the keto diet have centered on its effects on “body composition”, which usually refers to one of two things:

- Your body’s fat mass and lean mass (the latter being “everything that isn’t fat”).
- Your body’s fat mass, protein mass, mineral mass, and water mass.

Figure 1: Components of body composition



Keto can have varying effects — be they positive, negative, or neutral — on these four major components. The evidence can get really complicated and nuanced, notably because of wildly different study designs; so it can seem confusing, and even conflicting.

In this chapter, we’ll take a practical approach and walk you through the evidence for the effects a keto diet can have on your body weight, [water weight](#), [fat mass](#), and [muscle mass](#).

🔍 Digging Deeper: Mass vs. weight

Mass. A measure of how much matter is in an object. Unlike its weight, the mass of an object is constant. If an object’s mass is 1 gram on Earth, its mass is 1 gram on the Moon.

Weight. An object’s relative mass. Unlike mass proper, weight is affected by gravity: it will be different on Earth and on the Moon; it can even vary on Earth (a given object is slightly heavier at sea level than at the top of a mountain, and at the equator than at the poles).

For our purpose, mass and weight are pretty much interchangeable. It just happens that people usually talk of “body weight” and “water weight” but of “fat mass” and “muscle mass”.

This chapter will look at the effects a keto diet can have on three aspects of your body composition: your water weight, fat mass, and muscle mass.

Water weight

The effects a keto diet will have on your body's water stores are well documented.^{1,2,3,4} When you go keto, you'll likely lose a lot of weight in the first week or so — and that weight will mostly be water, for two reasons:

1. **You empty your carb stores.** When you eat carbs, your body can transform them into glucose, to be burned for energy or stored as glycogen. To store 1 gram of glycogen, you need about 3 grams of water. If you stop eating carbs, your body uses your stored glycogen, and the freed water gets excreted.
 2. **You excrete ketones.** As your ketone levels rise, your kidneys begin the process of excreting them in the urine, together with some additional salt (sodium) and water.
-

In your first week on a keto diet, you'll likely lose a lot of weight — and that weight will mostly be water.

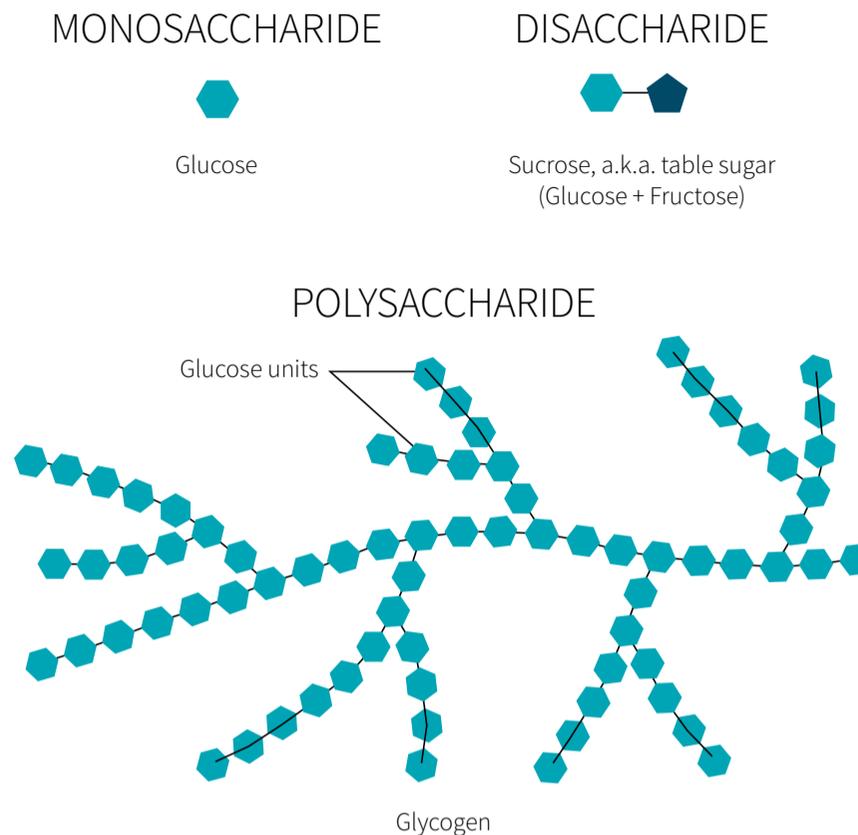
How your body stores carbs

Your body has two ways to store carbs:

- Your body can convert carbs into **glucose**, to be stored as **glycogen**.
- If your glycogen stores are full, your body can transform carbs into fat, in a process called *de novo lipogenesis* (DNL).

Glucose is a simple sugar. More precisely, it's a **monosaccharide** (*mono-* meaning “single” and *saccharide* meaning “sugar”). To store glucose molecules, your body combines them into a **polysaccharide** (*poly-* meaning “several”): glycogen. Glycogen gets stored in your [liver](#) and [muscles](#).

Figure 2: Depiction of glycogen



You store some carbs as glycogen in your liver

Glycogen cannot get stored on its own: it must be bound to water. Each gram of liver glycogen is paired with about 2.4 grams of water.⁵ When the liver’s glycogen stores are full, the average, healthy, male liver is heavier by 289–432 grams (0.6–1.0 lb), whereas the average, healthy, female liver is heavier by 241–364 grams (0.5–0.8 lb).^{6,7,8}

Table 1: Weight and glycogen capacity of a healthy liver

LIVER		MALE	FEMALE
Weight	Normal range	968–1,860 g (2.1–4.1 lb)	603–1,767 g (1.3–3.9 lb)
	Average	1,414 g (3.1 lb)	1,185 g (2.6 lb)
Glycogen (average liver, full)	Without water	85–127 g (0.19–0.28 lb)	71–107 g (0.16–0.24 lb)
	With water	289–432 g (0.64–0.95 lb)	241–364 g (0.53–0.80 lb)

References: Molina and DiMaio. *Am J Forensic Med Pathol.* 2015. PMID:[26108038](#) • Molina and DiMaio. *Am J Forensic Med Pathol.* 2012. PMID:[22182984](#) • Cahill. *Annu Rev Nutr.* 2006. PMID:[16848698](#)

Your liver is used for the short-term storage of glycogen. When you haven’t eaten in a while, your body starts breaking down your liver’s glycogen to release glucose into your bloodstream. This glucose is preferentially used to feed your brain.

When its glycogen stores are full, a typical female liver is heavier by 241–364 grams (0.5–0.8 lb) and a typical male liver by 289–432 grams (0.6–1.0 lb).

You store more carbs as glycogen in your muscles

As we saw, the average male liver weighs 1,414 grams and can store 106 grams of glycogen, whereas the average female liver weighs 1,185 grams and can store 89 grams of glycogen. In other words, you can store about 75 grams of glycogen in a kilogram (2.2 lb) of liver. By contrast, you can store less than 12 grams of glycogen in a kilogram of muscle.

Table 2: Grams of glycogen stored in a kilogram of healthy muscle (g/kg)

MUSCLE	AVERAGE	RANGE
Shoulder (deltoid)	9.8	5.3–4.0
Thigh (quadriceps)	13.5	9.5–20.0
Both muscles	11.7	5.3–20.0

Reference: Hultman. *Scand J Clin Lab Invest.* 1967. PMID:60579979

You still carry more glycogen in your muscles than in your liver, however, because you carry a lot more muscle mass than liver mass. Typical muscle mass is 15–30 kg (33 – 66 lb) for females and 22–40 kg (49 – 88 lb) for males.¹⁰ By combining those numbers with an estimation of the muscles' average glycogen content (11.7 g/kg in the table above), we can further estimate that, in their muscles, females carry 175–350 grams of glycogen, and males 256–466 grams.

As discussed earlier, however, **glycogen cannot be stored on its own: it must be bound to water.** In your muscles, each gram of glycogen comes with at least 3 grams of water (which can become 17 if you co-ingest a lot of fluid and a lot of carbs after exercising in a hot, dry environment).¹¹

Therefore, in normal circumstances, **females** who carry 23 kg (51 lb) of muscle also carry in their muscles 268 grams of glycogen and 804 grams of water (0.6 and 1.8 lb), so they are heavier by 1,072 grams (2.4 lb).

Likewise, in normal circumstances, **males** who carry 31 kg (68 lb) of muscle also carry in their muscles 361 grams of glycogen and 1,083 grams of water (0.8 and 2.4 lb), so they are heavier by 1,444 grams (3.2 lb).

Note that, unlike liver glycogen, muscle glycogen never gets released back into your bloodstream. Instead, it gets broken down to supply energy to the muscles it resides in.

When their muscles' glycogen stores are full, females with 23 kg of muscle (51 lb) are heavier by about 1.1 kg (2.4 lb), whereas males with 31 kg of muscle (68 lb) are heavier by about 1.4 kg (3.2 lb).

Your glycogen stores can weigh quite a bit

When their liver's *and* muscles' glycogen stores are full, **females** who carry 23 kg of muscle (51 lb) are heavier by about 1.4 kg (3.0 lb), of which 357 grams is glycogen and 1,018 grams is water, whereas **males** who carry 31 kg of muscle (68 lb) are heavier by about 1.8 kg (4.0 lb), of which 467 grams is glycogen and 1,338 grams is water.

How much glycogen your body can store mostly depends on three factors: muscle mass, level of physical activity, and strategic manipulation of carb intake.

The interplay of these factors is illustrated by a small study of three male collegiate athletes.¹² Compared to sedentary people, athletes have more muscle and can better synthesize and store glycogen, which makes them ideal candidates for testing the upper limits of glycogen storage.^{13,14}

This study found the maximal glycogen storage capacity of its subjects to be 629–1,146 grams (1.4–2.5 lb), with an average capacity of 810 grams (1.9 lb). That's a lot more than the 341–593 grams (85–127 [in the liver](#), 256–466 [in the muscles](#)) carried by the typical male.

There's an important caveat here. To reach these higher levels of glycogen storage (in other words, to reach *glycogen supercompensation*), the athletes had to follow a specific protocol:

- The first three days, they depleted their glycogen stores with exercise and a low-carb diet.
- The next seven days, they consumed 3,500–5,000 calories, of which 80–90% came from carbs (760–990 g).

At the end of this week-long binge, the athletes had gained 4.6 kg (10.1 lb) on average, of which 1.1 kg (2.5 lb) was fat. If these athletes had gone on a keto diet right after maximizing their glycogen stores, they likely would have experienced roughly 3.5 kg (7.7 lb) of weight loss in the first week!

With full glycogen stores, females with 23 kg of muscle (51 lb) are heavier by about 1.4 kg (3.0 lb), males with 31 kg of muscle (68 lb) are heavier by about 1.8 kg (4.0 lb), and male athletes using a method of *glycogen supercompensation* can be heavier by over 3.5 kg (7.7 lb).

Glycogen depletion and partial recovery

When you transition to a keto diet, you're removing the primary macronutrient (carbs) used to replenish your liver's and muscle's glycogen stores.

After 12–16 hours without food, your liver's stores are 25–50% depleted. Full depletion usually occurs after 3 days.⁸ If, rather than depriving yourself of all foods, you just reduce your carb intake to under 50 g/day, you can expect significant depletion to occur in less than a week¹⁵ (the more physically active you are, the faster the depletion¹⁴).

Your muscles' stores may take a bit longer to deplete, because each muscle's store can only be used by this muscle. Your liver can break down its glycogen and export it to other parts of the body, but your muscles lack the necessary enzyme (glucose 6-phosphatase) to do that. For that reason, how quickly you'll deplete a given muscle's store will depend on how much you exercise this muscle; and on the whole, how quickly you'll empty your muscles' stores will depend on the intensity, frequency, and duration of your physical activities.¹⁴

We saw that, if you exercise enough and don't eat much carbohydrate, you can deplete your muscles' stores in just 3 days.¹²

As we discussed, when your body wants to store glycogen, it needs to bind it to water: each gram of glycogen is paired with about 2.4 grams of water in the liver⁵ and at least 3 grams of water in the muscles.¹¹ When you empty your glycogen stores, you free this water, which then gets excreted.

How full or empty your glycogen stores are when you go keto will dictate just how dramatic your weight loss will be in the first week. If you are muscular and come from a high-carb diet, you may lose 3.5 kg (7.7 lb).¹² If you have little muscle and come from a low-carb diet, you may lose a kilogram, or even just a pound. Reported losses average about 2 kg (4.4 lb) but range from almost nothing to over 4 kg (8.8 lb).^{1,2,3,4} Those losses were reported as water losses but probably include some glycogen losses.

This phenomenon helps explain why so many people sing the praises of low-carb and keto diets when they first try them. Glycogen stores get quickly depleted, and the resulting weight loss convinces people that *this is working*. Except that, of course, the dramatic weight loss is mostly water, with some glycogen ... and very little fat. Appreciable fat loss will take more time.

Even when your body has learned to run mostly on ketones, it'll want to have some glycogen at hand for emergencies. So while you empty yourself of glycogen when you switch to a keto diet, both your liver and muscle stores will, in 1–4 weeks, can undergo a partial recovery.¹⁶ This may help explain two things:

- The “rebound” weight gain sometimes seen on keto: you may be regaining some of the water + glycogen you lost.
- The “rebound” exercise performance often seen on keto: during your first 1–2 weeks on the diet, your body isn’t yet optimized for burning ketones, *and* your glycogen stores are running low; later, your body is optimized for burning ketones, *and* your glycogen stores are getting partially refilled (though at a slower rate than on a higher-carb diet), providing you with a secondary reserve of available energy (secondary to your fat stores, that is).

Digging Deeper: Gluconeogenesis

Ketones can *partly* replace glucose as brain fuel.

More precisely, ketones can fulfill as much as 70% of your brain’s energy needs.¹⁷ That means that glucose must still fulfill the last 30%, even when you don’t eat carbs. How does your body manage this feat?

Your body breaks down the carbs you eat into glucose, but it can also make new glucose out of amino acids, lactate (produced through [lactic glycolysis](#)), or glycerol (a derivative of fat breakdown).¹⁸

This process of making new glucose is called **gluconeogenesis**.

After extensive glycogen-depleting exercise, small amounts of gluconeogenesis occur in the absence of any nutrition (i.e., during continued fasting).¹⁹ This glucose can serve to **feed your brain** and slowly **replenish your glycogen stores**. After all, it wouldn’t be very advantageous, from an evolutionary point of view, to not be able to feed your brain or replenish your glycogen stores when the hunt fails!

In people who fast for several weeks (obese individuals under medical supervision), gluconeogenesis amounts to about 80 grams per day: 35–40 grams from recycled lactate and pyruvate, 20 grams from fat-derived glycerol, 15–20 grams from protein-derived amino acids, and 10 grams from ketones.^{8,2}

To store glycogen, your body binds it to three times as much water. When you go keto, you empty your glycogen stores and excrete the unbound water. In a week, the average person thus loses about 2 kg (4.4 lb). However, over 1–4 weeks, your glycogen stores can partly recover as your body adjusts to its new low-carb environment.

Keto diets increase sodium excretion

After carbs, the main factor affecting your water weight is salt (sodium), which pulls water with it wherever it goes and thereby causes water retention or depletion.^{21,22}

There’s a bit of interplay between sodium and ketones, and this will contribute to your water loss, though to a lesser degree than the depletion of your glycogen stores.²³

- The more ketones you produce, the more get filtered through your kidneys, so the more your kidneys’ ketone concentration increases.

- The more your kidneys' ketone concentration increases, the more your kidneys' sodium concentration increases.
- The more your kidneys' sodium concentration increases, the more your kidneys' water concentration increases.

In the end, as the ketones pass from your kidneys to your bladder for excretion, some sodium and water get carried along.

People who sweat a lot, such as athletes and other people who exercise regularly, are already prone to sodium and water loss. When added to a keto diet, these factors may increase your sodium needs from 1.5 g/day to 3–5 g/day,²⁴ as we'll see in more detail in the Salt section of the Warnings chapter. You'll also need to monitor your fluid intake a bit more closely, to make sure that you don't end up dehydrated. By ensuring you get enough sodium and fluid, you can also fend off or mitigate the side effects some people experience when transitioning to a keto diet (notably nausea, constipation, and fatigue).

Table 3: Signs and symptoms of hypohydration and hyponatremia

HYPHYDRATION <i>(less body water than is optimal)</i>	HYPONATREMIA <i>(low blood levels of sodium)</i>
Apathy	
Dizziness or lightheadedness	
Dyspnea (shortness of breath)	
Headache	
Nausea or vomiting	
Acute weight loss	Acute weight gain
Cramping	Muscle twitching or weakness
Flushed skin	Swelling of hands, feet, or both
Heat sensations or chills	Acting "out of sorts"
Thirst	Altered mental state
	Disorientation or confusion
	Mood changes

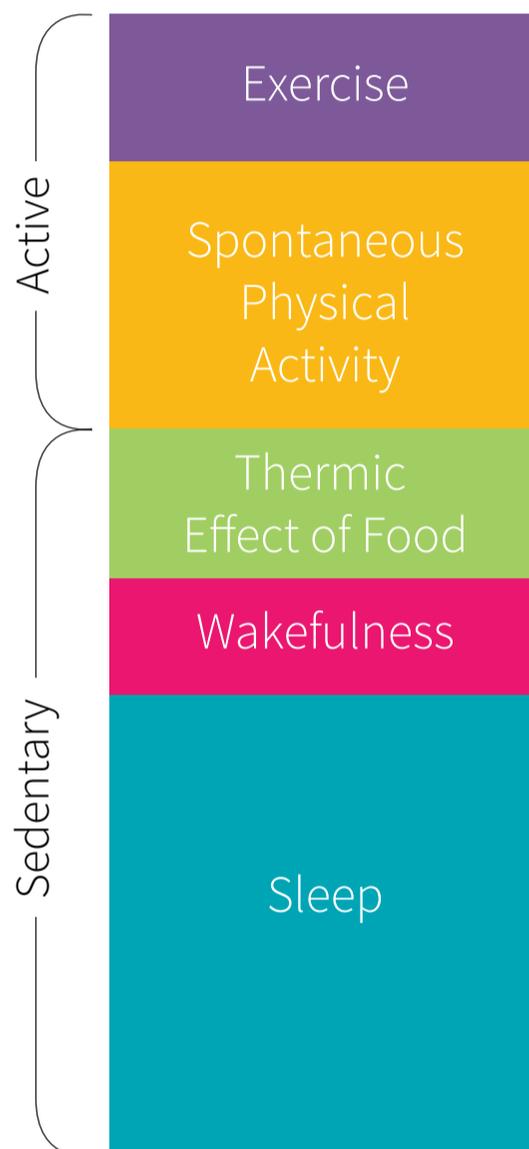
Adapted from McDermott et al. *J Athl Train*. 2017. PMID:2898512825

In your kidneys, more ketones bring more sodium, and more sodium brings more water. When your ketones pass from your kidneys to your bladder for excretion, some of the extra sodium and water get excreted too.

Fat mass

One of the most hotly debated aspects of the keto diet is its effect on fat mass. The oft-repeated claim is that, by cutting carbs and shunning sugars, a keto diet provides a “metabolic advantage” by boosting your metabolism in a manner that accelerates fat loss. To get a complete picture of what the evidence has to say on this question, we’ll review [metabolic-ward studies](#) then [free-living studies](#) — two types of studies that, through their respective advantages and downsides, complement each other.

Figure 3: Components of total energy expenditure



For **metabolic-ward studies**, participants are confined to a ward (a secure area) where the researchers can control and tally caloric intake (food) and expenditure (detailed in the side figure).

It is not unusual for the research staff to monitor meal consumption and all interactions between the participants and their visitors, to ensure that no food is consumed that isn’t part of the study. As for energy expenditure, it can be measured by placing the participants into *metabolic chambers* (enclosed rooms that can measure all energy output).

A metabolic-ward study has the **advantage** of letting us examine the mechanisms of how different diets affect metabolism. Its **downside** is that, because the participants are placed in this artificially strict environment, it can't really tell us how the studied diets would play out in real-world situations.

For **free-living studies**, participants are often coached on a specific eating style but are otherwise allowed to live their typical lives.

By taking diets out of the lab, a free-living study has the **advantage** of giving us a better idea of how those diets might fare in the real world, where people usually have to monitor themselves. Its **downside** is that it doesn't let us accurately measure and control "calories in" (caloric intake — i.e., food) and "calories out" (energy expenditure — e.g., breathing, exercising); we normally have to rely on self-reported values.

The two study types are complementary: **free-living studies** let us check if the observations made during the tightly controlled **metabolic-ward studies** produce the same effect when participants are being influenced by their natural environment.

So, does the evidence indicate that keto is king for fat loss? Let's take a look.

Here's the hypothesis for why keto should have a fat loss advantage:

- Keto dieters burn 300–600 more kilocalories per day, all in the sedentary components of their total energy expenditure (see figure above).^{26,27}
- Thanks to this **metabolic advantage**, they lose more fat than do people on non-keto diets.

Note that weight loss isn't (always) fat loss. The weight you lose may come from your fat mass, of course, but also from your lean mass, including your muscles and, as we saw in the previous section, your water and glycogen.

And here are the critical questions to answer:

1. In metabolic-ward studies, do keto diets display a metabolic advantage for fat loss?
2. If they do, does this advantage translate into greater fat loss in free-living studies?

A metabolic-ward study, being tightly controlled, allows us to test what happens when people *actually* follow a given diet. A free-living study is less rigorous but better reflects how a diet performs in the real world. Combined, the two study types can let us test the hypothesis that people burn more fat on a keto diet.

Metabolic-ward studies

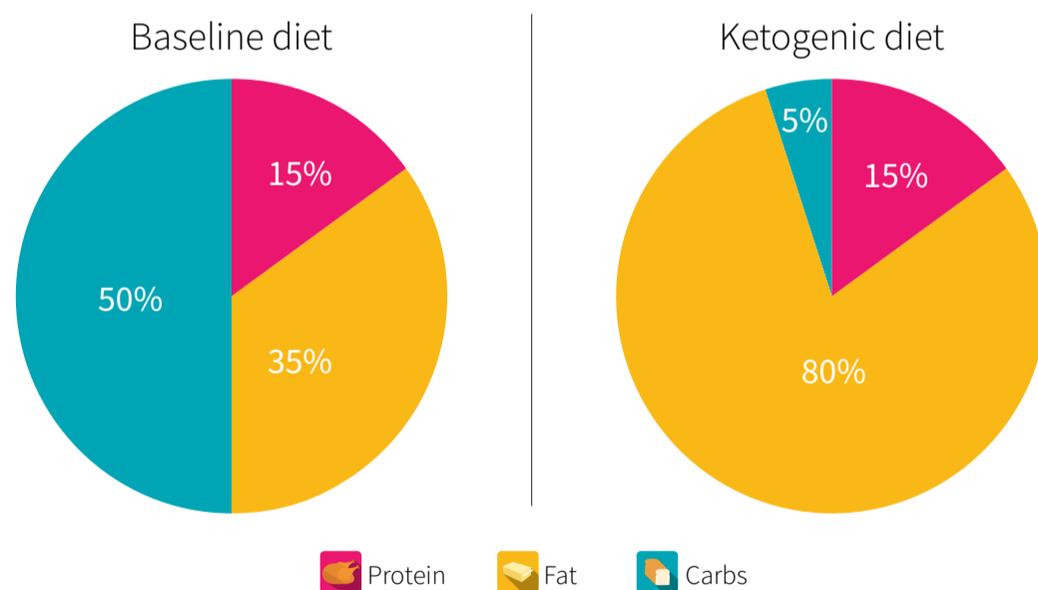
One of the more informative metabolic-ward studies to date was a 2-month trial that compared a keto diet to a high-carb/high-sugar diet.¹⁵ Seventeen overweight or obese males consumed a **high-carb diet for the first month**, then a **keto diet for the second month**.

Caloric intakes were altered weekly for the first two weeks of the high-carb diet, so as to ensure that each participant's weight stayed stable. Then no more adjustments were made: the caloric intake of each participant stayed fixed for the rest of the study.

Therefore, over the last two weeks of the high-carb diet and the month of the keto diet, **caloric and protein intakes stayed the same**, but carb intake (and thus fat intake) differed greatly.

- During the **high-carb month**, the participants ate **300 grams of carbs** per day, of which 147 grams (49%) was sugar. For reference, the average American adult consumes 244 grams of carbs per day, of which 106 grams is sugar, of which 71 grams is added sugar.^{28,29} This is already a lot more than the generally recommended added-sugar limit of 5–10% of daily calories^{30,31,32,33} (100–200 kcal for someone consuming 2,000 kcal, so 25–50 grams).
- During the **keto month**, the participants ate only **31 grams of carbs** per day. Sugar consumption dropped by 93%, from 147 to 10 g/day.

Figure 4: Macronutrient contents of the diets



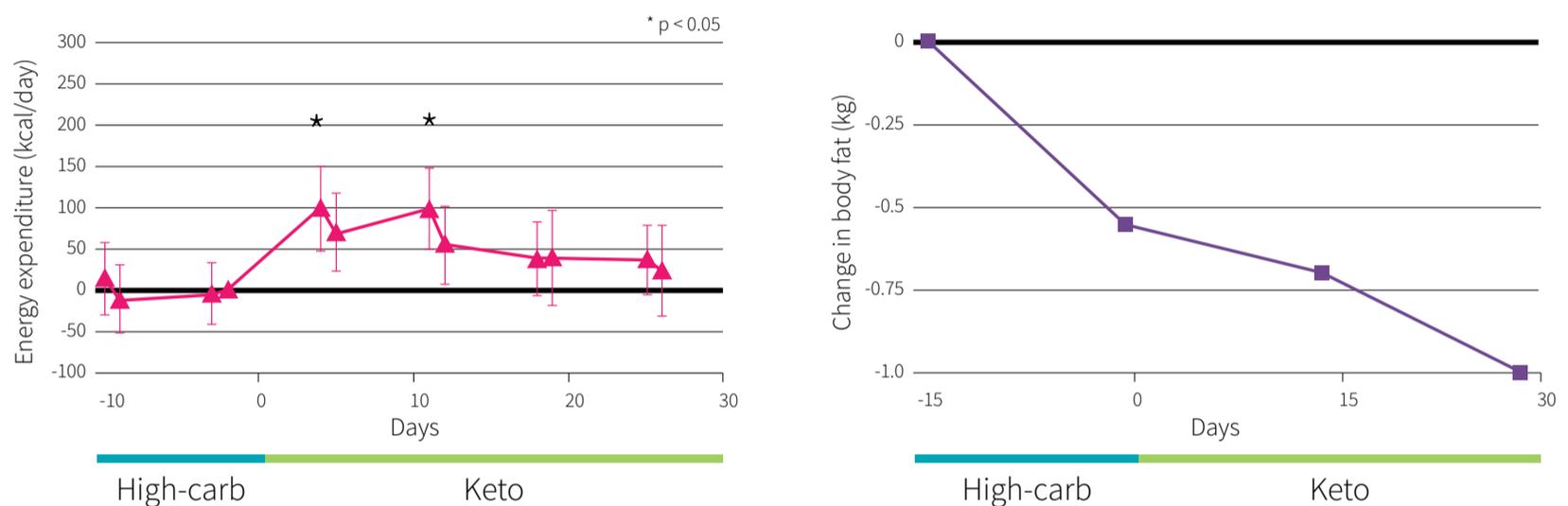
Reference: Hall et al. *Am. J. Clin. Nutr.* 2016. PMID:27385608

When the participants switched from a high-carb to a keto diet, they began burning about 100 more kilocalories per day. Hurray! Keto wins for fat loss!

Well, it's not quite that simple. The participants *did* see an increase in energy expenditure when they switched to keto, but it only lasted 10 days. Then it just disappeared. So, what happened?

Take a look at the figure below. You can see that, from the high-carb to the keto phase, energy expenditure increases (left graph) while, surprisingly, fat-loss rate decreases (right graph). In fact, the participants lost as much fat in their last two weeks of the high-carb diet (rate of 0.55 lb/wk) as they did over their entire four weeks of the keto diet (rate of 0.27 lb/wk). This indicates that, even though the participants were temporarily burning more calories on the keto diet, those calories weren't coming from their fat stores.

Figure 5: Energy expenditure** and fat mass



** The analysis was statistically adjusted for weight and fat loss.

Adapted from Hall et al. *Am. J. Clin. Nutr.* 2016. PMID:27385608

So, does this mean the keto diet is *worse* for fat loss? Again, not so simple.

When the participants switched from high-carb to keto, their energy expenditure jumped but so did their protein use. It is likely that *gluconeogenesis*, an energy-intensive process during which fat or protein is transformed into glucose, accounted for part of the increase in both protein use and energy expenditure. Higher protein use while transitioning to a low-carb diet has been seen in other metabolic-ward studies as well.³⁴

Also, when the participants switched from a high-carb to a keto diet, their glycogen stores were still full. There's a transition period during which your body still relies a lot on glucose (from its glycogen stores or from gluconeogenesis) while it adapts to running on ketones. During this transition, it isn't energy efficient. Once it has fully transitioned to running on ketones, though, it stops wasting energy on gluconeogenesis — and so, the extra energy expenditure disappears.

(Note that gluconeogenesis never fully stops. Your brain cannot be fueled *entirely* by ketones; it needs some glucose; so if you eat too little carbohydrate, as happens on a keto diet, your body *will* make the necessary glucose out of protein or fat.)⁸

It helps explain why, after the first two weeks on keto, the participants saw their rate of fat loss pick up a bit, despite their having lost their “+100 kcal burned per day” advantage. Their bodies, having become adjusted to the keto diet, were no longer trying to create a lot of glucose out of protein (which would lead to lean-mass loss, not fat loss); instead, they created a lot of ketones out of fat. It is possible that, had the study lasted longer, the rate of fat loss would have been comparable during both phases (high-carb and keto).

But not forever, anyway, because the caloric intake was fixed. If you keep consuming the same amount of calories, what starts as a hypocaloric diet (fewer calories than you need to maintain your weight) becomes a eucaloric diet (just enough calories to maintain your new weight). So the longer the diet lasts, the slower your fat loss becomes, until it stops.

This also means that, because the keto diet took place during the second month, when the participants had already lost weight, it could have been at a disadvantage compared to the high-carb diet — but the researchers took this possibility into account and statistically adjusted weight and fat loss for their analysis.

No study is perfect, though, which is one reason for there being so many — new studies are often designed to compensate for flaws in others.

And so, what about other metabolic-ward studies?

To date, only one published meta-analysis has looked at the effects that varying carb-to-fat ratios in the diet have on energy expenditure.³⁵ For this purpose, it reviewed 32 studies pitting a high carb-diet against a low-carb diet or a keto diet. All 32 studies, which totaled 563 participants, shared the following characteristics:

- They were relatively short (typically lasting less than a month), like all metabolic-ward studies tend to be.
- The participants were either inpatient (i.e., they were confined to ward) or partially inpatient (i.e., their time was split between free-living and ward confinement).
- The participants could eat only the food provided by the research staff.
- Within each study, the diets differed in calories from carbs and fat but were equal in total calories (they were *isocaloric*) and calories from protein.
- Body fat was measured objectively — usually via *dual-energy x-ray absorptiometry* (DXA/DEXA),³⁶ isotope dilution,³⁷ or metabolic fat balance.³⁴

- Daily energy expenditure was measured objectively — via metabolic chamber, as we discussed, or doubly labeled water.³⁸

🔍 Digging Deeper: Doubly labeled water and DXA: how do they work?

Doubly labeled water contains non-radioactive isotopes that can be measured when excreted in the urine. Their rate of excretion closely correlates with **energy expenditure**. Unlike metabolic-chamber measurements, doubly labeled water can be used in free-living studies, and it allows to measure energy expenditure more objectively than self-reports.

DXA scans are one of the more accurate ways to estimate changes in lean mass, body fat, and bone density. When you get a DXA scan, you lie down on a bed while a robotic arm moves up and down the length of your body, emitting very low-level X-rays and measuring how many get absorbed.

A DXA scan is fairly quick, usually taking 3–10 minutes, and it delivers measurements that are, on average, within 3 percentage points of those you'd obtain from the 4-component method — a more accurate but impractical, time-consuming, and expensive method restricted to scientific and medical studies.^{39,40} This means that if your body-fat percentage is 20% as measured by the 4-component method, you can expect a DXA scan to report a number between 17% and 23% (on average, remember: individual variations can reach 8 percentage points, which would give results between 12% and 28%).

Originally, DXA scans were employed to measure bone density, in order to detect (or track the development of) osteoporosis. Modern DXA machines can use equations to estimate body fat and lean mass. It means that modern DXA is a “3-component method”: it gives readings for fat, lean soft tissue, and bone mineral. It is important to note here that what many studies report as “lean mass” from a DXA scan is more often “lean soft tissue” mass — i.e., the lean mass minus the bone mass.

The meta-analysis found that, over all 32 studies, low-fat diets led to greater energy expenditure (+26 kcal/day) and fat loss (–16 g/day). When examining just those studies where participants were confined to a metabolic-ward, the low-fat diets still had a slight advantage. When examining just those studies where participants were confined to a metabolic ward *and* were on a keto diet, the low-fat diets again had a slight advantage.

Do numbers so small actually matter? Not really. Even the authors of the meta-analysis concluded that these differences are clinically meaningless.

In short, based on the findings from metabolic-ward studies, we can say that neither low-fat nor low-carb (including keto) produces a metabolic advantage for fat loss. But what do the free-living studies have to show us?

Metabolic-ward studies suggest that neither low-fat nor low-carb (including keto) produces a metabolic advantage for fat loss in the short term.

Free-living studies

Whereas metabolic-ward studies tell us about the diet itself (i.e., what happens when people *actually* follow the diet), free-living studies tell us about the diet prescription (i.e., what happens when people are *told* to follow the diet).

While free-living studies have the **advantage** of letting us test a diet prescription in a natural setting, they suffer from one core **downside**: imperfect adherence to the dietary intervention. Many participants start off strong in their assigned diet, but by the end of the study, they have often returned to their usual, prestudy eating habits.⁴¹ For keto diet studies, this means that carb intakes tend to creep back up to non-keto levels.

With free-living studies, we can never be 100% sure of what foods were eaten and in what amounts: we usually have to rely on self-reported values, and those values are never entirely accurate (they are more or less *inaccurate* depending on the self-assessment method⁴²). At least, with keto studies, we can track if the participants have been consuming low amounts of carbs by measuring their blood or urine levels of ketones; but unfortunately, most long-term trials (≥ 1 year) forgo ketone measurements.

In 2013, a meta-analysis of 11 *randomized controlled trials* (RCTs) compared the effects of keto and low-fat diets (defined in this study as under 30% of energy from fat) on body weight.⁴³ Together, the 11 studies involved 1,415 participants, and each study lasted at least a year. At the 1-year mark, the participants in the keto groups had lost more weight: an additional 0.91 kg (2 lb), on average. Updating the meta-analysis with an additional year-long keto RCT published in 2014⁴⁴ changes very little: the average becomes 1.1 kg (2.4 lb).

Note that all these studies shared two weaknesses:

- Fat-loss differences weren't assessed.
- Many participants didn't stick to their diet: over the course of the studies, they increased their carb intake to non-keto levels. (As we discussed, declining adherence to the dietary intervention is a disadvantage of free-living studies, compared to the tightly controlled metabolic-ward studies.)

So the results of these studies are imperfect reflections of the real effects of long-term ketosis, and they're all the more inconclusive since the average weight-loss difference between the keto groups and low-fat-but-not-keto groups is very small: just one kilogram over a whole year, and even less over two years.

Usually, to be considered *clinically relevant* (i.e., to benefit your health if you're overweight), your weight loss must reach 5% of your starting weight.^{45,46} For perspective, 1 kg (2.2 lb) is only 1.4% of 70 kg (154 lb). Put in another way, if you're overweight at 70

kg, you need to lose 3.5 kg for your weight loss to be considered clinically relevant. Most importantly, this weight loss needs to be *sustained* to have lasting effects.

So, all in all, metabolic-ward studies and free-living studies lead to the same conclusion. As *incredible* as it would have been to discover that a keto diet could blast your metabolism into the stratosphere, the current body of evidence doesn't indicate that it can, on average, meaningfully outperform non-keto diets with regard to fat loss.

Don't feel too bad for the keto diet, though. Metabolic-ward studies and long-term free-living studies of just about every type of diet — from extremely low-carb to extremely low-fat — have always produced unimpressive *average* weight-loss results.^{47,48}

But these unimpressive average weight losses don't tell the full story...

Free-living studies suggest that keto diets have a *small* weight-loss advantage in the long-term — too small to be clinically relevant. Diet adherence was often poor, though, and fat losses weren't consistently measured.

The best-kept secret of diet trials is individual variability

In diet studies, be they metabolic-ward studies or free-living studies, the **average weight loss of one group** tends to be similar to the average weight loss of the other group(s). However, the **weight loss of one individual** can differ greatly from the weight loss of another individual. In other words, whereas differences **between** groups tend to be small, differences **within** each group can be much greater.^{41,49}

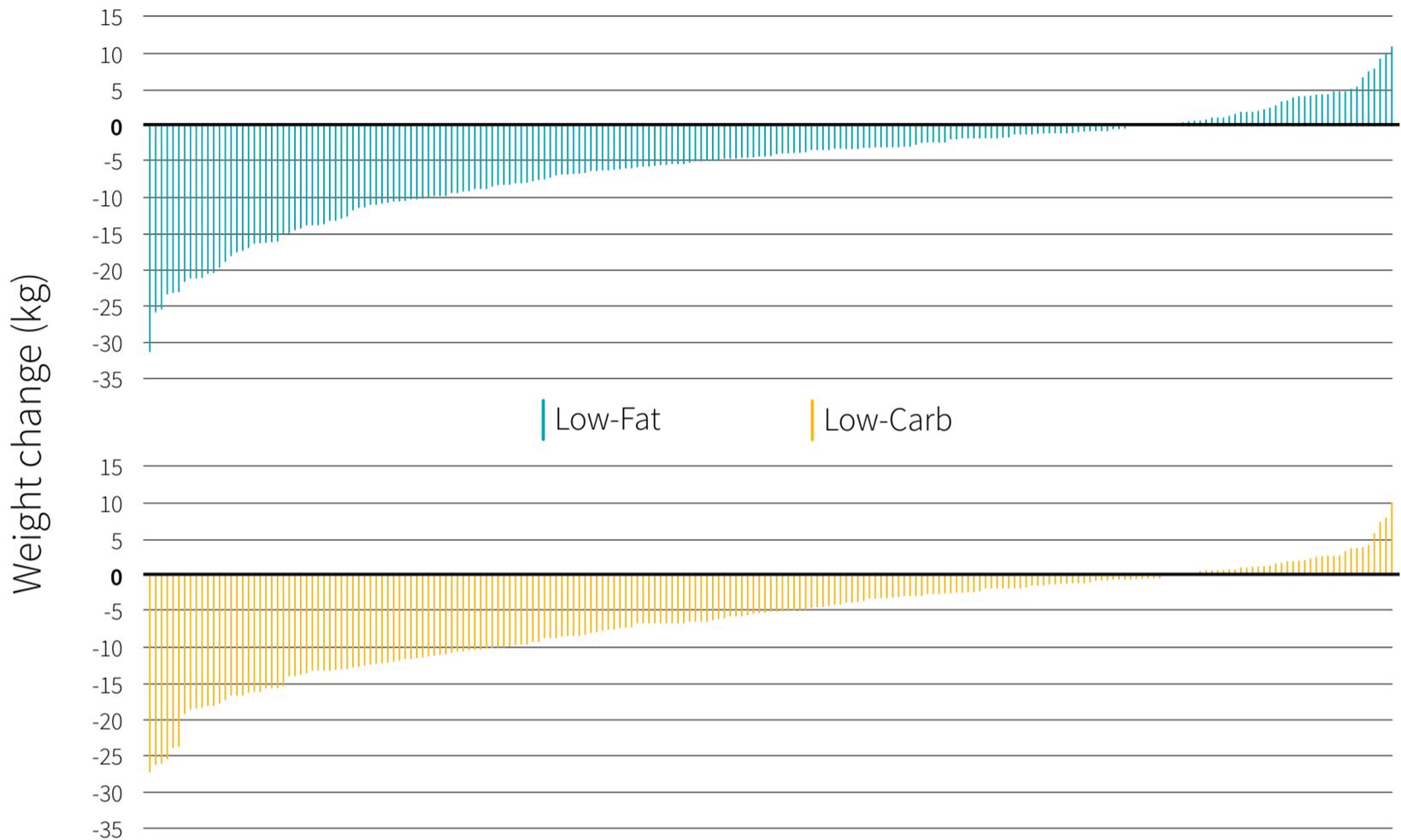
Individual variability is one of the most important aspects of diet trials — yet it is often overlooked. Let's use as an example the Diet Intervention Examining The Factors Interacting with Treatment Success (DIETFITS) RCT, for which 600 participants were randomly assigned to either a low-fat or low-carb (non-keto) diet for 1 year, with intensive support from dietitians and research staff.⁵⁰ (You can read our detailed review of this 2018 study [here](#).)

Impressively, the combined weight loss by the end of the study reached 3,000 kg (6,600 lb) — and that number takes into account the ≈10% of participants who gained weight. Here were the reported weight-loss averages:

- 5.3 kg (11.7 lb) in the **low-fat** group
- 6.0 kg (13.2 lb) in the **low-carb** group

Not bad, actually. But, as you can see in the figure below, in which each bar represents the weight change of a single participant, individual changes were all over the place in *both* groups: they ranged from -32 kg (-70 lb) to $+11$ kg ($+24$ lb).

Figure 6: 12-month weight change for each DIETFITS participant



Adapted from Gardner et al. *JAMA*. 2018. PMID:[29466592](https://pubmed.ncbi.nlm.nih.gov/29466592/)

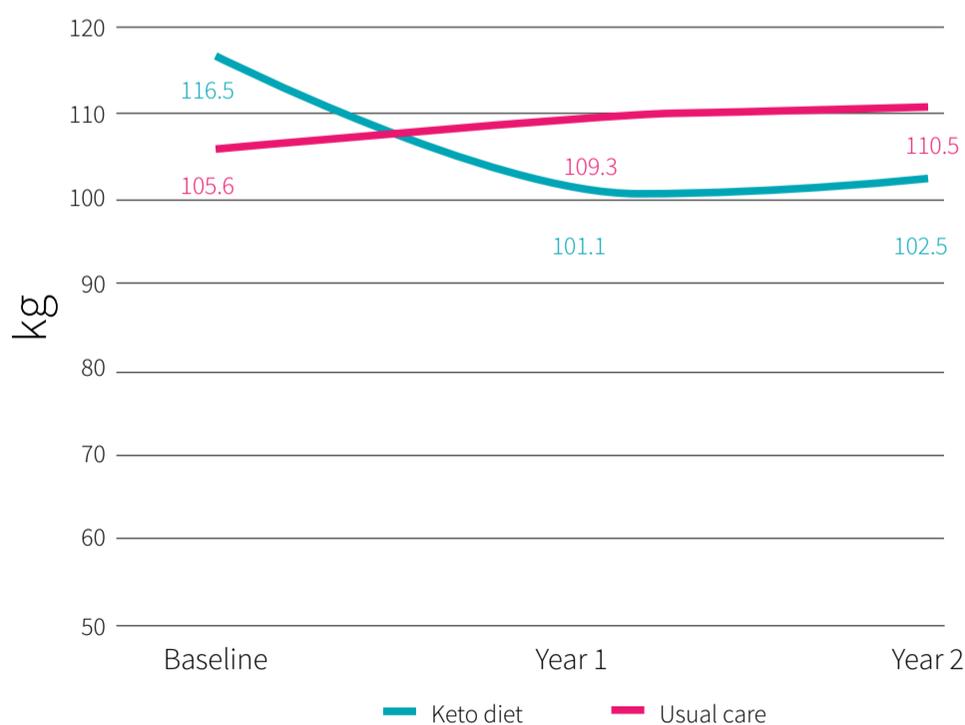
Although not an RCT, the Virta Health study is also informative.⁵¹ This trial provides an insight into what can be accomplished on a keto diet with intensive support and supervision. Here are the study's basics:

- 2-year, open-label (i.e., not blinded), non-randomized, controlled trial, funded by the Virta Health Corporation.
- 349 overweight or obese volunteers with type II diabetes chose which group they wanted to be in.
 - 262 chose the keto intervention.
 - 87 chose the usual-care intervention.
- The **usual-care group** had minimal contact with and support from the research staff.
 - Medical care was provided by the participants' personal physicians, and no modifications were made to the care they were receiving for their diabetes.

- The **keto intervention group** received intensive support and monitoring. Each participant was provided with the following:
 - Blood-pressure cuff, blood-glucose-and-ketone meter, and cellular-connected body-weight scale. Each participant had to monitor and regularly report the readings via an online app.
 - Individualized nutrition recommendations and behavioral strategies.
 - Dietary supplements as needed ([fish oil](#), [vitamin D](#), [multivitamin](#), [magnesium](#), sodium, etc.).
 - Online educational material.
 - Online access to the medical-care team (a health coach and physician or nurse practitioner). The participants also kept their personal physicians, who coordinated with the research staff as needed.
 - An online community for social support.

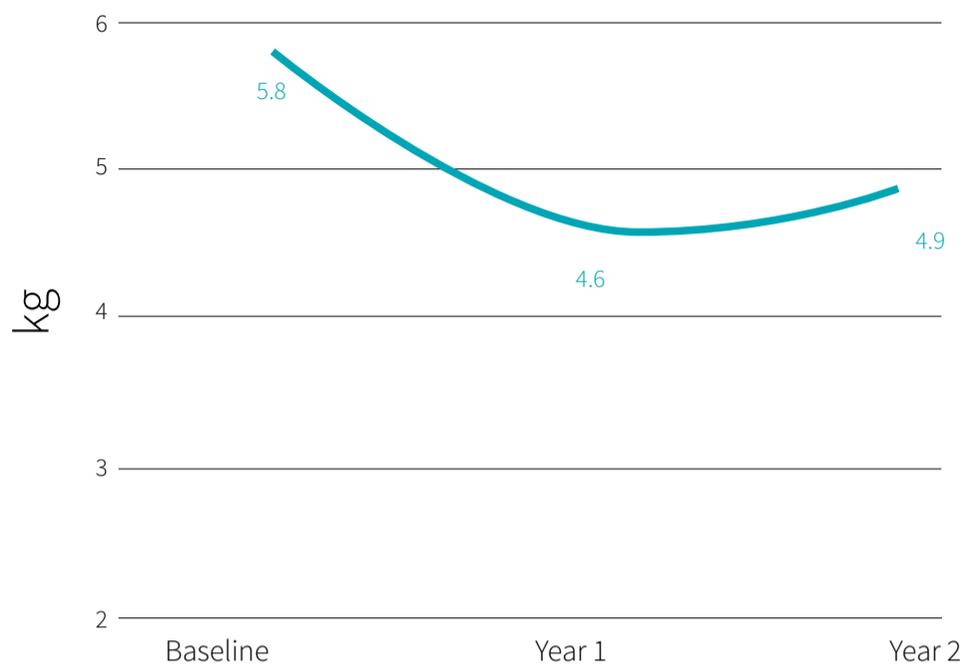
In the keto group, at the end of the 2-year study, the average weight loss was 14 kg (30.9 lb) and the typical weight-loss range was 9.1–19.0 kg (20.1–41.9 lb). [As we saw](#), to be considered *clinically relevant* (i.e., to benefit your health if you're overweight), your weight loss must reach 5% of your starting weight. At the end of the Virta Health study, the weight loss of 74.5% of the keto participants was clinically relevant. The figures below chart the changes in body weight and central abdominal fat, which is thought to be particularly harmful to health.

Figure 7: Changes in body weight in the Virta Health study



Reference: Athinarayanan et al. *Front Endocrinol (Lausanne)*. 2019. PMID:[31231311](#)

Figure 8: Changes in central abdominal fat in the keto group* in the Virta Health study



* Changes in central abdominal fat in the usual-care group were not reported.

Reference: Athinarayanan et al. *Front Endocrinol (Lausanne)*. 2019. PMID:[31231311](https://pubmed.ncbi.nlm.nih.gov/31231311/)

At the two-year mark, the dropout rates were 35% for the keto group and 28% for the usual-care group. That's actually pretty good for a diet study of this duration.

Unfortunately, it doesn't mean that 65% of the keto participants faithfully adhered to their diet. They'd been instructed to keep their blood levels of β -hydroxybutyrate (β HB, the predominant ketone in your blood) between 0.5 and 3.0 mmol/L, but ≥ 0.5 mmol/L was seen in only 32.8% of all reported measurements **over** the two years, and in only 14.1% of reported measurements **at the end of** the two years. Those ending measurements ranged from 0.05 to 2.7 mmol/L, with 0.3 mmol/L being the average.

The Virta Health and DIETFITS studies share two themes:

- Intensive and continual support from the research and healthcare staff for the entire duration of the trial (which can be very expensive!)
- A wide range of responses to the different treatments

Taken together, the results from both studies suggest that a support network is very helpful for adherence and that some diets may work better for certain individuals than for others. The reasons for these individual variances, however, are not well understood. If anything, all these trials show that there are multiple viable ways to achieving fat loss, including keto.

🔍 Digging Deeper: Can genetics predict success or failure on a low-carb diet?

Forget about The Best Diet; the holy grail of personalized medicine is Your Best Diet. Genetic differences are one possible explanation for the individual variations seen in diet studies, and so we have to ask:

Does your genotype predispose you to losing more weight on a certain type of diet?

This is one question the DIETFITS study sought to answer.⁵⁰ All participants were screened for 15 genetic patterns, identified in a previous study,⁴¹ that might influence weight change on low-fat or low-carb diets:⁵²

- Five “low-fat” patterns (hypothesized to be characteristic of people who do better on a low-fat diet)
- Nine “low-carb” patterns (hypothesized to be characteristic of people who do better on a low-carb diet)
- One “neutral” pattern (hypothesized to be characteristic of people who lose as much weight from low-fat as from low-carb diets)

At the end of this year-long RCT, the genetic patterns tested had failed to predict success or failure on either type of diet. The genes studied were relatively few, though, and the genetic variants selected were only supported by preliminary evidence, so we cannot conclude that gene-based diets in general are destined to fail.⁵³ Numerous genetic variations have yet to be tested — many with greater evidence for their potential predictive power.^{54,55}

How do you assess the predictive power of a given genetic variant? In short, it works like this: the more studies find a correlation between a genetic variant and weight loss on diet X, and the stronger this correlation is, the greater the evidence that this genetic variant might predict how the people having it will fare on diet X.

The genetic variants tested in the DIETFITS study had been correlated with greater fat loss on either a low-fat or a low-carb diet *in only one or two studies*. Other genetic variants have been correlated with greater fat loss on either a low-fat or a low-carb diet *in multiple studies* — therefore, they have greater evidence for their potential predictive power.

As genetic testing becomes more common, new correlations are being found at an increasing rate, whereas the evidence for the potential predictive power of known correlations get strengthened or weakened.

The story of diet and genotype interactions for weight loss is far from over.

Usually, in diet studies, weight loss differs little between diet groups but a lot between individuals within each group. In other words, *individual results will vary*. One reason is simply that some people stick to their diets while others don't (a support network is very helpful for diet adherence), but another may be that some diets do work better for some people than for others, for reasons that aren't completely understood.

Fat mass: the bottom line

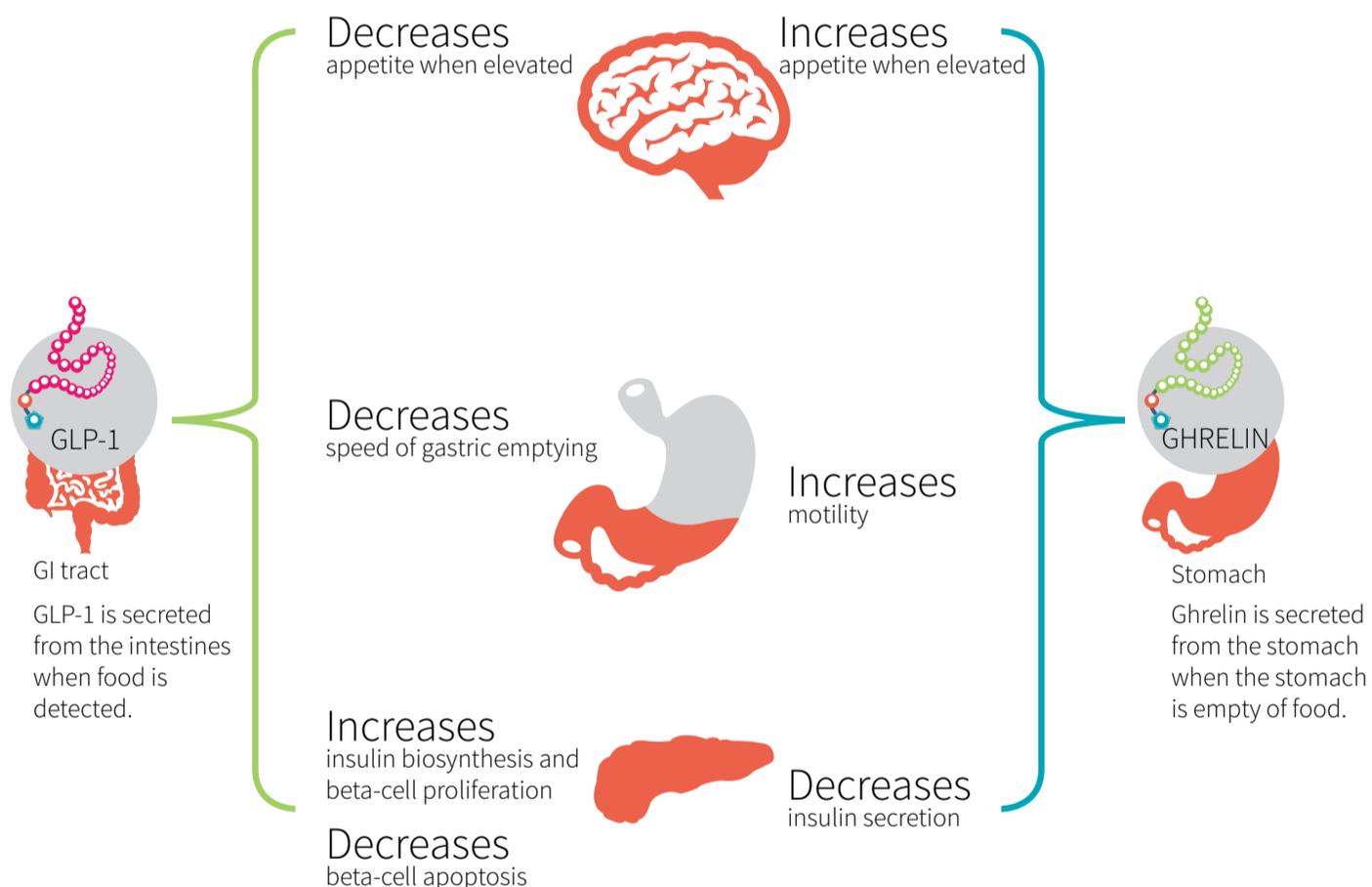
If there's one takeaway to keep in mind after looking at all the evidence, it is that neither low-fat nor low-carb is *inherently* superior. Choose a dietary pattern that fits your lifestyle, health goals, and food preferences. For some of you, a keto diet will check all these boxes.

Still, there's no denying that keto diets are especially restrictive, so what explains their growing popularity? What explains not only that more and more people try them but that more and more people actually stick to them long enough to lose weight?

Hunger. Or, the lack thereof. It isn't uncommon for people in the real world to report feeling less hungry on a low-carb or keto diet. Among the possible explanations, two stand out as especially plausible:

- People who start eating less carbohydrate start eating more protein ([the most satiating](#) of the three macronutrients⁵⁶).
- Ketones may also have an appetite-reducing effect by influencing hormones like GLP-1 and ghrelin, although to what degree is still an open question.⁵⁷

Figure 9: Influences of GLP-1 and ghrelin on appetite



Whatever the reason, if a diet makes you feel more satisfied on fewer calories, then you're on the right track, because any successful weight-loss diet has two main qualities, one objective, one subjective: it's **hypocaloric** (it makes you eat less than your burn) and it's **sustainable** (it isn't so unbearable that you give it up).

In other words, [the best weight-loss diet is the hypocaloric diet you can stick to](#). If you want to find out for sure if keto is Your Best Diet, the only way is to give it a serious try. A word to the wise, though: don't try and force yourself into an eating pattern you can't reliably sustain, else your chances of failure are high.

When it comes to weight loss, low-carb isn't *inherently* superior to low-fat — and vice versa. In the end, the best hypocaloric diet is simply the one you can stick to.



Tip: The three types of diets based on caloric intake

Calorie-wise, there are only three types of diets:

- A **hypocaloric diet** feeds you fewer calories than you burn. If you want to lose weight, that's the diet for you. If you want most of your weight loss to be in the form of fat, not muscle, you'll also need to get [enough protein](#) and preferably to exercise.
- A **hypercaloric diet** feeds you more calories than you burn. If you want to gain weight, that's the diet for you. If you want most of your weight gain to be in the form of muscle, not fat, you'll need to get enough protein and engage in resistance training (by lifting weights, for instance).
- A **eucaloric diet** feeds you as many calories as you burn. It is also called a *maintenance diet*, since your *weight* won't change much, but you can gain or lose fat or muscle, depending on how much protein and exercise you get.

Muscle mass

You'll often see the marketing material of muscle-gain supplements refer to studies reporting increases in lean mass, so it isn't surprising if many people think that "lean mass" is just a sciencey way to say "muscle mass". Strictly speaking, though, your lean mass can best be described as your [body weight](#) minus your [fat mass](#) — in other words, your lean mass is your muscle mass *plus* the mass of your [water](#), bones, ligaments, tendons, skin, and other organs.

So why don't studies on muscle building simply report on muscle mass? Because, whereas total fat mass is [relatively easy to assess](#), total muscle mass isn't.⁵⁸ To assess your total muscle mass accurately, the best way would be to kill you, extract all your muscles, and weigh them (any volunteers?). Other, less deadly methods have been devised, but they're [cost-prohibitive](#).

Therefore, in most studies on muscle building, lean mass is assessed, then *changes* in muscle mass are estimated. **In the long run**, if hydration levels and carb intake are kept fairly constant from measurement to measurement, then a *change* in lean mass should mostly be a *change* in muscle mass. **In the short run**, though, changes in lean mass and muscle mass are less likely to correlate, especially at the start of a keto diet, when, [as we saw](#), you can lose a lot of weight in the form of water and glycogen. This is a problem we'll see exemplified in some of the studies we'll analyze in this section.

For the moment, just remember that, if you're like most people, **improving your body composition means increasing your lean-to-fat ratio** (your ratio of lean mass to fat mass). So you'll need to decrease your fat mass or increase your lean mass, or both. And to increase your lean mass, you'll preferably want to increase your muscle mass (not just your water weight).

Digging Deeper: Muscle protein synthesis and breakdown

[Muscle protein synthesis](#) (MPS) is the process of building skeletal muscle, whereas *muscle protein breakdown* (MPB) is the process of breaking it down. MPB is a necessary part of muscle growth,⁵⁹ but for your muscle mass to increase, you need your MPS to exceed your MPB (overall, in the long term).

Whether you exercise or not, your body is going to break down old or damaged muscle fibers to reuse what it can of their constituent *amino acids* (AAs — the components of proteins) to make new muscle fibers, enzymes, hormones, etc. When it comes to using AAs, MPS is among your body's lowest priorities; if your body needs AAs to serve as neurotransmitters, for instance, and you haven't eaten for a long time, it will scavenge even healthy muscle fibers.

MPS is stimulated primarily by the *essential amino acids* (EAAs), the nine AAs your body cannot synthesize and thus needs to get from food.⁶⁰ The [quality of a protein](#) is often assessed based on EAA content. Among the EAAs, the three [branched-chain amino acids](#) (BCAAs — [leucine](#), [isoleucine](#), and [valine](#)) are the most potent MPS stimulators,^{61,62} with leucine being the most potent of all.⁶³

Your heart and stomach are muscular organs, but when you think of muscle building, you think of building your *skeletal* muscle — the type of muscle that moves your skeleton. **To promote muscle growth or preservation, you need to both stimulate your muscles and feed them.** In other words, you need to follow two rules:

- Eat enough [high-quality protein](#) daily.
- Engage in resistance training regularly.

In conclusion, we can already infer two ways in which a keto diet might affect your muscle mass: by affecting your **protein intake** and by affecting your **ability to exercise** (notably, to perform resistance training). So, will a keto diet help or hurt your gains? The time has come to place your bets, as we're now going to examine studies that looked at changes in muscle mass from [keto with prescribed exercise](#) and from [keto without prescribed exercise](#).

You can improve your body composition by decreasing your fat mass or increasing your muscle mass. The latter requires resistance training and high-quality protein. By affecting your protein intake and your ability to exercise, a keto diet might affect your muscle mass.

Keto with prescribed exercise

To be included in our analysis, a study had to meet the following criteria:

- It was a controlled trial (a study in which one group receives the tested treatment while the other receives a placebo, a reference treatment, or nothing at all).
- It measured blood or urine ketone levels to ensure that the participants really followed their keto diet (≤ 50 grams of carbs per day).
- It assessed body composition objectively, through methods such as *dual-energy x-ray absorptiometry* (DXA/DEXA), *bioelectrical impedance* (BIA), or air-displacement plethysmography (as used by BOD POD®).
- Its keto and higher-carb control groups were both prescribed an exercise regimen.

To date, 10 studies qualify.^{64,65,66,67,68,69,70,71,72,73} They lasted anywhere from 6 to 12 weeks (9.8 weeks on average).

Table 4: Controlled trials of keto with prescribed exercise

Study	Participants			Duration (weeks)	Assigned exercise	Diet ^(a) (keto / control)			Ketone measures	
	Age (avg.)	Sex (M / F)	Training status			Energy (kcal/day)	Carbs (g/day)	Protein (g/kg)	Blood(b) (keto / control)	Urine (keto)
LaFountain (2019)	26	25 / 4	Military personnel	12	RT: 2x/wk ET: 1x/wk	NR	NR	NR	1.0 / NR	—
Greene (2018)	35	9 / 5	Olympic & power lifters	12	Maintain 4.3x/wk avg	2,071 / 2,056	39 / 223	1.59 / 1.5	0.4 / NR	—
Kephart (2018)	31	9 / 3	Recreational CrossFit	12	Maintain 2.5x/wk avg (KG) or 1.7x/wk avg (CG)	1,950 / 2,500	15 / 164	1.08 / 1.48	1.5 / 0.2	—
McSwiney (2018)	33	20 / 0	Recreational ET	12	RT: 2x/wk HIIT: 2x/wk ET: 7+ hr/wk	3,020 / 2,640	41 / 400	1.6 / 1.2	0.5 / 0.1	—
Vargas (2018)	30	19 / 0	Recreational RT	8	Upper/lower split RT: 4x/wk	NR	NR	NR	—	Present
Gregory (2017)	35	5 / 22	Recreational CrossFit	6	CrossFit: 4x/wk	1,580 / 1,750	45 / 187	1.2 / 1.08	—	Present
Wilson (2017)	22	26 / 0	Recreational RT	8	Total body RT: 3x/wk	2,610 / 2,550	31 / 318	1.7 / 1.7	1.0 / 0.15	—

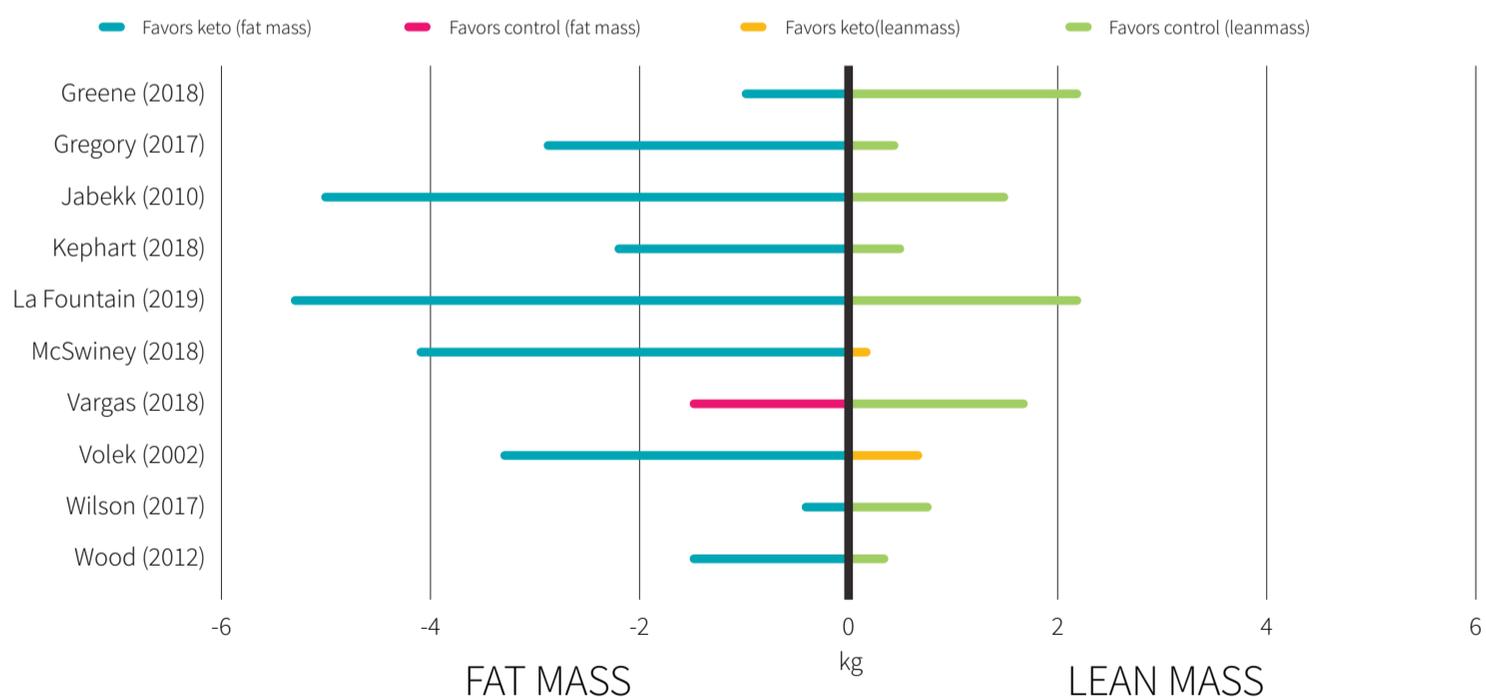
Wood (2012)	59	16 / 0	Untrained	12	Total body RT: 3x/wk	1,575 / 1,590	52 / 235	1.11 / 0.74	—	Present
Jabekk (2010)	20 to 40 ^(c)	0 / 16	Untrained	10	Total body RT: 2x/wk	1,755 / 1,970	23 / 196	1.05 / 0.91	—	Present
Volek (2002)	36	20 / 0	Recreational ET & RT	6	Habitual + ET: 2–5x/wk RT: 2–6x/wk	2,330 / 1,950	46 / 283	2.29 / 0.93	>0.2 / NR	—

CG: control group | ET: endurance training | HIIT: high-intensity interval training | KG: keto group | NR: not reported | RT: resistance training | avg: average | hr: hour | wk: week | (a) *energy* in kilocalories per day (kcal/day), *carbohydrate* in grams per day (g/day), and *protein* in grams per kilogram of body weight per day (g/kg/day) | (b) micromoles of β -hydroxybutyrate (β Hb) per liter of blood (mmol/L) | (c) This study provided a range but no average.

Let's look at some results in the figure below:

- **The keto groups lost more fat in 9 of the 10 studies.**^{66,70,72,73,74,75,76,77,78} On average, the keto groups lost 3.4 kg (7.5 lb) while the control groups lost 1.0 kg (2.2 lb).
- **The control groups had better lean mass preservation or gain in 8 of the 10 studies.**^{66,70,71,72,73,75,76,77} On average, the keto groups lost 0.3 kg (0.66 lb) while the control groups *gained* 0.6 kg (1.3 lb).

Figure 10: Difference in fat-mass loss (left) and lean-mass gain (right) between keto group and control group



Post-test Δ in keto and in control compared. The bars don't represent absolute losses or gains, but the difference in fat loss (left side) or lean-mass gain (right side) between the keto and control groups, for each study. Within each study, both groups were prescribed the same exercise regimen.

So it looks like a keto diet is great for fat loss but causes you to lose lean mass more often than not, right? Well, it's not quite so simple. **Three big caveats** come into play:

First, if your diet is strongly hypocaloric, then getting enough protein (as did the keto groups in 5 of the 8 studies that reported protein intake) may not *prevent* muscle loss, but it will at least *lessen* muscle loss, thus ensuring that most of your weight loss will be in the form of fat. And that's generally what we see in keto-group results: much greater fat loss than lean-mass loss.

Second, the studies used either DXA (9 studies) or BIA (1 study) to assess changes in body composition. This is an issue here because the lean- and fat-mass measurements from both methods are greatly affected by large changes in water + glycogen,^{79,80,81} and we saw that you lose a lot of water + glycogen when you switch from high-carb to keto.

Third, remember that your lean mass isn't just your muscle mass; it includes, notably, your water weight — which you quickly shed when you go keto. The resulting **loss of lean mass can be erroneously interpreted as a loss of muscle mass.**⁸⁰

As we saw, water + glycogen losses within the first week of a keto diet average about 2 kg (4.4 lb) and range from almost nothing to over 4 kg (8.8 lb).^{1,2,3,4} In other words, the losses in lean mass seen in many of the keto studies could be entirely in the form of glycogen and water. In fact, if we subtract a hypothetical 2 kg water + glycogen loss from an average 0.3 kg (0.66 lb) lean-mass loss (the average lean-mass loss in the keto groups over the 10 studies), we end up with a gain of 1.7 kg (3.7 lb).

So, is it possible these keto dieters actually *gained muscle*? Again, we can't say for sure: the only study that measured water losses also used a measurement method, BIA, susceptible to measurement errors when fluid status shifts. Does that mean that all 10 studies are hopelessly confounded? Can *anything* be learned from them?

What we *can* say is that the keto groups **engaged in regular exercise** in all 10 studies and **consumed enough protein** (≥ 1.2 g/kg) in 5 of the 8 studies that reported protein intake — **two factors** that help preserve or even increase muscle mass. The three studies that showed substantial *decreases* of fat mass while *increasing* lean mass while on a keto diet shared the following three qualities:^{64,69,76}

- High daily protein intakes (1.6–2.3 g/kg)
- High exercise volumes and intensities
- Caloric intake levels that did not create drastic caloric deficits (so as to better preserve muscle mass)

Bottom line: we need more studies that take steps to reduce the body-composition measurement errors introduced by sudden shifts in hydration status and glycogen levels.

Doing so will provide us with a better picture of how lean and fat masses actually change while on a keto diet.

🔍 Digging Deeper: Pitfalls of measuring body comp on keto

Just how susceptible DXA estimates are to changes in glycogen and hydration status is well demonstrated in a study of 26 resistance-trained male athletes placed on either a keto or a high-carb diet.⁷⁶

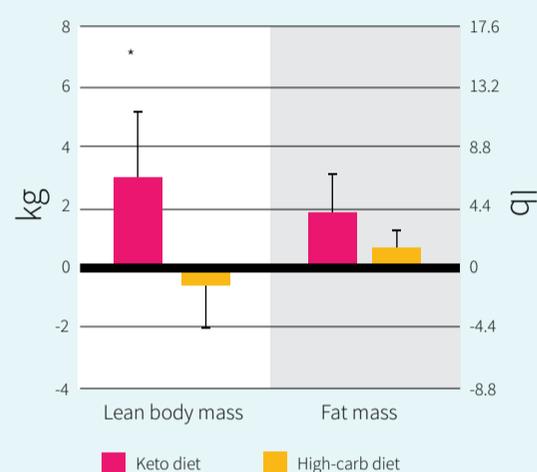
Three DXA scans were taken of the athletes: at baseline, at week 10, and at week 11. After 10 weeks, both groups saw an increase in lean mass (similar in both groups) and a decrease in fat mass (greater in the keto group). Between week 10 and 11, the keto group was switched to a high-carb diet (3 grams of carbs per kilogram of body weight per day) for 7 days before the third and final DXA scan.

As you can see in the figure below, from the second scan (week 10) to the third (week 11), the keto group saw unrealistically large average increases in both lean mass (≈ 3 kg, so ≈ 6.6 lb) and fat mass (≈ 2 kg, so ≈ 4.4 lb).

The large increase in lean mass can easily be explained. [As we saw](#), when you switch from keto to high-carb, you can store more water + glycogen than normal — a phenomenon called *glycogen supercompensation*. You can gain more than 3 kg that way.¹²

As for the large increase in fat mass, it may be partly due to a measurement error. When you switch from keto to high-carb, the resulting increase in water weight causes an increase in the BIA and DXA readings of your *body-fat percentage* (BF%).

Figure 11: DXA body-composition changes after a 1-week high-carb diet



* Significant change from week 10

Adapted from Wilson et al. *J Strength Cond Res.* 2017. PMID:28399015

- With BIA, a $\approx 3\%$ increase in hydration could result in a ≈ 2 percentage-point increase in your BF% reading (e.g., from 20% to 22%), as seen in a study of recreational endurance athletes.⁸²
- With DXA, a $\approx 5\%$ change in hydration could result in a ≈ 2.7 percentage-point change in your BF% reading (an increase in hydration leading to an increase in BF% reading, and a decrease in hydration leading to a decrease in BF% reading).⁸³

There are three methods to measure body composition more accurately:

- **Computerized tomography (CT)** is a complex, computer-processed combination of X-ray measurements taken from different angles. CT can be very expensive — about \$1,000 per scan in the US⁸⁴ — which is why you don't see it used very often in research studies.
- **Magnetic resonance imaging (MRI)** uses a series of radio waves and large magnets to peer inside the structures of your body. Like CT, MRI can be cost-prohibitive: in the US, scanning a single limb costs around \$1,000 on average, with prices ranging from \$500 to \$3,500;⁸⁵ scanning the whole body, which is very rarely done, would cost even more, and could also take hours, depending on the machine.
- **Autopsy.** The parts we want to weigh are cut out of the body. This method is cheaper than the two others, and even more accurate, but it requires you to be dead, so we don't recommend it.

Accurately measuring changes in body composition is surprisingly challenging. When you go keto, you lose a lot of water + glycogen, so a lot of lean mass — and this loss of lean mass can easily be misinterpreted as a loss of muscle mass (and fat mass, to a lesser extent).

Keto without prescribed exercise

To be included in our analysis, a study had to show the following qualities:

- It was a controlled trial (a study in which one group receives the tested treatment while the other receives a placebo, a reference treatment, or nothing at all).
- It measured blood or urine ketone levels to ensure that its participants really followed their keto diet (≤ 50 grams of carbs per day).
- It assessed body composition objectively, by using methods such as *dual-energy x-ray absorptiometry* (DXA/DEXA), *bioelectrical impedance* (BIA), or *air-displacement plethysmography* (as used by BOD POD®).
- Neither its keto group nor its control group was prescribed exercise, but some were allowed to maintain their normal levels of physical activity (as noted in the table below).

To date, 11 studies qualify.^{2,86,87,88,89,90,91,92,93,94} They lasted 2 to 24 weeks (8.5 weeks on average).

Table 5: Controlled trials of keto without prescribed exercise

Study	Participants			Duration (weeks)	Activity Level	Diet ^(a) (keto / control)			Ketone measures	
	Age (avg.)	Sex (M/F)	Health status / BMI			Energy (kcal/day)	Carbs (g/day)	Protein (g/kg)	Blood ^(b) (keto / control)	Urine (keto)
Choi (2018) (4:1)^(c)	30	9 / 4	Healthy / 30 (obese)	2	Habitual ^(d)	1,159 / 1,357	9.7 / 190	0.22 / 0.64	1.44 / 0.22	Present
Choi (2018) (1.7:1)^(c)	25	8 / 5	Healthy / 28 (overweight)	2	Habitual ^(d)	1,280 / 1,357	14.5 / 190	0.62 / 0.64	0.86 / 0.22	Present
Urbain (2017)	37	11 / 31	Healthy / 23 (normal)	6	Sedentary	2,223 / 2,321	40 / 233	1.54 / 1.06	—	Present
Brinkworth (2009)	49	22 / 38	Metabolic risk factors / 33 (obese)	8	Habitual ^(d)	1,558 / 1,547	20 / 183	1.45 / 0.96	0.49 / 0.14	—
Johnstone (2008)	38	17 / 0	Healthy / 35 (obese)	4	NR	1,733 / 1,900	22 / 170	1.13 / 1.24	1.52 / 0.28	Present

Noakes (2006)	48	12 / 55	2 CVD risk factors / 33 (obese)	12	NR	1,480 / 1,449	32.5 / 245	1.39 / 0.80	0.35 / 0.05	—
Johnston (2006)	38	4 / 15	Healthy / 35 (obese)	6	Sedentary	1,500 / 1,500	33 / 157	1.30 / 1.18	0.33 / 0.20	—
Brehm (2005)	45	0 / 40	Healthy / 33 (obese)	4	Habitual ^(d) (moderate activity)	1,531 / 1,422	92 / 171	2.20 / 1.25	—	Present
Volek (2004)	33	15 / 0	Healthy / 34 (obese)	≈7	Majority sedentary	1,855 / 1,562	42 / 226	1.44 / 0.83	—	Present
Meckling (2004)	41	9 / 22	Healthy / 32 (obese)	10	Habitual ^(d)	1,534 / 1,452	59 / 225	1.11 / 0.77	≈0.40 / 0.33	—
Brehm (2003)	43	0 / 42	Healthy / 34 (obese)	24	Habitual ^(d)	1,156 / 1,245	56 / 169.4	0.95 / 0.62	0.42 / 0.10	Present

BMI: body mass index | CVD: cardiovascular disease | NR: not reported | avg: average | (a) **energy** in kilocalories per day (kcal/day), **carbohydrate** in grams per day (g/day), and **protein** in grams per kilogram of body weight per day (g/kg/day) | (b) micromoles of β-hydroxybutyrate (βHB) per liter of blood (mmol/L) | (c) caloric ratio of fat to carbs + protein in the diet of the keto group | (d) The participants maintained their usual level of physical activity, which was overall pretty low (e.g., walking 6,000 steps a day).

We see that, within each study, the keto and control groups consumed similar amounts of calories — and both groups lost lean mass.^{2,86,87,88,89,90,91,92,93,94}

- The keto groups' lean-mass losses ranged from 0.6 to 3.4 kg (1.3 to 7.5 lb) and averaged 1.9 kg (4.3 lb).
- The control groups' lean-mass losses ranged from 0.25 to 2.2 kg (0.6 to 4.9 lb) and averaged 1.3 kg (2.8 lb).

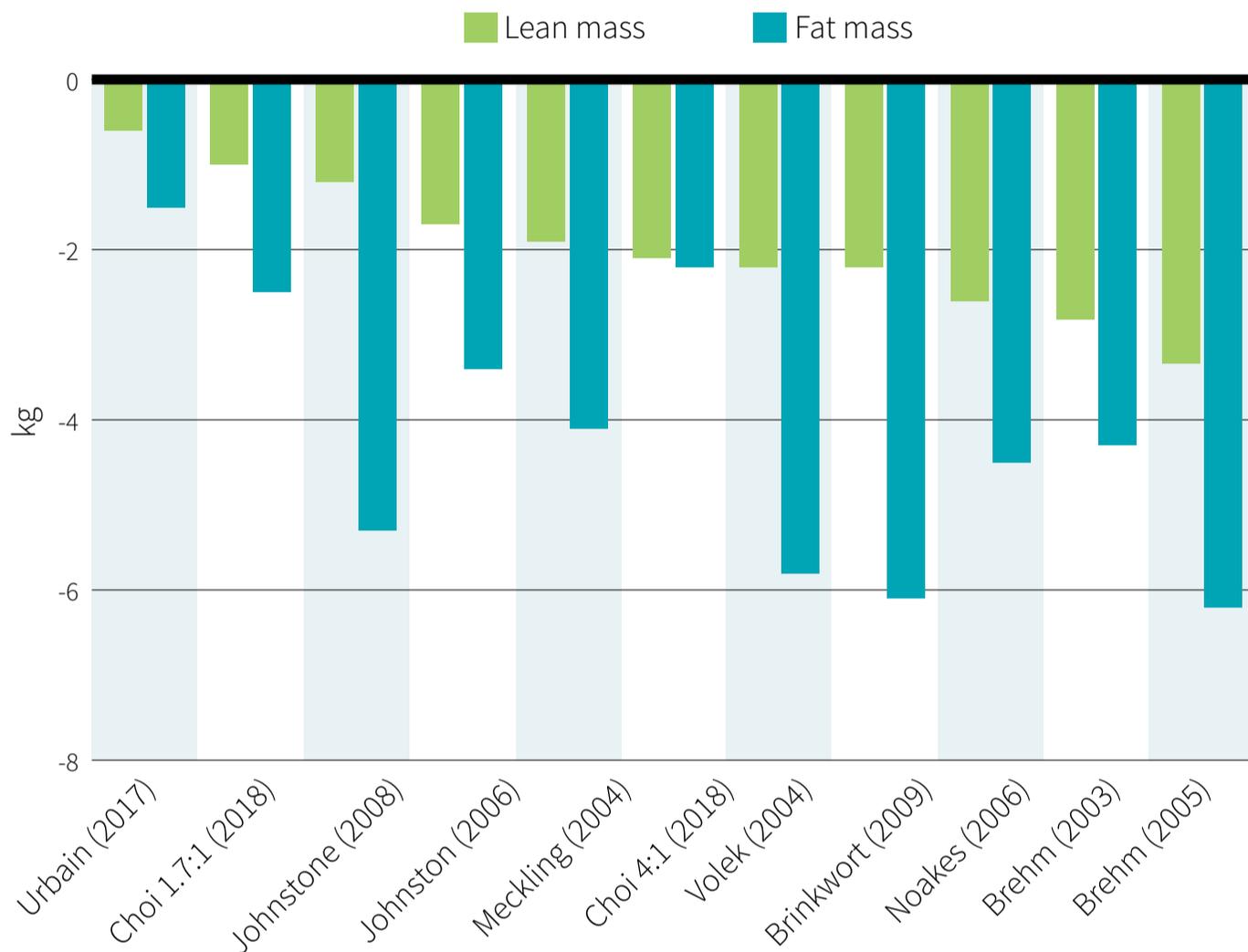
By contrast, in the studies [with prescribed exercise](#), the keto groups lost only 0.3 kg (0.66 lb), while the control groups *gained* 0.6 kg (1.3 lb), on average.

The large difference we see between the two types of studies (with and without prescribed exercise) is a testament to the importance of regular exercise for the preservation of muscle mass, particularly while on a caloric deficit. (Even taking [measurement errors](#) into account, the fat loss experienced by the keto groups in the two types of studies is indicative of a caloric deficit.)

An additional factor explaining the difference is that, in the studies without prescribed exercise, fewer participants managed to achieve the [recommended minimum protein intake](#) of 1.2 g/kg. Still, the keto groups consumed 0.31 g/kg more than the control groups, on average, so they *may* have actually lost less muscle mass than the control groups, even though they lost more lean mass (for reasons [previously explained](#), we can't ascertain how much of the lean mass lost by the keto groups was muscle and how much was water + glycogen).

Finally, we should note that, even without prescribed exercise and even considering that a lot of their lost mean mass may be water + glycogen, not muscle, the keto groups still managed to lose more fat mass than lean mass:

Figure 12: Changes in lean and fat mass on a keto diet without prescribed exercise



Tip: How to accurately measure body-composition changes on keto

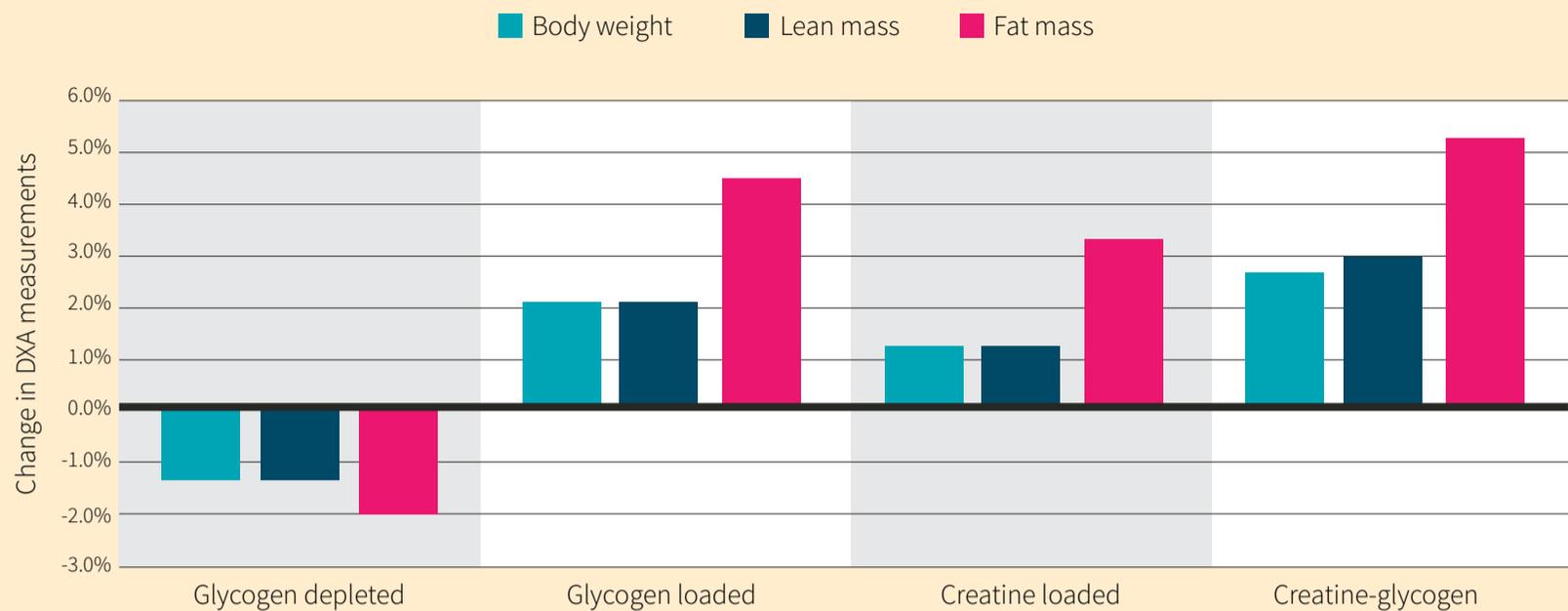
[In the first week or so](#) on a keto diet, you will drop water weight and use up much of your glycogen stores. Because these changes can throw off tools used to measure fat mass and lean mass (DXA, BIA, BOD POD®), it's best not to use pre-keto measurements as your baseline.

Instead, wait 1–2 weeks for your body to adapt to your keto diet ([metabolic-ward studies](#) suggest that it may take 10 days for your body to fully switch from being glucose-efficient to being fat-efficient). *Then* have measurements taken and use **these** as your baseline against which to compare future measurements.

This advice still applies if, instead of the tools listed above, you use calipers or [calculations from limb measurements](#). It also applies if you decide to switch from keto back to a higher carb diet: eat your higher-carb diet for 2 weeks, then take new baseline measurements.

Another confounding factor you need to be aware of: [creatine](#).⁹⁵ Like glycogen, creatine causes water retention and thus can mess up with your measurements. So if you want to be able to compare two measurements, use creatine consistently or not at all.

Figure 13: Different methods of body-water manipulation lead to different DXA readings



Reference: Bone et al. *Med Sci Sports Exerc.* 2017. PMID:[28410328](https://pubmed.ncbi.nlm.nih.gov/28410328/)

Creatine is just one example of a general rule: to reduce potential sources of error, try to **standardize the conditions under which measurements are taken**.

- Test at the same time of day (e.g., at 9 a.m.).
- Test under the same feeding conditions (e.g., before breakfast).
- Test under similar hydration statuses. Don't be over- or under-hydrated. Empty your bladder.
- Don't make any major dietary changes for 3 days before the test.
- Don't exercise for 24 hours before the test.
- Don't apply moisturizing lotions for 24 hours before the test.
- Wear the same or similar clothing (or none, if practicable).
- Use the same equipment, software/algorithm, technician, and body position.

Whether you're on a keto diet or not, getting enough protein and exercise will help you increase or at least spare your muscle mass. This is especially important if your diet is hypocaloric (i.e., if you burn more calories than you eat).

Improving body composition on keto

The basics of [fat loss](#) and [muscle building](#) — and indeed, of [fitness](#) in general — stay the same, regardless of which diet you follow. We'll briefly cover these basics before addressing some keto-specific considerations. First up ...

Protein

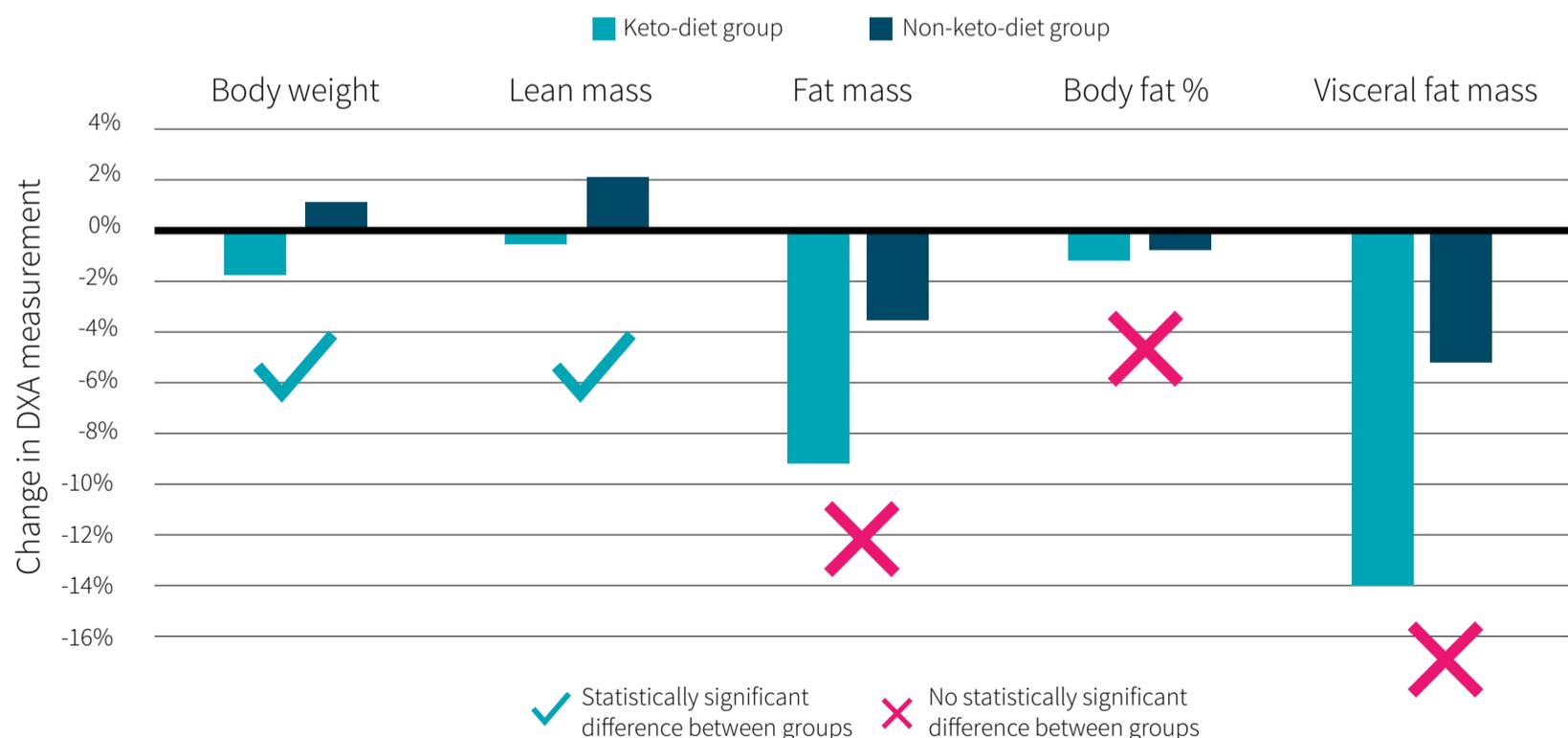
The US Recommended Dietary Allowance (RDA) for protein is 0.8 g/kg of bodyweight (0.36 g/lb),⁹⁶ but what most people misunderstand is that the RDA numbers don't represent *optimal* intakes; rather, they represent the *minimal* intakes needed by healthy, sedentary adults to avoid deficiency-related health issues.⁹⁶

And even for that purpose, 0.8 g/kg may not suffice; more recent research points to 1.2 g/kg (0.54 g/lb) as a better *minimum*.^{97,98,99,100} Your own protein requirements can easily be higher, depending on your circumstances (genetics, age, level of physical activity ...). So, does your being on a keto diet play a role in determining your protein intake?

It's hard to say; there simply isn't a lot of data on this topic. In an 8-week *randomized controlled trial* (RCT), 24 resistance-trained males were divided between three groups: keto, non-keto, and control.⁷¹ The keto and non-keto groups followed the same resistance-training program, and each group received dietary advice structured to cause a moderate caloric surplus with an optimal protein intake (2.0 g/kg). Meanwhile, the control group maintained their regular diet and training.

If we compare the changes in body weight and composition experienced by each group at the end of the 8 weeks, we see that even a calorie- and protein-adequate (2.0 g/kg) keto diet was suboptimal for increasing lean mass during resistance training.

Figure 14: Percentage change from baseline in the KD and NKD groups



Reference: Vargas et al. *J Int Soc Sports Nutr.* 2018. PMID:29986720

This study, however, suffered from three limitations that reduce the confidence we can place in its findings:

- The diets were imperfectly controlled. Meals weren't provided by the researchers to the participants, who were left to control their own diets. Urine ketones were measured weekly, however, and data analysis was restricted to the participants whose ketone levels indicated compliance with a keto diet.
- Body composition was assessed by DXA. [We saw](#) that changes in water weight can alter DXA readings for both fat and lean mass, and [we saw](#) that people who switch to a keto diet lose a lot of water as they empty their glycogen stores.
- There was no training-performance assessment.

A safe assumption is that you need to consume *at least* 1.2 g/kg (0.54 g/lb) a day if you are not in a caloric deficit. However, many people seem to spontaneously decrease their caloric intake when they switch to a keto diet. If that's your case, you'll probably need more than 1.2 g/kg to increase or at least preserve your muscle mass.

Here's a quick rundown of how much protein you may need in different situations. If you want to know more, check out our [in-depth article on protein needs](#).

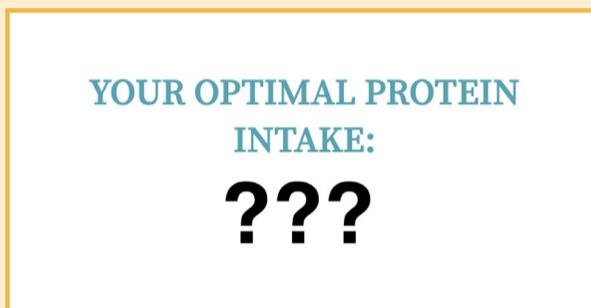
- If you're **sedentary**, aim for at least 1.2 g/kg (0.54 g/lb). Keep in mind that your body composition will improve more if you add consistent activity, especially resistance training, than if you merely hit a protein target.
- If you're of **healthy weight, active, and wish to keep your weight**, aim for 1.4–1.6 g/kg (0.64–0.73 g/lb). People who are trying to keep the same weight but improve their body composition (more muscle, less fat) may benefit from the higher end of the range.
- If you're of **healthy weight, active, and wish to build muscle**, aim for 1.4–2.4 g/kg (0.64–1.09 g/lb). If you're an experienced lifter in a bulking phase, intakes of up to 3.3 g/kg (1.50 g/lb) may help you minimize fat gain.
- If you're of **healthy weight, active, and wish to lose fat**, aim for 1.8–2.7 g/kg (0.82–1.23 g/lb), skewing toward the higher end of this range as you become leaner or if you increase your caloric deficit (by eating less or exercising more).
- If you're **overweight or obese**, aim for 1.2–1.5 g/kg (0.54–0.68 g/lb). This range, like all the others in this list, is based on your *total* body weight (most studies on people who are overweight or obese report their findings based on total body weight, but you'll find some calculators that determine your optimal protein intake based on your lean mass or your *ideal* body weight).

- If you're **pregnant**, aim for 1.66–1.77 g/kg (0.75–0.80 g/lb).
- If you're **lactating**, aim for at least 1.5 g/kg (0.68 g/lb).
- If you're **vegan or obtain most of your protein from plants**, then your protein requirements may be higher because plant-based proteins are usually inferior to animal-based proteins (with regard to both bioavailability and amino acid profile).



Tip: Use our Protein Intake Calculator

Your protein needs hinge on many factors — notably your weight, health goals, and level of physical activity. Based on our research and the data you input, we can calculate your optimal daily protein intake. Click on the image below to get started!



Getting enough protein on a keto diet shouldn't be too hard, unless you're vegetarian or vegan: fish, seafood, meat, and eggs have little to no carbs. Eggs, for instance, are 13% protein, 11% fat, and 1% carbs. And of course, many protein powders have very little carbs, notably the whey protein isolates advertised as lactose-free.

Is there a risk from *too much* protein?

If you eat more protein than your body needs, won't it be turned into glucose through gluconeogenesis, thus kicking you out of keto?

Medical ketogenic diets (used notably to help manage epilepsy) require high levels of blood ketones, so protein intake must be restricted alongside carbohydrate intake because eating a lot of protein can reduce ketone formation. There is a persistent belief that gluconeogenesis is responsible, but this may not be biochemically correct, for two reasons:

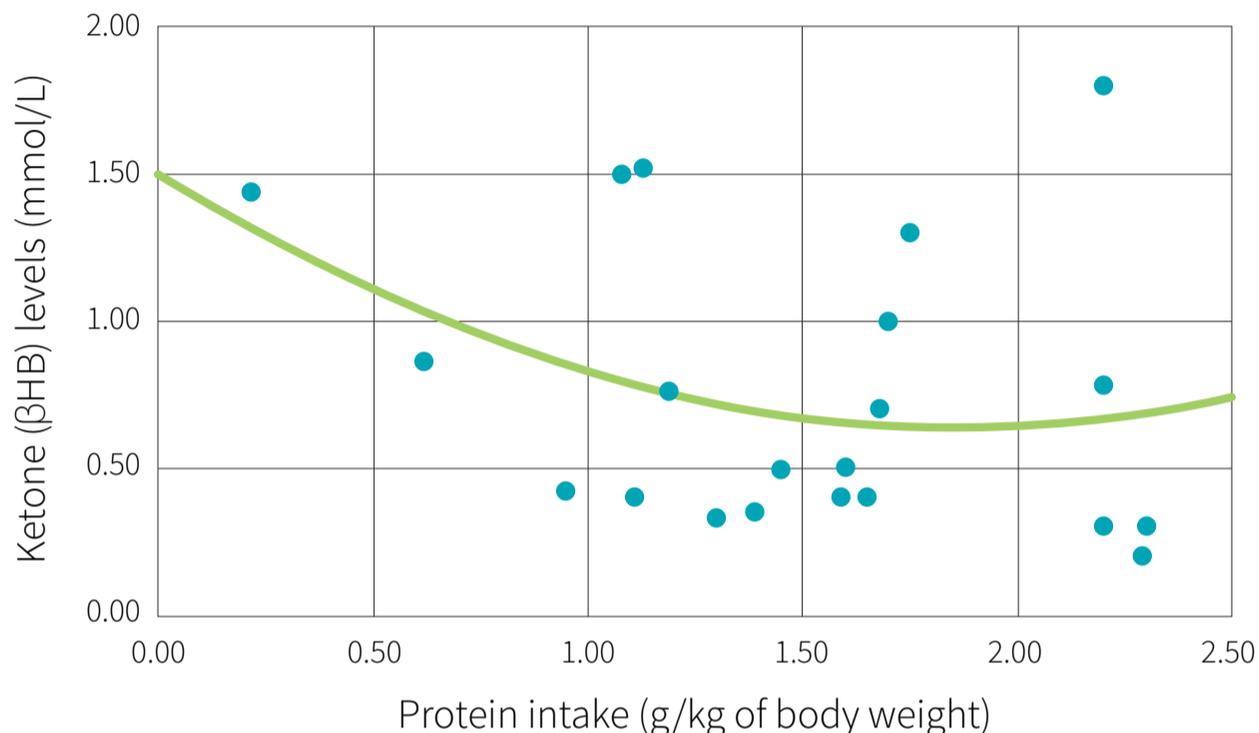
First, it doesn't make sense that gluconeogenesis would interfere with ketogenesis because both processes are up-regulated during fasting and occur simultaneously.

Second, gluconeogenesis is largely driven by demand, not supply. For example, on a standard diet (55% carbs), gluconeogenesis led to the creation of 145 grams of glucose per day, which increased to 171 grams — a mere 24 grams more — when carbohydrate intake was dropped to zero and protein intake was tripled.¹⁰¹ Note that both numbers are very high because gluconeogenesis was measured after an intense, glycogen-lowering exercise session; in sedentary people, the numbers would likely be lower.

Importantly, eating protein doesn't prevent ketosis — it only reduces the extent of ketosis. But to what degree? And, if you are not on a medical ketogenic diet, does it matter?

Based on a comprehensive review of ketone metabolism, blood ketone levels of 0.2–7.0 mmol/L indicates a state of ketosis (although 0.5 mmol/L is the more commonly used cutoff).¹⁰² The below chart compares various protein intakes to levels of blood ketones in 19 studies.^{2,15,64,70,72,76,78,87,88,90,91,92,93,103,104,105,106,107}

Figure 14: Protein intakes and blood ketone levels



An analysis of these studies suggests that a higher protein intake *might* reduce ketogenesis (i.e., your body's production of ketones), but to what extent is uncertain. The evidence is all over the place, ranging as it does from one study correlating a protein intake of 1.3 g/kg with ketone levels of 0.33 mmol/L⁹¹ to a study correlating a protein intake of 2.1 g/kg with ketone levels of 1.80 mmol/L.¹⁰⁴

Does that mean that different protein intakes affect different people differently? That's a possibility, but there could also be other factors at play. Notably, we saw how difficult it is, in free-living studies, to ensure that people really consume as little carbohydrate as they should, and a few grams too many can easily kick you out of ketosis.

Note that, of the studies analyzed, the study that correlated a protein intake of 2.1 g/kg with ketone levels of 1.80 mmol/L was one of the best controlled: rather than a free-living study per se, it was a study in elite race walkers undergoing a 3-week training camp, during which they ate only the food provided by the researchers.¹⁰⁴ (You can read our detailed review of this study [here](#).) But *the* best controlled of the studies analyzed was a metabolic-ward study, and it correlated a protein intake of 1.19 g/kg with ketone levels of 0.76 mmol/L.¹⁵

Of course, the levels of fitness and physical activity of elite race walkers at a training camp were very different from those of obese individuals confined to a metabolic ward. So levels of fitness and physical activity may matter more for ketone production on a keto diet than protein intake. Yet that doesn't mean that protein intake doesn't matter at all.

So, in the end, there's only one way for you to make sure: try. Start with a protein intake of 1.2 g/kg, measure your ketone levels for at least a week (starting from no earlier than the third week after you switched to a keto diet, to ensure that your body has had time to adapt), then augment your protein intake little by little each week, and see how it affects your ketone levels (preferably via blood testing, given the low accuracy of urine testing).

Keep in mind that, whereas keeping ketones levels consistently elevated is critical for *medical* keto diets (such as are used to treat epilepsy), a modest 0.5 mmol/L is enough to ensure a state of ketosis (in which your primary fuel has switched from glucose to fat).

Your protein needs increase based on various factors (notably your weight, health goals, and level of physical activity), but we don't know if a keto diet is one of them. Your body can make glucose out of protein; high protein intakes might reduce your blood ketone levels, but to what extent is uncertain. Note that, unless you follow a *medical* keto diet, ketone levels of 0.5 mmol/L should suffice to provide you with the benefits you seek (your primary fuel source will have switched from glucose to fat).

Calories

Your caloric needs will fluctuate depending on your goal: weight gain, loss, or maintenance.

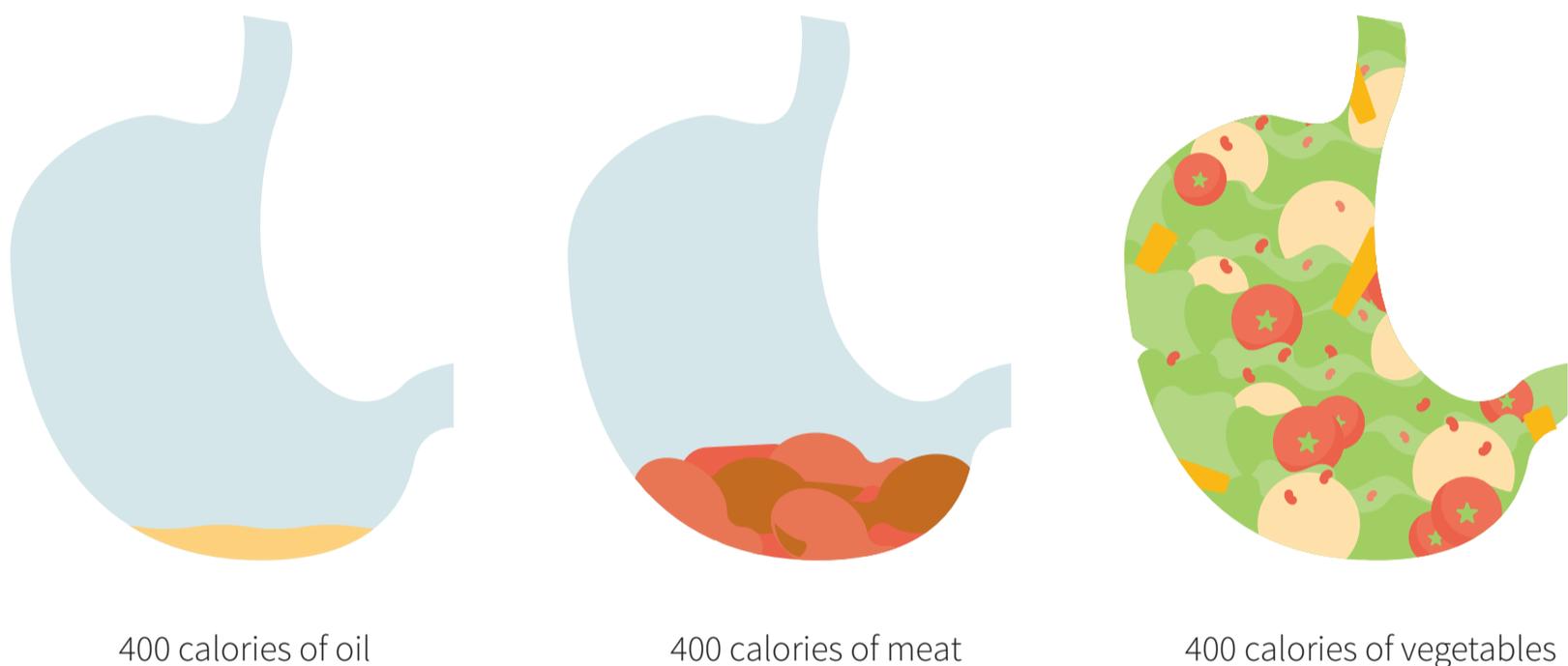
If building muscle is your priority, you'll want to eat more calories than your burn (you'll want your diet to be **hypercaloric**). Note, however, that most people need only some 200 kcal/day above maintenance to maximize muscle growth.¹⁰⁸ If you eat too much above maintenance, you risk accumulating too much fat, which you'll later have to lose.

If losing fat is your priority, then you'll want to eat fewer calories than you burn (you'll want your diet to be **hypocaloric**). Note, however, that the greater your caloric deficit, the more tired you may feel, and the harder you may find it to exercise (though some people find themselves with *more* energy when they switch from a diet high in junk foods to a keto diet, even when they eat less on the latter). Also, the greater your caloric deficit, the harder it becomes to build muscle — but *harder* doesn't mean *impossible*. At the very least, don't eschew exercising entirely; if you do, you risk *losing* muscle, which you'll later have to regain.

While people tend to decrease their calorie intake when they go low-carb or keto, this does not seem to last forever (most people go back to eating as much as they did before going keto, or slightly less).⁴² A more conscious effort to keep calories in check may be needed the longer you are on a keto diet. And remember, what starts as a caloric deficit will eventually become caloric maintenance — the amount of calories needed to maintain your new, lower weight.

Importantly, if you go keto, keep in mind that fat is the most energy-dense macronutrient. If much of your fat intake comes from oils and other processed sources rather than whole foods, don't be surprised if fat loss doesn't happen. A ketogenic diet isn't magic — it just tends to help people eat less.

Figure 15: The concept of energy density



Foods with lower caloric density are sometimes called *higher-volume foods*. By making you feel fuller, they can help you keep your body weight in check. Favor meat over oil, for instance, and don't eschew all vegetables. [Lettuce](#), for instance, is 1% protein and 0% fat, which doesn't look too keto, but it is also 95% water and 2% fiber, and so has very low caloric density. It can easily fill your stomach, which is why simply eating a salad at the start of a meal can reduce your caloric intake for the whole meal.¹⁰⁹

A keto diet has to be low in carbs, but it doesn't have to be all fat. It is worth remembering that foods rich in [water, fiber, and protein](#) are especially satiating.

**Tip: Calculating your caloric needs**

Your height, weight, age, and level of physical activity all contribute to your caloric needs. There are many calorie calculators out there, but one does stand above the rest:



This calculator has been tested and validated against real-world data.¹¹⁰ It can estimate the number of calories *you* need to reach then maintain a specific weight. Click on the image above to get going!

Keto isn't magic: it does tend to make people less hungry, at least in the short term, but it won't allow you to lose fat if you eat more calories than you burn and don't exercise. Even on a keto diet, prioritizing whole foods is important to promote satiety.

Training

On the whole, training on keto isn't different from training on any diet, so we'll start by reviewing a few basic principles; but if you want, you can skip directly to the [specific recommendations for keto dieters](#).

General recommendations

Your protein needs are dialed in, and so are your caloric needs. But to take full advantage of a well-tuned diet, you need to engage in regular exercise. As you decide on a training regimen, keep in mind these **four tips**:

Tip 1 — Don't overdo it. Don't *underdo* it, either. You must exercise hard enough to stimulate muscle growth, but not so hard as to injure yourself or impair your recovery.

Tip 2 — Include resistance training in your exercise regimen. To preserve your muscle mass, and even more to increase it, you need *at least* two resistance workouts a week.¹¹¹

Tip 3 — Cardio can increase blood flow to the muscles, feeding them the nutrients they need for recovery, and it can limit fat gain on a [hypercaloric](#) diet by keeping fat-burning pathways active. But too much cardio can also hinder muscle growth by burning up calories, cutting into recovery, and interfering with anabolic signaling pathways. If your primary goal is muscle building, take it easy on the cardio.¹¹² If your primary goal is weight maintenance or fat loss, some more cardio can help you create a caloric deficit or balance.

Tip 4 — Don't quit! Consistency is the name of the game. Be aware that you're likely to meet three major obstacles in your training journey:

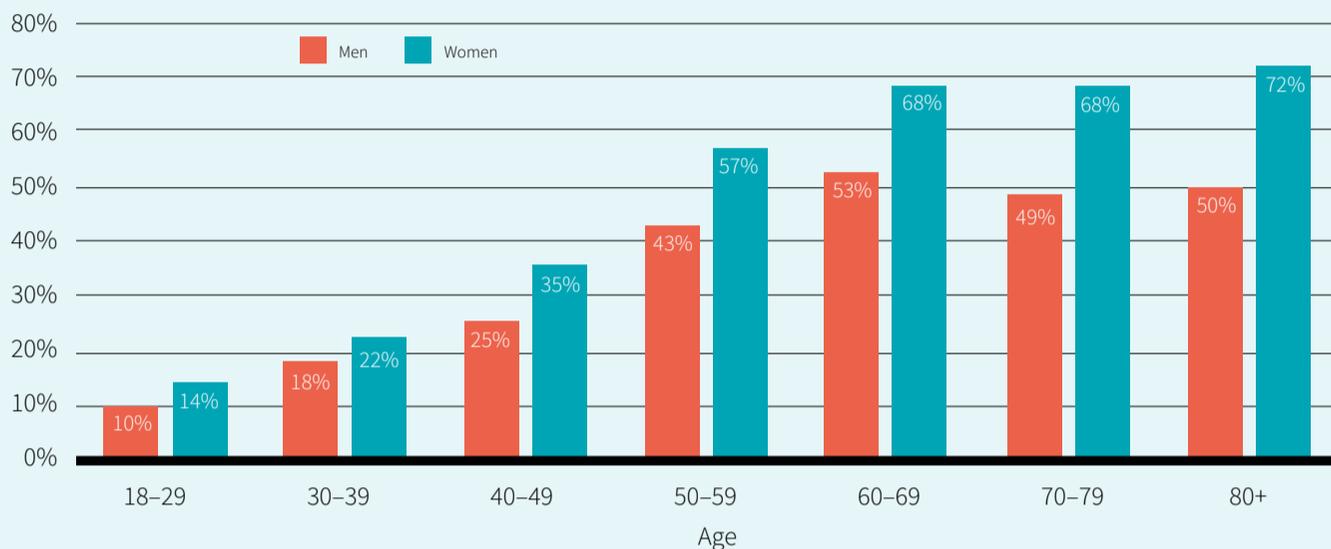
- **When you start.** When you start an exercise regimen, it's hard. You get tired easily and are sore the next day. It's all too easy to quit, or more often to push back today's workout to tomorrow ... every day. To avoid falling into this trap, willpower is key, as is not lying to yourself ("I'll do it tomorrow, yes, I promise, *this time* it's true") or searching for excuses (yes, you do need to buy your groceries, wash your car, do this, to do that ... but let's face it: we *always* have something left to do, especially if there's something else we'd rather *not* do). You could also make yourself accountable to an online support group or simply to a friend — especially one who can go to the gym with you.
- **When you hit a plateau.** You exercise consistently and seriously, but sooner or later, you hit a plateau. Your fat loss or muscle growth slows down to a crawl, or stops entirely. You may need to tweak your diet or your exercise regimen ... or you may just need a break. But if you take a break, make sure you have a plan to get back into action, otherwise you risk pushing back your return to the gym indefinitely.
- **When life intrudes.** Life *always* intrudes. But some times more than others. You may have a deadline coming at work, or a cold, or a family vacation ... the possibilities are endless. The good news is: a short break from exercising isn't in itself a big deal.^{113,114} The bad news is: once your exercise routine gets interrupted, it can be hard to resume — especially if enough time has passed that you fear your performance has decreased (nobody wants to face *that*). Again, in such a case, willpower is key, and having a plan to get back into action will help a lot.

🔍 Digging Deeper: Age-related muscle loss

“Don’t quit!” isn’t just a tip; it’s a lifetime — and life-saving — recommendation. The older you get, the greater becomes your muscles’ anabolic resistance (i.e., their resistance to growth), and so the greater the exercise volume and protein intake you’ll need to ward off sarcopenia (i.e., age-related muscle loss).

In the US, **sarcopenia** affects more than 40% of men and 55% of women over the age of 50.¹¹⁵ It may be the primary cause of physical frailty,¹¹⁶ which is associated with a higher risk of fractures,¹¹⁷ falls,¹¹⁸ hospitalizations,¹¹⁹ disabilities that affect daily activities,¹²⁰ and having to go to a nursing home.¹²¹

Figure 16: Prevalence of sarcopenia in the US



Reference: Janssen et al. *J Am Geriatr Soc.* 2002. PMID:12028177.

Training on keto

The [four tips](#) above are all general recommendations, and the supplements likely to promote [muscle gain and exercise performance](#) are mostly the same whether you’re on a keto diet or not (carb-heavy supplements, such as weight gainers and most energy drinks, being the obvious exception). But aren’t there considerations specific to people who **train on keto**?

Well, yes, there are. In the next two chapters, we’ll see how keto diets affect [strength](#) and [endurance](#); in the [Ketone Supplements](#) chapter, we’ll cover MCTs and ketone supplements; and in the [Warnings](#) chapter, we’ll address your salt intake (a topic we touched upon [earlier](#) in the current chapter). Right now, though, we’ll just cover two items that may serve to improve your training experience while on a keto diet: **creatine monohydrate** and **targeted carb intake** (also known as a *targeted ketogenic diet*, or TKD).

[Creatine monohydrate](#) is backed by strong evidence for its safety and its ability to increase power output and anaerobic endurance. It does so by increasing your muscle stores of creatine phosphate, which can be used to generate energy through the phosphagen system, as we’ll see in the Strength chapter. The standard dose is 5 g/day. People with more muscle

mass may benefit from as much as 10 g/day, but this claim is not fully supported by the evidence. To supplement 10 g/day, take 5 grams twice a day.

And if you're on keto, creatine monohydrate has an added advantage, though a theoretical one. Your muscles need sufficient water and glucose to perform at their best, but when you're on keto, they have less of both. Creatine can alleviate this problem: it can draw glucose into your muscles during contractions (so especially during exercise), and both the extra glucose and the creatine itself can increase muscle hydration.

However, if your keto diet is rich in meat, which is relatively rich in creatine,^{122,123,124} the effect of creatine supplementation on exercise performance may not be as pronounced.

Table 6: Approximate creatine content of select meats (g/kg)

RAW MEAT*	CREATINE CONTENT
Beef	4.7–5.5
Chicken	3.4
Rabbit	3.4
Cardiac tissue (ox)	2.5
Cardiac tissue (pig)	1.5
Kidney	0.2
Liver	0.2
Lung	0.2

* The creatine content may decrease with cooking time.

References: Harris et al. *Res Vet Sci*. 1997. PMID:[9160426](#) • Dahl. *J Sci Food Agric*. 1965. PMID:[5841078](#) • Harris et al. *Clin Sci (Lond)*. 1992. PMID:[1327657](#)

Targeted carb intake, also known as *targeted ketogenic diet* (TKD), is another strategy to consider. Compared to a regular keto diet, TKD allows you to get a bit more carbs, but those extra carbs must be eaten around the time you exercise. TKD has **three potential advantages**:

- TKD can make it easier for your body to refill your glycogen stores between workouts (though your glycogen stores get at least partially refilled [even if you don't eat carbs](#)).
- While no study has looked at the effect of TKD on exercise performance, studies on periodized carbohydrate intake for athletes suggest that **TKD could improve exercise performance**.¹²⁵
- **TKD could make it easier to stick to a keto diet** by offering times when getting a “carb treat” is allowed. As a bonus, the prospect of a treat can also help motivate you to go to the gym.

To try TKD, consume 10–50 grams of carbs during your workout or up to 30 minutes before. Finding how much extra carbohydrate your body can tolerate without getting kicked out of ketosis will be a trial-and-error endeavor requiring that you measure your ketone levels (preferably via blood testing, given the low accuracy of urine testing).

Training considerations are mostly the same for keto dieters as for everyone else. Keto dieters, however, may need to increase their intakes of salt and fluids, and they might benefit from creatine more than other people. Also, some keto dieters swear by the *targeted ketogenic diet* (TKD), which lets them consume 10–50 grams of extra carbs per day, taken during their workout or up to 30 minutes before.

Sleep

Sleep, or the lack thereof, affects just about every aspect of your health. [Getting enough quality sleep](#),¹²⁶ with or without the help of [supplements](#), may notably alleviate some of the unpleasant side effects (including nausea, constipation, and fatigue) some people experience when they transition to and from a keto diet.

Table 7: Recommended hours of sleep, by age

AGE	RECOMMENDED	MAY BE APPROPRIATE	NOT RECOMMENDED
0–3 months	14–17	11–19	<11 or >19
4–11 months	12–15	10–18	<10 or >18
1–2 years	11–14	9–16	<9 or >16
3–5 years	10–13	8–14	<8 or >14
6–13 years	9–11	7–12	<7 or >12
14–17 years	8–10	7–11	<7 or >11
18–25 years	7–9	6–11	<6 or >11
26–64 years	7–9	6–10	<6 or >10
≥65 years	7–8	5–9	<5 or >9

Adapted from Hirshkowitz. *Sleep Health*. 2015. PMID:[29073412](#)

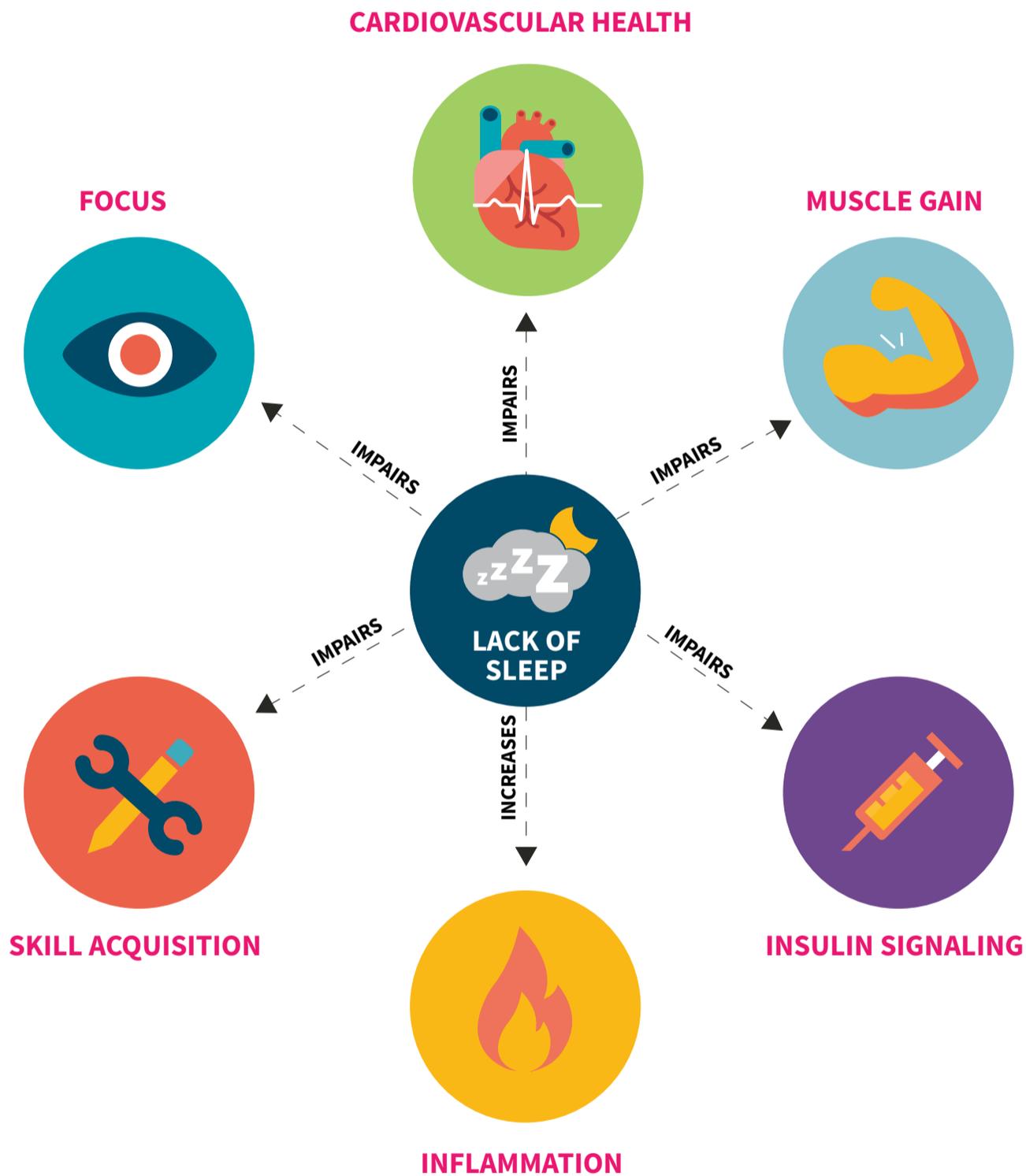
Conversely, lack of sleep impairs exercise performance in the short and long term.^{127,128} Part of the reason it impairs exercise performance in the long term is that it promotes inflammation, thus delaying muscle repair and increasing the risk of injury.¹²⁹

Since lack of sleep delays muscle repair, it also hinders muscle gain. While we can't say that more muscle mass is built during sleep than during waking hours, we do know that

lack of sleep can inhibit the creation of new muscle fibers,¹²⁸ possibly because of the aforementioned increase in inflammation, possibly because of a drop in [testosterone](#) levels, and possibly because of an increase in fatigue decreasing the quality of the workouts.^{130,131,132,133,134}

And to add insult to injury, lack of sleep also leads to fat gain.¹³⁵

Figure 17: How lack of sleep affects you



Yet there's a positive side to this discussion. [Exercise itself can actually improve sleep quality.](#)¹³⁶ That makes for quite a nice synergistic relationship, right? So, if you're looking to improve your sleep, try following these three steps:

1. Schedule enough time for sleep each day. Yes, we do mean *schedule*. Think of this as a very important meeting with your body that you *cannot* afford to miss.
2. Try going to bed at the same time every day, even during the weekend, as this habit both reduces the time it takes to fall asleep and improves sleep quality.
3. For at least one hour before bed, relax and avoid sources of blue light. No emails, text messaging, and no overly stimulating television shows or games. Look for something that allows you to tune out and turn your brain off.

Lack of sleep promotes fat gain, hinders muscle gain, and may worsen some of the unpleasant side effects some people experience when they transition to and from a keto diet. Make sure you get enough *quality* sleep, especially during times when your body is stressed (as when you're sick, or training hard, or transitioning from one diet to another).

Chapter 9: Strength

Summary

Resistance training requires that you convert glucose to lactic acid. Since the easiest way for your body to make glucose is to break down the carbs you eat, it wouldn't be surprising if keto diets impaired strength performance and gains — but they don't. If you don't eat carbs, your body will synthesize enough glucose to fuel your next workout.

- Glucose (“blood sugar”) is stored as glycogen notably in your muscles. Your muscles can break down this glycogen back into glucose, which it can then use to generate energy through either **aerobic** or **anaerobic** metabolism.
- **Aerobic metabolism** is the use of oxygen to oxidize (i.e., “burn”) glucose, fat, and ketones. It can generate energy indefinitely (it powers not only marathons but also most of the basic functions of your body) but is too slow to fuel bursts of effort (such as sprints).
- **Anaerobic metabolism** produces energy with no oxygen. It is fast, so it can produce a lot of energy quickly — but not for long. Bursts of effort are fueled with creatine phosphate (through the phosphagen system) for the first few seconds, then with glucose (through anaerobic glycolysis).
- **Anaerobic glycolysis** produces energy by converting glucose to lactic acid (quickly buffered to lactate). It peaks after 10–20 seconds of all-out effort, then sharply declines. It is the main method of energy production for resistance training — the kind of training you need to build muscle and strength. In other words, resistance training requires glucose.
- Since the easiest way for your body to produce glucose is to break down the carbs you eat, **in theory** keto diets should impair resistance training; but **in practice**, they don't, because your body will replenish your glycogen stores between workouts — even if you don't eat any carbs.
- **Gluconeogenesis** is, literally, the generation (the synthesis) of new glucose — from ketones, fat-derived glycerol, protein-derived amino acids, and recycled pyruvate and lactate. The more you exercise, the more of these substrates you produce, and thus the more you can recycle. In short, if you're on keto, your body will make sure to synthesize enough glucose to feed your brain and, by replenishing your glycogen stores, fuel your next workout.

No carbs, no strength: the theory

“No carbs, no strength.” The common belief is that keto promotes endurance (as we’ll see in the next chapter) but impairs strength. To understand the basis for this belief, you need to know a little about your body’s methods of energy production.

In your body, energy takes the form of molecules of *adenosine triphosphate* (ATP). For that reason, ATP is often called “the energy currency of the cells” or simply “life’s energy currency”. Your body can produce ATP with or without oxygen:

- **Aerobic metabolism** uses oxygen (*aero-* means “air”). Your body aerobically produces ATP by oxidizing — i.e., by using oxygen to “burn” — glucose, fat, or ketones.
- **Anaerobic metabolism** uses no oxygen. Your body can anaerobically produce ATP in two ways: anaerobic glycolysis and the phosphagen system.

Aerobic metabolism is slow. Anaerobic metabolism is fast. Feats of strength require that you produce ATP quickly. For that purpose, you need creatine (to fuel the phosphagen system) and glucose (to fuel anaerobic glycolysis).

The **phosphagen system** (a.k.a. ATP-CP system) is the process of using *creatine phosphate* (CP, a.k.a. phosphocreatine) to recycle “used ATP”. It is the main reason why supplementing with [creatine monohydrate](#) can enhance strength performance. (There’s also a reason why creatine supplementation may benefit keto athletes specifically, as [we saw](#) in the “Body Composition” chapter.)

Anaerobic glycolysis is the process of converting glucose into lactic acid. It is the reason why a diet rich in carbs (which your body breaks down into glucose) has long been considered necessary for optimal strength performance. In fact, a daily intake of 4–7 grams of carbs per kilogram of body weight (4–7 g/kg, so about 1.8–3.2 g/lb) has been recommended to strength athletes, based on data showing that resistance training relies primarily on anaerobic metabolism to produce energy.¹

Digging Deeper: What happens when you go all out?

How much energy you derive from either **aerobic** or **anaerobic** metabolism depends on energy demand, which is determined by your exercise intensity (usually expressed as a percentage of your VO_2 max, as [we’ll see](#) in the “Endurance” chapter).

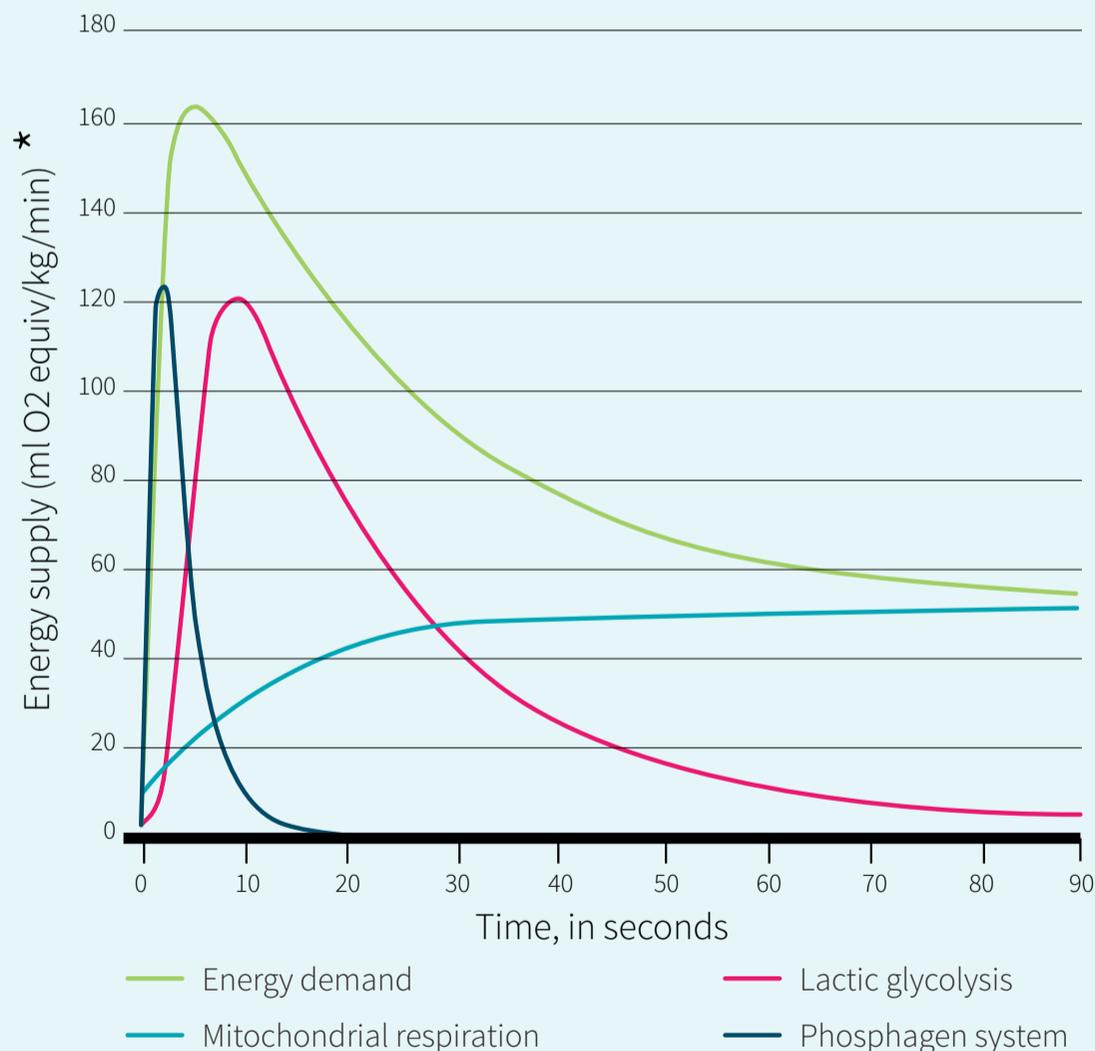
Your oxygen supply (VO_2) can increase through short-term adaptations to exercise, such as increases in breathing rate and heart rate (your heart beats faster to make your blood circulate faster, to bring more oxygen-rich red blood cells to your muscles). Likewise, the maximal rate at which you can use oxygen during exercise (VO_2 max) can increase through long-term adaptation to exercise.

But even then, compared to anaerobic metabolism, aerobic metabolism is slow — too slow to support high-intensity bursts of effort. In other words, when you sprint or lift heavy weights, your exercise intensity exceeds 100% of your VO_2 max. When that happens (which is to say, when your energy demand exceeds the energy available through aerobic metabolism), you cross the anaerobic threshold. To increase your energy expenditure beyond this threshold, you need to rely on the two kinds of anaerobic metabolism: **anaerobic glycolysis** and the **phosphagen system**.

When you go all out, immediate energy demands are met by the **phosphagen system**, which can produce a lot more energy than any other method. It is so efficient that ATP levels barely decrease during the first seconds of all-out exertion. CP levels, however, drop by 75% to 85% within the first ten seconds, and after 20 seconds the phosphagen system contributes little to no energy.² **Anaerobic glycolysis** picks up the slack for a short time, but its contribution to energy production peaks after 10–20 seconds and sharply declines thereafter.^{2,3,4}

This decline in energy supply causes a decline in exercise intensity, which in turn causes a decline in energy demand, so that supply and demand quickly balance each other out. Assuming all-out exertion from the get-go, the relative contributions of each of the three methods of energy production can be seen in the figure below.

Figure 1: The relative contributions of three methods of energy production to the total energy supply during 90 seconds of all-out cycle exercise



* The amount of energy produced by Y milliliters of oxygen per kilogram of body weight per minute, or the equivalent for anaerobic metabolism.

Adapted from Gatin. *Sports Med.* 2001. PMID:11547894

Strength is the ability to overcome resistance; but what does that mean, in practical terms? Who was stronger: Minoru Yoshida, when he plowed through 10,507 nonstop push-ups in 1980? Or Ryan Kennelly, when he bench-pressed 487.6 kg (1,075 lb) once in 2008? Minoru Yoshida was ropy; Ryan Kennelly was bulging; neither could have done what the other did. So?

So, the point is apparently moot, with regard to **anaerobic glycolysis**. Yoshida used mostly **aerobic metabolism** (which, actually, within the context of this guide, makes his feat less a feat of strength than one of endurance — the topic of our next chapter) and Kennelly used mostly the **phosphagen system**.

However, when people speak of growing stronger, they usually speak of lifting heavier weights, rather than doing more push-ups. To lift heavier weights, you need to develop bigger muscles. And to develop bigger muscles, you need to train so as to use anaerobic glycolysis.

In other words, resistance training requires that you lift weights that are neither so light that you can just use aerobic metabolism to lift them nor so heavy that you can lift the weight but once (thus leaving little time for anaerobic glycolysis to kick in). Kennelly didn't develop his bulging muscles by performing just one repetition of the heaviest weight he could lift then waiting a few minutes for his CP stores to refill before doing just one rep again, and so on and so forth.

There's a clear interaction between the weight you lift, the number of reps you can reach with that weight, and the time it takes you to complete a set. The ideal weight and reps for a given exercise — and even the speed at which that exercise should be performed — are still hotly debated. Further, whereas the ideal number of reps may be about the same for everyone, the ideal weight will vary greatly between individuals and will increase as your strength increases. Which is to say that the ideal weight can only be expressed as a *relative* weight.

Relative to what? Relative to the heaviest weight you can lift.

Your *1 repetition maximum* (1RM) is the heaviest weight you can lift at least once. Of course, your 1RM for one exercise will differ from your 1RM for another. In studies on strength, you will often see weights expressed either as an "X"RM (where X is the maximum number of reps you can reach with that weight) or as a percentage of 1RM.

- **1RM.** The maximum weight you can lift.
- **25% of 1RM.** If your 1RM is 80 kg, then 25% of your 1RM is 20 kg.
- **15RM.** The maximum weight you can lift a maximum of 15 times.

So what do studies tell us?

This guide isn't the place for a long exploration of the field, but we'll note a similar, small rise in blood levels of lactate under the following circumstances:

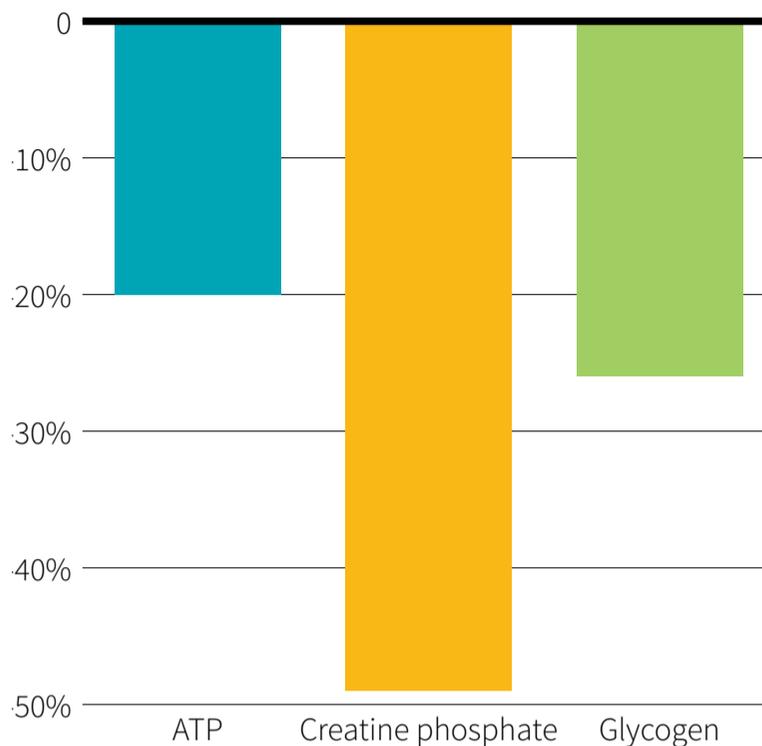
- 30 repetitions with 25% of 1RM on the back squat.^{5,6,7}
- 20 repetitions with 30% of 1RM on the leg press.⁸
- 5 repetitions with 10RM on the leg press.⁹

In all three cases, anaerobic metabolism is clearly involved, but only to a small extent, so those exercises aren't ideal to increase strength or muscle mass. Now compare this last exercise (5 reps of 10RM on the leg press) with a much more demanding exercise: 10 reps of 10RM on the leg press (doing 10 reps of your 10RM means that you're doing the set "to failure" — with this weight, you couldn't do even one more rep). You end with much higher blood levels of lactate: 2.4-fold higher than by lifting the same weight only 5 times.⁹

Most of the energy used in those 10 muscle contractions came from anaerobic metabolism: 64–71% from anaerobic glycolysis; 29–36% from the phosphagen system.¹⁰ The contribution from anaerobic glycolysis increased from 46–54% during the first 5 reps to 81–85% during the last 5 reps, as the CP stores dwindled. Further support for a strong reliance on anaerobic glycolysis during resistance training comes from studies documenting significant reductions in muscle glycogen after a weightlifting session. The extent of the depletion depends on the intensity and volume of the session:

- In **bodybuilders**, a to-failure set of 12 reps of one-arm biceps curls reduced the muscle's glycogen by 12%. In **other bodybuilders**, three to-failure sets of 12, 9, and 7 reps (the last two numbers being group averages and lower due to fatigue) reduced the muscle's glycogen by 24%.¹¹
- In **bodybuilders**, an exercise session consisting of four to-failure sets of 6–10 reps of front squat, back squat, leg press, and leg extension (sixteen sets in all) reduced the quadriceps' glycogen by 26%.¹² Glycogen depletion in other muscles contributing to the effort (such as the hamstrings and glutes) wasn't measured.

Figure 2: The effect of resistance exercise* on the quadriceps' ATP, creatine phosphate, and glycogen



* Competitive and recreational bodybuilders completed four to-failure sets of 6–10 reps of front squat, back squat, leg press, and leg extension.

Reference: Tesch et al. *Eur J Appl Physiol Occup Physiol*. 1986. PMID:3758035

- In **untrained adults**, eight sets of 10 reps on the leg-press and leg-extension machines (sixteen sets in all) depleted the quadriceps' glycogen by 33%. The decrease was significantly greater in **type II fibers** than in **type I fibers**.¹³

🔍 Digging Deeper: Types of muscle fiber

Your heart and stomach are muscular organs, but when you think of muscle building, you think of building your *skeletal* muscle — the type of muscle that moves your skeleton. This type of muscle is made of two types of fibers: **type I** (slow-twitch) and **type II** (fast-twitch). Type II fibers are further classified into *type IIa* and *type IIx*, but that's not relevant here.

Slow-twitch muscle fibers have more mitochondria, myoglobin, and capillaries, in order to process more oxygen. In other words, they're optimized for aerobic metabolism and thus slow but consistent energy production. They don't fatigue easily, but are relatively weak; they can't support high-intensity efforts (i.e., efforts above the anaerobic threshold). These are the fibers we use for prolonged submaximal exercise activities and postural control.

Fast-twitch muscle fibers have less mitochondria, myoglobin, and capillaries — they'd rather use the space to store more glucose (as glycogen, as [we saw](#) in the “Body Composition” chapter). They are also larger and more contractile (being larger makes them more contractile, which in turn gives them more growth potential, creating a positive feedback loop). Their being more contractile is part of what makes them ideal for short, powerful bursts of effort. However, their reliance on anaerobic metabolism makes them fatigue quickly. These are the fibers we use for weightlifting or sprints.

The “no carb, no strength” theory in a nutshell:

There is a statistically significant relationship between a reduction in the rate of ATP production and a reduction in power output.⁹ During all-out efforts, your body needs to **produce a lot of ATP quickly**. For that purpose, it relies on the **phosphagen system** for a few seconds, then on **anaerobic glycolysis**. But you can't have anaerobic glycolysis without glucose; therefore, if a muscle's glycogen stores are low, then it has less **strength** (defined as the ability to overcome resistance) and **power** (defined as the ability to overcome resistance in the shortest period of time).^{14,15,16} The easiest way for your body to make glucose is to break down carbs, and so, in theory, a diet high in carbs will lead to optimal strength and power.

Feats of strength require that you generate energy quickly. Since aerobic metabolism is slow, your muscles must rely on anaerobic metabolism (which needs no oxygen to produce energy). For that purpose, they can use the phosphagen system for a few seconds, then anaerobic glycolysis — the conversion of glucose to lactic acid. If a muscle is low on glucose, it will quickly run out of strength.

Your strength on keto: from theory to practice

The effect of keto diets on strength hasn't been studied as much as their effects on [endurance](#) and [body composition](#), yet maintaining your strength is an important part of healthy aging, and increasing your strength is necessary to excel in most sports, not just powerlifting or Olympic weightlifting. Relatedly, by allowing you to move heavier weights, greater strength can promote muscle growth and maintenance — a goal not just for bodybuilders but for all of us who intend to stave off [sarcopenia](#) (i.e., age-related muscle loss).

So, are carbs really necessary to use and develop strength? To answer this question, a study should have the following qualities:

- It should include a keto group and a control group. The two groups should undergo the same exercise regimen, prescribed and monitored by the research staff.
- It should provide all meals to both groups and monitor the extent of the keto group's ketosis through regular blood-ketone testing.
- It should measure strength at different points in time (at least at baseline and at the end of the study), so that the *change* in strength in the keto group can be compared with the *change* in strength in the control group.

Alas, there are no such studies. In all studies, the participants were simply told how to follow a keto diet and write food logs (a less-than-perfectly-accurate method of assessing

caloric and macronutrient intake, and thus adherence to a keto diet).¹⁷ However, some studies did measure blood-ketone levels.

Let's have a look at the studies whose participants exercised (studies in participants who did *not* exercise are less interesting since exercise is a necessary stimulus for strength gains):

- Two studies measured **blood ketones** to confirm ketosis in military personnel (1.0 mmol/L on average)¹⁸ and recreational weightlifters (1.0 mmol/L on average)¹⁹ whose **exercise regimen was prescribed and monitored**.
- Two studies measured **blood ketones** to confirm ketosis in powerlifters and Olympic weightlifters (0.4 mmol/L on average)²⁰ and recreational CrossFitters (1.5 mmol/L on average)²¹ who just pursued their usual exercise regimen.
- Two studies measured **urine ketones** to confirm ketosis in recreational CrossFitters²² formerly untrained adults²³ whose **exercise regimen was prescribed and monitored**.
- One study measured neither blood nor urine ketones in elite gymnasts who just pursued their usual exercise regimen.²⁴

In all seven studies, changes in strength performance over the course of the study were similar between the keto and high-carb groups. The outcomes reported included measures of **performance** (on calisthenic exercises to failure, vertical and countermovement jumps, and powerlifting and Olympic weightlifting competition lifts) and **maximal strength** (such as 1RM for back squat, bench press, and power clean).

The only study to report a statistically significant difference in strength performance involved 31 resistance-trained men and women who all followed their usual diet for one week then switched to a keto diet for one week.²⁵ Handgrip strength, vertical jump, and 1-RM for back squat (but not for bench press) were higher at the end of the keto week.

What does that mean? Actually, very little. First, one week is barely enough to empty your glycogen stores, and neither blood nor urine ketone levels were ever measured. Second, strength wasn't measured at the start of the study, so we can't compare the change in strength over the usual-diet week with the change in strength over the keto week. All we know is that strength performance was higher at the end of the second week than at the end of the first week, but that could be just due to ... one more week of strength training.

So, at best, this study suggests that a keto diet doesn't destroy your strength — but the seven other studies mentioned in this section already taught us that.

In theory, a diet low in carbs should hinder strength performance, and thus strength gains. In practice, a keto diet neither hinders nor enhances strength performance or gains.

Theory vs. practice: why the disconnect?

In theory, a lack of carbs should hinder strength performance. In practice, a keto diet doesn't seem to hinder strength performance.

This disconnect between theory and practice (which also affects endurance on keto) helps explain why you can find reference-laden blog posts that defend opposite points of view on the effects of a keto diet. To get the whole picture, you need to parse the whole body of evidence, and you need to do so with an open mind.

Of course, apparently contradictory studies can make this “whole picture” seem maddeningly confusing. When that happens, it does help if you can call on a disinterested team of scientists with diverse expertise, ranging from sports nutrition to molecular biophysics. They'll tell you that, when it comes to keto diets and strength performance, there are **three main reasons** why we see a disconnect between theory and practice:

First, most studies on keto and strength defined strength as the ability to perform a very short, explosive effort, such as jumping as high as possible or doing one rep with the heaviest weight you can lift. Such efforts last only a few seconds, and so rely primarily upon the phosphagen system (which uses creatine phosphate) to generate energy; anaerobic glycolysis (which uses glucose) doesn't have time to take over.

However, while this reason could suffice to explain why strength *performance* is unaffected, it doesn't explain why strength *gain* is unaffected too. After all, to build muscle and gain strength, you need resistance *training*; you can't just stop by the gym, do one rep with the heaviest weight you can lift, wipe your brow, smile, and go home. You need to perform multiple sets of multiple reps — and if a set lasts more than a few seconds, you run out of creatine phosphate; you have to rely on anaerobic glycolysis. You need glucose.

And you'll have some. You'll have *enough*. Because ...

Second, you won't lose that much glycogen.

Wait, really? But we saw that **one** to-failure set of one-arm biceps curls was enough to reduce the muscle's glycogen by 12%.¹¹ Logically, shouldn't **three** to-failure sets reduce the muscle's glycogen by 36%, and **nine** to-failure sets empty the muscle entirely?

No, your body doesn't work that way. The same study showed that **three** to-failure sets (with fewer reps in the second set, and even fewer in the third set, due to fatigue) only reduced the muscle's glycogen by 24%, not 36%.

The biceps is a relatively small muscle, though. Even if you depleted it entirely, you still wouldn't lose a significant percentage of your total (i.e., whole-body) glycogen. So, what if you exercised a bigger muscle? Or even (hypothetically) *all* your muscles, from the mighty quadriceps down to the tiny muscles moving your toes?

A study reported that even a heavy leg workout performed by bodybuilders depleted quadriceps glycogen by only 26%.¹² Of course, during a leg workout, other muscles are working hard — notably the glutei maximi, the largest muscles in the human body (one per buttock). So let's say that all your leg muscles also lose 26% of their glycogen. In fact, let's imagine that, in a single workout, you manage to exercise *all* your muscles as hard as bodybuilders exercise their quadriceps during a leg workout. In other words, let's imagine that you lose 26% of all your muscles' glycogen. How many grams of glycogen would you lose?

For the sake of argument, let's use the same numbers as in the “Body Composition” chapter:

- A **female** who carries 23 kg of muscle (51 lb) typically carries in her muscles 268 grams of glycogen. By losing 26% of all her muscles' glycogen, she loses 70 grams.
- A **male** who carries 31 kg of muscle (68 lb) typically carries in his muscles 361 grams of glycogen. By losing 26% of all his muscle glycogen, he loses 94 grams.

Fine, so you don't lose a lot of glycogen. But you still lose some, during each workout, and so, sooner or later, you'll no longer have enough glycogen to break down into glucose to fuel your workout — right?

Wrong. Fortunately. Because ...

Third, your glycogen stores get refilled *even if you don't eat carbs*.

In the previous chapter, we saw that, whereas your body breaks down the carbs you eat into glucose, it can also make new glucose out of amino acids, lactic acid, or glycerol.²⁶ We called this process of making new glucose **gluconeogenesis**.

In people who fast for weeks (obese individuals under medical supervision), gluconeogenesis amounts to about 80 grams per day: 35–40 grams from recycled lactate and pyruvate, 20 grams from fat-derived glycerol, 15–20 grams from protein-derived amino acids, and 10 grams from ketones.^{27,28}

So you gain 80 grams of glucose. However, your brain gets first dibs on any glucose in your body, and 80 grams is probably more than enough to satisfy its needs (*when* it runs mostly on ketones). What happens, then, if you spend, let's say, 100 grams of glycogen during a grueling leg workout? Since you are on a keto diet, will your muscles' glycogen stores stay depleted?

No, [as we saw](#) in the “Body Composition” chapter, when you stick to a keto diet, your glycogen stores undergo at least a partial recovery, even if you keep exercising. In fact, a study in ultra-marathoners and ironman distance triathletes [we'll review](#) in the “Endurance” chapter found similar levels of muscle glycogen between the athletes on a high-carb diet and the athletes self-reporting adherence to a keto diet for an average of 20 months.²⁹

Why? Probably because, when you exercise, a large proportion of your muscle glycogen is [converted into lactic acid](#) (which is quickly buffered into lactate), and because, in humans and across all animal species studied to date, there is strong evidence that lactate gets recycled into glucose, which can then serve to replenish glycogen stores.³⁰ Not only that, but exercise increases the availability of ketones (through an increase in ketogenesis), glycerol (through an increase in fat breakdown), and amino acids (through an increase in protein breakdown), all three of which your body can also use to make glucose. Therefore, while sedentary people might produce only 80 grams of glycogen from ketones, glycerol, amino acids, and recycled lactate and pyruvate, people who exercise can produce a lot more.

This explains why keto diets don't impair strength gains: yes, glucose is necessary for optimal resistance training, but your body can synthesize enough after a workout (notably by recycling the lactate produced during said workout) to fuel the next workout.

Keto diets neither hinder nor enhance strength performance or gains, for two reasons. First, because tests of strength commonly last only a few seconds, during which your muscles use creatine phosphate as fuel, rather than glucose. Second, because when you don't eat carbs, your body still produces enough glucose to refill your glycogen stores between workouts, thus ensuring that you can train just as hard as someone on a high-carb diet.

Chapter 10: Endurance

Summary

Since being keto-adapted means being better at burning fat **and** your body can store more fat than carbs, **then** keto diets should enhance endurance performance; **but in practice**, they seem to have either no effect or a negative one, in both elite and recreational athletes. This is because, at the exercise intensities seen in endurance events, what matters is how efficiently you burn carbs, not fat.

- *Exercise intensity* is commonly expressed as a percentage of VO_2 max (the maximal rate at which you can use oxygen during exercise). Above a certain intensity, your rate of fat oxidation plateaus: you have reached your *peak rate of fat oxidation*.
- Keto-adapted athletes burn fat faster: one study found their peak rate of fat oxidation to be 1.54 g/min at 70% of VO_2 max, compared to 0.67 g/min at 55% of VO_2 max for other endurance athletes.
- Competitive intensities in endurance sports (>80% of VO_2 max) force even keto-adapted bodies to turn from fat to a less oxygen-dependent fuel: carbs.
- Your body converts carbs into glucose (“blood sugar”), which it either burns for energy or stores as glycogen. Being less oxygen-dependent than fat, glucose allows you to produce energy 2.5 to 5 times faster.
- It is how efficiently you burn glucose, not fat, that determines your performance at competitive intensities. In one experiment, the performance of athletes chemically prevented from using fat as fuel while running at a competitive half-marathon pace (80% of VO_2 max) was unimpaired.
- By making you more reliant on fat and impairing your ability to burn glucose, a keto diet can hurt your performance. The best controlled study to date found that, at the end of a 3-week training camp, a high-carb group showed improved performance (their time on a 10 km walk improved), whereas a keto-adapted group didn't.
- Recreational athletes perform at intensities where keto-adaptation should theoretically improve performance (<70% of VO_2 max), but studies in these athletes show a keto diet to have no effect or a negative one on performance.

Keto-adaptation: the theory

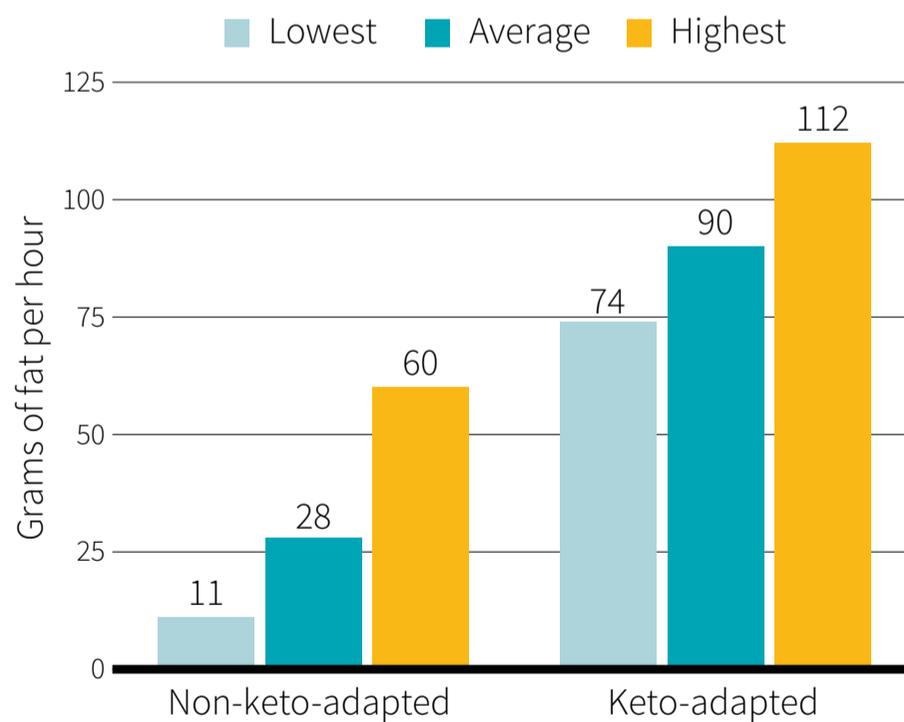
You store carb-energy (glucose, or “blood sugar”) as glycogen (in your [muscles](#) and [liver](#)) and fat-energy as body fat. Compared to your fat stores, however, your glycogen stores are quick to deplete. So wouldn’t a diet that makes you better at burning fat also make you better at winning prolonged athletic events? In simpler words, **is a keto diet ideal for endurance athletes?**

Such is the hypothesis developed in “Rethinking fat as a fuel for endurance exercise”.¹ This 2015 paper starts with a historical perspective presenting four arguments:

- Even lean humans store a lot more fat-energy (as body fat) than carb energy (as glycogen).
- Dietary fat was humans’ main energy source until the advent of agriculture.
- Arctic explorers have performed feats of prolonged exercise while eating hardly any carbs.
- Keto-dieters have proven successful in endurance competitions.

The paper proceeds to compare a study in elite cyclists on a keto diet²) with a study in trained and untrained men and women *not* on a keto diet.³

Figure 1: Rates of fat oxidation during exercise



Left: peak rates of fat oxidation in 300 people (including highly trained individuals)
Right: rates of fat oxidation during exercise at 64% VO_2 max in keto-adapted elite cyclists

Adapted from Volek et al. *Eur J Sport Sci.* 2015. PMID:[25275931](#)

Among the non-keto subjects, *peak* fat oxidation rate averaged 0.46 g/min (with 1.01 g/min as the highest recorded value). Among the keto athletes, fat oxidation rate averaged 1.50 g/min (at 64% of VO_2 max, so their *peak* fat oxidation rate may have been even higher; in a 2016 study, the fat oxidation rate of keto athletes peaked at 70% of their VO_2 max⁴).

In sum, keto athletes can produce three times as much fat-derived energy in the same amount of time. This ability to burn fat faster is called **keto-adaptation**.

🔍 Digging Deeper: VO_2 max, fitness, and performance

VO_2 stands for *ventillary oxygen* and represents the amount of oxygen you're inhaling.

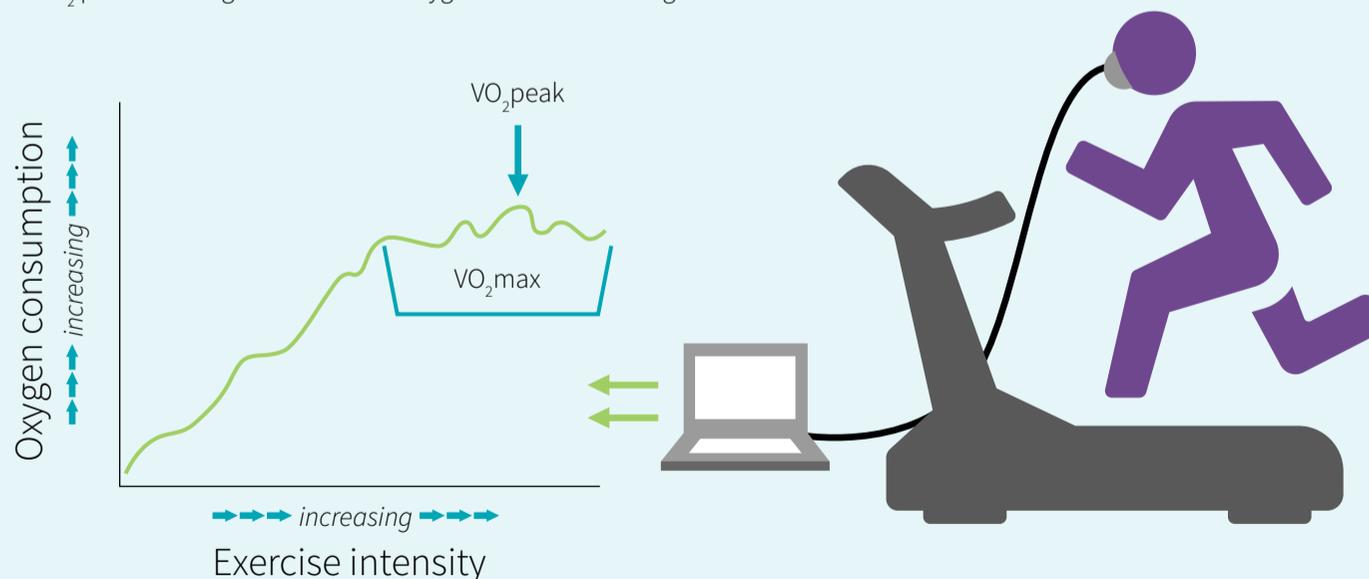
VO_2 max represents the maximal rate at which you can use oxygen during exercise.

Three things to remember:

- VO_2 max is a common parameter of cardiorespiratory fitness because, as you become fitter, you become better at using oxygen to produce energy (your VO_2 max increases).
- Moreover, **exercise intensity** is often reported as a percentage of VO_2 max based on the assumption that more intense exercise requires that you consume more oxygen to fuel your mitochondria (the power generators in your cells).
- Nevertheless, higher VO_2 max doesn't always translate as better sports performance, because sports often involve moments when energy requirements are so great (the intensity is so high) that oxygen isn't used to generate the required energy.

Figure 2: How to measure VO_2

- 1 – A mask is worn to help measure the amount of oxygen inhaled and exhaled.
- 2 – The test is usually conducted on a treadmill or bike that increases in speed or resistance at regular intervals.
- 3 – As exercise intensity increases, the amount of oxygen exhaled decreases as your cells begin to use more of it.
- 4 – VO_2 max is reached when oxygen consumption reaches a maximum and plateaus.
- 5 – A typical VO_2 max ranges between 30 and 60 milliliters of oxygen per kilogram of body weight per minute (ml/kg/min)
- 6 – VO_2 peak is the highest amount of oxygen consumed during exercise.



Further evidence in support of keto-adaptation came the following year, in 2016, from the Fat-Adapted Substrate use in Trained Elite Runners (FASTER) study.⁴ This study involved ultra-distance triathletes and runners, some on a high-carb diet and others self-reporting adherence to a keto diet for an average of 20 months.

Exercise intensity was again expressed as a percentage of VO_2 max, and the keto-adapted athletes displayed two advantages: their **fat oxidation rates were higher at any intensity and plateaued at higher intensities**. More precisely, fat oxidation rate peaked/plateaued at 1.54 g/min (at 70% of VO_2 max) for the average keto athlete and at 0.67 g/min (at 55% of VO_2 max) for the average high-carb athlete.

Surprisingly, high-carb athletes and keto athletes had similar levels of muscle glycogen (stored glucose), both before and after exercise, despite the keto athletes consuming little carbohydrate.

The authors of the FASTER study interpreted its results as a challenge to the superiority of the conventional high-carb diet for endurance athletes:

The enhanced ability to oxidize fat during exercise across a range of intensities is striking, as is the ability to maintain “normal” glycogen concentrations in the context of limited carbohydrate intake. Keto-adaptation provides an alternative to the supremacy of the high-carbohydrate paradigm for endurance athletes.

Think of it this way:

- You can store a lot more fat (as body fat) than carbs (as glycogen in your muscles and liver).
- During endurance events, your glycogen stores can get depleted.
- When your glycogen stores are depleted, you start burning more fat.
- When you have to rely on fat for fuel, being able to burn fat more efficiently gives you an advantage.

Sticking to a keto diet allows you to become keto-adapted, so to burn fat faster. In the same timeframe, keto-adapted athletes can produce three times as much energy from fat as other athletes. This is a potential advantage during endurance events, since your body can store more fat than carbs.

Keto-adapted athletes: from theory to practice

Studies in elite athletes

Of the studies that compared keto diets with high-carb diets for endurance performance, only two involved complete dietary control by the research staff. This control ensured that the keto and high-carb diets were equal in both protein and calories, and that the athletes on a keto diet would really be in a state of nutritional ketosis (so would really become keto-adapted). In other words, it helped minimize the risk of interference from factors such as dietary adherence and energy availability.

For the first of these two studies, whose results were published in 1983, 5 competitive cyclists were recruited. Cycling time to exhaustion (at a constant pace of 62–64% of VO_2 max) was statistically equivalent before and after four weeks on a keto diet.²

This finding seems to support the idea that a keto diet is a suitable alternative to a high-carb diet for endurance athletes, but its real-world relevance is questionable, because most endurance sports require training and competing at intensities well above 62–64% of VO_2 max.

- Professional cyclists train and compete at or above 90% of VO_2 max for more than one hour at a stretch.^{5,6}
- Elite marathon competitors run at speeds requiring 80–90% of VO_2 max for the entirety of the race.⁷
- During 20 km and 50 km events (12.4 and 31.1 miles), elite race walkers go at speeds requiring 70–75% and 85–90% of VO_2 max, respectively.⁸

Furthermore, endurance races are similar to other races in that they require that you cover long distances as fast as possible; they don't require that you run at a set pace until you drop from exhaustion. It is therefore not surprising if the time-to-exhaustion test has shown low reliability for estimating performance during endurance events, with some researchers arguing that it should not be used as a stand-alone measure.^{9,10}

🔍 Digging Deeper: Assessing endurance performance

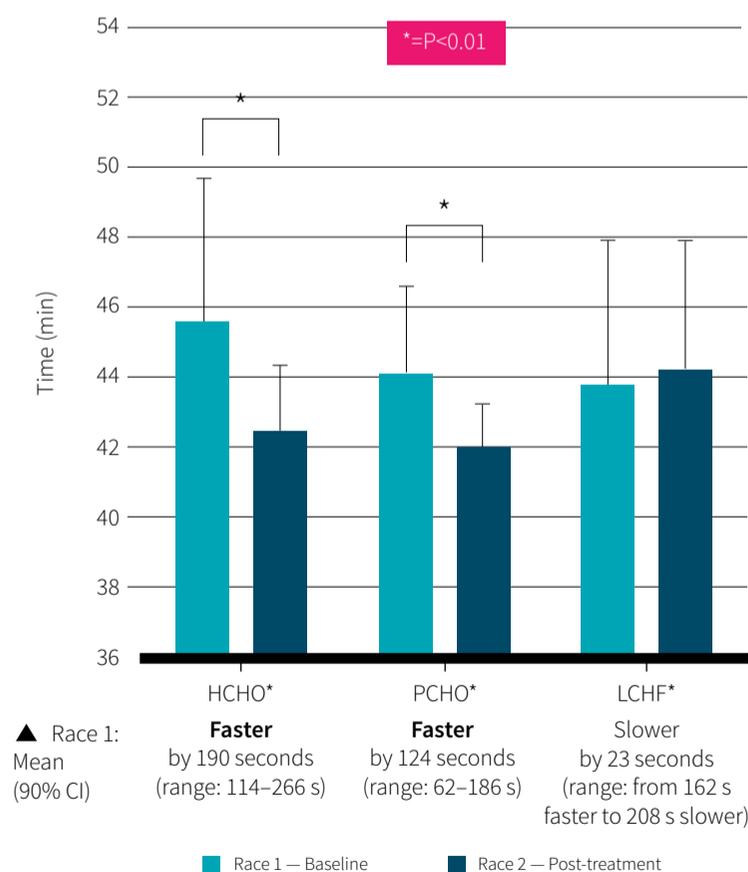
Endurance performance can be assessed in three main ways:

- You can measure **time to completion** for a set distance (if you run the same distance in less time, your endurance performance has improved). Of course, the distance needs to be long enough for the race to be considered an endurance race.
- You can measure **distance crossed** in a set time (if you run farther in a set time, your endurance performance has improved). Of course, the time needs to be long enough for the race to be considered an endurance race.
- You can measure **time to exhaustion** at a set pace (if you run longer/farther at a set pace, your endurance performance has improved). Of course, the pace needs to be slow enough for the race to be considered an endurance race.

In this guide, unless otherwise specified, endurance performance means **time to completion**, which reflects how most endurance races work in the real world.

That brings us to the second diet-controlled study, published 34 years later, in 2017.⁸ It involved 19 elite race walkers undergoing a 3-week training camp while following a keto diet or a carb diet (periodized-carb or high-carb). As its main outcome, it used performance on a 10 km (6.2 miles) race-walking event both before and after the training camp:

Figure 3: Performance on a 10 km race-walking event before and after a 3-week training camp



* HCHO: high-carb | PCHO: periodized carb | LCHF: low-carb, high-fat

Adapted from Burke et al. *J Physiol*. 2017. PMID:28012184

From one event to the next, the two carb groups became faster, but the keto group didn't. This study, with its tightly controlled intervention — training camp, controlled diet — and its measures of actual performance, has “real-world” credibility.

Importantly, keto-adaptation was achieved in both the 1983 and 2017 diet-controlled studies. In the 1983 study, the cyclists burned an average of 1.5 grams of fat per min.^{1,2} In the 2017 study, the keto group also burned 1.5 g/min — three times as much as the high-carb group!⁸ (You can read our detailed review of the 2017 study [here](#).)

The fat oxidation rate of keto-adapted athletes peaks at 70% of VO₂ max, but endurance competition requires intensities well above this level. A diet-controlled study looked at actual performance before and after a training camp and saw a general improvement in the carb groups but not in the keto group.

Studies in recreational athletes

[As we saw](#), elite marathoners run at 80–90% of VO₂ max,⁷ but even keto-adapted elite athletes cannot increase their rate of fat oxidation beyond 70% of VO₂ max.⁴

Recreational marathoners, however, average only about 60% of VO₂ max¹¹ — an intensity very close to the fat-burning peak of even non-keto-adapted trained individuals: 59–64% of VO₂ max.¹²

It is therefore possible that being keto-adapted benefits *recreational* athletes more than *elite* athletes. This theory is put to the test in seven studies, but alas, unlike the two studies in elite athletes we've just reviewed, those seven studies in recreational athletes didn't involve complete dietary control by the research staff — the participants (recreational athletes) controlled their own diets, which notably led to the keto groups consuming more protein and fewer calories than the high-carb groups.

- Five studies reported no differences between the keto and high-carb groups for performance on a 5 km (3.1 miles) running time-trial,¹³ a 100 km (62.1 miles) cycling time-trial,¹⁴ a graded treadmill run to exhaustion,^{15,16} a shuttle run sprint test to exhaustion,¹⁶ and a run to exhaustion at a constant pace of 70% VO₂ max.¹⁷
- Two studies reported that keto diets reduced performance on a 45-minute cycling ride¹⁸ and a yo-yo intermittent recovery test that evaluated the participants' ability to perform repeated bouts of high-intensity sprints.¹⁹

Only three of the seven studies reported fat oxidation rates, however, and those averaged a mere 0.8–1.1 g/min (compared to 1.5 g/min in the diet-controlled studies). [We saw](#) earlier

that even people not on a keto diet can reach 1.0 g/min,³ so it is possible that the “keto” athletes in those three or all six studies failed to reach true keto-adaptation (despite their fat oxidation rates being higher, on average, than those seen in the high-carb groups).

There is another possibility, though. [As we saw](#), keto-adaptation was originally defined based on the fat oxidation rates of *elite* athletes,^{2,4,8} and we don't currently know if those rates hold up for individuals with less training. As of yet, no study has compared fat oxidation rates between elite athletes and recreational athletes eating the same keto diet.

In other words, fat oxidation rates of 0.8–1.1 g/min may be the norm for keto-adapted *recreational* athletes — but we cannot be sure, and the hypothesis being that keto diets benefit endurance performance through keto-adaptation, it cannot be tested using studies whose participants might not have been fully keto-adapted.

(In those seven studies, the blood ketone levels of the keto athletes averaged 0.3–0.9 mmol/L, compared to 1.3–1.8 mmol/L in the two diet-controlled studies. Keep in mind, though, that keto-adaptation is based on fat oxidation rates, not blood ketone levels.)

Finally, three case studies looked at the effects of keto diets on endurance performance in recreationally active adults. One reported a negative effect on a graded cycling test to exhaustion;²⁰ the other two reported no effect on a graded treadmill run to exhaustion.^{21,22}

Fat oxidation rates were reported in two of the studies: from before to after the ketogenic dietary intervention, they rose from 0.6 to 0.8 g/min in one study²⁰ and from 0.55 to 0.6 g/min in the other.²² These numbers are far from the 1.5 g/min seen in keto-adapted elite athletes.

(Remember that, since case studies have no control or comparison group, they constitute weak evidence from which to draw conclusions.)

Ultimately, the evidence we have so far suggests that a keto diet has either a neutral or negative effect on endurance exercise performance. This conclusion seems to hold true for both elite and recreational athletes, and for the [three definitions of endurance performance](#).

A word on the studies we haven't mentioned

Even as you read these words, some of you are probably pointing to a handful of studies we didn't mention. Did we just miss them?

Probably not. While we can't assert with 100% confidence that we found every study ever published on keto diets and endurance performance, we can state that we reviewed

many studies we decided not to mention because they didn't meet the criteria we set for inclusion in this guide.

For instance, a 1994 study reported that a keto diet improved cycling time to exhaustion when riding at 60% of VO_2 max and preserved performance when riding at 90% of VO_2 max.²³ However, diet wasn't controlled or reported and neither group entered into a state of nutritional ketosis based on blood ketone levels.

Likewise, three other studies found no differences in blood ketone levels between the keto and high-carb groups, making any outcomes irrelevant to the “keto diet versus high-carb diet for endurance performance” question.^{24,25,26}

To mention one last example, a study in female cyclists reported that a low-carb diet reduced performance at 80% of VO_2 max, but carbohydrate intake was just above the usual cut-off point for a keto diet (the participants were averaging 72 g/day) and no measures of ketosis were taken.²⁷

Most studies reviewed for this guide didn't make the cut, whether because of quality concerns or because a study's “keto group” wasn't shown to be eating a ketogenic diet (as suggested by blood ketone levels).

Theory vs. practice: why the disconnect?

The crossover concept

As we've seen, a keto diet makes you better at burning fat, in two ways:

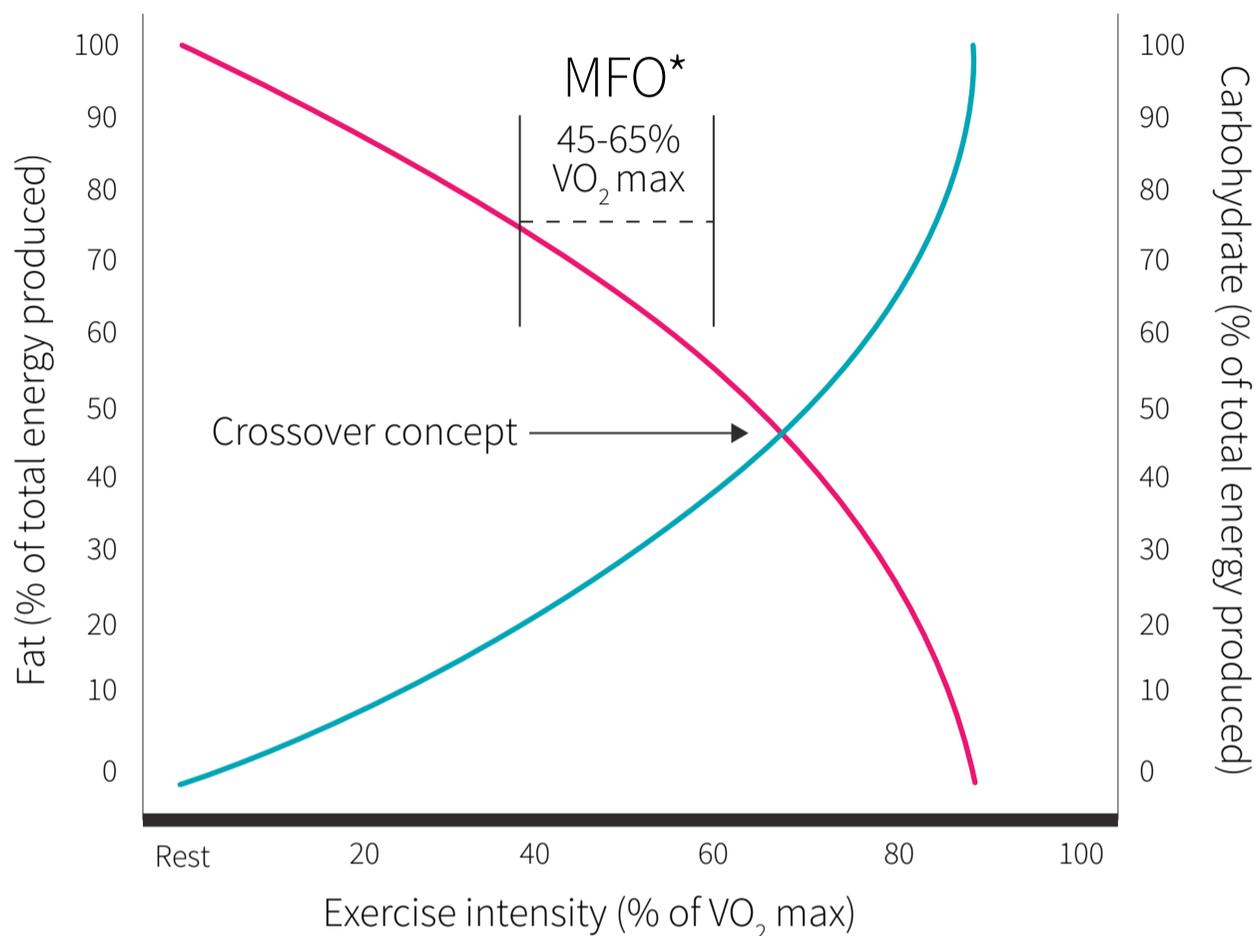
- **It increases your fat oxidation rate at any exercise intensity.** So, for instance, at an intensity of 64% of VO_2 max, keto-adaptation could *triple* your fat oxidation rate (from 0.46^3 to 1.5^2 g/min), meaning that you could generate thrice the fat-derived energy in the same timeframe.
- **It raises the exercise-intensity threshold above which your fat oxidation rate stops increasing.** So, for instance, you could keep increasing the speed at which you convert fat to energy until you reach 70% of your VO_2 max, instead of seeing it plateau above 55% of your VO_2 max.⁴

So why don't we see actual benefits in studies that assess endurance performance?

Because of the erroneous assumption that fat oxidation matters for endurance performance — it doesn't.

Fat is the dominant fuel during exercise of submaximal intensity. Fat oxidation keeps increasing only up to exercise intensities of 59–64% of VO_2 max in athletes and 47–52% of VO_2 max in untrained adults.¹² Even in keto-adapted athletes, it can keep increasing only up to 70% of VO_2 max.⁴

Figure 4: The crossover concept



* Maximal fat oxidation

Adapted from Purdom et al. *J Int Soc Sports Nutr.* 2018. PMID:29344008.

Above this exercise-intensity threshold, carbs play an increasingly important role as fuel. In fact, it is your ability to efficiently burn glucose (“blood sugar”, which your body stores as **glycogen**) that determines your ability to reach the levels of exercise intensity needed to win races — fat oxidation is irrelevant.

Irrelevant, really? Isn't that an exaggeration?

You'd think so, but no. A study tested the concept by preventing athletes from using fat as fuel (by chemically inhibiting the release of fat from fat tissue). The athletes' performance while running at a competitive half-marathon pace ($\approx 80\%$ of VO_2 max) was unimpaired.²⁸

Even for entire marathons, glucose is the near-exclusive fuel at real-world race intensities; but even at lower intensities (under 80% of VO_2 max), competitive marathon and half-

marathon runners still rely on glucose to provide up to 80% of their energy needs.²⁹ Of course, recreational marathon runners average only about 60% of VO_2 max,¹¹ so they may very well see an increase in performance or reduction in perceived effort with a keto diet. Keep in mind, however, that they are not running to win.

The higher your exercise intensity, the more your body fuels chooses to burn glucose instead of fat. Above a certain intensity threshold (70% of VO_2 max in keto athletes, 59–64% in other athletes, 47–52% in untrained adults), your fat oxidation rate stops increasing entirely.

Why this progressive switch from fat to glucose?

The main reason is the relative oxygen cost of burning either fuel — in other words, the oxygen cost of extracting energy from fat or glucose. **You need a lot more oxygen to burn fat than glucose.** Therefore, at low exercise intensities, with plenty of oxygen available, your body would rather burn fat, of which it has greater stores than glucose; but as the intensity ramps up and it becomes harder and harder to get enough oxygen, your body turns from fat to glucose.

That's it, in a nutshell. The complete truth is a little more complex, so the rest of this subsection explores the science in more detail. If you don't fear getting a tad technical, then put your nerd hat on; we'll guide you step by step. Otherwise, you can skip to [the next subsection](#).

Still with us? Then let's begin!

Mitochondria, which we called [earlier](#) the power generators in your cells, produce [adenosine triphosphate](#) (ATP), life's energy currency.

We've known since 1920, based on pure chemistry, that the oxygen cost of ATP production is greater for fat than glucose.^{30,31,32} A single molecule of fat generates more ATP than a single molecule of glucose, but a molecule of glucose is smaller and requires less oxygen to generate ATP — in fact, it can even generate ATP *without* any oxygen. Consequently, whereas a molecule of fat generates 0.4 moles of ATP per minute (via aerobic metabolism), a molecule of glucose generates 1.0–2.0 mol/min (via aerobic and anaerobic metabolism).²⁵

More simply, we could say that glucose generates 2.5 to 5 times more energy than fat in the same timeframe — or, more simply still, that glucose allows you to produce energy 2.5 to 5 times faster. Since most endurance events require that you sustain high-intensity exercise performance by rapidly generating energy, we begin to understand why keto diets seem to hurt rather than help competitive performance.

So **oxygen cost** (burning glucose requires less oxygen than burning fat) and **speed of energy production** (burning glucose generates energy faster than burning fat) are two related reasons why your body turns from fat to glucose as exercise intensity increases. We'll now explore the third and last reason, which has to do with the regulation of **fat metabolism during exercise**.

⚠ **Warning: Spot reduction doesn't work — here's why**

[Spot reduction](#) refers to the claim that fat in a certain area of the body can be targeted for reduction through exercise of specific muscles in that desired area. For example, exercising the abdominal muscles in an effort to lose weight in or around one's midsection.

It doesn't work. Your fat isn't a self-service restaurant; your muscles can't simply get energy from the nearby fat cells. Instead, fatty acids must be released from fat cells into the bloodstream, which can swiftly carry them wherever they're needed.

Therefore, when you do crunches, it doesn't matter to your body if the fatty acids delivered by the bloodstream originate from fat cells situated above your abs or elsewhere.

One reason keto-adaptation makes you faster at burning fat is that it makes you faster at releasing fatty acids from fat cells, thus making the fat available as fuel. Since increases in fat oxidation parallel increases in fat availability during exercise of low to moderate intensity,³³ we understand why, compared to other athletes, keto athletes can get three times as much energy from fat in the same timeframe.^{1,2,8}

During high-intensity exercise, however, fat release gets inhibited in two ways:³⁴

- As oxygen becomes harder to get, your body uses more and more glucose *without* oxygen, in a process called [anaerobic metabolism](#). As a result, your blood levels of lactate rise, serving as a signal to decrease fat release.
- You produce more epinephrine (a.k.a. adrenaline), which activates alpha-adrenergic receptors on fat cells, thus decreasing fat release.

Decreased **fat release** means decreased **fat availability** means decreased **fat burning**.

Evidence shows, however, that decreased fat availability isn't the *only* cause of decreased fat burning during high-intensity exercise. If we infuse fat into endurance athletes during exercise of high intensity (85% of VO_2 max), the increase in fat oxidation is smaller than the increase in fat availability.³⁵

In other words, during exercise of high intensity, increases in fat oxidation *do not* parallel increases in fat availability, as we saw they do during exercise of low to moderate intensity. That's because the entry of fat into the mitochondria, the power generators in your cells, is inhibited by the stimulation of glycogen breakdown and the use of glucose for energy.³³

In short, fat oxidation is of little importance during high-intensity exercise, and thus during endurance events, because (1) energy demands are too high to be supported by aerobic metabolism only, (2) fat release from fat cells (and therefore fat availability) is limited, and (3) the transport of fat into the mitochondria, where it would be oxidized for energy, is inhibited.

Therefore, keto-adaptation (the ability to burn fat faster you acquire by sticking to a keto diet) doesn't increase [endurance performance](#). But still, why would your exercise performance *decrease* if you're on a keto diet? Read on to find out!

As exercise intensity increases, energy requirements increase; as energy requirements increase, oxygen requirements increase; as oxygen requirements increase and begin to exceed oxygen availability, your body turns from fat to a less oxygen-dependent fuel: glucose.

Why do keto diets hurt endurance performance?

For one, a greater reliance on fat as a fuel lowers your exercise efficiency — i.e., you need more oxygen at any given pace.⁸ If you could compensate for this lower efficiency simply by taking in more oxygen, all would be well, but you can't: the exercise intensity of endurance events is too high for that. This may explain why exercise efficiency is a critical determinant of performance³⁶ — indeed, why it is a much better determinant of performance than VO_2 max (which [we saw](#) is the maximal rate at which you can use oxygen during exercise).³⁷

For two, a keto diet probably reduces your ability to turn glucose into energy — a process that requires an enzyme complex known as *pyruvate dehydrogenase* (PDH). At exercise intensities above 80% of VO_2 max, PDH naturally increases its activity to support the increased demand for energy,^{38,39} but low-carb, high-fat diets inhibit this increase,^{40,41} even with carbohydrate loading and glycogen restoration before exercise.⁴⁰

This helps answer a frequent question: **Would it be beneficial to follow a keto diet during training and then use a high-carb intake on race day?**

And the answer is **no**. Upregulating your fat oxidation via keto training and then fueling with carbs when performance matters most sounds good in theory: the idea is that, if you can burn fat better, you can use less glucose, and so have more glucose ready when you need it most. The problem, however, is that when you become better at burning fat, you become worse at burning glucose (because of the aforementioned decrease in active PDH), and being worse at burning glucose will hurt your performance.

So, in short, you can't have your cake and eat it. Switch to keto, and you'll become better at burning fat *and* worse at burning glucose. Switch to high-carb, and you'll become better at burning glucose *and* worse at burning fat.

But isn't it **possible** that, having been through keto, your body is now at least a bit better at burning fat even out of keto? Well, yes, it is *possible* — but we don't have studies testing this hypothesis, and even *if* you keep a fat-burning advantage, it won't help you win races, because at competitive intensities, [as we saw](#), what matters is how efficiently you burn glucose.

Also, it is true that, if you switch from keto to high-carb some days before a race, your muscles will store more glucose than they would usually be able to (a phenomenon known as glycogen supercompensation). So it is **possible** that, if you time your switch just right, you'll be efficient again at burning glucose *and* have more glucose in your muscles. But there again, no study has tested this hypothesis, so we have no idea if the idea is valid, let alone how long before a race the switch should occur. Furthermore, since most races allow you to consume sugar (in fluids, gels, sweets ...) during the event, the potential advantage of having more glucose stored in your muscle is very small.

To conclude, at the exercise intensities required to compete in endurance sports, what matters is how efficiently you burn glucose, not fat. A keto diet makes you more reliant on fat as fuel and may even impair your ability to burn glucose for energy; this explains why, as we saw [earlier](#), field studies have found that keto diets have either no effect or a negative effect on [endurance performance](#).

At the exercise intensities seen in endurance events, what matters is how efficiently you burn glucose, not fat. By making you more reliant on fat and impairing your ability to burn glucose, a keto diet can hurt your performance.

Does keto have any endurance-related benefits?

After reading this chapter, you may expect a resounding *no*. But the truth is, we don't yet know. We know that a keto diet isn't likely to help you win races, but what if winning a race isn't your goal? What if you like to go jogging or hiking, for pleasure or to keep in shape? Can keto help you then?

Possibly.

Endurance races are similar to other races, [we said](#), in that they require that you cover long distances as fast as possible; they don't require that you run at a set pace until you drop

from exhaustion. But what if your main goal isn't to win a race but just to be able to keep going for a few more minutes or a few more hours, so you can spend more time enjoying the endurance activity of your choice?

The first study we mentioned in this chapter pointed out that even lean humans store a lot more fat-energy (as body fat) than carb-energy (as glycogen),¹ so if you spend the day hiking, it could be advantageous for your body to be geared toward burning fat rather than carbs.

Of course, you can simply snack on carbs while you hike. That's what most hikers do. But if you wish to lose fat, then being able to keep hiking without eating means that your body has to use its fat stores.

We saw in the “Body composition” chapter that people who exercise tend to lose more fat when they're on a keto diet, whether they undergo endurance or resistance training. Would the “keto advantage” be greater if the training took place in a fasted state? Likewise, would people who already train in a fasted state lose more fat if they followed a keto diet?

Without dedicated studies, we can only hypothesize. Fortunately, keto dieting is a popular field of research, so we may get answers sooner than later. We'll keep you updated!

Although keto dieting seems to impair endurance *performance*, as defined by *time to completion* of an endurance race, it might help you keep going (i.e., it might help you sustain non-competitive exercise intensities) without food, and in such a way facilitate fat loss.

Chapter 11: Ketone and MCT Supplements

Summary

There are two types of supplements that can help put you in ketosis: exogenous ketones and medium-chain triglyceride (MCT) oil. Both supplements bypass normal feedback loops that limit our ability to enter ketosis. They must be taken every several hours if a state of ketosis is to be maintained from supplements alone. But there are also differences, discussed below.

Exogenous ketones:

- Exogenous ketones are absorbed as is, causing a rapid and notable rise in blood ketone concentrations.
- Exogenous ketones reduce serum levels of potassium and bicarbonate, making the body somewhat more acidic. Levels of free fatty acids and triglycerides are reduced as well.
- The effects of exogenous ketones on endurance performance are unclear, with no effect seen in recreational athletes, and mixed evidence on elite athletes.
- There is potential for exogenous ketones to have a benefit within the realm of brain disorders and diseases, such as epilepsy, glioblastomas, cognitive decline, and psychiatric conditions, based on promising evidence using a ketogenic diet. However, this remains theoretical due to a lack of data.

MCTs:

- Fatty acids from MCT oil promote ketogenesis because they fairly directly make their way to the liver, as opposed to typically consumed longer chain fats, which do not.
- MCTs have shown promise in the management of epilepsy as an alternative to a traditional ketogenic diet, with the MCT oil diet allowing for a greater intake of carbohydrates while still maintaining ketosis.
- There is also some evidence that consuming MCTs in place of long-chain fats can reduce food intake and facilitate weight loss, although the evidence is mixed.

Why supplement in the first place?

The ability to produce ketones and be in a state of ketosis evolved to help prolong life during a famine, by providing an alternate energy source for the brain and slowing the breakdown of carbohydrates and protein stores.^{1,2} We can also achieve a state of ketosis with a low-carbohydrate diet, even when calories are sufficient.

Today, we have the ability to bypass the “natural” production of ketones through ketone supplements. *Exogenous ketones* are supplements of ketone salts and esters that are identical to the ketones the body naturally produces. *Medium-chain triglycerides* (MCTs) are a unique type of fat that bypasses certain regulatory mechanisms designed to prevent ketosis, leading to the rapid generation of ketone bodies.

Basically, you can be in a state of ketosis, regardless of what you eat.

There are definite advantages and disadvantages to using exogenous ketones or MCTs. We'll explore those next.

Exogenous ketones are supplements that allow us to be in a state of ketosis, regardless of what we eat.

Exogenous ketones

Exogenous ketones exist in two forms: salts, where the ketone is bound to sodium, potassium, or calcium, and esters, where the ketone is bound to an alcohol compound. Notably, ketone salts are usually a mixture of D- and L-isoforms of beta-hydroxybutyrate (β HB), despite the L-isoform not being produced by the liver and having a poorly understood metabolism.^{3,4} Ketone esters are purely the D-isoform.

The whole idea of taking exogenous ketones is to put the body in a state of ketosis, regardless of anything else going on. A 280 milligram (mg) dose of β HB per kilogram (kg) of body weight, or 20 grams of β HB for a 70 kg (154 lb) adult, is able to push blood ketone levels up to 3 millimolar (mmol/L) after just 30–60 minutes for esters or 60–90 minutes for salts.⁵ Of course, that's the peak, and levels only remain above 1 mmol/L for about 2.5 hours, returning to baseline by 4 hours.

Importantly, blood ketone levels are not the only thing affected. Serum electrolytes seem to take a hit. If we look just at ketone esters, thus removing the interfering effects of the attached molecule of a salt in ketone salts — sodium, potassium, and calcium — we see

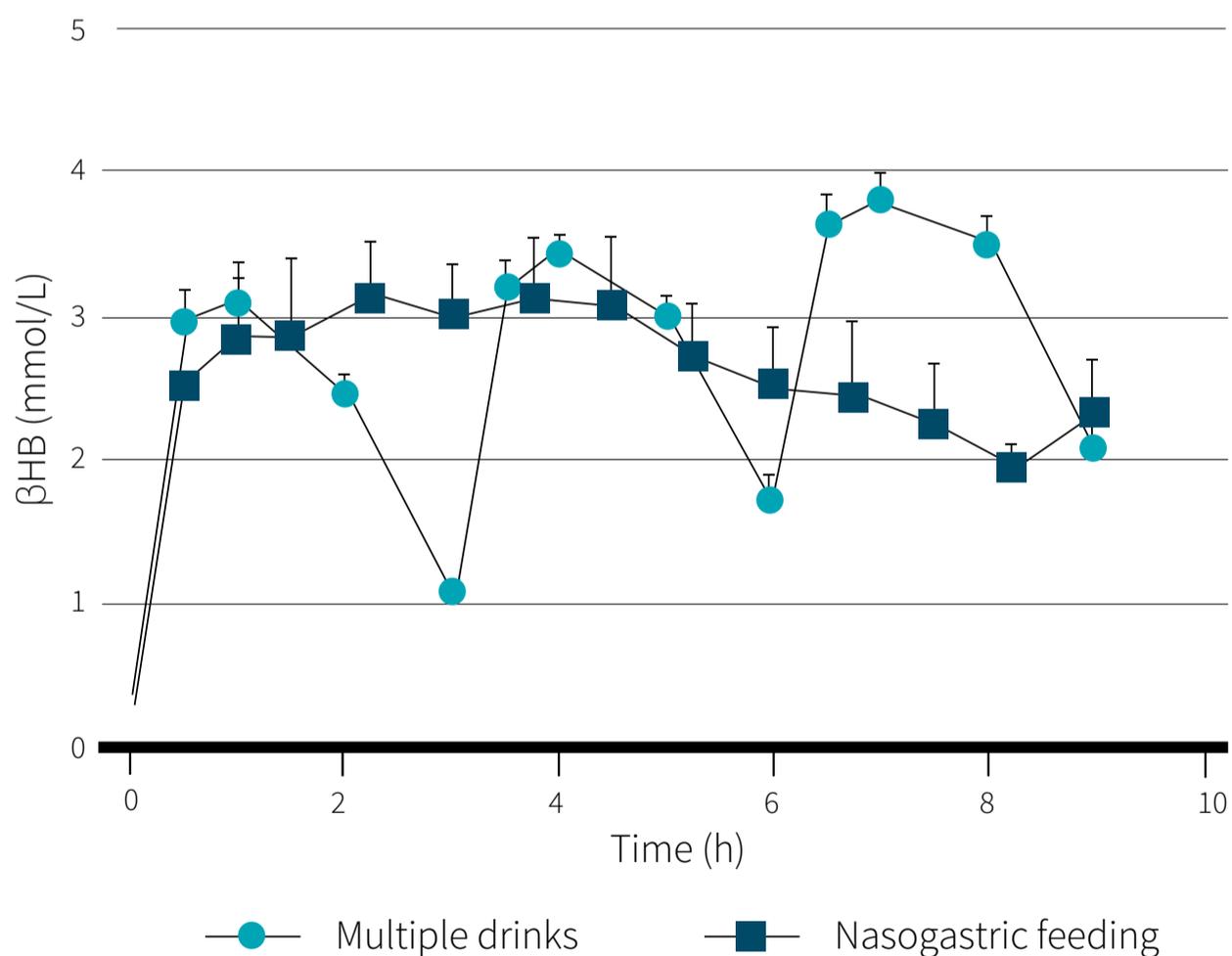
that taking exogenous ketones lowers potassium and bicarbonate levels, which are buffers against acidity.⁵ Blood pH also takes a drop, indicating that the body does indeed become more acidic.

Free fatty acids and triglycerides are reduced,⁵ which confirms the anti-lipolytic effects of ketones as part of their negative feedback loop.⁶ Blood glucose levels are also marginally lowered, which could be owed to the small insulin spike caused by the exogenous ketones — again, part of their negative feedback mechanisms.⁷

Now, all that is in the fasted state. If you take the same dose of ketones with a high-carbohydrate meal, then blood ketone levels hit only 2 mmol/L on average.⁵ There are, of course, variations between people in either situation: the D- β HB peak values ranged from 1.3 to 3.5 mmol/L when fed and 2.3 to 4.7 mmol/L when fasted.

Logically, you could consume exogenous ketones every 3 hours to maintain a state of ketosis, as is the case in research studies.⁵

Figure 1: Blood β HB levels, after ketone ester delivery via drinks or feeding tube



Multiple drinks: blood β HB following three ketone ester drinks consumed following a fast
 Nasogastric feeding: blood β HB while ketone ester was continuously delivered via a feeding tube

Adapted from Stubbs et al. *Front Physiol.* 2017. PMID:[29163194](https://pubmed.ncbi.nlm.nih.gov/29163194/)

Given what we discussed about exercise performance in the [Endurance](#) chapter, the ability of exogenous ketones to enhance exercise performance may be enticing. After all, you don't need to restrict carbohydrates to obtain a state of ketosis and have ketones available as an alternative fuel source.

At least one review article has suggested that exogenous ketones may be a more effective option for enhancing exercise performance, as opposed to nutritional ketosis.⁸

Exogenous ketones are a type of ketone supplement that supplies beta-hydroxybutyrate (one of three types of ketones) directly to the body. They cause a dose-dependent increase in blood ketone levels that can last for several hours.

For performance

Overall, eight placebo-controlled studies have investigated how supplementing with exogenous ketones before and/or during exercise affects endurance performance.^{9,10,11,12,13,14,15,16} Three involved elite or highly trained athletes;^{9,10,12} the others, recreationally competitive athletes.^{11,13,14,15,16}

Only one small crossover study of eight highly trained endurance athletes found a benefit for exogenous ketones.⁹ On two separate testing days, either carbohydrate (80 g) or ketone esters (48 g) with carbohydrates (50 g) were consumed before immediately completing a 30-minute time trial, where the athletes try to ride as far as possible in the set time. The ketone + carb group rode 2% further than the carb-only group on average, about 0.4 km or 0.2 miles. However, there was a wide amount of distance variability in the overall results. Since the study was so small and we don't have data for what happened to each individual athlete, we can't conclude anything concrete from this study.

The only other study to combine exogenous ketones was also a crossover trial that involved eight endurance-trained runners who completed a 10 km (6.2 miles) timed run.¹⁶ The trial compared the effect of ketone esters (40 g) with carbohydrates (60 g) to carbohydrates alone (60 g). Half of the supplement was consumed before the run and the other half was split equally during the run. No statistical or meaningful difference was seen between groups.

There are several possible reasons for the discrepancy in findings, such as the training status of the participants (highly trained vs recreational), type of exercise (cycling vs running), performance test (30-min time trial vs 10 km time trial), and method of supplementation (immediately pretest vs divided across an hour pretest). This makes it difficult to tease out why one study found a benefit but the other didn't.

Of the remaining studies, none found a benefit to using exogenous ketones for exercise performance.^{10,11,12,13,14,15}

Overall, it seems there *might* be a benefit to combining exogenous ketones with carbohydrates for elite endurance athletes. But the data here are too preliminary to make any firm conclusions. When looking at the effects of exogenous ketones alone for elite athletes, they do not seem to provide a performance benefit. For recreational athletes, exogenous ketones alone *or* with carbohydrates do not seem to provide a performance benefit.

It is too early to say if exogenous ketones, with or without carbohydrates, could provide an endurance performance benefit in elite athletes. Many more trials with larger samples will be needed to help answer this question. Based on the handful of trials we have, there does not appear to be a benefit from exogenous ketones for recreational athletes, regardless of whether they are taken with carbohydrates or alone.

For other health endpoints

Arguably the biggest potential benefit for exogenous ketones is within the realm of brain disorders and diseases, such as epilepsy, glioblastomas, cognitive decline, and psychiatric conditions.^{17,18}

Epilepsy

A high-fat, low-carbohydrate, low-protein ketogenic diet has been in clinical use for over a century as a treatment option for children with epilepsy, and current evidence suggests that it can be quite effective for seizure reduction, with the caveat that only a small number of high-quality studies have been conducted.¹⁹ The anticonvulsant property of ketones is not completely understood but is likely related to neurotransmitter modulation, reduced neuronal excitability, enhanced energy production, and a direct antioxidant effect on the brain.^{20,21} However, adverse events, such as stunted growth and bone loss,^{22,23} as well as a low palatability of the diet, may limit adherence and the length of time that children may safely follow it.¹⁹

At least one study in rats has suggested that exogenous ketones can provide a benefit for epilepsy, without requiring dietary restriction. Using exogenous ketones to maintain serum ketone levels of 1.35–2.37 mmol/L nearly doubled the onset time of a seizure, compared with a normal diet without exogenous ketones.²⁴ It also prevented neuronal losses that were observed in the non-ketone group.

Migraines

Currently, a study in human adults is in the works, investigating how exogenous ketone supplementation affects migraine frequency and severity,²⁵ spurred by positive results of a ketogenic diet pilot study.²⁶

Brain cancer

For glioblastoma, or brain cancer, things are more complicated. Ketones have shown potential benefits for brain cancer in several rodent models,²⁷ but human trials are lacking.²⁸ While case studies have suggested that a ketogenic diet may be an effective addition to standard care,^{29,30} rigorous studies comparing a ketogenic diet or exogenous ketone use with standard therapy alone are nonexistent.²⁸

Theoretically, exogenous ketones may offer some advantages over a traditional ketogenic diet, based on some of the complaints of researchers who've attempted to implement ketogenic diets with their patients:²⁸

- Brain cancer patients frequently have problems in areas such as executive functioning, thinking, coordination, and vision that can make instituting and complying with an extremely restricted diet very difficult or almost impossible. Exogenous ketones are much easier to use than implementing a full-blown ketogenic diet.
- Because brain cancer is a life-threatening illness, it is often difficult for caregivers to restrict calorie intake or forbid foods that patients find comforting, familiar, and drawn to in a time of illness. Exogenous ketones would require neither calorie nor food restriction.
- The preparation and palatability of food can be a challenge to patients as well as caregivers. Again, exogenous ketones would eliminate this issue.
- Hospitalizations in an acute or chronic care facility — rehabilitation centers, assisted living, long-term care facilities — can be a problem, because many of these institutions may be ill-equipped to handle a ketogenic diet, with its strict weighing of food. Exogenous ketones would circumvent this issue.
- Because of the strictness and limitations of a ketogenic diet, socializing with friends and family around a meal can be difficult. Exogenous ketones make staying in ketosis easy, regardless of diet.

Cognition

For cognitive decline, one pressing issue is that the brain can fail to obtain enough energy to function properly, be it from vascular problems or insulin resistance.^{31,32,33} A ketogenic

diet is believed to provide a neuroprotective benefit by reducing oxidative stress and increasing mitochondrial respiration.³⁴

A ketogenic diet is also believed to provide benefit by reducing the oxidation of glucose and leading to calorie restriction,³⁴ neither of which would necessarily be present with the use of exogenous ketone supplements. The few studies available on patients with Alzheimer's disease suggest that a ketogenic diet may hold value but ultimately needs to overcome a brain energy deficit.³⁵ By this logic, exogenous ketones could be beneficial by allowing for both glucose and ketones as fuel sources for the brain.

Research investigating the role of exogenous ketones in neurodegenerative diseases and cognitive decline is lacking. What studies are available test cognitive function in healthy adults after a bout of endurance exercise. These studies suggest that supplementing with exogenous ketones either benefits cognitive function when combined with carbohydrates, as compared with carbohydrates alone,³⁶ or provide no benefit when taken alone, as compared with a noncaloric placebo.¹⁴

Psychiatric disorders

Finally, we have psychiatric disorders. Animal and human research have suggested that a ketogenic diet may have therapeutic potential in the treatment of several psychiatric conditions, including schizophrenia, anxiety, and depression, likely through bioenergetics, ketone metabolism, neuronal activity, neurotransmitter balance, and countering inflammation.¹⁸ However, research at this point is largely speculative and theoretical, with a need for further investigation via controlled trials in humans.

Exogenous ketones have been best investigated for their use in various brain disorders and diseases, such as epilepsy, glioblastomas, cognitive decline, and psychiatric conditions. Overall, research at this point is speculative and theoretical, and based on studies using a ketogenic diet rather than exogenous ketones.

MCTs

What are MCTs?

Medium-chain triglycerides (MCTs) are a type of saturated fat in which the fatty acids have only 6 to 10 carbons.³⁷ These individual fatty acids are referred to as medium-chain fatty acids (MCFAs) and include:

- Caproic acid (C6:0)
- Caprylic acid (C8:0)
- Capric acid (C10:0)

Although the 12-carbon saturated fatty acid lauric acid is commonly called an MCFA, it displays properties somewhere between MCFAs and long-chain fatty acids and is therefore not a true MCFA.^{38,39}

True MCTs are found primarily in coconut oil, palm kernel oil, and dairy fat.

Table 1: MCT content of select foods

FOOD	GRAMS OF MCT PER 25 g SERVING (≈2 Tbsp)
Coconut oil	3.17
Babassu oil	2.93
Dried coconut meat (unsweetened)	2.12
Palm kernel oil	1.80
Dried coconut meat (toasted)	1.54
Butter, without salt	1.43
Butter oil, anhydrous (Ghee)	1.38
Hard goat cheese	1.29
Coconut cream	1.14
Raw coconut meat	1.10
Semisoft goat cheese	1.09
Roquefort cheese	0.87
Coconut milk	0.78
Feta cheese	0.71

Reference: USDA Food Composition Databases. Accessed July 22, 2019.

<https://ndb.nal.usda.gov/ndb/nutrients/report/>

[nutrientsfrm?max=25&offset=0&totalCount=0&nutrient1=608&nutrient2=609&nutrient3=610&subset=0&sort=c&measureby=g](https://ndb.nal.usda.gov/ndb/nutrients/report/nutrientsfrm?max=25&offset=0&totalCount=0&nutrient1=608&nutrient2=609&nutrient3=610&subset=0&sort=c&measureby=g)

How are MCTs relevant to ketogenic diets?

Dietary fat is a mainstay of ketogenic diets, but not all fats are equal when it comes to ketogenesis. A diet with a higher proportion of MCTs can considerably increase ketogenesis, relative to diets high in long-chain triglycerides (LCT),^{40,41} and allow for increased carbohydrate consumption while maintaining comparable ketosis.⁴² A typical MCT ketogenic diet for childhood epilepsy contains around 19% carbohydrates, 10%

protein, and 71% fat, of which 60% are MCTs. There is also a modified version that uses 30% MCTs, and levels anywhere in between could theoretically be used.⁴³

MCT diets are sometimes used in the treatment of epilepsy and may be as effective as classical ketogenic diets.⁴⁴ Studies are scarce though, and more research is needed to compare these diets with other strategies. One obvious perk of an MCT ketogenic diet is that if more carbohydrates are allowed, the diet can be more diverse and nutritious, with more vegetables and fruits.

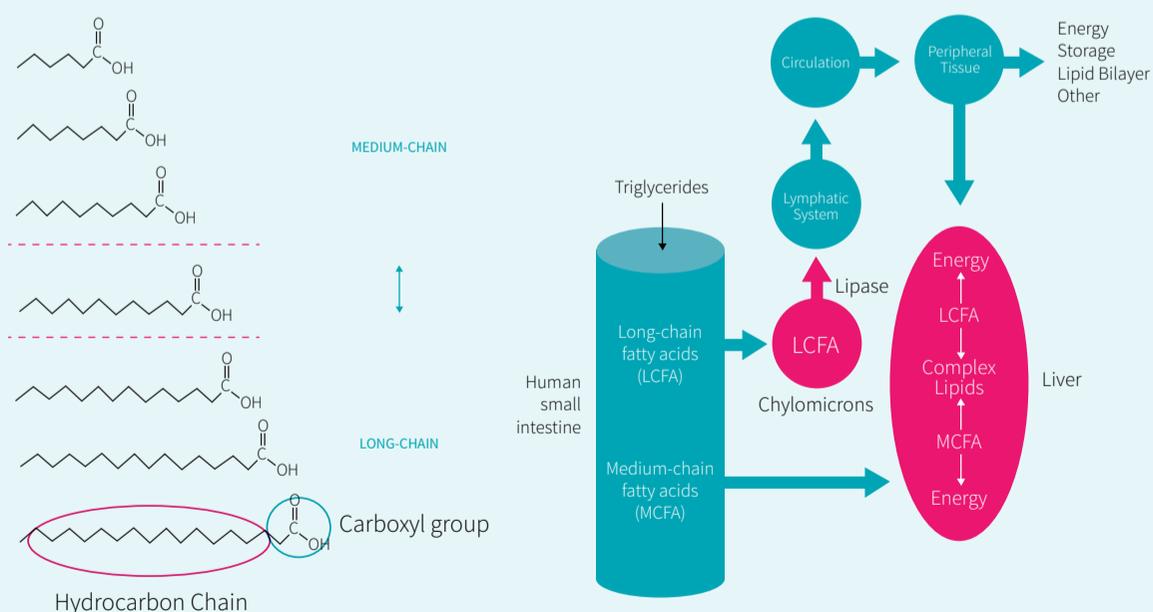
🔍 Digging Deeper: How does the digestion of MCTs and LCTs differ?

A triglyceride is chemically composed of three fatty acid tails attached to a glycerol backbone. These tails are comprised of a carboxyl group and a hydrocarbon chain. When the length of the hydrocarbon chains are 6 to 10 carbon atoms, the triglyceride is called a medium-chain triglyceride (MCT, shown in the figure below).⁴⁵ Triglycerides with longer chain lengths are called long-chain triglycerides (LCTs). The carboxyl group can interact with water, but the hydrocarbon chain can't. So the longer the hydrocarbon chain, the more insoluble the triglyceride is in water. This is why most oils, which are comprised mainly of LCTs, form droplets in water instead of dissolving.

Because our blood is mostly water, the body had to develop a way to break down LCTs and then shuttle their fatty acids to our tissues for energy or storage. Once LCTs enter the small intestine, bile breaks them into smaller groups (think smaller oil droplets in water), allowing for lipase (an enzyme) to have more surface area access to break the triglycerides into fatty acids. The fatty acids then enter the intestinal wall. Still insoluble in blood, they must then be packaged into a chylomicron, which has an outer coating that can interact with water. But the chylomicron must first go through lymph before it enters the blood, then circulates to take the fatty acids to tissues in need.

This roundabout process makes LCTs not as readily available as glucose for energy soon after digestion. Conversely, since MCTs have hydrocarbon tails that are relatively smaller than those of LCTs, they can just barely dissolve in water. This allows the MCTs to enter directly into the portal vein, where they head straight to the liver and can be converted into ketones.

Figure 2: The difference between LCTs and MCTs and their digestion



Reference: St-Onge and Jones. *J Nutr.* 2002. PMID:11880549

Medium-chain triglycerides (MCT) are digested differently than long-chain triglycerides (LCT), and one of the implications is that they're more liable to be turned into ketones in the liver. This has led to the use of large amounts of MCTs in ketogenic diets, allowing for a higher intake of carbohydrates than would typically be conducive to ketosis. MCT ketogenic diets have the added perk of allowing for more fruits and vegetables in the diet, but have received less research attention for disease treatment than standard ketogenic diets.

Does the difference between MCTs and LCTs have implications for weight loss?

The different transportation and metabolism of MCTs may make them more readily oxidized and less liable to be stored as body fat than LCTs, as numerous animal studies suggest.^{46,47,48,49,50,51,52}

When it comes to humans, studies have compared meals with a notable amount of MCTs against meals with LCTs having the same total caloric intake. They've generally found modest increases in postmeal fat oxidation and energy expenditure in the MCT groups,^{53,54,55,56,57,58,59} but not all have.^{60,61,62} This may translate to modest reductions in body fat, as seen in various studies,^{53,54,63,64} but the evidence is fairly inconsistent, with other studies not finding any effect.^{40,55,62,65} It's possible that a high proportion of fat intake has to come from MCTs to see a notable effect, but more research is needed.

A ketogenic diet is very high in fat, so there may be more room to make a high proportion of that fat MCTs. On the other hand, MCT oil is largely devoid of nutrients, and nutrient intake can be an issue on ketogenic diets. If you find yourself deficient in nutrients, focus on choosing more nutrient-dense meats, nuts, and vegetables. MCT oil lets you maintain ketosis while allowing you to get more carbohydrates from plant foods if you so choose.

MCTs are oxidized at a higher rate than LCTs after meals, and some evidence suggests that leads to a net increase in energy expenditure and a small reduction in body fat. More research is needed to properly measure differences in weight loss in the long term. MCT oil is also largely devoid of micronutrients, so MCT oil ketogenic diets may require extra attention to nutrient intake.

Don't MCTs provide greater satiety?

A possible additional perk of adding MCTs to a ketogenic weight loss diet is an increase in satiety. In a variety of studies, a meal high in MCTs reduced food consumption at subsequent meals when compared with the same amount of calories from other fats.^{60,66,67,68,69,70} Fats that were compared to MCTs included olive oil, lard, corn oil, unspecified vegetable oil, rapeseed oil, coconut oil (which contains some MCTs, but not nearly as much as pure MCT oil), and corn oil, and the fat doses compared were 10 g, 25 g, 30 g, 35 g, and 40 g. However, not all studies have found an effect,^{57,68} including studies comparing MCT oil with rapeseed and sunflower oil, at doses of 20 g and 68 g.

When it comes to subjective ratings of hunger and satiety, some studies have found a reduction by MCT,^{40,66,67,70} but others haven't.^{54,57,60,69,71} Some studies found a reduction in calorie intake, but, oddly, no reductions in ratings of hunger and satiety. More research is needed to figure out why study results differ and what the mechanism is.

A number of studies suggest that the consumption of medium-chain triglycerides (MCTs) can lead to lower food intake at subsequent meals as compared with the same amount of long-chain triglycerides (LCTs). Most studies are short, and it's unclear what the implications for weight loss are in the long term. Research on subjective ratings of hunger and satiety is quite mixed, and although studies generally show less food consumption after consuming MCTs, it's unclear why.

Chapter 12: Blood Sugar

Summary

A ketogenic diet may be helpful for diabetes and blood sugar control. Specifically, it can reduce elevated glucose and insulin levels, although only notably so in those with diabetes or prediabetes. With keto, glucose levels after meals are often lower, and blood sugar may be more stable. More research is needed to determine how effective it is for weight loss, especially compared with other diets.

However, what we really care about is overall health, not just individual blood sugar related measurements. So it's important to keep in mind that:

- Cardiovascular health is an especially pressing concern when it comes to high blood glucose and obesity. It's not clear that a keto diet leads to an improvement in cardiovascular health, outside of the impacts from weight loss. Keto may also have some downsides when it comes to certain cardiovascular risk factors, although evidence is mixed, and may be balanced with improvements in other risk factors.
- Keto can also come with an increase in circulating free fatty acid levels, which can play a role in the perpetuation and harm of diabetes.
- The ketogenic diet isn't a panacea that's going to solve all health problems simply by restricting carbohydrates and producing ketones. Everything else still matters, from micronutrient intake to exercise.
- Supplementation may be helpful, and careful dietary planning is necessary. Future research will hopefully explore ketogenic diets from many more angles: the features of the best possible ketogenic diets, how they compare to the best possible higher carbohydrate alternatives for a wide range of cardiometabolic outcomes — and, ultimately, mortality and disease complications.

Diabetes: a major public health problem

Effective strategies for keeping blood sugar at a healthy level have never been more critical. The Centers for Disease Control and Prevention (CDC) reported in 2017 that the rate of diabetes in the US had increased steadily over the past 60 years and was at an all-time high.¹ A 2016 World Health Organization report estimated that 422 million people worldwide had type II diabetes and the percentage of people in the world with diabetes roughly doubled since the '80s.²

Figure 1: Trends in the prevalence of diabetes by region, 1980–2014

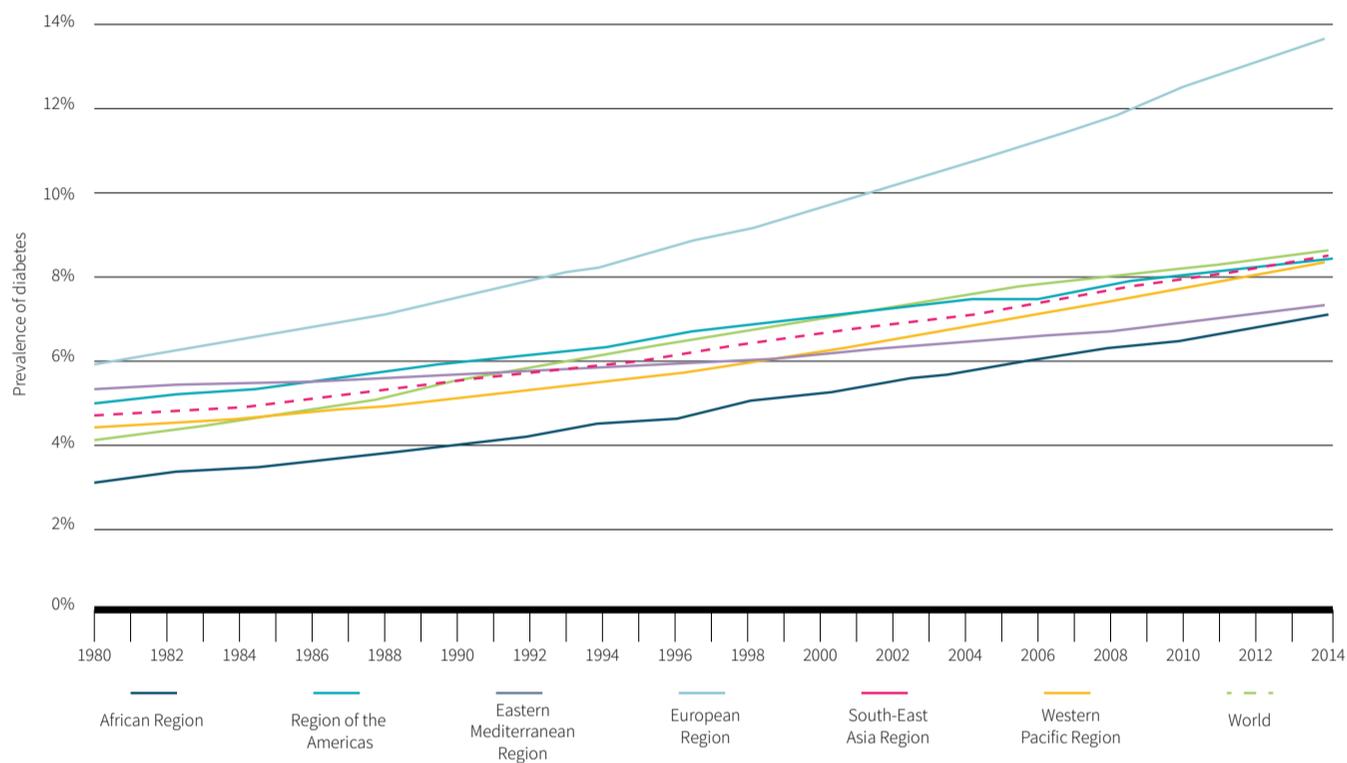
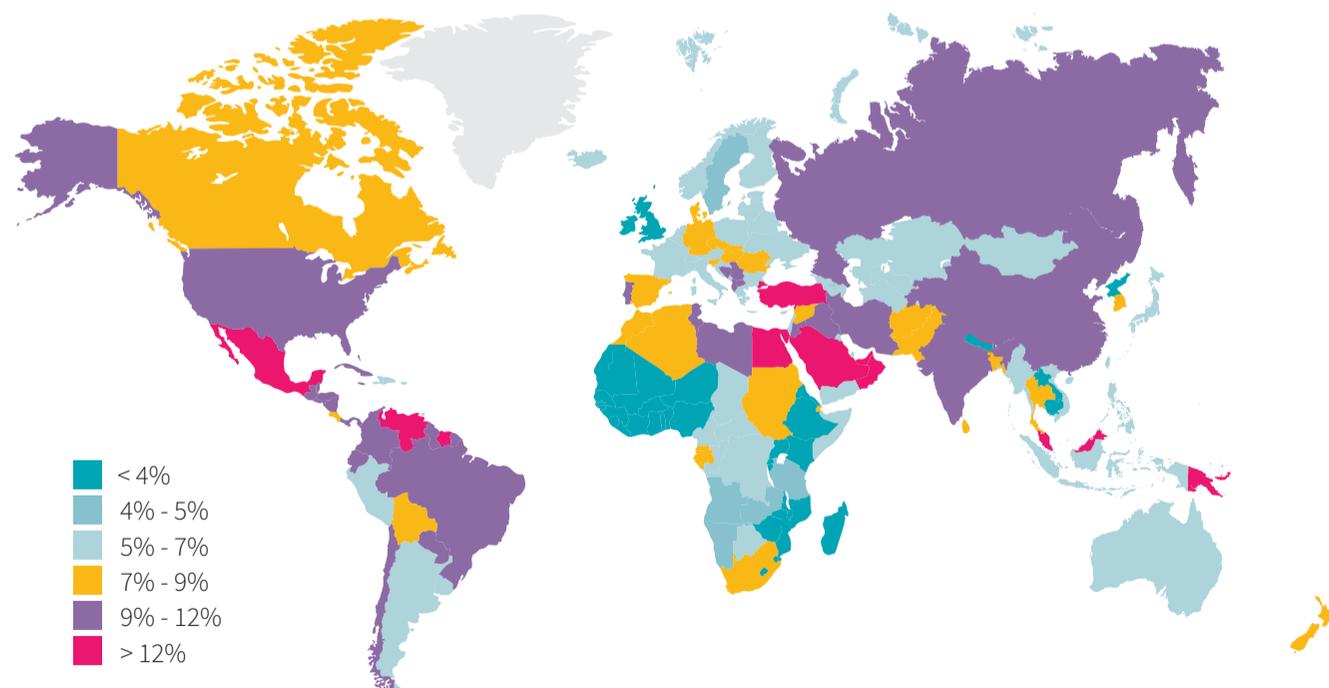


Figure 2: Worldwide diabetes prevalence



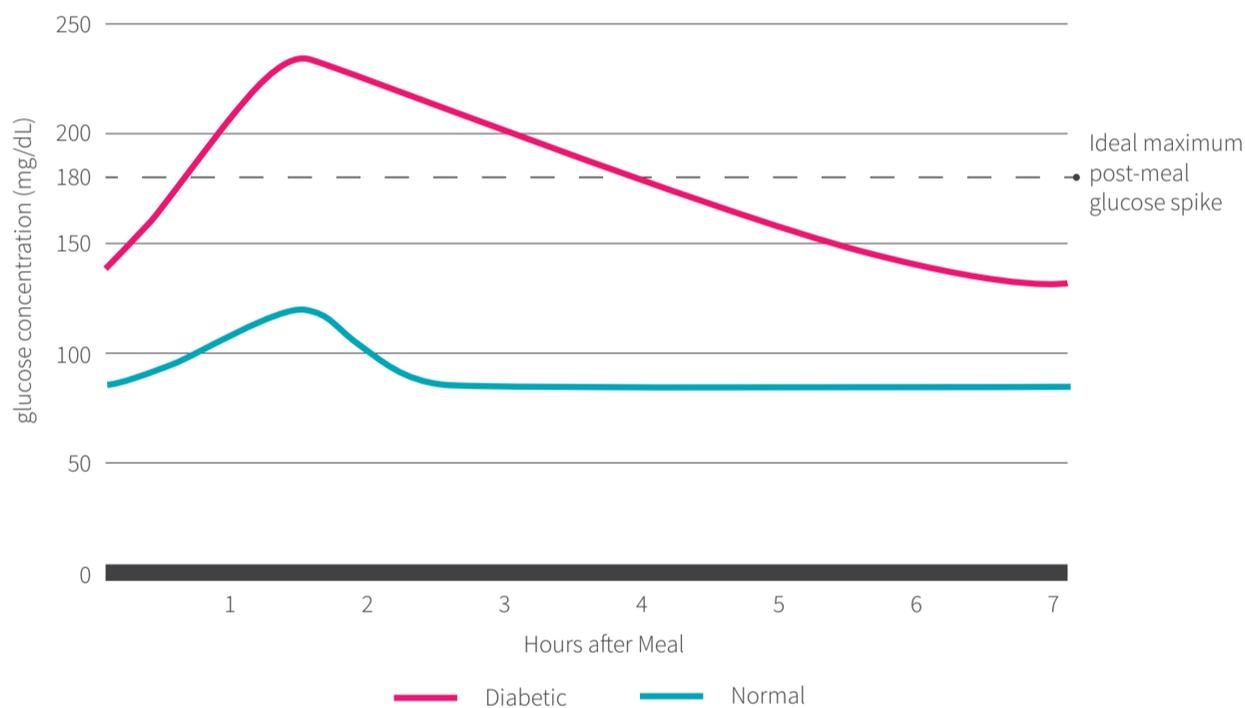
Adapted from Cho et al. *IDF Diabetes Atlas*. 2017. ISBN:978-2-930229-81-2

But that's only full-blown diabetes. There are also a lot of people with prediabetes: mildly elevated glucose that can also be harmful to health and lead to diabetes.³

Diabetes occurs when cells become resistant to insulin (type II diabetes) or the pancreas can't produce enough insulin (type I diabetes or type II diabetes in its later stages). When insulin signaling isn't working right, glucose can't be removed from the bloodstream quickly enough, producing very high levels of blood sugar (i.e., [hyperglycemia](#)).

Hyperglycemia and insulin resistance are damaging to the body through oxidative stress, inflammation, and malfunctioning of a variety of processes.^{4,5,6} Although diabetes itself can be fatal, the main danger is that it increases the risk of other diseases — most commonly cardiovascular diseases,⁷ but also likely cancer, Alzheimer's, and Parkinson's.^{8,9,10} to name a few. Additionally, damage to organs and the skin produces many other health problems, such as lesions and blindness.

Figure 3: Hyperglycemia: how diabetics and nondiabetics respond to a meal



Excess calorie intake and the resulting obesity are the major driving forces behind the new diabetes epidemic. Naturally, weight loss can help. One review found that weight loss from all kinds of interventions — surgery, appetite-suppressing medication, lifestyle interventions, or a combination¹¹ — improved diabetes

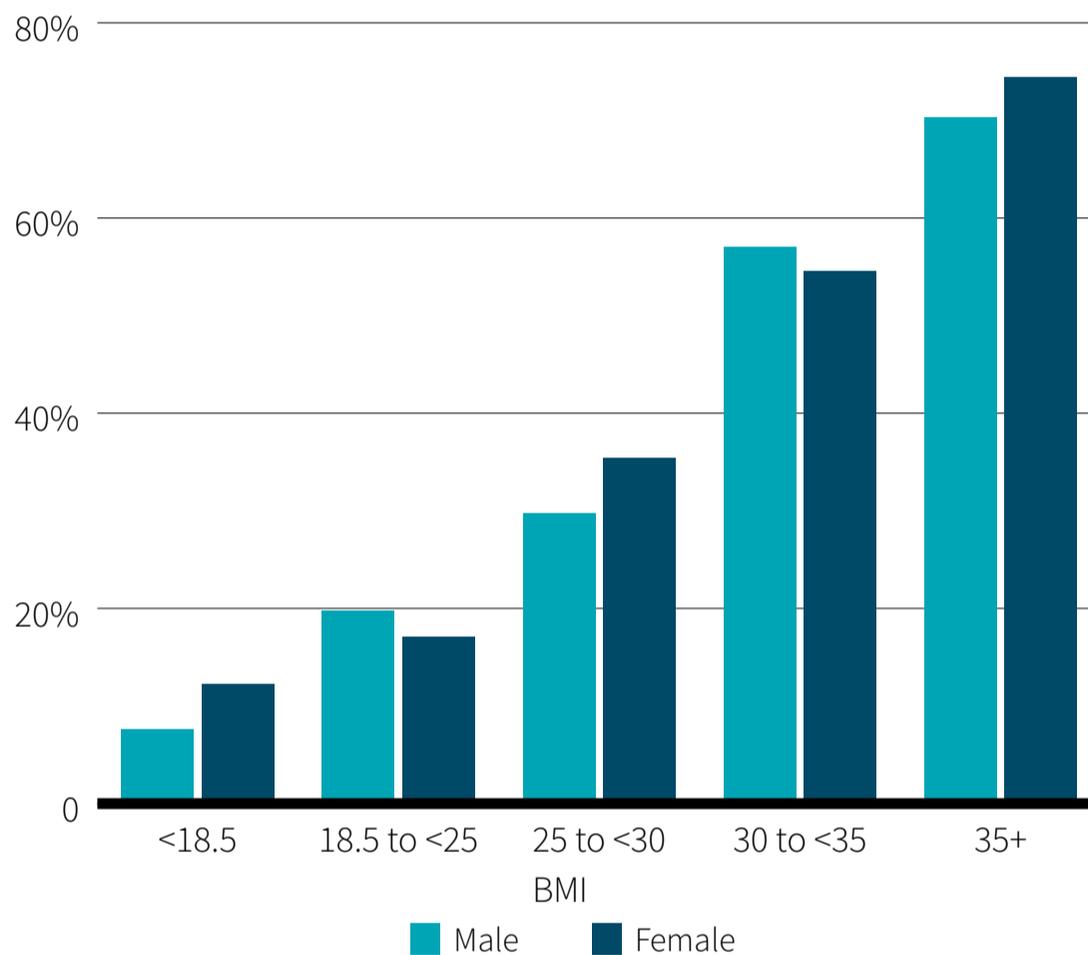
On the other hand, many long-term studies that use a diet to achieve weight loss tend to produce only modest improvements in diabetes. Why? Probably because they rarely achieve substantial long-term weight loss.¹²

Fortunately for those at high risk of diabetes, even mild weight loss can meaningfully reduce the risk, though not eliminate it.¹³ While helpful, weight loss isn't the only game in town, and

some people with diabetes aren't obese or even overweight. Quality of diet also matters, and the composition of macronutrients (carbohydrates, fat, and protein) is worth exploring.

The prevalence of diabetes, the harm that it does, and the difficulty of sustaining long-term weight loss have made many people more open to extreme measures, such as a ketogenic diet. Many people use it to try to lose weight and control their blood sugar when they haven't had success with other approaches. But does it have an independent effect on hyperglycemia, and can it improve health outcomes related to diabetes? Let's take a look.

Figure 4: Lifetime risk of diabetes for 18-year-olds in the US



Reference: Narayan et al. *Diabetes Care*. 2007. PMID:[17372155](https://pubmed.ncbi.nlm.nih.gov/17372155/)

Type II diabetes has become an enormous health problem in the world, fueled by the rise in the obesity rate. It causes considerable harm to the body and increases the risk of other diseases. Weight loss can help, but it's often difficult to achieve long term. However, weight loss isn't the only game in town. Dietary quality and the composition of macronutrients have been explored as part of a strategy for improving metabolic health.

The ketogenic diet has become popular among people who haven't had success with other approaches. It's an extreme diet, but when faced with an extreme problem like type II diabetes, extreme solutions are worth investigating.

Does keto for diabetes make sense?

Extreme carbohydrate restriction as a treatment for diabetes has garnered the interest of researchers recently, but the practice goes back hundreds of years, before the advent of insulin as a medication or insulin-sensitizing drugs.¹⁴ Back then, diabetes would only be diagnosed through symptoms and the presence of large amounts of sugar in the urine, which is when diabetes is at its most severe. Dating back to 1796, very low carbohydrate diets were used, and efficacy was measured by the reduction of sugar in the urine.

Throughout the 19th century, carbohydrate restriction was refined and became a prominent treatment, eventually falling out of favor due to the breakthrough of insulin medication.

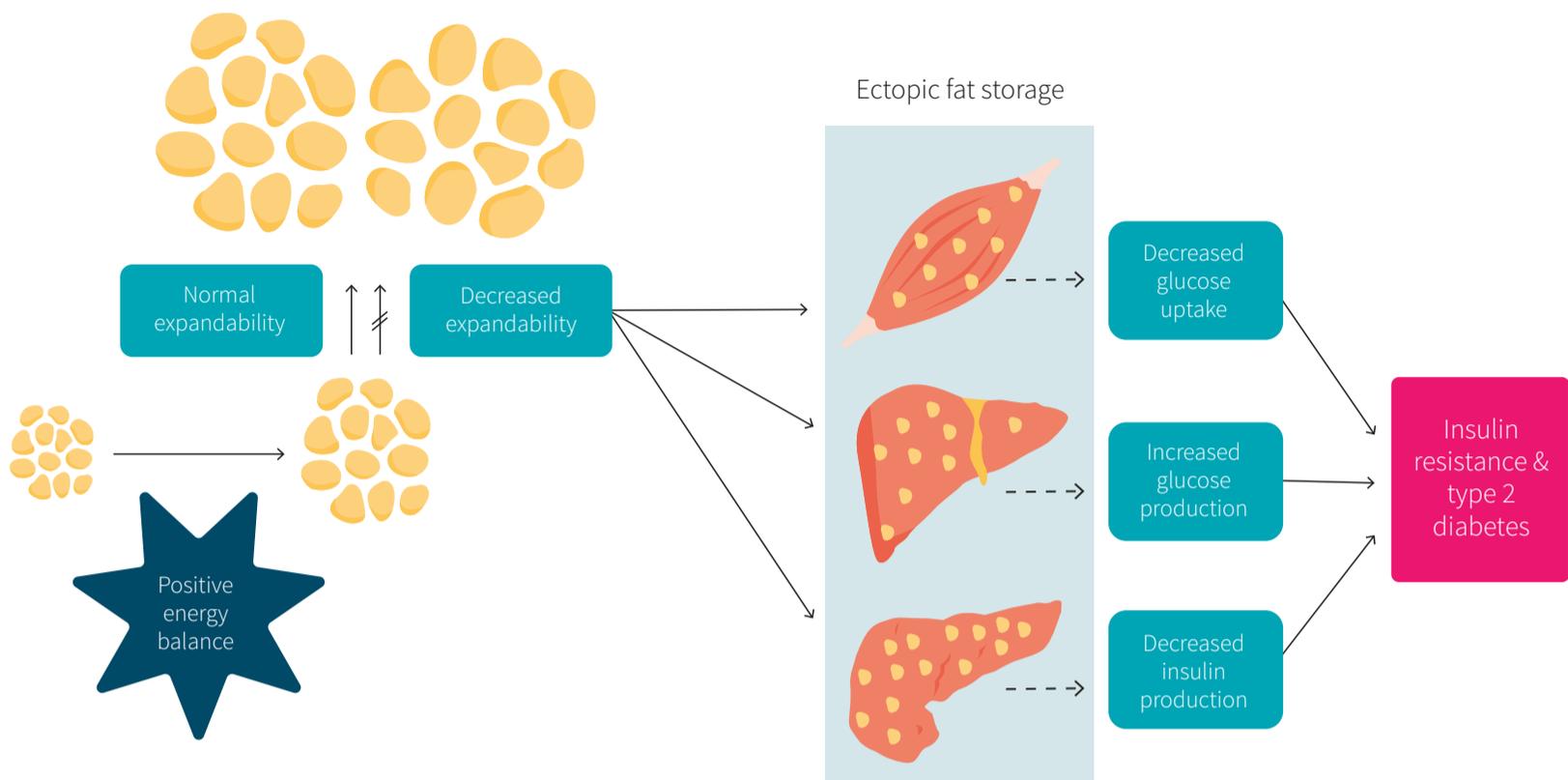
These days, thanks to accurate blood sugar measurements, we understand diabetes quite differently. Type II diabetes is diagnosed long before glucose is present in the urine, and unless the pancreas is severely damaged, the focus is on reducing blood sugar by improving insulin sensitivity. Modern reasoning for why a ketogenic diet might help with blood sugar makes some sense: if the cells are insulin resistant and can't use glucose efficiently, taking as much glucose out of the equation as possible and running mostly on fat can reduce glucose and insulin levels, thus reducing harm to the body.¹⁵ There is also the possibility that ketones themselves could help reduce glycemia and possibly have a therapeutic role.¹⁶

However, some caveats may apply.

Glucose is just one part of the picture. Fatty acids aren't benign to diabetics.

Free fatty acids (FFAs) are fatty acids that are not connected to glycerol, such as in triglycerides. They are used primarily for energy in the body or stored in fat cells when they're not needed — at least ideally. Much like glucose, their concentration in the bloodstream can become too high. When this happens, they accumulate in and damage pancreatic beta cells, which produce insulin;¹⁷ skeletal muscle and liver cells, where they cause insulin resistance;¹⁸ and endothelial cells, where they impair vascular function.¹⁹ They generally cause chaos in whichever tissues they accumulate.

Figure 5: Adipose tissue's expandability and the path to hyperglycemia



But if a ketogenic diet increases fat-burning, might it also help prevent excessive FFA accumulation? Not necessarily, since various studies have found that a ketogenic diet tends to raise FFA levels.^{20,21,22}

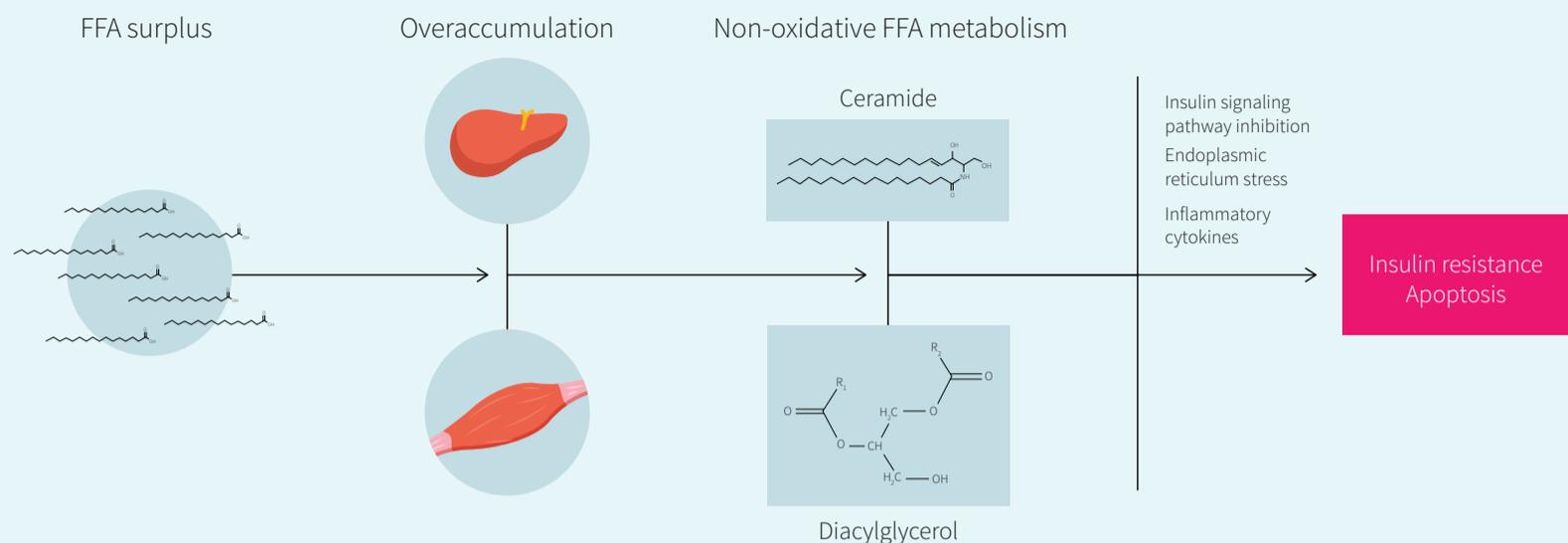
And it's possible that these higher FFA levels can lead to harm. Many studies have found worse flow-mediated dilation (the dilation of arteries in response to increased blood flow; impaired flow-mediated dilation is a risk factor for atherosclerosis) in people on a low-carbohydrate/high-fat diet than on a diet lower in fat.^{23,24,25} That is not to say that this will necessarily happen, and it doesn't across all studies; after all, hyperglycemia is also implicated in impaired vascular function. The point is that carbohydrates are not the only macronutrient that can become harmfully elevated and cause damage to the body.

🔍 Digging Deeper: Flow-mediated dilation, vascular function, and cardiovascular disease

Flow-mediated dilation is a common way to assess endothelial function, by measuring the ability of brachial artery endothelial cells to induce dilation in response to an increase in blood flow.²⁶ It's a simple test where a blood pressure cuff is used to restrict blood flow for 5 minutes, and then the artery's diameter is measured in response to the rush of blood after release.

Impaired flow-mediated dilation is associated with an increased risk of cardiovascular disease.²⁷ Although its exact relevance is difficult to discern, there's a rationale for a causal role of endothelial function in the development of atherosclerosis.²⁸ This measurement is commonly performed in clinical trials of the effects of diets and will come up multiple times when we look at ketogenic diet studies.

Figure 6: Potential role of free fatty acids in insulin resistance



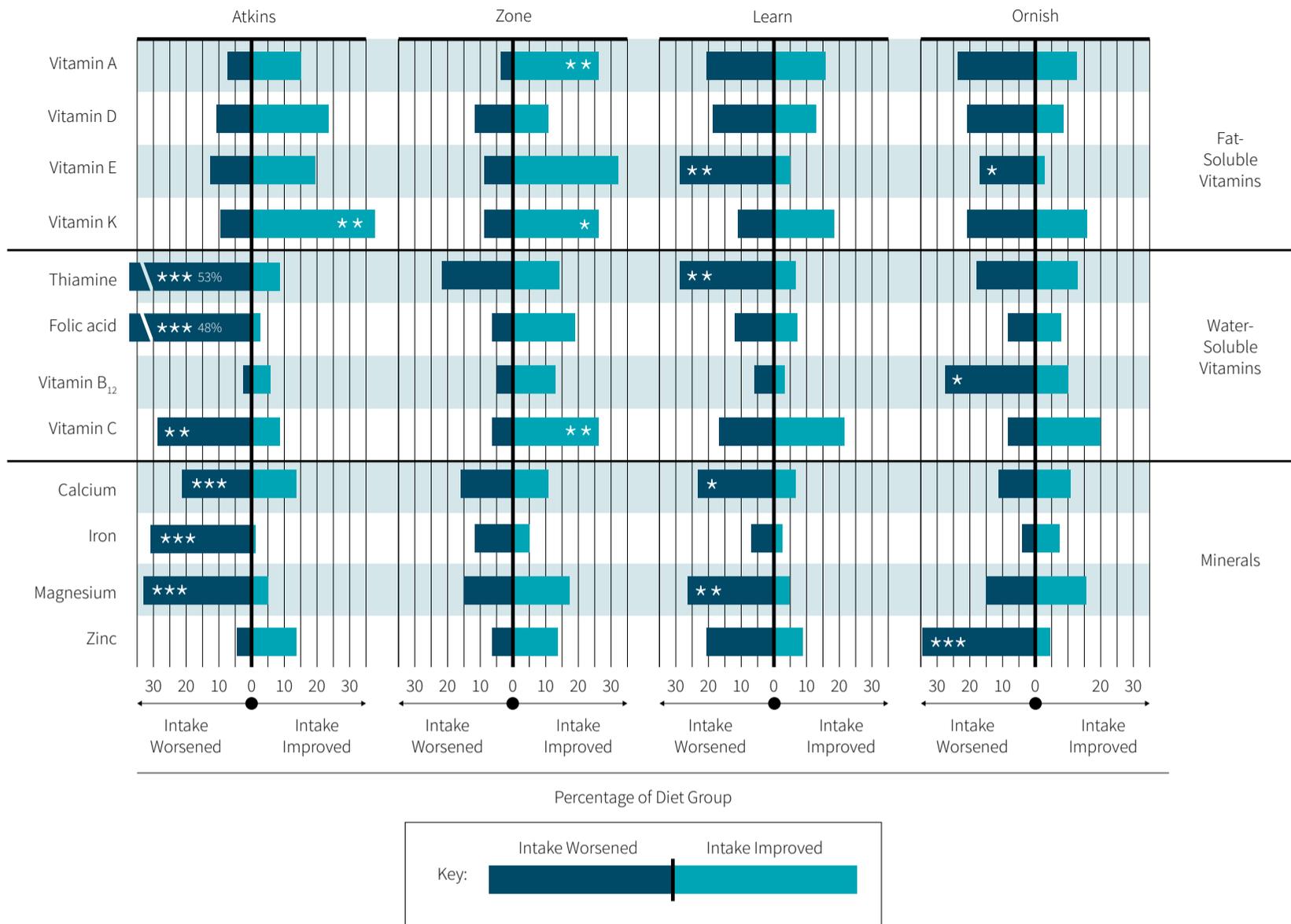
References: Castro et al. *Arq Bras Endocrinol Metabol.* 2014. PMID:[25211442](#) • Chaurasia et al. *Trends Endocrinol Metab.* 2015. PMID:[26412155](#)

While fasting glucose, glycated hemoglobin, and measures of insulin resistance are relevant and important things to look at in type II diabetes, the real endgame is the rate of disease and severity of complications.

Diets that restrict macronutrients may restrict healthy foods

While some off-limits foods will be no real loss nutritionally — notably, refined grains and sugars — there are many other foods, such as fruits, whole grains, and some vegetables, that will be severely restricted when carbohydrates are limited. This can lead to an insufficient intake of essential nutrients,^{29,30} which isn't unique to low-carbohydrate and ketogenic diets: restriction of macronutrients without careful dietary planning naturally reduces the intake of some micronutrients prevalent in restricted foods.

Figure 7: Risk of micronutrient inadequacy in differing diet patterns



* P < 0.01, ** P < 0.001, *** P < 0.0001

Adapted from Gardner et al. *Am J Clin Nutr.* 2010. PMID:20573800.

One of the nutrients that tends to be lacking in a ketogenic diet is magnesium, which people with type II diabetes are already low in.³¹ The resolution of low magnesium levels through supplementation likely confers a modest improvement in glycemia.^{32,33,34}

Micronutrient supplementation may bridge the gap, and careful planning to include large amounts of low-carbohydrate greens and nuts may assist in meeting nutritional needs. But without thorough and continuous diet tracking, a ketogenic diet may put people at risk for deficiency.

Additionally, low-carbohydrate diets — ketogenic diets even more so — tend to be lower in dietary fiber than other diets, and dietary fiber is associated with a lower risk of type II diabetes in observational studies and RCTs.^{35,36} Some types, such as psyllium and beta-glucan, notably lower fasting glucose, glycated hemoglobin, and insulin levels in controlled trials.^{37,38,39}

But glucose levels and carbohydrates are only one piece of the puzzle. Since cardiovascular disease is such a big problem for diabetics, and such a common cause of death and disability, a diet should be good not only for metabolic health but also for cardiovascular health. Will a ketogenic diet be a net positive? Only clinical trials can tell us.

A dramatically lower intake of carbohydrates makes sense to combat high glucose and insulin levels, but it comes with an increase in fat intake. Free fatty acids play a role in the harm and perpetuation of type II diabetes. When levels get too high, they can cause insulin resistance and damage the arteries, pancreas, and other organs — many of the same concerns as with hyperglycemia.

A ketogenic diet is restrictive and will also make it harder to get enough fiber and nutrients, which can help improve insulin sensitivity. Technically, that obstacle can be overcome through supplements and careful dietary planning, but it isn't always.

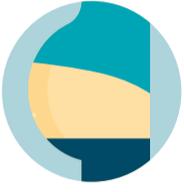
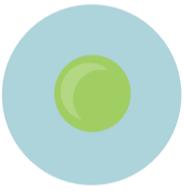
Further clinical trials will be needed to figure out which diets work best for different situations.

Do ketogenic diets uniquely help treat diabetes and prediabetes?

The question of whether ketogenic diets help keep blood sugar in check can be looked at in a couple of ways. Greater weight loss has been noted in many ketogenic diet studies, and that can help normalize blood sugar levels. But even though weight loss is effective for some people, it isn't a unique benefit of a ketogenic diet. For that reason, it's useful to look at studies where neither group lost significantly more weight than the other to know the specific effect of extreme carbohydrate restriction. We'll look at those first. These are at studies where blood and urine testing or provision of food by the researchers confirmed that a majority of participants in the ketogenic group were, in fact, in at least mild ketosis.

The caveat, as in all studies with large dietary changes, is that the diets participants end up following won't necessarily be the best versions of those diets. As already mentioned, many diets low in carbohydrates tend to be low in fiber and various other nutrients, but they don't have to be. Also, because type II diabetes increases the risk for cardiovascular disease, we'll take a look at any cardiovascular risk markers from the studies to see how well any change in metabolic health markers translates to improvements in cardiovascular health markers.

Figure 8: The definition of metabolic syndrome

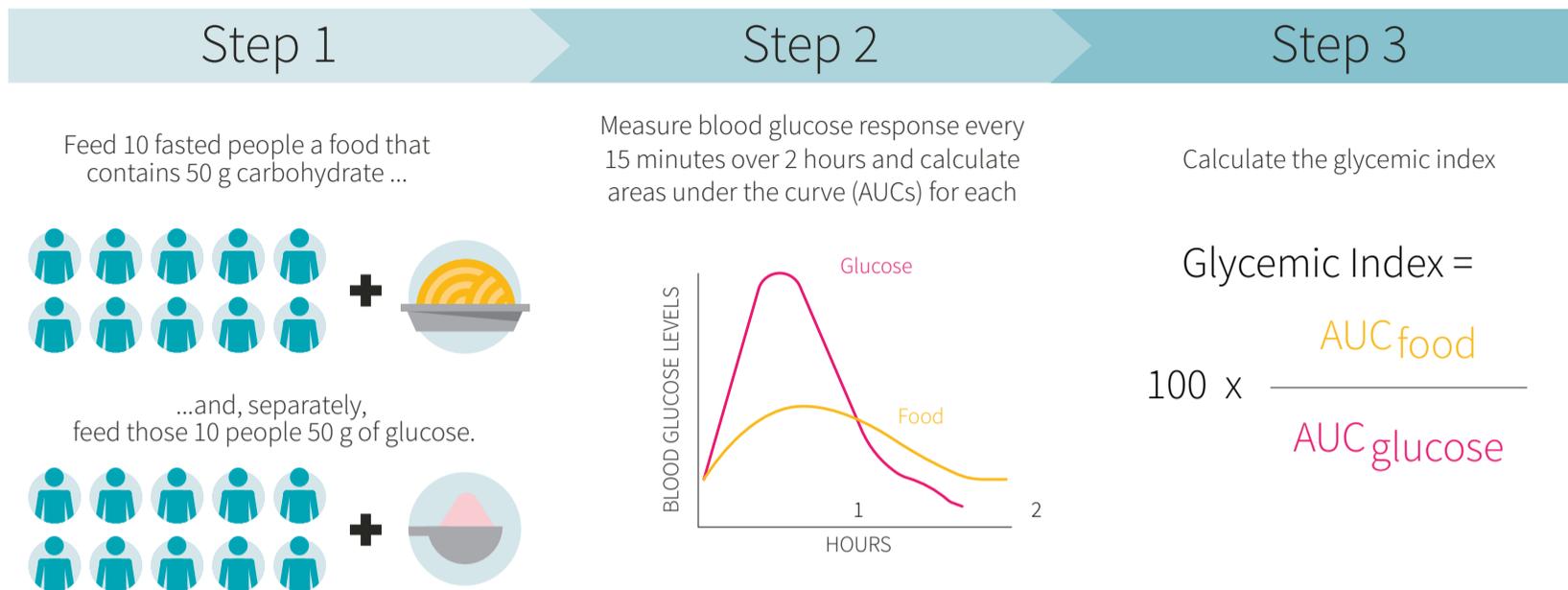
Three or more of the following criteria...													
Waist circumference 	NATIONALITY AND ETHNICITY-SPECIFIC <table border="1"> <thead> <tr> <th></th> <th>USA</th> <th>EUROPE, SUB-SAHARAN AFRICA, MIDDLE EAST</th> <th>ASIA, CENTRAL AND SOUTH AMERICA</th> </tr> </thead> <tbody> <tr> <td>♂</td> <td>≥ 102 cm</td> <td>≥ 94 cm</td> <td>≥ 90 cm</td> </tr> <tr> <td>♀</td> <td>≥ 88 cm</td> <td>≥ 80 cm</td> <td>≥ 80 cm</td> </tr> </tbody> </table>		USA	EUROPE, SUB-SAHARAN AFRICA, MIDDLE EAST	ASIA, CENTRAL AND SOUTH AMERICA	♂	≥ 102 cm	≥ 94 cm	≥ 90 cm	♀	≥ 88 cm	≥ 80 cm	≥ 80 cm
	USA	EUROPE, SUB-SAHARAN AFRICA, MIDDLE EAST	ASIA, CENTRAL AND SOUTH AMERICA										
♂	≥ 102 cm	≥ 94 cm	≥ 90 cm										
♀	≥ 88 cm	≥ 80 cm	≥ 80 cm										
Triglycerides 	≥ 150 mg/dL or drug treatment for elevated triglycerides												
HDL-C 	≤ 40 mg/dL or drug treatment for low HDL-C												
Blood pressure 	Systolic ≥ 130 or diastolic ≥ 85 mmHg or drug treatment for hypertension												
Fasting glucose 	≥ 100 mg/dL or drug treatment for elevated glucose												

Reference: Alberti et al. *Circulation*. 2009. PMID:[19805654](https://pubmed.ncbi.nlm.nih.gov/19805654/)

Ketogenic diets for diabetes and metabolic syndrome

One study assessed 11 people with type II diabetes, who completed two dietary interventions lasting 4 days each: a ketogenic diet, a low-fat diet rich in low-glycemic index carbohydrates, and the same ketogenic diet with daily walking.⁴⁰ Although this study is too short to provide very much information on diabetes, it offers some insight into the acute effects of carbohydrate restriction.

Figure 9: How the glycemic index is measured

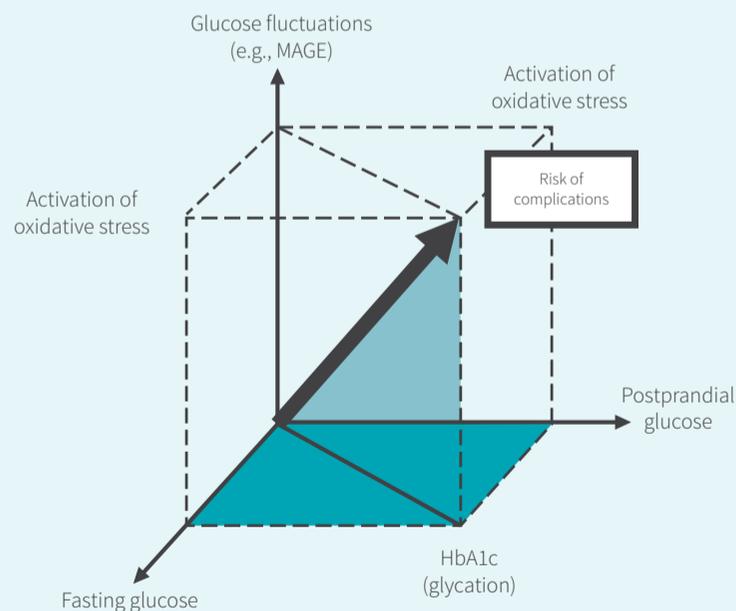


When the participants were on the ketogenic diet, they saw a larger reduction in fasting glucose and average glucose levels compared with the low-fat diet. They also had a lower mean amplitude of glycemic excursion (MAGE), a measure of glycemic variability. So, in short: this study supports the idea that a keto diet beats out a low-fat diet for glycemic control.

🔍 Digging Deeper: Glycemic variability

Research suggests that glucose fluctuation throughout the day is an independent risk factor for diabetic complications, beyond the average glucose level. Glucose swings lead to more oxidative stress than could be expected by the average glucose level.^{41,42,43}

Figure 10: Glucose fluctuations play a role in complications



However, outside the realm of glycemic control, the keto diet's advantages over the low-fat diet were less clear. There weren't any notable differences between the diets in fasting triglycerides, insulin, or inflammatory cytokines. In another paper based on the

same study, no notable effect on vascular flow-mediated dilation (the ability to widen in response to increased blood flow) was observed, but the ketogenic diet reduced endothelial microparticles, suggesting less susceptibility to atherosclerosis.⁴⁴ There was an increase in c-reactive protein (a marker of inflammation in the body and risk factor for cardiovascular disease) on the ketogenic diet, but it's unclear if it was a genuine effect of the diet.

Another study done on 107 overweight or obese people who were at risk for diabetes but still in the early stages of prediabetes raises some doubt on the efficacy of a ketogenic diet.⁴⁶ The ketogenic group consumed 35% of energy as protein, 61% as fat, and 4% as carbohydrate, while the high-carbohydrate group consumed 24%, 30%, and 46%, respectively. The diets lasted for 8 weeks, key foods were provided to the participants, and a dietitian educated them on the diets. Weight loss was slightly higher in the ketogenic group but not notably. Neither group saw a greater improvement in fasting glucose or insulin than the other, and the differences in total cholesterol, HDL, LDL, triglycerides, and vascular function weren't notable. The high-carbohydrate group saw a greater reduction in c-reactive protein. Importantly, measurements of plasma ketone concentrations suggested that the ketogenic group was in light ketosis during the study, though the only measurement was taken at 2 weeks. So, even though this study didn't strongly support keto's efficacy, it may be because the participants were barely in ketosis.

Tip: How can I tell if my blood sugars are normal?

Blood glucose (blood sugar) can be measured in a few ways, each with its own cutoff values indicating impaired glucose regulation.⁴⁷ Of these tests, [fasting blood sugar](#) and [HbA1c](#) are the most common. Fasting blood glucose is simply how much glucose is floating around in your blood during a fast. HbA1c (glycated [hemoglobin](#)) is a marker of blood glucose metabolism that estimates the average amount of glucose in your blood over the previous 3 months. The graphic below outlines the common methods for measuring and assessing your blood glucose levels.

Figure 11: Methods of blood glucose measurement

	Acute Methods			Chronic Methods
TEST	 Random or Casual Plasma Glucose Test	 Fasting Plasma Glucose (FPG)	 Oral Glucose Tolerance Test (OGTT)	 Hemoglobin A1c (HbA1c)
PROCEDURE	Droplet of blood is assessed via blood glucose meter Can be taken at any time	A check of blood glucose measures via glucose monitor after at least an 8-hour fast	A test that measures your blood glucose levels 2 hours after you consume a drink with a specific amount of glucose	A single blood test that can measure an average of your blood glucose levels over the past two to three months
RANGES	Variable depending on activity levels and last meal Above 200 mg/dL at any point is considered abnormal	Normal: < 100 mg/dL Prediabetes: 100-125 mg/dL Diabetes: ≥ 126 mg/dL	Normal: < 140 mg/dL Prediabetes: 140-199 mg/dL Diabetes: ≥ 200 mg/dL	Normal: < 5.7% Prediabetes: 5.7% - 6.4% Diabetes: ≥ 6.5%

A couple of other small trials in prediabetic subjects found an advantage in the ketogenic group when it came to glucose and insulin levels, but it was unclear if there was an overall benefit on cardiovascular risk markers and health. One of them found a slight increase in HDL, and there was a large reduction in triglycerides and an increase in LDL particle size, but no difference in total LDL or vascular function. Neither study measured markers of inflammation.^{48,49}

Overall, there's some mild evidence suggesting that a ketogenic diet can improve glycemic control compared to higher-carbohydrate diets, but its impact on other health markers compared to other diets is less clear. Much more research is needed to determine the many possible effects of a ketogenic diet on metabolic and cardiovascular outcomes.

Limited research suggests a reduction in fasting glucose and insulin by a ketogenic diet compared with roughly the same caloric intake from various higher-carbohydrate diets. Glucose concentrations throughout the day were also lower in one study, as was glucose variability, suggesting a reduced risk for hyperglycemia-induced complications. A ketogenic diet didn't lead to worse vascular function than the control groups, and one very short study found an advantage over the control group for one marker of susceptibility to atherosclerosis. There was an unclear effect on blood lipid profiles.

Ketogenic diets for the prevention of diabetes

There aren't very many studies that looked at the effect of a ketogenic diet on people with diabetic or prediabetic blood tests, but there are more studies on people who are overweight or obese who have yet to cross the prediabetic threshold. These studies speak to the overall healthfulness of ketogenic diets and whether they might prevent escalation to type II diabetes.

Figure 12: Effects of diabetes



The largest was a study on 83 obese participants, who were assigned to one of three weight-loss diets, each restricted by 30% of daily energy requirements, for 12 weeks.⁵⁰ The diets were either very low in fat with 70% carbohydrates; high in unsaturated fat with 50% carbohydrates; or ketogenic with 4% of energy as carbohydrates. Protein was higher on the ketogenic diet (35% of energy) compared with the other diets (20% of energy).

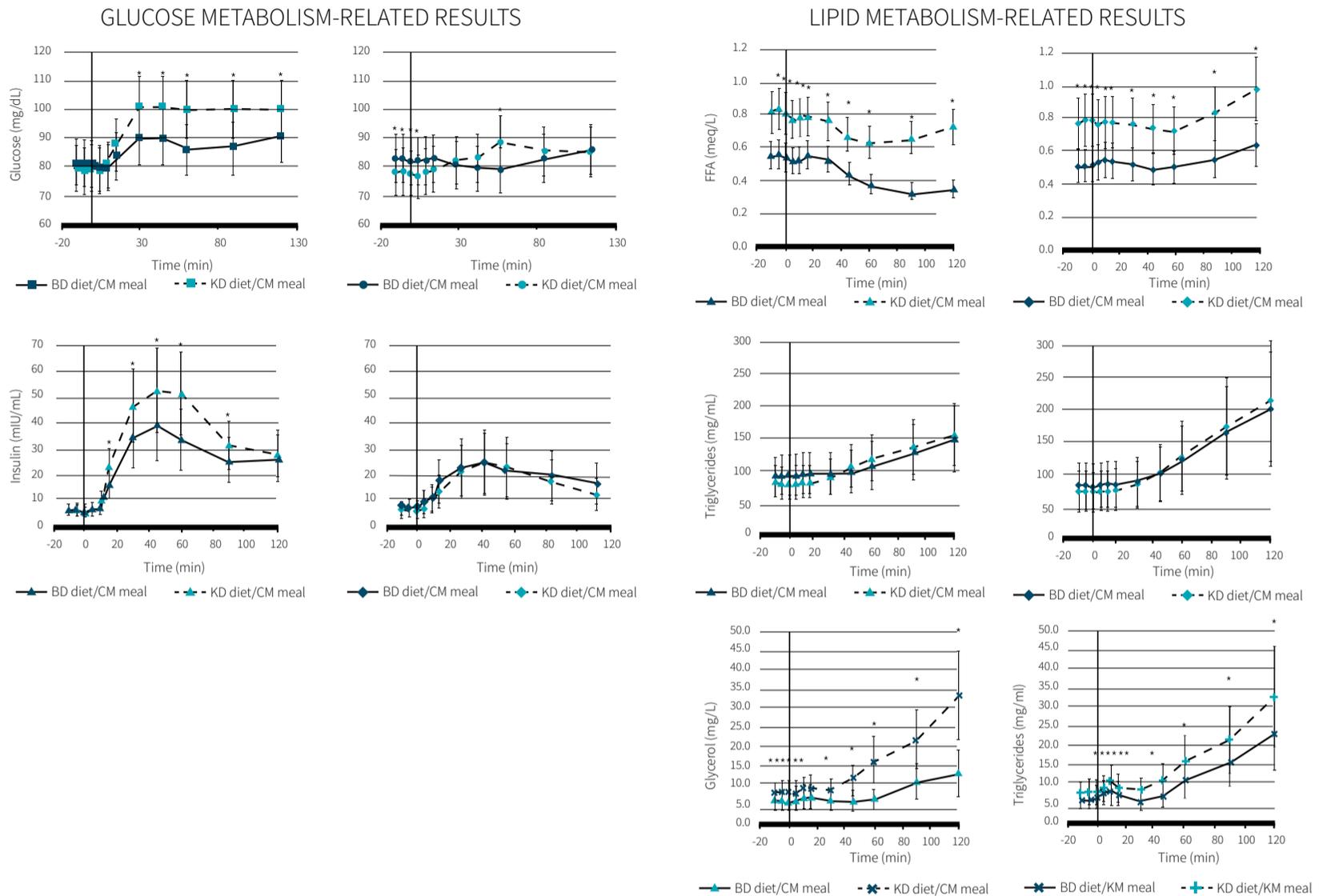
Participants in the ketogenic diet group were, on average, in mild ketosis for 8 of the 12 weeks, and at 8 weeks, there were no differences in fasting glucose between groups. The ketogenic group saw a greater reduction in insulin than the other groups but had considerably higher insulin at baseline, making it hard to know how it compared to the other diets. LDL and HDL cholesterol was unchanged in the keto group but reduced in the others. The reduction in triglycerides was greater for the ketogenic group. C-reactive protein was only evaluated at 12 weeks when the keto group was no longer eating a fully keto diet but a more moderate low-carbohydrate diet. The low-fat group saw a greater reduction than the ketogenic group, while the high unsaturated fat diet saw a smaller reduction, but it's unclear if any of this was a genuine effect of the dietary differences.

Another much smaller trial with 17 overweight/obese participants went to the greatest length of any study to ensure that the planned ketogenic diet was, in fact, ketogenic and calorically matched to the control (in this case, the baseline diet).²¹ Participants were confined to a metabolic ward, and all of their food intake was controlled by the researchers. Ketone levels were measured, and ketosis was confirmed. They underwent 4 weeks on a high-carbohydrate diet before switching to a ketogenic diet with the same number of calories and the same amount of protein but considerably less fiber. Although the study population was overweight/obese, their fasting glucose levels were well within the healthy range, so it's not surprising that there was no change in glucose levels on the ketogenic diet, but it did reduce insulin concentrations. This may imply that the reduced insulin concentrations seen in the first study we mentioned may indeed be genuinely due to the ketogenic diet. The ketogenic diet corresponded to increased c-reactive protein for the duration of the study and initially increased IL-6 (an inflammatory cytokine and cause of insulin resistance), but levels decreased slightly below baseline by the fourth week, again implying that there may be some inflammatory issues attributable to the keto diet.

The researchers also looked at metabolic outcomes in response to meal characteristics of both diets: a ketogenic meal and a high-carbohydrate meal were given to participants while on the high-carbohydrate diet and also when they were on the ketogenic diet. Compared with the baseline diet, the ketogenic diet saw greatly increased postprandial (postmeal) glucose levels in response to both meals, and much higher insulin after the high-carb meal but not after the ketogenic meal. The ketogenic diet increased the

clearance of free fatty acids from the bloodstream in response to the high-fat ketogenic meal. Triglycerides and glycerol were higher for the ketogenic diet during the keto meal, suggesting reduced clearance. Free-fatty-acid levels during fasting were also higher on the ketogenic diet.

Figure 13: Select glucose and lipid metabolism-related results



BD: baseline diet | CM: control meal | KD: ketogenic diet | KM: ketogenic meal

Adapted from Rosenbaum et al. *Obesity (Silver Spring)*. 2019. PMID:31067015

Additionally, a number of small, controlled studies found the following effects of a ketogenic diet:

- No apparent benefit to fasting glucose levels. However, since levels weren't very high, it doesn't indicate a lack of effect in those with higher levels.^{20,22,51,52,53}
- A moderate reduction in fasting insulin^{20,22,51,52,53}
- Generally higher LDL but larger particle size, and no difference in oxidized LDL in one study^{20,51,52,53}
- Somewhat higher HDL^{20,51,52,53}

- A reduction in triglycerides^{20,51,52,53}
- Lower systolic blood pressure in one study but not another, and no real difference in diastolic blood pressure^{51,53}
- Worse flow-mediated dilation in one study⁵¹
- No notable overall difference for c-reactive protein^{51,53,54}

Keep in mind, more high-quality research on each outcome is needed to know what the average expected effect will be.

Limited research failed to find a consistent reduction in fasting glucose for overweight/obese participants whose levels hadn't entered the prediabetic range or beyond, though a ketogenic diet coincided with lower fasting insulin. This doesn't indicate a lack of effect at higher glucose levels. Keto probably has an inherent triglyceride-reducing effect and can preserve HDL levels during low-calorie diets. LDL is generally increased by ketogenic diets, though particle size increased too. Only one study evaluated flow-mediated dilation and found that it was worse on a ketogenic diet than a low-fat diet. Once again, it's unclear if a ketogenic diet affected inflammation, but studies were more likely to find a modest increase.

What about when ketogenic diets are better for weight loss?

An ongoing study of a ketogenic diet for obese people with type II diabetes has published its 2-year follow-up findings.⁵⁵ Participants were educated about the ketogenic diet, and fewer than 30 g of carbohydrates was planned, with 1.5 g/kg of protein per day. Participants were encouraged to consume sufficient amounts of vitamins and minerals, with supplementation as needed. The control group didn't receive dietary counseling and continued their usual diet. Ketone levels were measured, and the ketogenic group, on average, maintained mild ketosis over 2 years.

There were 262 people on the ketogenic diet, to begin with, 218 of whom completed the first year, and 194 of whom completed the second year. After the first year, the ketogenic diet group lost a staggering 14.3 kg (31.5 lb) on average, from a starting point of 114.6 kg (252.7 lb), though there was no further weight loss in the second year and a little bit of weight gained back. The control group saw no notable changes in weight at any time. As keto participants' body weight changed, so did markers of type II diabetes, with a notable improvement in glycated hemoglobin, fasting glucose, and insulin and a reduction in

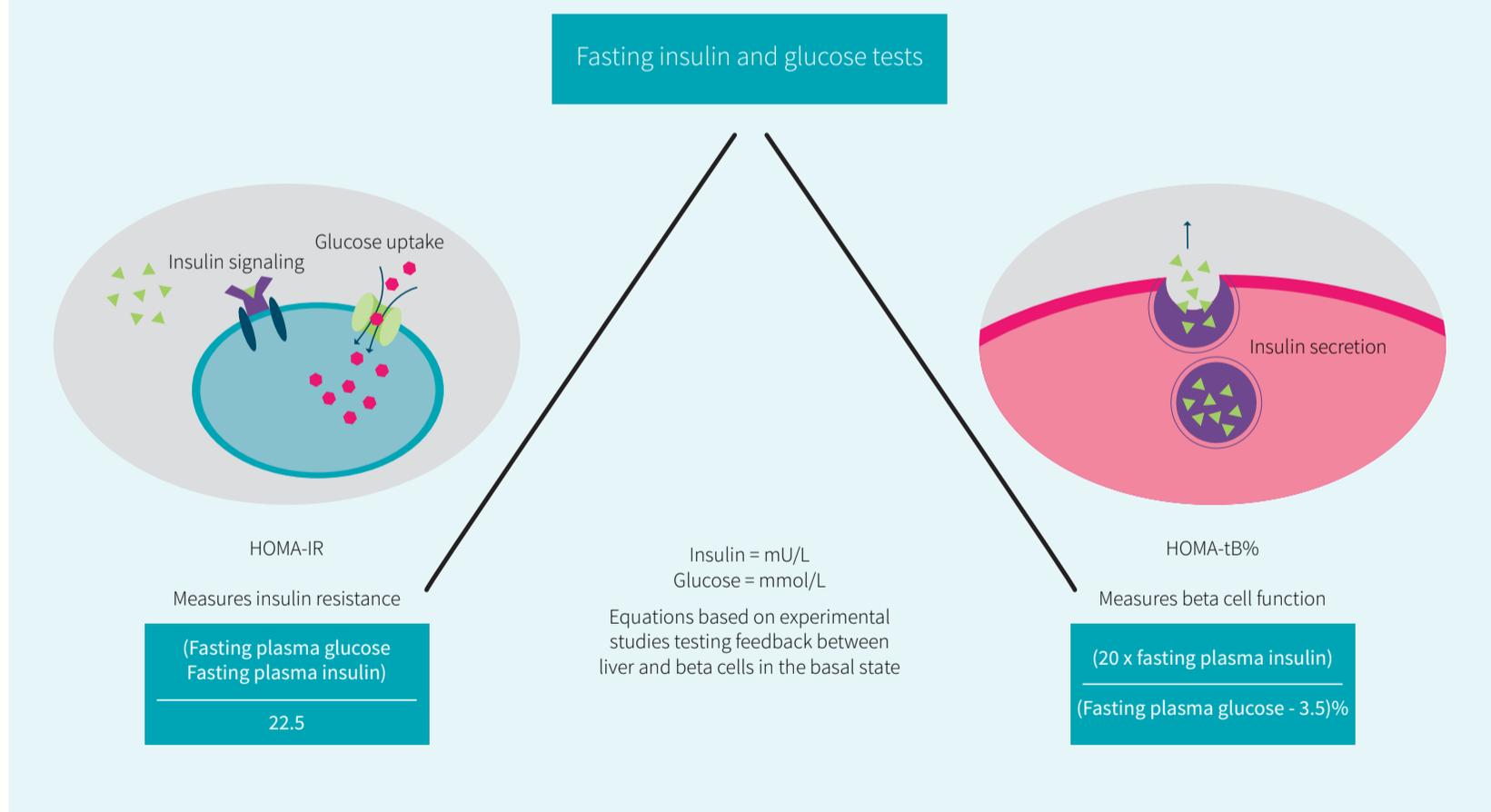
HOMA-IR — a formula that estimates insulin resistance using glucose and insulin levels — at one year compared with the control group. Blood pressure, c-reactive protein, HDL, and triglycerides improved, while LDL cholesterol increased in the ketogenic group and decreased in the control group. There were also some notable improvements in terms of liver health. In the second year, improvements generally stopped, and there was a slight worsening in most of them as they gained back a small amount of weight. Impressively, no instances of ketoacidosis or severe hypoglycemia were reported by the ketogenic group.

🔍 Digging Deeper: What is HOMA, and what does it measure?

[Insulin](#) is a hormone produced by the pancreas that increases when blood glucose (blood sugar) rises and acts to reduce glucose by putting it into cells and increasing its usage for energy production. [Insulin sensitivity](#) is a function of how well your body can handle glucose through insulin secretion. Relatively speaking, you are insulin sensitive if a small amount of insulin is needed to process blood glucose and insulin resistant if a larger amount is needed to process the same dose of glucose.

Insulin sensitivity is commonly measured in a few ways, each with their own cutoff values indicating insulin resistance. HOMA-IR is the Homeostatic Model Assessment of Insulin Resistance and is calculated using fasting glucose and insulin levels. Scores of ≥ 2 and ≥ 2.5 have been used to indicate the presence of insulin resistance.⁵⁶ HOMA-B assesses the health of the beta cells in your pancreas by indirectly measuring insulin secretion. Low HOMA-B values have been associated with an increased risk of developing diabetes.⁵⁷

Figure 14: Basic HOMA calculations



From this study, it can be said that 2 years on a ketogenic diet was safe and beneficial to the health of type II diabetics, most likely due primarily to the weight loss. On the other hand, this study doesn't tell us if a ketogenic diet is the ideal choice, because the control group didn't receive a rival dietary intervention nor intensive support from a healthcare team.

Various other studies where a ketogenic diet group lost more weight than the control group generally found:

- A consistent modest reduction in glucose levels^{58,59,60,61,62,63,64,65,66}
- A modest reduction in glycated hemoglobin from limited research^{61,64}
- Generally greater reductions in insulin^{58,59,60,61,63,64,65,66}
- A reduction in triglycerides^{58,59,61,63,64,65,66}
- A modest increase in LDL cholesterol but a shift toward a larger particle size and no apparent difference in oxidized LDL^{58,59,60,61,63,64,65,66}
- A small advantage when it comes to HDL^{58,59,60,61,63,64,66}
- No real difference in c-reactive protein, though the weight loss difference was only modest in these studies^{63,64}
- A reduction in blood pressure in one study, but it's not close to consistent across studies, and none measured flow-mediated dilation^{58,60,64,66}

When ketogenic diets lead to more weight loss than the control group, it's unsurprising that they often lead to improvements in metabolic outcomes, but the effects on inflammation and vascular function aren't clear from the research we have.

Even with greater weight loss, a ketogenic diet still tends to have a disadvantage when it comes to LDL cholesterol but a probable advantage when it comes to HDL and triglycerides. The results from the largest and longest study suggest that the benefits of a ketogenic diet probably depend largely on the amount of weight people lose while on it.

Type I diabetes

type I diabetes occurs when the pancreatic beta-cells are too damaged to produce sufficient insulin, usually due to an autoimmune problem. It's primarily managed through self-administration of insulin in combination with blood sugar testing and carbohydrate counting. Although insulin injections are a lifesaver, type I diabetes still increases the risk of cardiovascular disease.⁶⁷ Naturally, carbohydrate restriction sounds appealing as a way to better control blood sugar. Unfortunately, there isn't very much research on ketogenic diets and type I diabetes. But this may be changing.

One recent study randomized 10 type I diabetics using an insulin pump to a high-carbohydrate (≥ 250 g/day) or ketogenic (≤ 50 g/day) diet with the same number of

calories for 1 week before crossing over to the other diet.⁶⁸ The study was far too short to accurately assess glycated hemoglobin levels, and unsurprisingly, there was no difference between the diets. There was also no difference in the average glucose levels, as measured by the glucose monitor, but the ketogenic group had less variability, spent more time in euglycemia (normal blood sugar), and spent less time in hypoglycemia or hyperglycemia, though the last one wasn't statistically significant. Overall, the ketogenic group required considerably less insulin. Other notable findings were higher levels of FFAs and glucagon in the keto group. There was no notable effect on lipid profile or blood pressure.

Besides that, there are reports of favorable effects of ketogenic diets on blood sugar when used long term by people with type I diabetes.^{69,70} While not controlled, these reports suggest low HbA1c. It's possible that some of this effect is merely due to reduced energy intake, but it's plausible that by cutting down on carbohydrates, overall glycemia is reduced. When it comes to overall health, there doesn't seem to be any clear red flag besides the usual elevated LDL-C, but more research is needed on the long-term effects, including complications and important health outcomes. There are reports of more instances of hypoglycemia, as defined by measurements of low blood glucose, but interestingly, participants self-reported it less frequently. The likely explanation is that, by providing energy for the brain, ketones prevent symptoms of hypoglycemia. This poses the question of whether ketogenic diets can prevent the adverse effects of hypoglycemia. While this is an intriguing idea, much more research is needed to verify the effects.

Not a lot can currently be said about ketogenic diets for type I diabetes, except that it's plausible that they can help keep blood sugar more stable and possibly reduce HbA1c. The overall effects on health in the long term are unknown, and more research is needed. Protection against the adverse effects of hypoglycemia by the use of ketones as fuel for the brain is possible, but requires more high-quality research to verify.

Commonsense keto

Some people treat a ketogenic diet as some sort of mythical diet unicorn: as long as your carbohydrate intake remains low, you'll be golden. From what we've seen, that's not accurate. Although the dietary variability in ketogenic diets is smaller than in many other kinds of diets, there are still many things that can differ between ketogenic diets. Commonsense keto would mean treating it like a diet with any other macronutrient composition: everything else still matters.

As has been mentioned, fat clearance after meals can have implications for cardiovascular health. Arteries may be more protected when a number of factors are present.

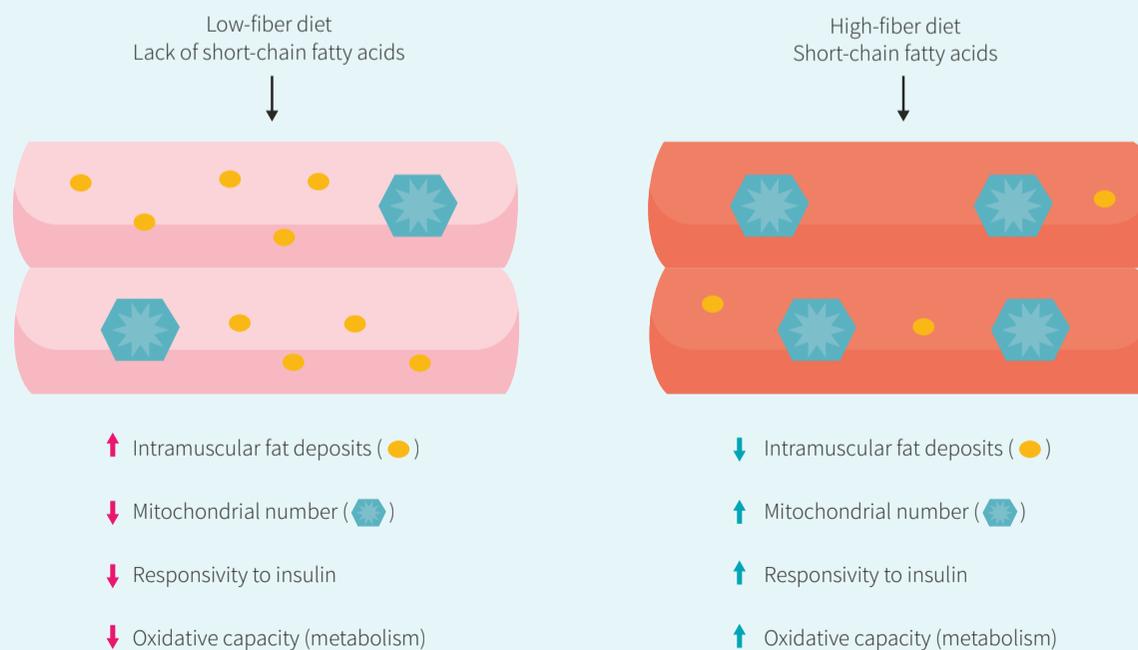
- Exercise after a meal protects arteries.^{71,72,73,74} Exercise is also good for insulin sensitivity and important regardless of diet.
- Fiber with a meal may be helpful.⁷⁵
- Omega-3 fatty acids may have a protective effect.⁷⁶
- Palmitic acid (the 16-carbon saturated fatty acid) seems to be more liable to cause insulin resistance than oleic acid (the major dietary monounsaturated fatty acid), which seems to have a protective effect.⁷⁷ The implication is that it's probably wise not to rely too heavily on animal fats or palm oil on a ketogenic diet and to vary fat sources.

Due to the elimination of grains and most legumes, ketogenic diets tend to be lower in dietary fiber than most other diets. As previously mentioned, dietary fiber can improve insulin sensitivity and is a general boon to people with elevated glucose levels. Some types are also quite good at improving lipid profiles; psyllium and beta-glucan supplements, in particular, may be a wise choice to compensate for the lack of dietary fiber and offset the increase in LDL cholesterol commonly seen on high-fat diets.^{78,79,80,81,82}

🔍 Digging Deeper: Fiber, the gut, and glucose

Fiber isn't just a component of food that slows the absorption of carbohydrates into the bloodstream. The more fermentable (able to be consumed by the bacteria in our guts) fiber is, the more short-chain fatty acids it will produce. These short-chain fatty acids have direct and indirect effects on metabolic and cardiovascular health.^{83,84}

Figure 15: Proposed way whole grain fiber benefits insulin sensitivity



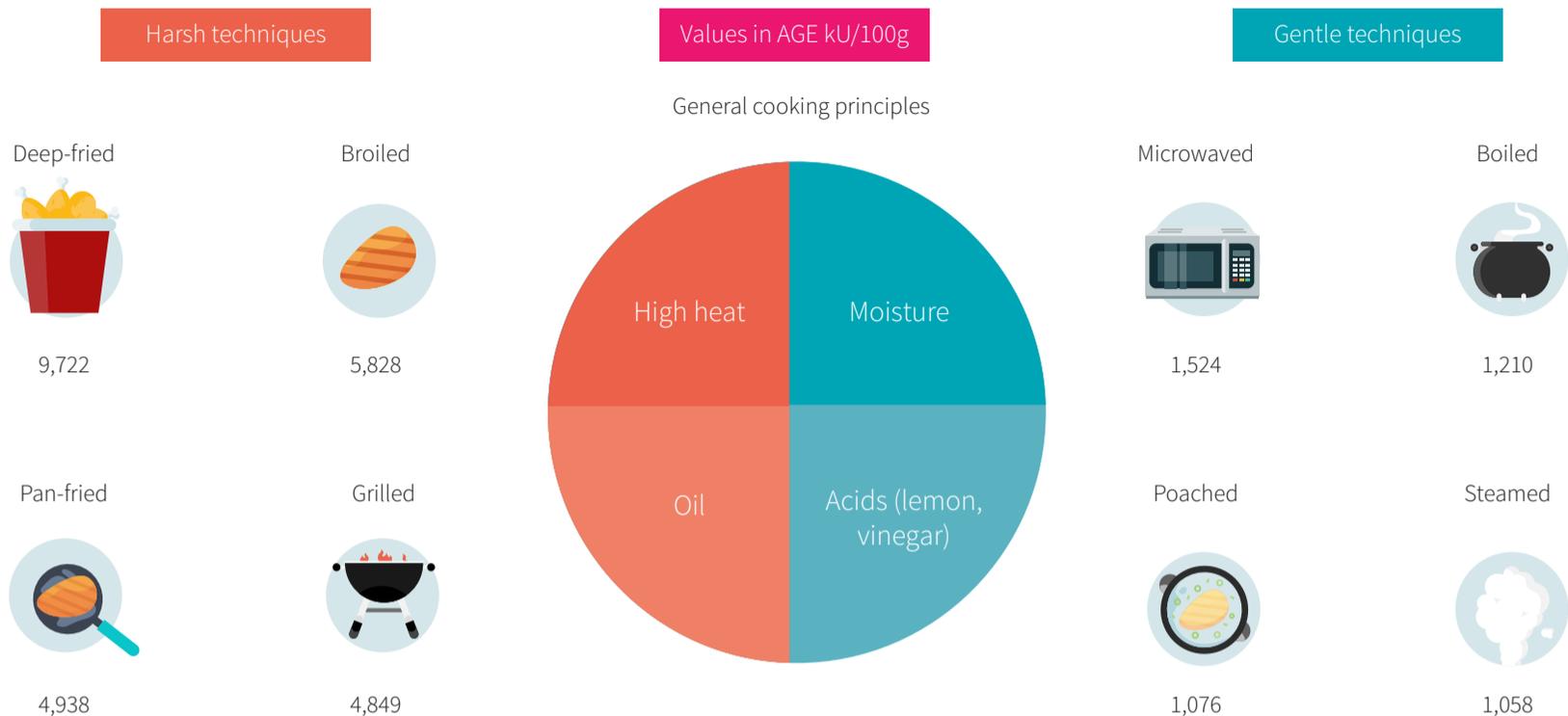
Adapted from McNabney and Henagan. *Nutrients*. 2017. PMID:29231905

They have a regulatory effect on inflammatory signaling by improving the function of the gut barrier, thus preventing inflammatory compounds from getting into the bloodstream; improving insulin sensitivity; and increasing the rate of fatty acid oxidation. A lack of dietary fiber on a ketogenic diet is one plausible reason for the higher c-reactive protein levels found in some studies, and fiber supplementation could even help curb increases in LDL.

Dietary fat, in particular, seems to be a catalyst for the absorption of lipopolysaccharides, which are harmful to the body and evoke an inflammatory response. One study compared the effects of glucose, cream, and orange juice on a variety of markers of inflammation after consumption.⁸⁵ Cream and glucose both led to notable increases in inflammation in the post-meal period, but interestingly, orange juice didn't. Also, cream led to an increase in lipopolysaccharides, but the others didn't, suggesting that fat is unique in that regard. In another study, pairing orange juice with a high-fat meal seemed to blunt some of the increase in lipopolysaccharides, inflammation, and oxidative stress.⁸⁶ A fruit extract containing resveratrol and other phytochemicals also seemed to suppress the negative effects of a high-fat meal in yet another study.⁸⁷ One possible takeaway is that the phytochemicals from plants are a good thing to consume with high-fat meals, yet a ketogenic diet can often be very low in plant foods, and it's unclear if low-carb vegetables have the same effects. When it comes to lipopolysaccharides, fermentable fiber is indeed protective, so this is another reason supplementation may be wise.

Ketogenic diets technically don't have to be high in meat, but they tend to be. Although it's still unclear how much it matters to health, the way meat is cooked may influence its healthfulness, particularly for people with metabolic syndrome. In general, high-heat cooking tends to produce more compounds suspected of being harmful to the body than low-heat cooking, and some human trials suggest that this may matter for cardiometabolic health.^{88,89} In general, cooking meat and other foods with as little browning and blackening as possible, while still practicing food safety principles, is healthier.

Figure 16: Carboxymethyl-lysine content of chicken breast by cooking technique



Reference: Uribarri et al. *J Am Diet Assoc.* 2010. PMID:[20497781](https://pubmed.ncbi.nlm.nih.gov/20497781/)



Tip: Nutrient tracking is essential

As mentioned earlier, highly restrictive diets can put people at risk for nutrient deficiencies. A keto diet can be simple to follow: just eat meat, eggs, and fats, and go easy on the higher-carb vegetables, and you're on a keto diet. But to get it right, tracking nutrient intake and careful dietary planning are essential.

Ultimately, we want to know what makes for the best possible ketogenic diets, and how those approaches compare to the best possible high-carbohydrate alternatives. There is probably no such thing as a perfect diet, especially when there are multiple health concerns, as with metabolic syndrome. But there's much more research underway that will shed light on when specifically keto may be uniquely beneficial.

Chapter 13: Warnings

Summary

Before starting a ketogenic diet, there are a few things safety considerations you should learn about. Additionally, you can face some hurdles as you start and maintain a ketogenic diet. This chapter provides guidance on how to navigate and manage these as they arise.

- First and foremost, you will want to decide if a ketogenic diet is appropriate and safe for you, given your health status and history. There are cases where a keto diet can cause harm and other scenarios where **medical supervision may be warranted**. Take these into consideration before beginning the diet.
- You may want to consider monitoring certain health parameters via blood tests. Monitoring is not mandatory, but you should have a conversation with your physician to see which blood tests may be appropriate for you.
- Both drugs and supplements can contain “hidden” carbohydrates used as fillers, stabilizers, or sweeteners that may not be readily disclosed on the label. Half the battle is knowing what to look for, so we have provided a table of common carbohydrate names that may appear on labels.
- The keto diet eliminates a fair number of foods and food groups, which can make it challenging — but not impossible — to meet all your nutrient needs. **The nutrients that may drop below recommended levels** on a keto diet include [calcium](#), [fiber](#), [folate \(B₉\)](#), [iodine](#), [iron](#), [magnesium](#), [potassium](#), sodium, [thiamine \(B₁\)](#), [vitamin A](#), [vitamin C](#), and [vitamin D](#). These may need to be monitored to ensure you get adequate intake through food or supplementation.
- **In the first 1–4 weeks of trying the keto diet**, you may experience fatigue, nausea, bad breath, intestinal discomfort, brain fog, or other various ailments. This collection of symptoms is commonly referred to as the “low-carb flu” or “keto flu”. Luckily, they are often temporary, and we tell you what steps to take to help prevent or alleviate some of these unwanted side effects.

Why does keto warrant safety considerations?

While it's not a great idea to just jump headfirst into any new diet, the keto diet in particular requires a bit more planning before starting. As a rule of thumb, the more foods and food groups a diet restricts, the more attention to detail is required to prevent insufficient nutrient intake and unwanted side effects or adverse events.

In this chapter, we're going to walk you through considerations you should understand when starting a keto diet to help keep it safe and enjoyable. We'll also give you guidance on how to manage some of the common side effects of the diet.

⚠ **Warning: An important note**

First, speak with your physician before starting the keto diet.

Second, the warnings herein are aimed at people who have not been prescribed a ketogenic diet for medical conditions, such as epilepsy. Medically directed keto diets often require more specialized considerations and monitoring and are outside the scope of this guide.

When is keto not recommended?

There are specific conditions where the ketogenic diet may not be appropriate, as its use may cause harm.^{1,2} Please take note of these before beginning a ketogenic diet.

- **Absolute contraindications.** If you have any of these conditions (many of which are identified in early childhood), then a keto diet — of any type — just isn't for you.
- **Possible contraindications.** If you have any of these conditions, then a keto diet may be contraindicated, but you might still be able to start one — under medical supervision. Talk to your physician first.

ABSOLUTE CONTRAINDICATIONS	POSSIBLE CONTRAINDICATIONS
Carnitine palmitoyltransferase I or II deficiency	Cancer (a keto diet may alter treatment efficacy or cause possibly undesirable weight loss)
Carnitine translocase deficiency	Concurrent use of propofol (a keto diet may increase the risk of propofol infusion syndrome)
Fatty acid beta-oxidation defects	Diabetes (medication adjustment/monitoring may be required; see below the table for more information)
Long-chain 3-hydroxyacyl-CoA deficiency	Dysphagia (difficulty swallowing)
Long-chain acyl-dehydrogenase deficiency	Eating disorders
Medium-chain 3-hydroxyacyl-CoA deficiency	Familial hyperlipidemia (a genetic disorder that increases blood lipids)

Medium-chain acyl-dehydrogenase deficiency	Gallbladder disease or no gallbladder (because of issues with digesting fat)
Organic acidurias	<i>Gastroesophageal reflux disease (GERD)</i>
Porphyria	Gout
Primary carnitine deficiency	History of kidney stones
Pyruvate carboxylase deficiency	Hypertension (high blood pressure; medication adjustment/monitoring may be required)
Short-chain acyl-dehydrogenase deficiency	If 18 years old or younger (or still growing, as a general rule)
	Kidney disease or failure
	Liver disease or failure
	Multiple food allergies
	Pregnancy or breastfeeding
	Received bariatric surgery (because of possible issues with digesting fat)

There are situations where undertaking a keto diet is not advisable and may even cause harm to your health. Before beginning, check the list above and consult your physician if you have any concerns or questions.

Do you need to monitor health biomarkers?

Some of the best data we have on the short- and long-term effects of the keto diet come from children with epilepsy, a neurological disorder that can cause seizures. For these kids — and some adults — the keto diet may be medically prescribed to help decrease seizure frequency, particularly if drug therapies have failed to do so.

There is some overlap between medically prescribed keto diets and those commonly used for nonmedical purposes, but there are also important differences. Notably, a medical keto diet can include the following.

- Very strict limits on total carb intake
- Somewhat restrictive protein intake
- Intake rules on which types of fat are eaten

By contrast, the style of nonmedical keto diets more popular to the general public look more like this.

- Relatively more flexible total carb intake
- Not particularly restrictive protein intake
- Which types of fat are eaten is left to personal discretion

The list below is one used for monitoring the health parameters of children on a medically supervised keto diet expressly for the treatment of epilepsy. Do you *have* to get all these tests? Probably not — in fact, certainly not. This list just gives you a starting point of things to be aware of. Keep in mind that the very long-term (>2 years) effects of a keto diet on overall health status have not been well-studied (though more research is coming), so some level of monitoring may be prudent. Any concerns you have and questions about testing are best addressed with your healthcare provider.

Table 1: Recommended blood testing during medical ketogenic dietary therapy for the treatment of epilepsy

ESSENTIAL	RECOMMENDED	OPTIONAL
Calcium, phosphate	LDL and HDL	Clotting screen
Cholesterol and triglycerides	Uric acid	Copper
Free and acylcarnitine profile		Ferritin
Full blood count with platelets		Folate
Glucose and HbA1c		Vitamin B12
Liver profile		Vitamin C
Magnesium		
Renal profile*		
Selenium		
Vitamins A, D, E		

* Includes sodium, potassium, urea, creatinine, bicarbonate, and albumin

Adapted from Neal. Monitoring and Side Effects (Chapter 18 of Dietary Treatment of Epilepsy: Practical Implementation of Ketogenic Therapy, ed. Neal. 2012). <https://doi.org/10.1002/9781118702772.ch18>

The very long-term effects (>2 years) of a keto diet are a bit of a mystery, so some active monitoring of health markers may be needed. You certainly will not need all of the above-mentioned tests, which are intended for children with epilepsy who are undergoing medical ketogenic diet therapy. But this list can serve as a reference point when talking over your concerns with a healthcare provider.

Medications

Hidden carbs

Medication issues don't always involve the medication's active ingredients. For example, keto dieters aren't always aware of hidden carbs in medications.

Be aware that drugs — prescription or over-the-counter — can use carbs as fillers, stabilizers, or sweeteners, and these amounts may not be readily disclosed on the label.³ In some cases, carb contents may be high enough to kick you out of ketosis. Among the worst offenders are chewable and liquid medications, which may contain several grams of carbs per serving.^{4,5} In many cases, these medications can also be found in tablet or capsule form, which tend to have a lower carb count.



Tip: Do I need to care about carbs in drugs?

This really depends on your situation. Here are three different scenarios you may encounter.

You were medically prescribed a keto diet.

There's a good chance you need your ketone levels to stay above a certain threshold, and keeping your carb intake very low will be important for this. You must ensure the drugs you take don't contain enough carbohydrates to kick you out of ketosis.

You were *not* medically prescribed a keto diet but take certain drugs regularly.

If you opted to go on a keto diet, staying in ketosis may not be critical, but it could be important to you. So, if you take any drugs regularly, check their carbohydrate content. If it's high, check with your physician to see if alternatives are available.

You were *not* medically prescribed a keto diet and only take drugs occasionally.

The amount of attention you pay to the carb content of drugs in this scenario really depends on how important staying in ketosis is to you. If you take drugs only occasionally and have no medical reason for being on the diet, it may not be worth the extra time and effort to fish for this information.

Whatever you decide, **if you are taking drugs that have been prescribed, do not stop taking them before speaking with your physician.**

It may be difficult to obtain the exact carbohydrate content of certain drugs, but here are a few options when trying to do so.

- **Drug package inserts:** This usually comes with the drug packaging, but most are also readily available online. They list what type of carb is in the medication, if any, but don't regularly list the exact carb count.
- **Medical databases:** If you do not have access, your physician most likely does. Check to see if they can find an up-to-date list in one of these databases.
- **Manufacturer's website:** If you can't find carb information anywhere on their site, emailing or calling them may be useful.
- **Assume the inactive ingredients are carbs:** This is more of a conservative workaround than an exact science. All drugs list the exact amounts of active ingredients. Subtract this number from the weight of the drug and assume the difference is all carbohydrates. For example, a 1,000-milligram (mg) pill may contain 100 mg of the active ingredient. In this case, you would then assume the other 900 mg (0.9 g) is carbohydrate.

A word to the wise: **sugar-free does not mean carbohydrate-free!** These products may still contain other carbs, such as sugar alcohols, which are simply another class of sweeteners (sugar substitutes) that contain fewer calories per gram compared with glucose or table sugar.⁶ The six sugar alcohols most used as sweeteners, and their calorie content, are listed below.

Table 2: Calorie content of sugar alcohols

SWEETENER	CALORIES (KCAL/G)
Glucose or sucrose (table sugar)	4.0
Erythritol	0.2
Lactitol	2.4
Maltitol	3.0
Mannitol	1.6
Sorbitol	2.6
Xylitol	3.0

Reference: Chattopadhyay et al. J Food Sci Technol. 2014. PMID:[24741154](https://pubmed.ncbi.nlm.nih.gov/24741154/).

Drugs can contain carbs that are often not disclosed on the label. Typically, chewable and liquid medications have a higher carb content. However, unless you have been medically prescribed a keto diet, you probably don't need to be concerned with this. Do *not* stop taking a prescribed drug simply because it has some carbs. Speak with your physician if you wish to switch to a lower-carb version.

Supplements

Carb content

As with drugs, supplements may contain undisclosed carbs as fillers, stabilizers, or sweeteners. Keep an eye out for the below ingredients in your dietary supplements. Additionally, ingredients ending in “-ose” or “-ol” — excluding cellulose, an indigestible fiber — are typically carbohydrates. Remember, ingredients must be listed from most to least abundant, so you can get a general idea of how many carbs a product contains, but only if you can guess the amounts of the surrounding ingredients. The closer to the beginning of the ingredient list a carb is, the more carbs that product may contain, and vice versa.

Table 3: Names of carbohydrates on food and supplement labels

Agave nectar or syrup	Demerara sugar	Grape sugar	Polyglycerol
Arabitol	Dextrin	High-fructose corn syrup (HFCS)	Polysaccharide
Barley malt	Dextrose	Honey	Raw sugar
Beet sugar	Diastatic malt	Hydrogenated starch hydrolysate (HSH)	Refiner's syrup
Blackstrap molasses	Disaccharide	Icing sugar	Ribitol
Brown rice syrup	Dulcitol	Iditol	Ribose
Brown sugar	Erythritol	Invert sugar	Rice malt
Buttered sugar/ buttercream	Ethyl maltol	Isomalt	Rice syrup
Cane juice crystals	Evaporated cane juice	Lactitol	Saccharose
Cane sugar	Florida crystals	Lactose	Sorbitol
Caramel	Fructose	Malt syrup	Sorghum syrup
Carob syrup	Fruit juice	Maltitol	Sucanat
Castor sugar	Fruit juice concentrate	Maltodextrin	Sucrose

Coconut nectar	Galactose	Maltose	Sugar (granulated or table)
Coconut sugar	Glucose	Mannitol	Sweet sorghum
Confectioners' sugar (powdered sugar)	Glucose syrup solids	Maple syrup	Threitol
Corn syrup	Glycerol	Molasses	Treacle
Corn syrup solids	Glycol	Monosaccharide	Turbinado sugar
Crystalline fructose	Golden sugar	Muscovado sugar	Xylitol
Date sugar	Golden syrup	Panela sugar	Yellow sugar

And remember, **sugar-free does not mean carbohydrate-free!** These products may still contain other carbs, such as sugar alcohols.

Tangential to supplements, self-care products like mouth rinses and toothpaste can also contain some carbs. However, this is likely only of concern to those with medically prescribed keto diets who must keep their carb intake very low.

Carbs can go by many names when listed on food and supplement labels. Check the labels of all the supplements you are taking. You may be unknowingly ingesting carbs!

Supplements and blood glucose interactions

People with diabetes should be aware of the effects some supplements may have on blood glucose, particularly when paired with a keto diet. While low blood sugars (i.e., [hypoglycemic events](#)) have not been strongly linked to the supplements listed below, many simply lack long-term trials that track this outcome. It is possible these supplements may cause hypoglycemia, so some caution is warranted, particularly when taken with medications that can lower blood sugar, such as [antidiabetic drugs](#). **If you have diabetes or prediabetes and are taking any of these supplements, suddenly and drastically dropping your carb intake may put you at greater risk of hypoglycemia.** Talk to your physician to see if they want you to stop or continue using them before attempting a keto diet.

- [Berberine](#)
- [Chromium](#)
- [Cinnamon](#)
- [Creatine](#)
- [Inositol](#)
- [Magnesium](#)
- [Salacia reticulata](#)
- [Zinc](#)

If you have diabetes or prediabetes, some supplements may put you at greater risk of low blood sugars (i.e., hypoglycemia) when paired with a keto diet or antidiabetic drugs. It is advisable to talk to your physician about stopping or continuing these supplements as you transition to a keto diet. They can be reintroduced, slowly, after your body has had time to adjust to its new low-carb regimen.

Nutrients you should monitor

Net carbs

While wandering the grocery aisle, you may have seen some food labels displaying “net carbs”, “impact carbohydrate”, or “digestible carbohydrate” on their labels. Let’s see why and when net carbs might matter for keto dieters.

What is a net carb?

If you look at a food label, you’ll notice that fiber is included as part of the total carbohydrate count. But our bodies can’t absorb fiber and the calories it contains. For example, if you eat a sandwich that has 25 grams (g) of carbs but 5 g is fiber, then the amount of carbohydrate available for absorption by your body is 20 g.

Enter the term “net carbs”, which is a simple way to split out non-fiber carbs: net carbs equals total carbs minus fiber. The problem is that the term isn’t regulated by the U.S. Food and Drug Administration (FDA), and companies have been cited in the past for providing inaccurate carbohydrate counts when using the net carbs label.⁷ For example, some companies opt to subtract sugar alcohols from total carbs as well, even though they can still provide some calories (≈ 2 calories per gram).

Beware of *isomalto oligosaccharide* (IMO), though. This ingredient, common notably in protein bars, was once thought to be fiber, since it dissolves neither in the mouth nor

Figure 1: Fiber on a nutrition facts panel

Nutrition Facts	
8 servings per container	
Serving size	2/3 cup (55g)
Amount per serving	
Calories	230
% Daily Value	
Total Fat 8g	10%
Saturated Fat 1g	5%
Trans Fat 0g	
Cholesterol 0mg	0%
Sodium 160mg	7%
Total Carbohydrate 37g	13%
Dietary Fiber 4g	14%
Total Sugars 12g	
Includes 10g Added Sugars	20%
Protein 3g	

in the stomach. It later surfaced, however, that IMO gets mostly absorbed in the small intestine, providing 2.7–3.3 calories per gram (compared with 4 kcal/g for fully digestible carbohydrates).^{8,9,10}

What are the benefits of counting net carbs?

For keto dieters, it may be better to count net carbs, because counting fiber as part of your total carb intake may have some inadvertent negative effects such as causing you to underconsume fiber and further restricting your food choices.

In a hypothetical scenario, where you **do** count fiber toward your carb allotment of ≈ 50 g per day, you may ingest 35 g from carbohydrates and the remaining 15 g from fiber — well under the Adequate Intakes (AIs) set for total fiber seen in the table below. Because you've been trying to keep your carb + fiber intake below 50 g/day, you have further restricted foods that may have increased your daily fiber intake, such as low-carb vegetables, seeds, and nuts.

In another hypothetical scenario, where you **do not** count fiber toward your carb allotment, ≈ 50 g of nonfibrous carbs can be consumed, and your daily fiber intake could subsequently increase to 25 g (still under the AI for males, but a lot closer).

Table 4: Adequate Intakes (AIs) for Total Fiber (g)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	—	—	—	—
7–12 months	—	—	—	—
1–3 years	19	19	—	—
4–8 years	25	25	—	—
9–13 years	31	26	—	—
14–18 years	38	26	28	29
19–30 years	38	25	28	29
31–50 years	38	25	28	29
51–70 years	30	21	—	—
>70 years	30	21	—	—

Reference: Institute of Medicine. *The National Academies Press*. 2005. DOI:[10.17226/10490](https://doi.org/10.17226/10490)

As we will discuss in the [fiber](#) section below, low-carb and keto dieters are more likely to underconsume this nutrient. So counting net carbs may allow you a bit more dietary freedom and more opportunities to increase fiber intake. A win-win!

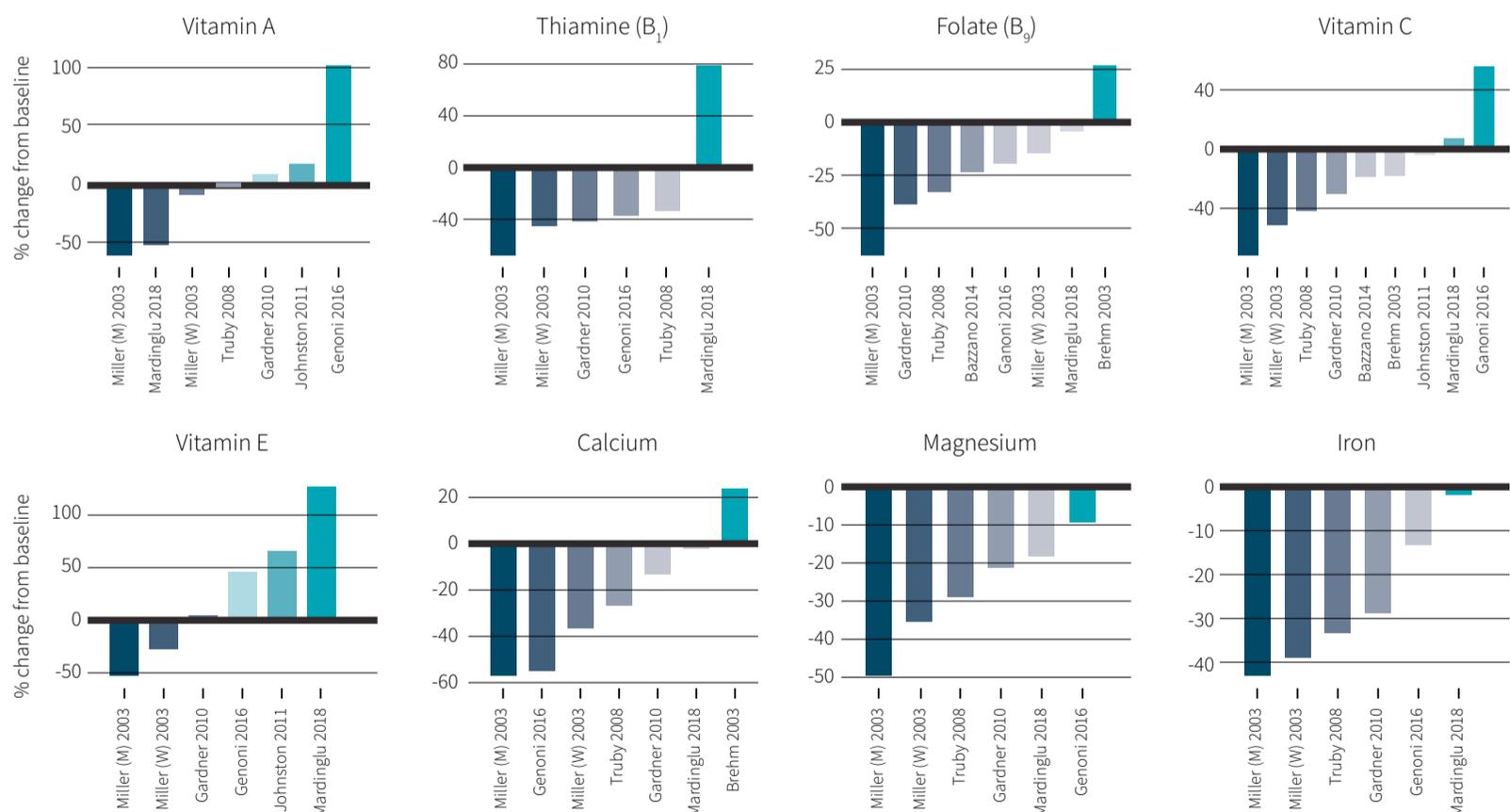
Net carbs are simply the total carbohydrate content of a food serving minus the indigestible fiber content. For those on a keto diet, counting net carbs rather than total carbs may allow for a little more food flexibility and help increase overall daily fiber intake.

Low nutrient intakes

Diets that restrict many foods or food groups — keto, vegan, gluten-free, paleo, low calorie — make it more challenging to meet all your nutrient needs.^{11,12,13,14,15} When it comes to keto, it is possible to meet nearly all your vitamin and mineral requirements with food,¹⁶ but it does require a fair amount of effort and attention to dietary planning.

Even the best-formulated diet plans could use a little help sometimes. In these cases, supplementation can help fill nutrient gaps. In the sections below, we'll cover nutrients that are commonly underconsumed while on a keto or low-carb diet and show you which foods or supplementation protocols may be used to help ensure sufficient intake. A list of low-carb foods rich in the nutrients discussed below can be found in [Appendix B](#).

Figure 2: Changes in micronutrient intakes in carb-restricted diets*



* Percentage changes from baseline intakes at the end of each study by micronutrient.

Adapted from Churuangasuk. *Obes Rev.* 2019. PMID:[31006978](#)

⚠ Warning: Don't overload on vitamins and minerals

High-dose, long-term consumption of any supplement can potentially interact with your biochemistry in unexpected ways. If you plan to supplement with vitamins or minerals on a keto diet, try not to exceed the Tolerable Upper Intake Levels (UL) from both food and supplement sources. More is not always better!

You can find UL tables here for [vitamins](#) and [minerals](#).

Any diet pattern that limits many foods or food groups can increase the risk of underconsuming important nutrients. While a keto diet can meet all your vitamin and mineral needs, it does take some thought and planning. Where practical, supplements may be used to help fill any nutrient gaps.

B Vitamins

The B vitamins [thiamine](#) (B₁) and [folate](#) (B₉) may be underconsumed on a reduced-carb diet. Thiamine is involved heavily in glucose production, so ensuring sufficient intake is very important, particularly if you are trying to improve your glucose control.

Thiamine supplements are frequently available in doses well above the RDA — commonly in the 100–300 mg range. If you supplement with thiamine while on a keto diet, aim for 50% of the RDA.¹⁷ This would be about 0.6 mg for male adults and 0.5 mg for female adults who are not pregnant or lactating. However, finding supplements that provide these doses may be difficult. As a backup plan, find the lowest dose supplement you can that also puts you at or below the RDA. If inclined, you can cut the supplement tablet in half to help reduce your dose.

When using thiamine at these doses, timing does not matter much; you can take one dose in the morning or divide your doses throughout the day. Thiamine does not need to be taken with food to be absorbed.

A list of low-carb foods rich in thiamine can be found in [Appendix B](#).

Table 5: Recommended Dietary Allowances (RDAs) for Thiamine (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	0.2*	0.2*	—	—
7–12 months	0.3*	0.3*	—	—
1–3 years	0.5	0.5	—	—
4–8 years	0.6	0.6	—	—

9–13 years	0.9	0.9	—	—
14–18 years	1.2	1.0	1.4	1.4
19–50 years	1.2	1.1	1.4	1.4
>50 years	1.2	1.1	—	—

* Adequate Intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 1998. DOI:[10.17226/6015](https://doi.org/10.17226/6015)

Folate is the essential vitamin sometimes referred to as Vitamin B₉, but more commonly known by its synthetic supplemental form, folic acid.¹⁷ Supplements tend to be in one of three forms; folate, folic acid, or L-methylfolate. Of these, folic acid is the most common.

When supplementing with **folate**, take 200–320 mcg (200–320 dietary folate equivalent [DFE]) for male adults and female adults who are not pregnant or lactating.

When supplementing with **folic acid**, take 100–160 mcg (200–320 DFE) for male adults and female adults who are not pregnant or lactating.

When supplementing with **L-methylfolate**, take 7.5–10 mg a day.

A list of low-carb foods rich in folate can be found in [Appendix B](#).

Table 6: Recommended Dietary Allowances (RDAs) for Folate (mcg DFE*)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	65	65	—	—
7–12 months	80	80	—	—
1–3 years	150	150	—	—
4–8 years	200	200	—	—
9–13 years	300	300	—	—
14–18 years	400	400	600	600
>18 years	400	400	600	600

* Dietary folate equivalents (DFE)
1 mcg DFE =

- 1 mcg food folate
- 0.6 mcg folic acid from fortified foods or dietary supplements consumed with foods
- 0.5 mcg folic acid from dietary supplements taken on an empty stomach

Reference: Institute of Medicine. *The National Academies Press*. 1998. DOI:[10.17226/6015](https://doi.org/10.17226/6015)

⚠ Warning: High-dose B vitamins

When it comes to B vitamins, the scientific literature hints at some danger when consistently taken in high doses. An [observational study of 77,000 participants](#) showed that long-term supplementation with B₆ or B₁₂ increased lung cancer risk in current male smokers, especially in those supplementing with high doses of either vitamin. It is uncertain if other B vitamins may have adverse effects when taken in doses above the RDA for extended periods or in nonsmokers.

Calcium

[Calcium](#) is a macromineral due to the relatively large amounts required in the diet — at times exceeding a gram a day. It is predominantly found in dairy products and, to a lesser extent, some vegetables. Since calcium is one of the major mineral components of bone,¹⁸ a deficiency can lead to osteopenia and osteoporosis, two diseases characterized by dangerously low bone density. On reduced-carb diets, calcium intake typically ranges from 40 to 70% of the RDA for adults ages 19–70.¹⁵ Incorporating more calcium-rich, keto-friendly foods into the diet or considering supplementation to make up for any deficit would be prudent.

⚠ Warning: Consuming calcium above the RDA may be harmful

When consistently taken at levels above the Recommended Dietary Allowance (RDA), which is most likely to occur with the use of supplements, calcium may increase the risk of dangerously high levels of calcium in the blood (i.e., [hypercalcemia](#)). Additionally, some studies have linked the use of higher-dose calcium supplements to an increased risk of cardiovascular disease.^{19, 20}

Supplementing with calcium is best considered only after a dietary evaluation. Track what you eat for a week and compare the calcium content of your diet with the RDA for your gender and age. If, on average, you are getting less than 80% of your RDA, supplementation becomes a possibility, but you should first consider tweaking your diet.

A list of low-carb foods rich in calcium can be found in [Appendix B](#).

Table 7: Recommended Dietary Allowances (RDAs) for Calcium (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	200*	200*	—	—
7–12 months	260*	260*	—	—
1–3 years	700	700	—	—
4–8 years	1,000	1,000	—	—
9–13 years	1,300	1,300	—	—
14–18 years	1,300	1,300	1,300	1,300
19–50 years	1,000	1,000	1,000	1,000

51–70 years	1,000	1,000	—	—
>70 years	1,200	1,200	—	—

* Adequate intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 2011. DOI:[10.17226/13050](https://doi.org/10.17226/13050)

Fiber

Dietary requirements for [fiber](#) have been established via Adequate Intake (AI) levels.²¹ The AI for fiber can be thought of as the minimum amount needed to ensure nutritional adequacy. While the recommended intake for adults ranges from 21 to 38 g/day, the US average intake is just ≈ 16 g — 23–58% below the AI.²²

Table 8: Adequate Intakes (AIs) for Total Fiber (g)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	—	—	—	—
7–12 months	—	—	—	—
1–3 years	19	19	—	—
4–8 years	25	25	—	—
9–13 years	31	26	—	—
14–18 years	38	26	28	29
19–30 years	38	25	28	29
31–50 years	38	25	28	29
51–70 years	30	21	—	—
>70 years	30	21	—	—

Reference: Institute of Medicine. *The National Academies Press*. 2005. DOI:[10.17226/10490](https://doi.org/10.17226/10490)

Fiber intake may be low in reduced-carb diets.^{23,24} Getting sufficient fiber may help improve blood glucose levels^{25,26,27} and blood lipid profiles,^{28,29} and help ensure your microbiome and intestinal barrier remain healthy.³⁰

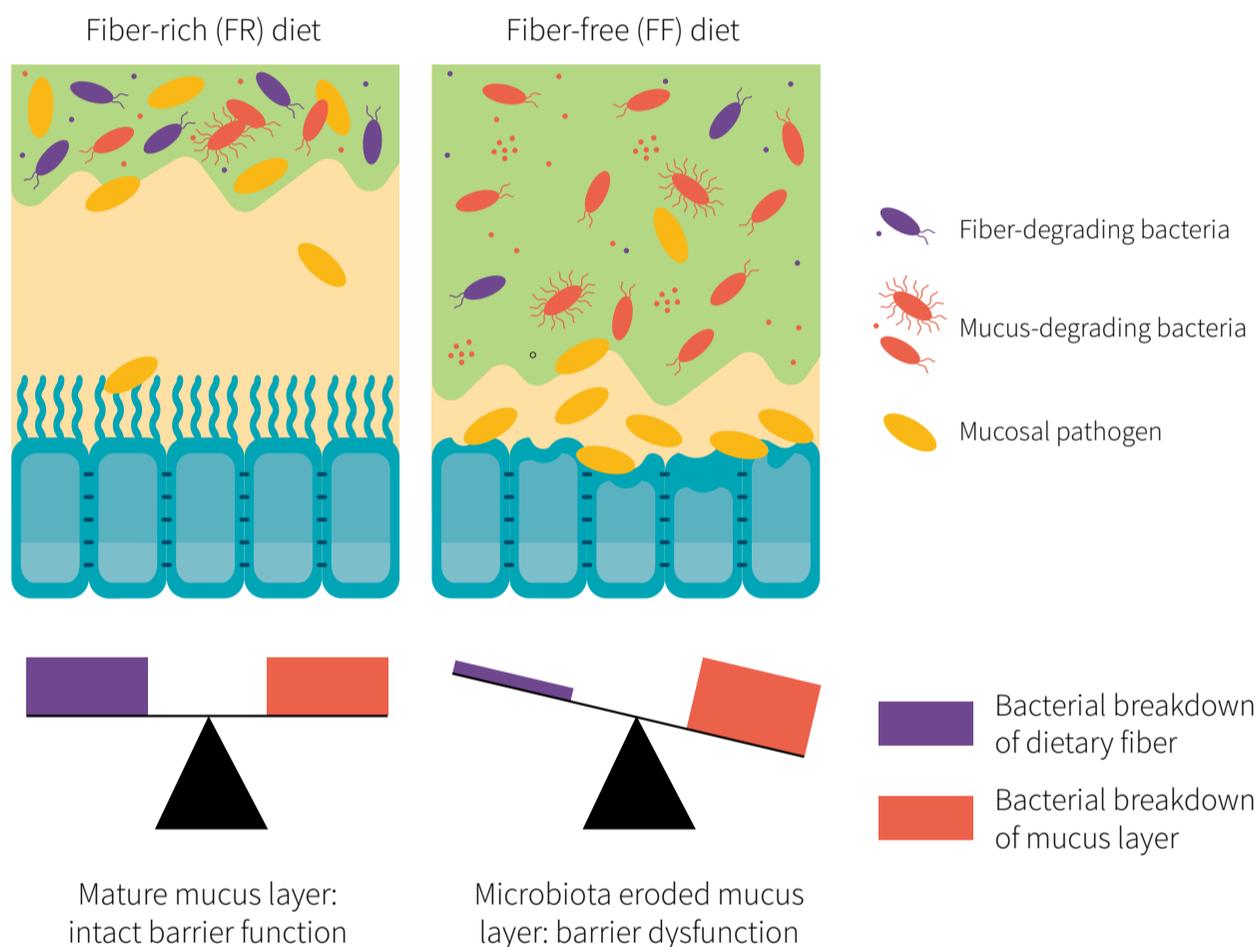
First try to obtain more fiber through foods, as isolated fiber supplements do not entirely replicate the health benefits obtained from fibrous foods. Focus on consuming more low-carb nuts, vegetables, and fruits.

A list of low-carb foods rich in fiber can be found in [Appendix B](#).

If you wish to add a fiber supplement, start slow and work your way up to 5–15 g/day. While fiber can be taken without food, it may be better tolerated and deliver better results when taken with food. If so, doses should be taken within 15 minutes before a meal — if not during — and split evenly across meals. Start with the lowest suggested dose to see how you react before increasing, if needed.

Psyllium may be a good option to start with. Due to its high viscosity, solubility in fluid, relatively neutral taste, and low degree of fermentation, this supplement is less likely to produce unwanted side effects, such as bloating. It has also been shown to produce very favorable effects on blood glucose control in those with elevated levels.³¹

Figure 3: Diet effects on the intestinal mucus layer



Adapted from Desai et al. *Cell*. 2016. PMID:[27863247](https://pubmed.ncbi.nlm.nih.gov/27863247/).

Regardless of where your fiber intake comes from (foods, supplements, or both), take the following steps to minimize unwanted side effects.

First, gradually increase your fiber intake over 1–2 weeks. This will allow your microbiome to adjust and help you identify your personal tolerance threshold. If taking a supplement, begin with 3–4 g a day for the first few days before increasing your dose.

Second, increase your fluid intake as you increase your fiber intake. Consuming fiber in the 40–70 g/day range can be generally well tolerated with sufficient fluid intake in healthy

adults without intestinal issues (e.g., IBD, IBS, celiac, Crohn's, ulcerative colitis, or low intestinal motility).²¹ Consuming at least 240 mL (8 oz) of fluids with 10 g of fiber should be enough, but individual results will vary. If you are particularly sensitive, drink 8 oz of fluid with every 5 g of fiber.

Third, split your fiber intake evenly across multiple meals to ease digestion.

Iodine

There is not much data on what occurs to iodine intake when dietary carbs are reduced, but what studies there are have shown consumption levels at 42% below¹⁴ to 18% above³² the RDA for adults, so iodine may need to be monitored on a keto diet.

For those who experience a drop in iodine intake, the exclusion of iodine-rich, higher-carb dairy products, such as milk³³ and some yogurts, may help explain it. Regular consumption of seaweed (no more than three grams per week) or iodized salt (at least 1.5 g/day) should be sufficient to ensure adequate iodine intake.³⁴ Due to [variability in seaweed iodine content](#), it's possible to inadvertently get too much or not much at all. The use of iodized salt will better ensure sufficient iodine intake.

A list of low-carb foods rich in iodine can be found in [Appendix B](#).

Table 9: Recommended Dietary Allowances (RDAs) for Iodine (mcg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	110*	110*	—	—
7–12 months	130*	130*	—	—
1–3 years	90	90	—	—
4–8 years	90	90	—	—
9–13 years	120	120	—	—
14–18 years	150	150	220	290
>18 years	150	150	220	290

* Adequate intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 2001. DOI:[10.17226/10026](https://doi.org/10.17226/10026)

Iron

Too little [iron](#) can cause [anemia](#), where blood does not carry enough oxygen, and extreme fatigue.³⁴ Premenopausal women are especially at risk, since they lose iron monthly during menstruation, as are vegetarians (and vegans even more so), since the iron in vegetable products is less easy for the body to use than the iron in animal products.

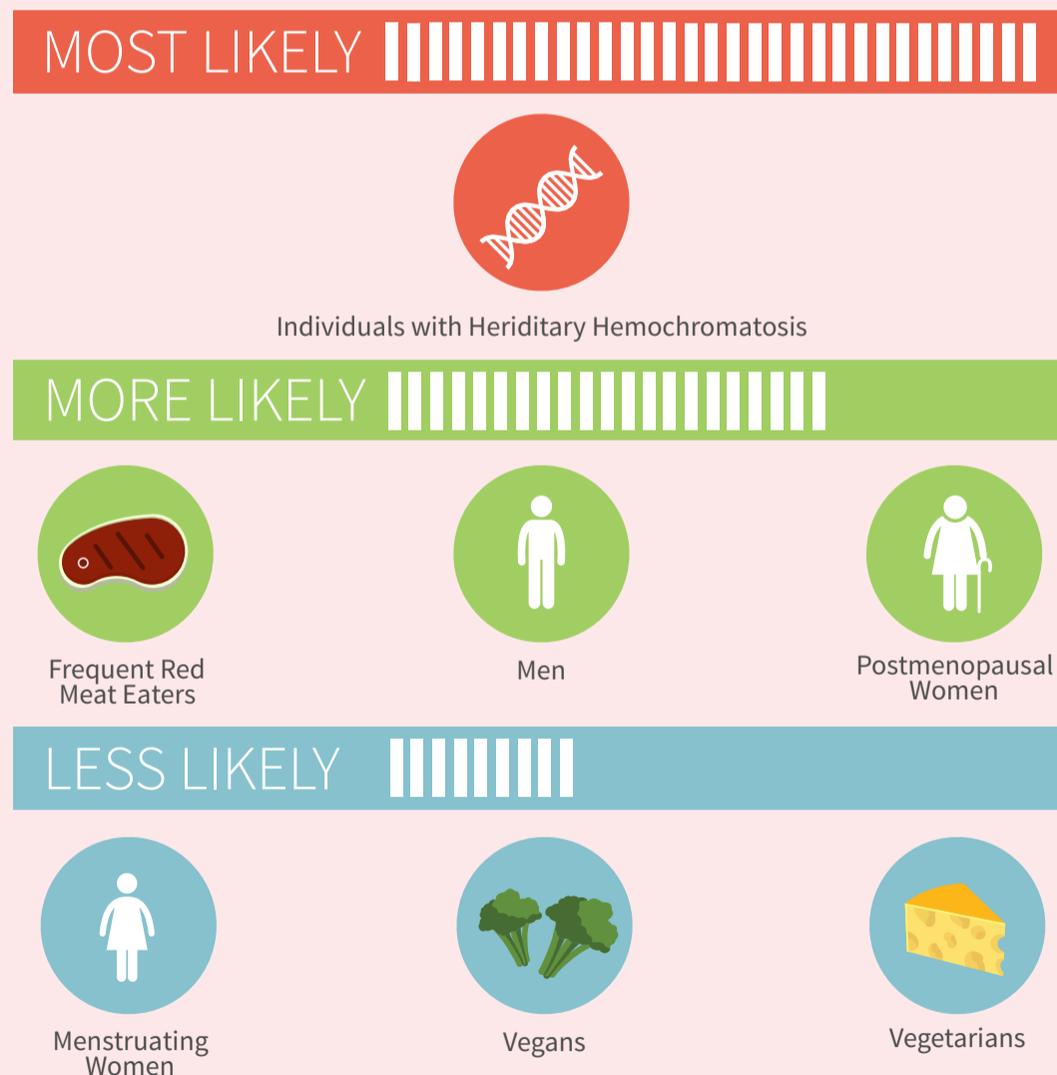
While iron intake may drop on carb-restricted diets, overall intake is usually still sufficient for adult males but regularly below the RDA for females, particularly if they are pregnant. But iron supplementation should not be undertaken haphazardly. Because iron can accumulate in the body, chronic poisoning is a possibility. Too much iron on a regular basis can increase unwanted oxidation in some organs for years before a problem becomes apparent. Men and postmenopausal women are especially at risk if they take a multivitamin or multimineral, as those often contain 18 mg of iron: the recommended daily intake for premenopausal women.

⚠ Warning: Iron overload

Too much iron has been linked, via prospective observational studies, to a wide variety of conditions, such as Alzheimer's, heart disease, and colorectal cancer.^{35,36,37} There are a couple of groups who don't have to worry as much about iron overload though: menstruating women and vegetarians/vegans. But for others, especially those who regularly eat red meat, it doesn't take much to push yourself into excess iron territory. And for the nearly one million Americans who have hereditary [hemochromatosis](#), a condition — which typically emerges in adulthood — where you absorb too much dietary iron, the risk is much more serious.

Those at risk for iron overload should also learn more about how to cook with cast iron safety. We provide full details on how to do so [here](#).

Figure 4: Chances of iron overload in different populations



Certain multivitamins and foods (especially breakfast cereals) also have added iron, which can contribute to iron buildup over time in those who don't get rid of iron once a month, namely all males and nonmenstruating females. One of the most popular multivitamins on pharmacy shelves has 100% of the iron RDA per pill. While breakfast cereals are of little concern on a keto diet, when eating red meat and iron-fortified energy bars a few times a week plus taking a multivitamin, the iron can quickly add up.

Take stock of your iron intake from supplements and your diet before adding an iron supplement.

A list of low-carb foods rich in iron can be found in [Appendix B](#).

Table 10: Recommended Dietary Allowances (RDAs) for Iron (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	0.27*	0.27*	—	—
7–12 months	11	11	—	—
1–3 years	7	7	—	—
4–8 years	10	10	—	—
9–13 years	8	8	—	—
14–18 years	11	15	27	10
19–50 years	8	18	27	9
>50 years	8	8	—	—

* Adequate intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 2001. DOI:[10.17226/10026](https://doi.org/10.17226/10026)

Magnesium

While [magnesium](#) intake tends to be moderately low on a keto diet, this may be doubly problematic in those with type II diabetes (who may be more likely to try a low-carb or keto diet) as they can already have lower levels.³⁸ However, ensuring sufficient intake can actually bring a modest improvement to blood glucose control.^{39,40,41}

A list of low-carb foods rich in magnesium can be found in [Appendix B](#).

Table 11: Recommended Dietary Allowance (RDA) for Magnesium (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	30*	30*	—	—
7–12 months	75*	75*	—	—
1–3 years	80	80	—	—
4–8 years	130	130	—	—
9–13 years	240	240	—	—
14–18 years	410	360	400	360
19–30 years	400	310	350	310
31–50 years	420	320	360	320
>51 years	420	320	—	—

* Adequate intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 1997. DOI:[10.17226/5776](https://doi.org/10.17226/5776)

Magnesium supplementation is generally safe if you don't exceed the *tolerable upper intake level* (UL) for supplemental magnesium (see table below),⁴² but adverse gastrointestinal effects, such as nausea and diarrhea, can occur when too little magnesium is absorbed.³⁹ Magnesium obtained from food has not been linked to adverse effects.⁴²

Table 12: Tolerable Upper Intake Level (UL) for Supplemental Magnesium (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–12 months	—	—	—	—
1–3 years	65	65	—	—
4–8 years	110	110	—	—
>9 years	350	350	350	350

Reference: Institute of Medicine. *The National Academies Press*. 1997. DOI:[10.17226/5776](https://doi.org/10.17226/5776)

If you **do not know your magnesium intake**, a reasonable standard dose for avoiding deficiency is 200 mg of *elemental magnesium* once a day.

If your **magnesium intake is very low** (<50% of the RDA), take up to 350 mg/day of elemental magnesium. (Remember, 350 mg is the UL for supplemental magnesium; there is no UL for magnesium obtained from food.)

**Tip: What is elemental magnesium?**

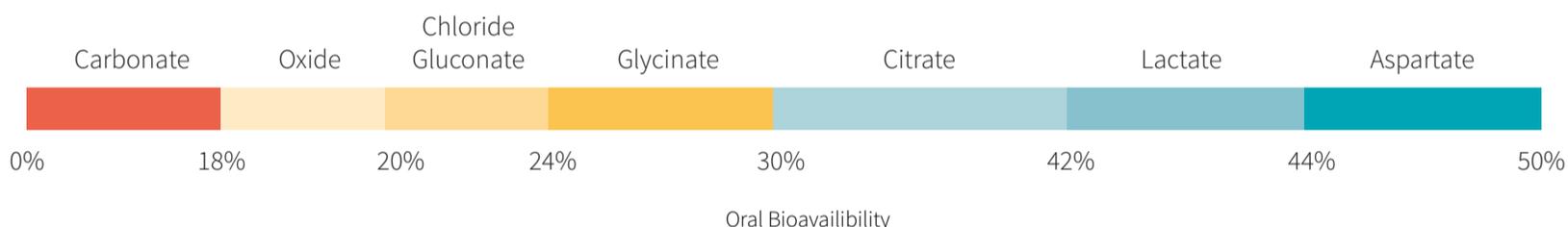
“Elemental” refers to the weight of the mineral by itself, separately from the compound bound to it. For instance, ingesting 500 mg of magnesium gluconate means ingesting 27 mg of elemental magnesium, whereas ingesting 50 mg of zinc gluconate means ingesting 7 mg of elemental zinc.

Product labels display the elemental dosage. On a 500 mg supplement label, “27 mg of magnesium (as magnesium gluconate)” means 27 mg of elemental magnesium (and 473 mg of gluconic acid); on a 50 mg label, “7 mg of zinc (as zinc gluconate)” means 7 mg of elemental zinc (and 43 mg of gluconic acid).

Common supplement forms include **oxide, citrate, gluconate, and glycinate**. To increase absorption, magnesium gluconate should be taken with a meal; citrate and glycinate can be taken on an empty stomach. Magnesium oxide is better avoided, as it has very low bioavailability, which can decrease how much your body absorbs, making it more likely to cause intestinal discomfort and diarrhea.^{43,44,45}

Magnesium **carbonate, lactate, and aspartate** are not commonly available on the market.

Figure 5: Oral bioavailability of various magnesium salts in humans



Reference: Ranade et al. *Am J Ther.* 2001. PMID:[11550076](https://pubmed.ncbi.nlm.nih.gov/11550076/)

Potassium

While [potassium](#) intake on a reduced-carb diet may be only 57–63% of the RDA,^{11,23,24} the use of potassium supplements has been linked to adverse abdominal symptoms (e.g., nausea, vomiting, and abdominal pain).⁴⁶ Worse, too much potassium at once on an empty stomach can lead to [hyperkalemia](#) (very high levels of potassium in the blood), which can lead to dangerous changes in heart rhythm; to avoid this, in the United States, supplemental potassium is limited to 99 mg per serving⁴⁷ a very low amount. Too low to be useful as a supplement.

Additionally, potassium might be excreted in the urine more readily if your sodium intakes are low, which can further compound the problem of insufficient potassium intake. If you suspect you have low potassium levels, you can incorporate more potassium-rich foods into your diet.

A list of low-carb foods rich in potassium can be found in [Appendix B](#).

Table 13: Recommended Dietary Allowances (RDAs) for Potassium (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	400	400	—	—
7–12 months	860	860	—	—
1–3 years	2,000	2,000	—	—
4–8 years	2,300	2,300	—	—
9–13 years	2,500	2,300	—	—
14–18 years	3,000	2,300	2,600	2,500
19–50 years	3,400	2,600	2,900	2,800
>50 years	3,400	2,600	—	—

* Adequate Intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 2019. DOI:[10.17226/25353](https://doi.org/10.17226/25353)

Salt (sodium)

As discussed in the Body Composition chapter, as ketones get filtered and pass from your kidneys to your bladder for excretion, some extra sodium gets carried along with them. This may temporarily increase your sodium intake needs as your body adjusts to the keto diet over a period of 1 to 4 weeks, or possibly even longer. Additionally, people who sweat a lot, such as athletes and other people who exercise regularly, are already prone to sodium loss. When you add a keto diet to the mix, your sodium needs may increase from 1.5 g/day to 3–5 g/day.⁴⁸ Remember, this is a *total* of 3–5 g/day from foods plus added sodium, not just from added sodium.

Table 14: Signs and symptoms of hyponatremia

HYPONATREMIA (low blood levels of sodium)
Acting “out of sorts”
Acute weight gain
Altered mental state
Apathy
Disorientation or confusion
Dizziness or lightheadedness
Dyspnea (shortness of breath)

Headache
Mood changes
Muscle twitching or weakness
Nausea or vomiting
Swelling of hands, feet, or both

Adapted from McDermott et al. *J Athl Train*. 2017. PMID:[28985128](#)

In studies looking at a reduced-carb diet, sodium intake has been seen to range from 2,200–4,400 mg a day.^{11,24} Whether you personally need to add sodium to your diet can depend on your activity level and sweat loss, and if you experience symptoms of the “low-carb flu”, such as fatigue, constipation, nausea, or headaches.

Those who have high blood pressure or have been prescribed a low-sodium diet, such as the Dietary Approaches to Stop Hypertension (DASH) diet, should use extra caution before adding more sodium.

A list of low-carb foods rich in sodium can be found in [Appendix B](#).

Table 15: Adequate Intake (AI) for Sodium (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	110	110	—	—
7–12 months	370	370	—	—
1–3 years	800	800	—	—
4–8 years	1,000	1,000	—	—
9–13 years	1,200	1,200	—	—
14–18 years	1,500	1,500	1,500	1,500
19–30 years	1,500	1,500	1,500	1,500
31–50 years	1,500	1,500	1,500	1,500
51–70 years	1,500	1,500	—	—
>70 years	1,500	1,500	—	—

* Adequate intake (AI)

Reference: Institute of Medicine. *The National Academies Press*. 2019. DOI:[10.17226/25353](#)

Vitamin A

[Vitamin A](#) is a catch-all term that refers to a number of related compounds. Retinol is one of the most abundant types of vitamin A⁴⁹ and can convert into biologically active (e.g., retinal and retinoic acids) or inactive forms (e.g., retinyl esters⁵⁰).

Intake of vitamin A on reduced-carb diets can be variable, with some studied dieters increasing their intake by 100% while others experienced a 67% decrease.¹⁵ Low intake can lead to impaired vision, dry skin, and poor immunity, but high intake is not risk-free. Being fat-soluble, vitamin A can accumulate in your body and lead to toxicity ([hypervitaminosis A](#)).

Note that this toxicity concern applies to preformed vitamin A, which consists of retinol and retinyl esters, found mainly in the liver and concentrated fish oils.³⁴ Provitamin A carotenoids, such as beta-carotene, have not been readily associated with toxicity or notable adverse events unless taken at very high doses (>20,000 mcg or 3,300 mcg RAE).⁵¹

Getting enough vitamin A on a keto diet may not require any supplementation or dietary adjustment, but your intake should be monitored to ensure adequate amounts are being consumed while.

A list of low-carb foods rich in vitamin A can be found in [Appendix B](#).

Table 16: Recommended Dietary Allowances (RDAs) for Vitamin A (mcg RAE)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	400*	400*	—	—
7–12 months	500	500*	—	—
1–3 years	300	300	—	—
4–8 years	400	400	—	—
9–13 years	600	600	—	—
14–18 years	900	700	750	1,200
19–50 years	900	700	770	1,300
>50 years	900	700	—	—

* Adequate Intake (AI)

1 IU retinol = 0.3 mcg retinol activity equivalents (RAE)

1 IU beta-carotene from dietary supplements = 0.15 mcg RAE

1 IU beta-carotene from food = 0.05 mcg RAE

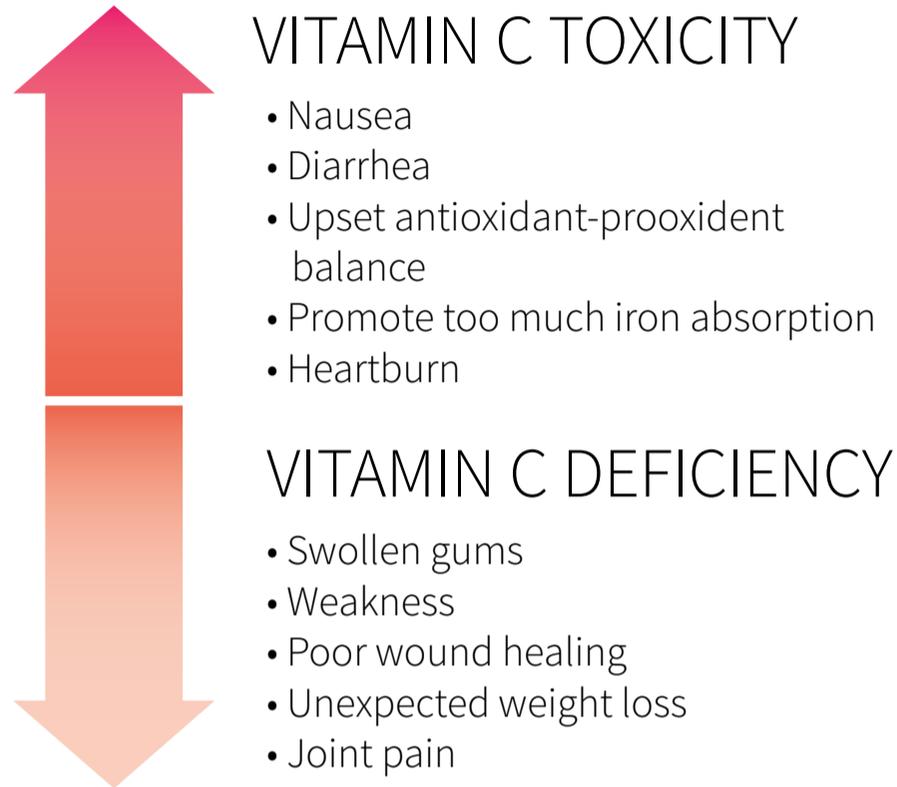
1 IU alpha-carotene or beta-cryptoxanthin = 0.025 mcg RAE

Reference: Institute of Medicine. *The National Academies Press*. 2001. DOI:[10.17226/10026](https://doi.org/10.17226/10026)

Vitamin C

Vitamin C (L-ascorbic acid) is a water-soluble essential vitamin. It is a very popular dietary supplement due to its antioxidant properties, safety, and low price.

Figure 6: Symptoms of vitamin C toxicity and deficiency



People on a reduced-carb diet typically consume $\approx 50\%$ of the RDA for vitamin C.¹⁵ Because the Tolerable Upper Intake Levels (ULs) for vitamin C are quite high — 2,000 mg for adults 19 and over — supplementing with a dose up to the RDA can be safe for long-term use.⁵²

A list of low-carb foods rich in vitamin C can be found in [Appendix B](#).

Table 17: Recommended Dietary Allowances (RDAs) for Vitamin C** (mg)

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–6 months	40*	40*	—	—
7–12 months	50*	50*	—	—
1–3 years	15	15	—	—
4–8 years	25	25	—	—
9–13 years	45	45	—	—
14–18 years	75	75	80	115
>18 years	90	75	85	120

* Adequate intake (AI)

** Smokers may require 35 mg/day more than nonsmokers

Reference: Institute of Medicine. *The National Academies Press*. 2000. DOI:[10.17226/9810](https://doi.org/10.17226/9810)

Vitamin D

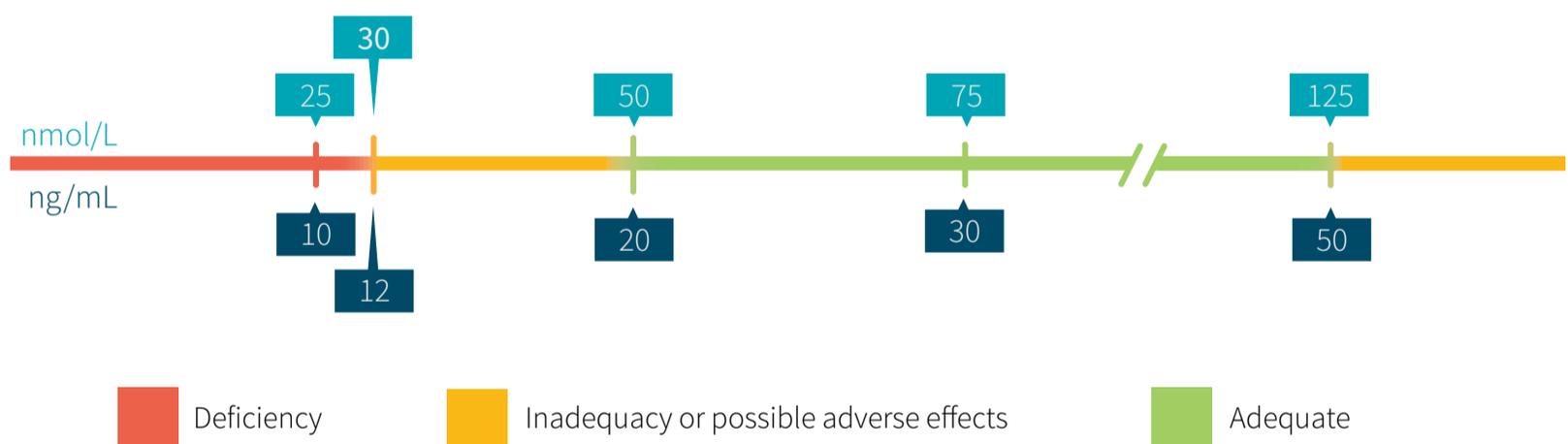
Vitamin D, a fat-soluble vitamin, has two main dietary forms: ergocalciferol (D₂) and cholecalciferol (D₃). Vitamin D is of interest in public health, as it has been identified as an underconsumed nutrient whose low intake levels have been associated with poor health outcomes.⁵³

While vitamin D is most well known for aiding in the formation of healthy bones,¹⁸ increasing attention has been given to its potential role in nonskeletal health factors. Vitamin D receptors (VDRs) have been found in over 35 tissues throughout the body: cardiac, muscle, intestinal, pancreatic, colon, skin, kidney, lung, thyroid, prostate, and brain tissue, among others, possess VDRs.^{54,55} The presence of VDRs across numerous tissues implicates vitamin D in many health functions.⁵⁶

The evidence on what happens to vitamin D intake on a reduced-carb diet is somewhat mixed.^{11,24,57,58} It does appear that a subset, ≈30%, may see a drop in vitamin D intake when changing to a lower-carb diet. However, most experience no change or an increase.

Before considering supplementation, you should first determine if you really need to by checking your current **blood levels of 25(OH)D**, the common indicator of vitamin D status. Those with darker skin pigmentation may be at increased risk of having low vitamin D levels, as vitamin D synthesis in the skin can be reduced in these skin types.⁵⁹ As such, these populations may want to give extra attention to their vitamin D levels.

Figure 7: Blood levels of vitamin D status



Vitamin D should always be taken with food that contains fat to aid in absorption, or with a fat-containing supplement (e.g., [fish oil](#)).

In case of **deficiency**, a medically supervised intervention will be needed. **Do not begin any intervention without discussing it with your physician.** Common medical interventions include taking 50,000 IU (1,250 mcg) of D₂ or D₃ at least three times a week for six to eight weeks, but people with a borderline deficiency may not need as high a dose.

At the end of this intervention, if vitamin D levels are above 30 nmol/L (12 ng/mL), a daily dose of 800–1,000 IU (20–25 mcg) is usually prescribed for maintenance.

In case of **inadequacy**, 800–2,000 IU (20–50 mcg) of D₃ per day is likely to raise vitamin D levels to an adequate level, at which point 800–1,000 IU (20–25 mcg) per day should suffice for maintenance.

In case of **adequate** vitamin D levels, a vitamin D supplement may not be necessary, especially if you spend a lot of time outside or live near the equator (or both). However, taking 400–600 IU (10–15 mcg) of D₃ per day may help keep vitamin D levels in the adequate range, particularly during the colder, darker months, when you are least likely to synthesize enough vitamin D from sun exposure.

In cases of **high vitamin D levels**, which may cause potential adverse effects, do not supplement with vitamin D, and cease use of any supplements containing vitamin D unless specifically instructed not to by a medical professional. Schedule a follow up with your physician to determine if these high levels may be problematic.

If you do not know your vitamin D levels and cannot get them tested but are still intent on taking a supplement, it would be prudent to limit yourself to a maintenance dose of 400–600 IU (10–15 mcg) of D₃ per day. Alternatively, you could track your food intake for a week to determine your average vitamin D intake and select an appropriate dose to achieve the RDA.

If the maintenance doses above prove insufficient, as could be the case in people with a BMI over 30 or who suffer from poor vitamin D absorption or processing — due to genetics, a problem in the kidney, liver, or gastrointestinal tract — you could switch to 1,000–2,000 IU (25–50 mcg) of D₃ per day.

A list of low-carb foods rich in vitamin D can be found in [Appendix B](#).

Table 18: Recommended Dietary Allowances (RDAs) for Vitamin D (IU^{**})

AGE	MALE	FEMALE	PREGNANT	LACTATING
0–12 months	400*	400*	—	—
1–13 years	600	600	—	—
14–18 years	600	600	600	600
19–50 years	600	600	600	600
51–70 years	600	600	—	—
>70 years	800	800	—	—

* Adequate intake (AI)

** 40 IU = 1 mcg

Reference: Institute of Medicine. *The National Academies Press*. 2011. DOI:10.17226/13050.

⚠ Warning: Supplementing with calcium and vitamin D

If you supplement with calcium daily, and this dose puts you over the RDA, consider reducing your dose before adding vitamin D. Taken with high doses of calcium, even modest doses of vitamin D (400 IU) may increase your risk of kidney stones, which can also be independently increased with a keto diet^{60,61}. If the calcium supplement you are taking was prescribed by your physician, consult them before making any changes.

If you take a [multivitamin](#), check to see if it contains vitamin D. It may already contain sufficient amounts for your needs. Check the calcium dose as well, to ensure it is not too high.

If you are taking a medically prescribed calcium supplement, do not make any changes without speaking to your physician.

Intake of certain nutrients may drop when consuming a lower-carb diet, notably calcium, fiber, folate (B9), iodine, iron, magnesium, potassium, sodium, thiamine (B1), vitamin A, vitamin C, and vitamin D. These may need to be monitored to ensure adequate intakes are consistently met.

Common adverse reactions and side effects

As we noted at the beginning of this chapter, a lot of the long-term data on the effects of a keto diet come from children with epilepsy who use it as a medical treatment. The table below shows some side effects and their reported incidence in these children.⁶² However, the structure of a nonmedical keto diet is more flexible, so side effect frequency and severity can be lower.

Many people who have tried the keto diet report experiencing fatigue, nausea, bad breath, intestinal discomfort, brain fog, or even vomiting in the first 1–4 weeks. This collection of symptoms is often called the “low-carb flu” or “keto flu”.

In the sections below, we’ll cover additional adverse reactions and side effects, and give you strategies to help cope — or hopefully get rid of them all together!

Table 19: Reported short- and long-term side effects of medical ketogenic diet therapy for the treatment of epilepsy

SIDE EFFECT	ONSET FROM DIET INITIATION	REPORTED INCIDENCE (%)
Acidosis	Early	0.8–1.9
Cardiomyopathy	Late	0.8
Dehydration	Early	0.3–46.5
Elevated liver enzymes	—	0.2

Gallstones	—	0.4
Gastrointestinal upset	Early and late	1.9–38.7
Hepatitis	Early and late	2.3–5.4
Hypoglycemia (low blood sugar)	Early and late	0.8–7.0
Hypomagnesemia (low magnesium)	Early and late	4.7–10.9
Hypoproteinemia (low protein)	Early and late	3.9–5.5
Increased infections	Early and late	0.8–20.9
Lipoid aspiration pneumonia	Early and late	0.3–4.7
Osteopenia and fracture risk	Late	14.7
Pancreatitis	Early and late	0.1–0.8
Protein loss enteropathy	—	0.20
Raised lipids/cholesterol	Early and late	2.6–27.1
Raised serum uric acid	Early and late	1.8–26.4
Renal stones (kidney stones)	Late	1.3–3.1

Adapted from Cross et al. *Arch Dis Child*. 2010. PMID:20418339

Acidosis

Metabolic acidosis is a condition in which your bodily fluids, such as blood, become too acidic. A sudden increase of acidic ketones and insufficient levels of agents to buffer them, such as bicarbonate — one component of **sodium bicarbonate**, aka baking soda — may cause mild acidosis. This can manifest as fatigue, nausea, or even vomiting. If you are experiencing symptoms of acidosis, try the following.

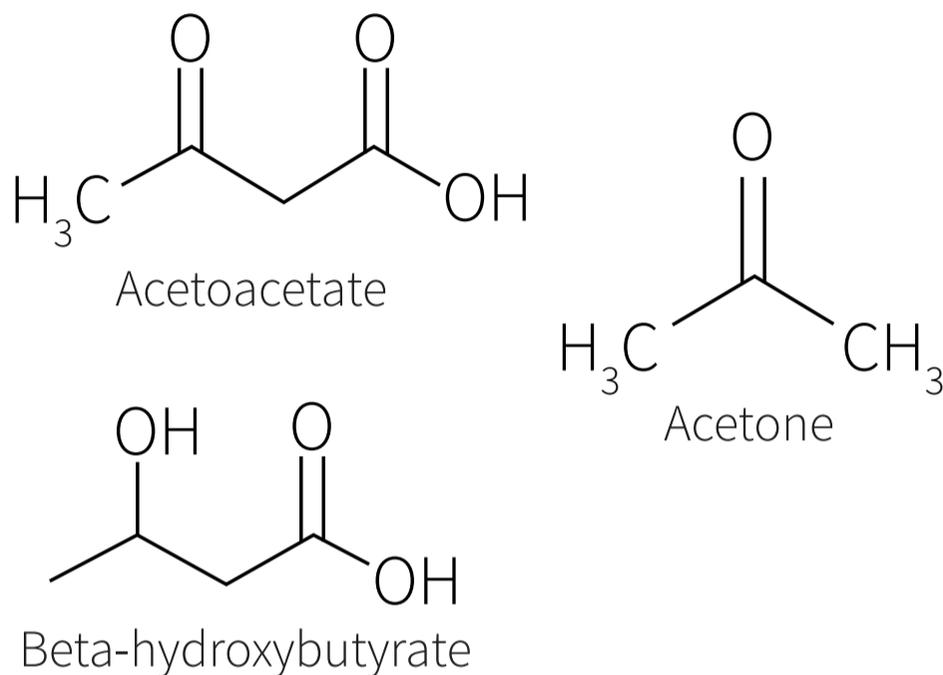
- Ensure you are staying hydrated. Fluids with electrolytes (sodium, potassium, magnesium) may provide greater benefit. Add at least 710 mL (24 oz) to your diet.
- Supplement with sodium bicarbonate. A 5–10 g daily dose should help. Be aware that taking sodium bicarbonate by itself can cause stomach upset, so it's best to split your total intake into 2–3 smaller doses taken with a meal.

Additionally, as 27.3% of sodium bicarbonate's weight is due to sodium, every 1,000 mg confers about 270 mg of sodium to the diet; this needs to be accounted for and severely limits usage by people with salt-sensitive hypertension (high blood pressure). However, those on a keto diet may experience some additional relief from “keto flu” by increasing their sodium intake, so sodium bicarbonate could be a win-win supplement.

Bad breath

In ketosis, your body will produce three types of ketones: acetone, acetoacetate, and beta-hydroxybutyrate (β HB).

Figure 8: Ketone bodies
Ketone Bodies



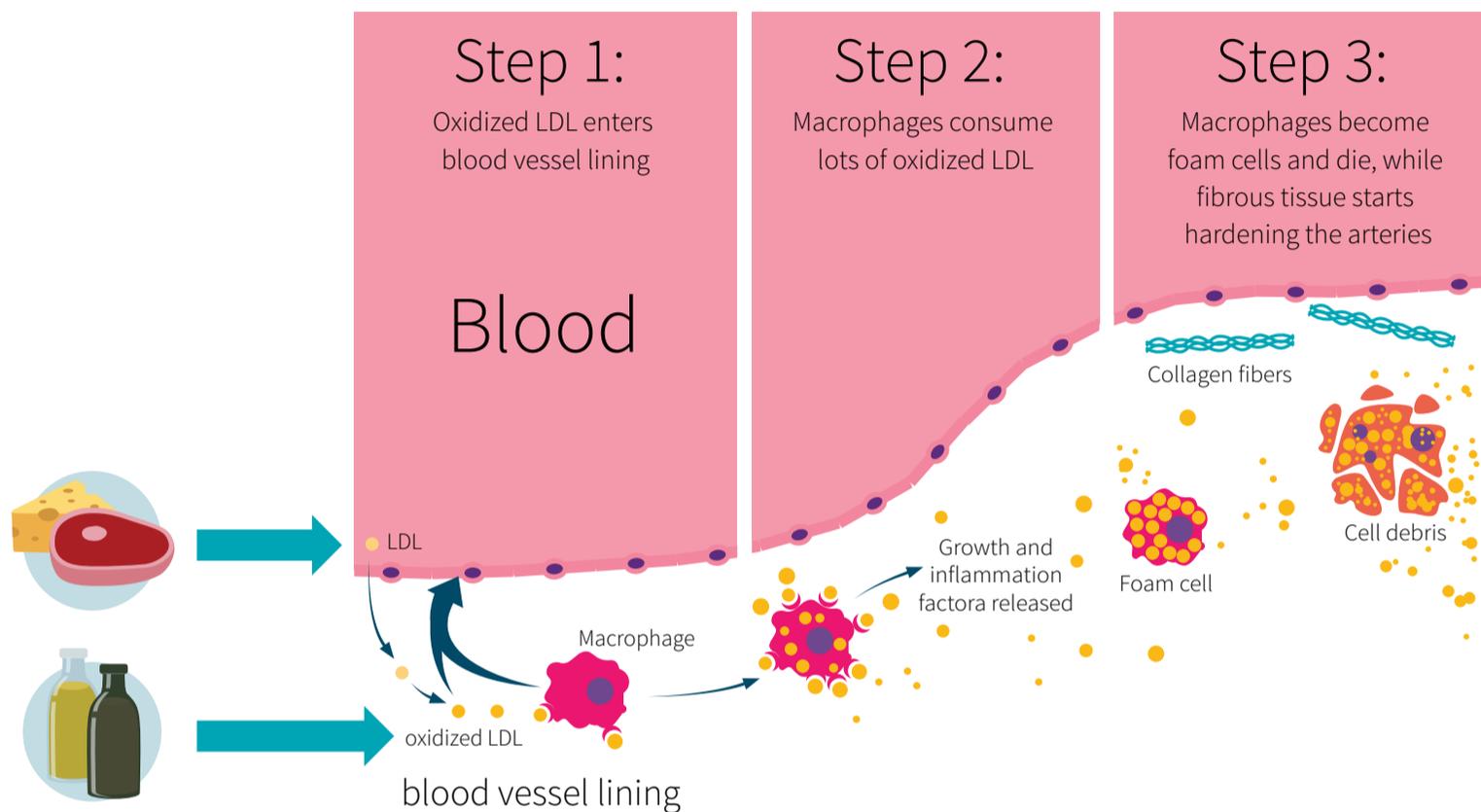
Acetoacetate can be converted into acetone, which can then be excreted through your urine or exhaled. This can give your breath an odor that has been described both as “nail polish remover” and “slightly fruity”. Whatever the smell, most people do not find this side effect desirable. Here are some potential solutions to your odorous problem.

- Wait it out. Sometimes this will go away once your body has adapted to the keto diet.
- Drink more fluids. Acetone can also be excreted in your urine. Theoretically, drinking more fluids may help decrease the amount excreted through your breath.
- Decrease ketone levels. If your breath is really bothering you, you can try to increase your daily carb intake to help decrease the formation of ketones. Try adding 10–20 g of carbs back into your diet and see what happens. Individual results will vary, so some trial and error will be needed.
- Mask the smell. Keep some sugar-free or low-carb mints, gum, mouthwash, strips, or oral spray on hand. Use as needed to keep “keto breath” at bay.

Cholesterol

Heart disease is most commonly the result of atherosclerosis, the buildup of plaque in arteries. This occurs when LDL particles penetrate arterial walls, become oxidized, and are attacked by white blood cells.^{63,64} HDL removes cholesterol from arteries and plaque, protects the lining of the arteries from damage, and inhibits LDL oxidation.⁶⁵

Figure 9: How oxidized LDL may contribute to atherosclerosis



Reference: Steinberg. *J Lipid Res.* 2009. PMID:[19011257](https://pubmed.ncbi.nlm.nih.gov/19011257/).

Those on a low-carb or ketogenic diet can experience shifts in their blood lipid and cholesterol profiles. Triglycerides commonly drop, while *high-density lipoproteins* (HDL) and *low-density lipoproteins* (LDL) usually increase.^{66,67} Both HDL and LDL particles have been seen to increase and remain elevated on a long-term keto diet (2 years).⁶⁶

LDL particles are one part of the heart disease picture. Several events need to occur for heart disease to develop. This helps explain why heart disease has numerous risk factors, such as diabetes, obesity, smoking, lack of exercise, and infection.⁶⁸ While the full implications of having elevated LDL while on a low-carb diet are not fully known, it would be prudent to keep an eye on measurements.

So before starting a ketogenic diet, get a baseline measurement of your current cholesterol profile. After a month, recheck to see what has changed. If you see an increase in your LDL, the following steps can be taken to try to bring it back down.

- Focus on adding more high-fiber, low-carb foods to your diet. (See [Appendix B](#) for some ideas.)

- Add a soluble fiber supplement. [Psyllium](#) can be a good option due to its high viscosity and low degree of fermentation, making it less likely to produce unwanted side effects. Take 10–15 g/day in divided doses across multiple meals.
- Do a fat swap. Replace butter, coconut oil, and ghee with foods like olives or olive oil, nuts, seeds, and avocados or avocado oil.

⚠ Warning: What if I already have high cholesterol?

If you currently have an unfavorable cholesterol profile, you should take extra caution before starting the keto diet. Speak with your physician and have a plan in place to implement the suggestions discussed in the above three bullet points. You should also have a plan in place to actively monitor changes in your cholesterol profile if you plan to stay on this diet for months or years.

Constipation

Constipation is one of the more common side effects in the initial weeks of the keto diet.⁶⁹ A decrease in food intake, fiber, and hydration may all be contributing factors. If you experience constipation, try taking the following actions.

- Increase your soluble fiber intake. [Psyllium](#) can be a good option due to its high viscosity and low degree of fermentation, making it less likely to produce unwanted side effects. Take 10–15 g/day in divided doses across multiple meals.
- Hydrate. Increase fluid intake throughout the day so you're adding at least 710 mL (24 oz) to your diet. Despite rumors to the contrary, the infamous cup of coffee may help with hydration and constipation.
- Strategically use oils. Adding 1–2 tablespoons (15–30 g) of MCT oil or mineral oil to your diet per day may help.
- If you need more immediate relief, over-the-counter [polyethylene glycol 3350](#) (MiraLAX®) can also be used.

If you have not experienced any improvement in two to three days, you may need to seek the help of a physician or temporarily cease your keto diet. You'll want to use increased fiber and fluid intake as a long-term strategy to stay regular, but MCT oil, mineral oil, or polyethylene glycol can be used as short-term solutions while you work out the kinks in your keto diet routine.

Dehydration

A keto diet can have a diuretic effect on your body. You'll likely lose a lot of weight in the first week or so, but that weight will mostly be water, for two reasons:

1. **You empty your carb stores.** When you eat carbs, your body can transform them into glucose, to be burned for energy or stored as glycogen. To store 1 g of glycogen, you need about 3 g of water. If you stop eating carbs, your body uses your stored glycogen, and the freed water gets excreted.
2. **You excrete ketones.** As your ketone levels rise, your kidneys begin to excrete them in your urine, along with some salt (sodium) and water.

Your body can adjust to this after a few weeks, but if you're feeling a little dehydrated⁷⁰, be sure to increase your daily fluid intake. Start with 710 mL (24 oz) of extra fluids per day.

Table 20: Signs and symptoms of hypohydration

HYPOHYDRATION (less body water than is optimal)
Acute weight loss
Apathy
Cramping
Dizziness or lightheadedness
Dyspnea (shortness of breath)
Flushed skin
Headache
Heat sensations or chills
Nausea or vomiting
Thirst

Adapted from McDermott et al. *J Athl Train*. 2017. PMID:[28985128](#)

Electrolyte levels

There are six main electrolytes: [calcium](#), chloride, [magnesium](#), phosphorus, [potassium](#), and sodium. Four of these — calcium, magnesium, sodium, and potassium — may be below recommended intake levels on a reduced-carb diet.^{11,15,23,24} These electrolytes are involved in numerous bodily functions, so obtaining adequate amounts is key to maintaining overall health.

On a keto diet, a drop in electrolyte intake may be responsible for some of the symptoms of the “keto flu” (nausea, fatigue, brain fog). To alleviate some of these symptoms, you can increase your magnesium, sodium, and potassium intake.

- For **sodium**, try increasing your daily intake to 3–5 g/day. Remember, this is a *total* of 3–5 g/day from foods plus added sodium, not just from added sodium. If you have been prescribed a low-sodium diet, speak with your physician before upping your intake.
- For **magnesium**, start with 200 mg of *elemental magnesium* once a day. If you don’t notice symptom improvement, you can increase your dose to 350 mg/day.
- For **potassium**, supplements will likely be insufficient for improving overall potassium intake, as most contain <100 mg (compare that with an RDA of 3,400 mg for adult males and 2,600 mg for adult females).⁴⁸ Incorporating more potassium-rich foods into your diet can help maintain adequate levels during your transition to the keto diet. Remember, too much potassium at once on an empty stomach can lead to [hyperkalemia](#) (very high levels of potassium in the blood).
- For **calcium**, supplementation is best considered only after a dietary evaluation, as overconsumption may have negative effects on cardiovascular health.^{19,20} Track what you eat for a week and compare the calcium content of your diet with the RDA for your gender and age. If, on average, you are getting less than 80% of your RDA, supplementation becomes a possibility, but you should first consider tweaking your diet.

Fatigue

You may feel oddly tired or have brain fog in the first few weeks of a keto diet. This may subside as your body keto-adapts, but here are a few strategies that may help you stave off these symptoms.

- Stay hydrated. Add at least 710 mL (24 oz) to your diet — preferably water, but other low-calorie, low-carb fluids may suffice.
- Increase your overall electrolyte intake. We discuss how to do this above, in [Electrolyte levels](#).
- Take a slower approach to getting into ketosis. Temporarily increase your carb intake to 100–150 g a day and then slowly decrease to below 50, as tolerated.
- Check your calories. Many people unknowingly decrease their caloric intake when they switch to a keto diet (hence, the potential for beneficial weight loss!), and a

hypocaloric diet — fewer calories than you need to maintain your weight — can cause you to feel tired. If it was not your intent to decrease your calories, add some more food to counter the fatigue.

 **Tip: Calculate your caloric needs**

Your height, weight, age, and level of physical activity all contribute to your caloric needs. There are many calorie calculators out there, but one stands above the rest:



This calculator has been tested and validated against real-world data.⁷¹ It can estimate the number of calories *you* need to reach and then maintain a specific weight. Click on the image above to get going!

Hypoglycemia (low blood sugar)

Hypoglycemic events may be experienced more in the initial weeks, while your body adjusts to your new low-carb diet. This may notably affect those with diabetes or prediabetes.

 **Warning: Diabetes and the keto diet**

If you have diabetes or prediabetes and are considering the keto diet, first speak to your physician, a registered dietitian, or both about your intentions. Because a keto diet drastically decreases carb intake, medications may need to be adjusted and monitored to make sure they do not cause any adverse events. Plans may also need to be put in place on how to deal with hypoglycemic events, should they occur.

Being prepared to deal with a hypoglycemic event takes a little forethought and planning. Essentially, you want to have a small amount of easily accessible food or candy or a sugary beverage that contains fast-digesting carbs (sucrose, glucose, dextrose). Sugar-containing fluids like juices tend to act faster in reversing low blood sugar, compared to solid sources of sugar. If you have low blood sugar, try the following.

- Consume 10–20 g of sugars initially, wait 10 minutes and see how you feel (or test your blood sugar, if possible).
- If no change has occurred in 10 minutes, consume another 10 g of sugars and retest in another 10 minutes. Repeat until blood sugars return to normal.

Nausea

Nausea may occur in the first two weeks or so due to your body adapting to your new high-fat diet. The reasons can vary widely from person to person, and exact causes are hard to pin down, but here are a few things to try to alleviate your symptoms.

- Stay hydrated. Drink as much as you can tolerate, preferably a fluid that provides some electrolytes (water mixed with some salt, Pedialyte®, etc).
- Increase your overall electrolyte intake. We discuss how to do this above, in “Electrolyte levels”.
- Take a slower approach to getting into ketosis. Temporarily increase your carb intake to 100–150 g a day and then slowly decrease to 50, as tolerated.

Vomiting

Vomiting is most likely to occur in the first weeks due to the sudden increase in ketones (aka [metabolic acidosis](#)).⁷² Ironically, vomiting can increase metabolic acidosis, which further promotes vomiting, and a vicious cycle is created. If you experience this problem, try taking the following steps.

- As soon as the vomiting starts or if you feel like you may vomit, have a drink that contains at least 30 g of carbs, such as fruit juice. This should kick you out of ketosis and may help prevent further vomiting.
- If you are unable to keep down fluids and continue vomiting, seek medical attention, as you may need an intravenous (IV) infusion to prevent dehydration.
- After vomiting ceases, temporarily increase your carb intake. If 50 g a day caused these symptoms, try starting with 100–150 g a day and then slowly decrease your intake, as tolerated.

The range and severity of adverse reactions and side effects you might experience on a keto diet are hard to predict. However, you can ease your transition by paying attention to common issues new keto dieters face, and noting potential solutions ahead of time.

Chapter 14: Closing remarks

Top 10 tips and takeaways to keep in mind

1. **Keto isn't your only option.** *Your* best diet will fit your lifestyle, eating preferences, and health considerations.
2. People tend to lose a lot of weight as water and glycogen in their first week on keto — 2 kg (4.4 lb) on average — but **for fat loss, keto isn't inherently superior** to higher-carb options, and vice versa. Different diets work differently for different people, but ***your* best weight-loss diet** will have at least two qualities: it'll be **hypocaloric** (it'll make you eat less than you burn) and **sustainable** (it'll fit *your* lifestyle and eating preferences well enough that you can stick to it).
3. **All diets see a decrease in adherence over time.** Although keto is very restrictive, adherence to keto doesn't seem worse than adherence to low-fat diets, vegan diets, or any kind of hypocaloric diet.
4. **In theory**, keto diets should impair resistance training, since the easiest way for your body to get the glucose it needs to fuel your workout is to break down the carbs you eat. Yet, **in practice**, they don't seem to. If you don't eat any carbs, your body will replenish your glycogen stores between workouts by making glucose out of different substrates (such as protein, fat, and ketones).
5. Being **keto-adapted** means being better at burning fat. Since your body can store more fat than carbs, then **in theory**, keto diets should enhance endurance performance, but **in practice**, they seem to have either no effect or a negative one. This is because, at the exercise intensities seen in endurance events, what matters is how efficiently you burn glucose, not fat. At lower exercise intensities, however, such as seen when hiking or jogging, keto-adaptation may still provide an advantage by allowing you to keep going longer without ingesting calories.
6. Exogenous ketones may provide an **endurance performance benefit** when combined with carbohydrates, but only in elite athletes (not in recreational athletes), and even then, the evidence is mixed.
7. A strict ketogenic diet might help with various brain-related diseases, such as epilepsy, gliomas, cognitive decline, and psychiatric conditions, but for most diseases the evidence is still preliminary. Exogenous ketones might also help, but no study to date has tested this hypothesis.

8. MCTs might lead to an **increase in energy expenditure** resulting in a small **increase in fat loss**, but only when they *replace* other fats in your diet — not when they're just an added source of calories. In any case, more research is needed to properly measure differences in weight loss in the long term.
9. A keto diet may **reduce high levels of insulin and blood sugar** in prediabetics and type II diabetics. In some cases, a well-formulated hypocaloric keto diet put type II diabetes into remission.
10. A keto diet is **not a cancer wonder-cure**, but it might help with select types of cancer, such as gliomas (a rare form of brain cancer). The evidence is *very* preliminary, however; much more research is needed before keto dieting can be recommended as a general cancer treatment.

Appendices

Appendix A: Tables of low-carb foods

Table 1: Low-carb alcohol

	DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
BEER	Low-carb beer	340	12 oz	96	0	3
	Light beer	340	12 oz	108	0	6
	Regular beer	340	12 oz	156	0	13
LIQUOR	100 proof (gin, rum, vodka, whiskey)	28	1 oz	82	0	0
	94 proof (gin, rum, vodka, whiskey)	28	1 oz	76	0	0
	90 proof (gin, rum, vodka, whiskey)	28	1 oz	73	0	0
	86 proof (gin, rum, vodka, whiskey)	28	1 oz	70	0	0
	80 proof (gin, rum, vodka, whiskey)	28	1 oz	64	0	0
	Sake	28	1 oz	39	0	1
	Coffee with cream liqueur, 34 proof	28	1 oz	102	0	7
	Coffee liqueur, 63 proof	28	1 oz	107	0	11
	Coffee liqueur, 53 proof	28	1 oz	117	0	16
	WHITE WINE	Pinot Blanc	170	6 oz	144	0
Pinot Gris (Grigio)		170	6 oz	144	0	4
Sauvignon Blanc		170	6 oz	144	0	4
Chardonnay		170	6 oz	150	0	4
Fume Blanc		170	6 oz	144	0	4
Table white		170	6 oz	144	0	5
Gewurztraminer		170	6 oz	144	0	5
Semillon		170	6 oz	144	0	6
Chenin Blanc		170	6 oz	144	0	6
Muller Thurgau		170	6 oz	132	0	6
Riesling		170	6 oz	144	0	7
Muscat		170	6 oz	150	0	9

Appendix A

RED WINE	Pinot Noir	170	6 oz	144	0	4
	Gamay	170	6 oz	138	0	4
	Carignane	170	6 oz	132	0	4
	Cabernet Franc	170	6 oz	144	0	4
	Lemberger	170	6 oz	144	0	4
	Merlot	170	6 oz	144	0	4
	Cabernet Sauvignon	170	6 oz	144	0	5
	Syrah	170	6 oz	144	0	5
	Table red	170	6 oz	150	0	5
	Sangiovese	170	6 oz	150	0	5
	Mouvedre	170	6 oz	156	0	5
	Petite Sirah	170	6 oz	150	0	5
	Barbera	170	6 oz	150	0	5
	Zinfandel	170	6 oz	156	0	5
	Claret	170	6 oz	144	0	5
Burgundy	170	6 oz	150	0	7	
OTHER WINE	Rose	170	6 oz	150	0	7
	Dessert wine, dry	170	6 oz	270	0	21
	Dessert wine, sweet	170	6 oz	282	0	24

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>.

Table 2: Low-carb beverages

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Club soda	226	8 oz (1.0 cup)	0	0	0
Water, non-carbonated and carbonated	226	8 oz (1.0 cup)	0	0	0
Tea	226	8 oz (1.0 cup)	0	0	0
Coffee	226	8 oz (1.0 cup)	5	0	0
Almond milk, unsweetened	226	8 oz (1.0 cup)	39	1	3
Soy milk, unsweetened	226	8 oz (1.0 cup)	80	1	3
Acerola juice	226	8 oz (1.0 cup)	56	1	11
Lemon juice	226	8 oz (1.0 cup)	54	1	16

Appendix A

Blackberry juice	226	8 oz (1.0 cup)	95	0	19
Lime juice	226	8 oz (1.0 cup)	60	1	19
Orange juice	226	8 oz (1.0 cup)	111	1	25

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 3: Low-carb dairy

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Port de salut	132	1.0 cup, diced	465	0	1
Mozzarella, part skim milk	28	1.0 oz	72	0	1
Cream cheese	15	1.0 tbsp	51	0	1
Caraway	28	1.0 oz	107	0	1
Monterey	132	1.0 cup, diced	492	0	1
Neufchatel	28	1.0 oz	72	0	1
Romano	28	1.0 oz	110	0	1
Half and half	30	1.0 fl oz	40	0	1
Cheshire	28	1.0 oz	110	0	1
Goat milk	31	1.0 fl oz	21	0	1
Muenster	132	1.0 cup, diced	486	0	1
Swiss cheese	132	1.0 cup, diced	519	0	2
Fontina	132	1.0 cup, diced	513	0	2
Pimento	140	1.0 cup, diced	525	0	2
Mozzarella, whole milk	112	1.0 cup, shredded	335	0	3
Provolone	132	1.0 cup, diced	463	0	3
Cottage, lowfat, 1% milkfat	113	4.0 oz	81	0	3
Colby	132	1.0 cup, diced	520	0	3
Heavy whipping	120	1.0 cup, whipped	408	0	3
Queso fresco	122	1.0 cup, crumbled	365	0	4
Cheddar cheese	132	1.0 cup, diced	532	0	4
Cottage, lowfat, 2% milkfat	113	4.0 oz	92	0	5
Queso asadero	132	1.0 cup, diced	470	0	5
Feta	150	1.0 cup, crumbled	398	0	6

Appendix A

Queso anejo	132	1.0 cup, crumbled	492	0	6
Ricotta, part skim milk	124	0.5 cup	171	0	6
Queso chihuahua	132	1.0 cup, diced	494	0	7
Greek yogurt, plain, low fat	200	7 oz	146	0	8
Yogurt, plain, whole milk	170	6 oz	104	0	8
Ricotta, whole milk	124	0.5 cup	186	0	9
Milk, whole	244	1.0 cup	156	0	11
Kefir, plain, low fat	243	1.0 cup	104	0	12
Buttermilk	245	1.0 cup	152	0	12
Yogurt, plain, low fat	170	6 oz	107	0	12
Gjetost	28	1.0 oz	132	0	12
Yogurt, plain, skim milk	170	6 oz	95	0	13
Sheeps milk	245	1.0 cup	265	0	13
Milk, 2%	246	1.0 cup	138	0	14
Milk, 1%	246	1.0 cup	118	0	14
Parmesan	100	1.0 cup	420	0	14
Chocolate milk	250	1.0 cup	208	2	24

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 4: Low-carb fruits

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Olives	3	1.0 olive	4	0	0
Longans	3	1.0 fruit	2	0	0
Litchis	3	1.0 fruit	7	0	2
Kumquats	19	1.0 fruit	13	1	2
Avocados	150	1.0 cup, cubes	240	10	3
Rhubarb	122	1.0 cup, diced	26	2	3
Limes	67	1.0 fruit	20	2	5
Carambola (starfruit)	132	1.0 cup, cubes	41	4	5
Blackberries	144	1.0 cup	62	8	6
Acerola (west indian cherry)	98	1.0 cup	31	1	6

Appendix A

Raspberries	123	1.0 cup	64	8	7
Clementines	74	1.0 fruit	35	1	8
Asian pears	122	1.0 fruit	51	4	9
Strawberries	152	1.0 cup, halves	49	3	9
Gooseberries	150	1.0 cup	66	7	9
Prickly pears	149	1.0 cup	61	5	9
Cranberries	110	1.0 cup, chopped	51	4	9
Casaba	170	1.0 cup, cubes	48	2	10
Figs	64	1.0 large	47	2	10
Currants, red and white	112	1.0 cup	63	5	11
Acerola juice	242	1.0 cup	56	1	11
Watermelon	154	1.0 cup	46	1	11
Mulberries	140	1.0 cup	60	2	11
Apricots	243	1.0 cup, halves	66	4	12
Quinces	92	1.0 fruit	52	2	12
Peaches	154	1.0 cup slices	60	2	12
Nectarines	143	1.0 cup slices	63	2	13
Pomegranates	87	0.5 cup arils	72	4	13
Cantaloupe	177	1.0 cup, balls	60	2	13
Blueberries	140	1.0 cup, frozen	80	6	13
Papayas	145	1.0 cup	62	3	13
Honeydew	170	1.0 cup, diced	61	1	14
Abiyuch	114	0.5 cup	79	6	14
Apricots	155	1.0 cup, halves	74	3	14
Apples, with skin	125	1.0 cup	65	3	14
Guavas	165	1.0 cup	112	9	15
Grapes, American type	92	1.0 cup	62	1	15
Maraschino cherries	40	8.0 cherries	64	2	15
Loquats	149	1.0 cup, cubed	70	3	16
Grapefruit, pink or red or white	230	1.0 cup sections, with juice	74	3	16

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Lemon juice	244	1.0 cup	54	1	16
Sour cherries	155	1.0 cup, without pits	78	3	16
Pummelo	190	1.0 cup, sections	72	2	16
Medjool dates	24	1.0 date, pitted	66	2	16
Elderberries	145	1.0 cup	106	10	16
Plums	165	1.0 cup, sliced	76	2	17
Pears	140	1.0 cup, sliced	88	4	17
Oranges	180	1.0 cup, sections	85	4	17
Pears	140	1.0 cup, slices	80	4	17
Blueberries	148	1.0 cup	84	4	18
Goji berries	28	5.0 tbsp	98	4	18
Sweet cherries	138	1.0 cup	87	3	19
Blackberry juice	250	1.0 cup	95	0	19
Lime juice	242	1.0 cup	60	1	19
Pineapple	165	1.0 cup, chunks	84	2	20
Japanese persimmons	34	1.0 fruit	93	5	20
Rowal	114	0.5 cup	127	7	20
Kiwifruit	180	1.0 cup, sliced	110	5	21
Feijoa	243	1.0 cup, pureed	148	16	21
Mangos	165	1.0 cup pieces	99	3	22
Tangerines (mandarin oranges)	195	1.0 cup, sections	103	4	23
Cherimoya	160	1.0 cup, pieces	120	5	24
Grapes, seedless	245	1.0 cup	98	2	24
Applesauce, unsweetened	244	1.0 cup	102	3	25

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 5: Low-carb legumes

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Tofu, extra firm	91	0.2 block	76	1	0
Tofu, soft	120	1.0 piece	73	0	1
Peanuts	28	1.0 oz	161	2	2

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Hummus	15	1.0 tablespoon	27	1	2
Soybeans	172	1.0 cup	296	10	4
Peanut butter	32	2.0 tbsp	191	2	6
Soy protein concentrate	28	1.0 oz	93	2	6
Peanut flour, low fat	60	1.0 cup	257	10	9
Lupin beans	166	1.0 cup	193	5	11
Natto	175	1.0 cup	369	10	13
Soy flour, defatted	105	1.0 cup	343	18	17
Soy flour, full-fat	84	1.0 cup	365	8	19
Refried beans, fat-free	231	1.0 cup	182	11	20
Mungo beans	180	1.0 cup	189	12	22
Chickpeas (garbanzo beans, bengal gram)	240	1.0 cup	211	11	22
Baked beans	259	1.0 cup	368	18	22
Refried beans, traditional style	238	1.0 cup	214	9	23
Broadbeans (fava beans)	170	1.0 cup	187	9	24
Lentils	198	1.0 cup	230	16	24
Cowpeas (blackeyes)	171	1.0 cup	198	11	24
Great northern beans	177	1.0 cup	209	12	25
Split peas	196	1.0 cup	231	16	25
Black beans	172	1.0 cup	227	15	26
French beans	177	1.0 cup	228	17	26
Lima beans	188	1.0 cup	216	13	26
Yellow beans	177	1.0 cup	255	18	26
White beans	179	1.0 cup	254	19	28
Pigeon peas (red gram)	168	1.0 cup	203	11	28
Cranberry bean	177	1.0 cup	241	15	28
Navy beans	182	1.0 cup	255	19	28
Kidney beans	177	1.0 cup	225	11	29
Pinto beans	171	1.0 cup	245	15	29
Yardlong beans	171	1.0 cup	202	7	30
Black turtle beans	185	1.0 cup	240	15	30

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 6: Low-carb nuts and seeds

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Flaxseed	35	1/4 cup	187	10	1
Pecans	35	1/4 cup	242	3	1
Hemp seed, hulled	35	1/4 cup	194	1	2
Pumpkin and squash seed kernels	35	1/4 cup	196	2	2
Macadamia nuts	35	1/4 cup	251	3	2
Raw coconut meat	35	1/4 cup	124	3	2
Hazelnuts or filberts	35	1/4 cup	220	3	2
Walnuts	35	1/4 cup	229	2	2
Butternuts	35	1/4 cup	214	2	3
Coconut meat, dried	35	1/4 cup	231	6	3
Chia sees	35	1/4 cup	170	12	3
Almond butter	35	1/4 cup	215	4	3
Almonds	35	1/4 cup	203	4	3
Pine nuts	35	1/4 cup	236	1	3
Sunflower seed kernels	35	1/4 cup	204	3	4
Sesame seeds	35	1/4 cup	201	4	4
Hickorynuts	35	1/4 cup	230	2	4
Ginkgo nuts	35	1/4 cup	39	3	4
Pistachios	35	1/4 cup	196	4	6
Sesame butter	35	1/4 cup	205	2	6
Cashew nuts	35	1/4 cup	194	1	9
Cashew butter	35	1/4 cup	213	1	10
Breadnut tree seeds	35	1/4 cup	128	5	23
Chestnuts	35	1/4 cup	131	4	23

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>.

Table 7: Low-carb vegetables

DESCRIPTION	GRAMS	SERVING	KCAL	FIBER	NET CARBS
Chives, raw	3	1.0 tbsp chopped	1	0	0
Endive, raw	25	0.5 cup, chopped	4	1	0
Coriander (cilantro) leaves, raw	4	0.25 cup	1	0	0
Epazote, raw	1	1.0 tbsp	0	0	0
Arugula, raw	2	1.0 leaf	0	0	0
Alfalfa seeds, sprouted, raw	33	1.0 cup	8	1	0
Watercress, raw	34	1.0 cup, chopped	4	0	0
Broccoli raab, cooked	85	1.0 cup	21	2	0
Malabar spinach, cooked	44	1.0 cup	10	1	0
Mushrooms, enoki, raw	5	1.0 large	2	0	0
Lettuce, red leaf, raw	28	1.0 cup shredded	4	0	0
Spinach, raw	30	1.0 cup	7	1	0
Escarole, cooked	150	1.0 cup	22	4	0
Pimento	12	1.0 tbsp	3	0	0
Ginger root, pickled	25	2.0 tablespoon	5	1	1
Lettuce, green leaf, raw	36	1.0 cup shredded	5	1	1
Dill pickles	35	1.0 spear, small	4	0	1
Lettuce, cos or romaine, raw	47	1.0 cup shredded	8	1	1
Asparagus, cooked	180	1.0 cup	32	3	1
Mushrooms, shiitake, raw	19	1.0 piece whole	6	1	1
Seaweed, wakame, raw	10	2.0 tablespoon	4	0	1
Mustard greens, raw	56	1.0 cup, chopped	15	2	1
Cloud ears, dried	28	1.0 cup	80	20	1
Seaweed, kelp, raw	10	2.0 tablespoon	4	0	1
Grape leaves, raw	14	1.0 cup	13	2	1
Cabbage, red, cooked	22	1.0 leaf	6	1	1
Nopales, raw	86	1.0 cup, sliced	14	2	1
Bamboo shoots, cooked	120	1.0 cup	14	1	1
Seaweed, irishmoss, raw	10	2.0 tablespoon	5	0	1

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Cabbage, kimchi	150	1.0 cup	22	2	1
Seaweed, laver, raw	26	10.0 sheets	9	0	1
Lettuce, iceberg (includes crisphead types), raw	72	1.0 cup shredded	10	1	1
Cabbage, chinese (pak-choi)	170	1.0 cup, shredded	20	2	1
Shallots, raw	10	1.0 tbsp chopped	7	0	1
Radicchio, raw	40	1.0 cup, shredded	9	0	1
Tomatillos, raw	34	1.0 medium	11	1	1
Peppers, hungarian, raw	27	1.0 pepper	8	0	2
Kale, cooked	118	1.0 cup	42	5	2
Mushrooms, morel, raw	66	1.0 cup	20	2	2
Mushroom, white, raw	70	1.0 cup pieces or slices	15	1	2
Cucumber	52	0.5 cup slices	8	0	2
Mushrooms, Chanterelle, raw	54	1.0 cup	17	2	2
Sauerkraut	142	1.0 cup	27	4	2
Radishes, raw	116	1.0 cup slices	19	2	2
Turnip greens, raw	55	1.0 cup, chopped	18	2	2
Mushrooms, portabella, raw	86	1.0 cup diced	19	1	2
Pepper, banana, raw	124	1.0 cup	33	4	2
Cabbage, common	75	0.5 cup, shredded	17	1	3
Gourd, white-flowered (calabash), cooked	146	1.0 cup	19	2	3
Balsam-pear (bitter gourd), leafy tips, cooked	58	1.0 cup	20	1	3
Balsam-pear (bitter gourd), pods, cooked	124	1.0 cup	24	3	3
Mushrooms, maitake, raw	70	1.0 cup	22	2	3
Broccoli, cooked	78	0.5 cup, chopped	27	3	3
Collards, cooked	190	1.0 cup, chopped	63	8	3
Peppers, serrano, raw	105	1.0 cup, chopped	34	4	3
Tomatoes, yellow, raw	139	1.0 cup, chopped	21	1	3
Cabbage, mustard	128	1.0 cup	36	4	3
Peppers, hot chili, red, raw	45	1.0 pepper	18	1	3
Peppers, jalapeno, raw	90	1.0 cup, sliced	26	3	3

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Sweet potato leaves, cooked	64	1.0 cup	22	1	4
Swiss chard, cooked	175	1.0 cup, chopped	35	4	4
Peppers, hot chili, green, raw	45	1.0 pepper	18	1	4
Fennel, bulb, raw	87	1.0 cup, sliced	27	3	4
Beet greens, cooked	144	1.0 cup	39	4	4
Cabbage, savoy, cooked	145	1.0 cup, shredded	35	4	4
Peppers, chili, green	139	1.0 cup	29	2	4
Garden cress	135	1.0 cup	31	1	4
Okra, raw	100	1.0 cup	33	3	4
Peas, edible-podded, raw	98	1.0 cup, chopped	41	3	5
Peppers, ancho, dried	17	1.0 pepper	48	4	5
Pickle relish, sweet	15	1.0 tbsp	20	0	5
Summer squash, all varieties, cooked	180	1.0 cup, sliced	36	3	5
Eggplant, cooked	99	1.0 cup	33	3	6
Mushrooms, oyster, raw	148	1.0 large	49	3	6
Edamame, cooked	155	1.0 cup	188	8	6
Turnips, raw	130	1.0 cup, cubes	36	2	6
Brussels sprouts, cooked	155	1.0 cup	65	6	7
Tomatoes, crushed	121	0.5 cup	39	2	7
Artichokes, (globe or french), cooked	120	1.0 artichoke, medium	61	7	7
Celeriac, cooked	155	1.0 cup pieces	42	2	7
Tomato juice	243	1.0 cup	41	1	8
Lotus root, cooked	60	0.5 cup	40	2	8
Tomatoes, cooked	240	1.0 cup	43	2	8
Leeks, (bulb and lower leaf-portion), cooked	124	1.0 leek	38	1	8
Carrots, raw	128	1.0 cup chopped	52	4	9
Rutabagas, raw	140	1.0 cup, cubes	52	3	9
Beets, raw	136	1.0 cup	58	4	9
Kohlrabi, cooked	165	1.0 cup slices	48	2	9
Tomato products, sauce	245	1.0 cup	59	4	9

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Mountain yam, hawaii, raw	68	0.5 cup, cubes	46	2	9
Eggplant, pickled	136	1.0 cup	67	3	10
Lotus root, raw	81	10.0 slices	60	4	10
Baked potatoes, flesh and skin	61	0.5 cup	57	1	12
Onions, raw	160	1.0 cup, chopped	64	3	12
Peas, green, raw	145	1.0 cup	117	8	13
Pumpkin	245	1.0 cup	83	7	13
Sweet yellow corn, cooked	150	1.0 cup	96	3	17
Parsnips, raw	133	1.0 cup slices	100	7	17
Sweet potato, cooked	114	1.0 medium	103	4	20
Carrot juice	236	1.0 cup	94	2	20
Sweet white corn, cooked	164	1.0 cup	110	3	20
Gourd, dishcloth (towelingourd), cooked	178	1.0 cup	100	5	20
Wasabi root, raw	130	1.0 cup, sliced	142	10	21
Seaweed, spirulina, dried	112	1.0 cup	325	4	23
Taro, raw	104	1.0 cup, sliced	116	4	23
Jerusalem-artichokes, raw	150	1.0 cup slices	110	2	24
Burdock root, cooked	125	1.0 cup	110	2	24
Cowpeas (blackeyes)	165	1.0 cup	160	8	25
Succotash (corn and limas), cooked	170	1.0 cup	158	7	27

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Appendix B: Tables of low-carb foods rich in commonly underconsumed nutrients

Table 1: Low-carb foods rich in calcium

DESCRIPTION	GRAMS	SERVING	CALCIUM (mg)	NET CARBS
Tofu	126	0.5 cup	861	1
Queso chihuahua	132	1.0 cup, diced	859	7
Parmesan	100	1.0 cup	853	14
Feta	150	1.0 cup, crumbled	740	6
Mozzarella, whole milk	112	1.0 cup, shredded	566	3
Sheeps milk	245	1.0 cup	473	13
Almond milk	240	8.0 fl oz	451	22
Almonds	138	1.0 cup whole kernels	370	14
Buttermilk	30	0.25 cup	355	15
Milk, fat-free	246	1.0 cup	352	14
Milk, 2%	246	1.0 cup	352	14
Orange juice, fortified	249	1.0 cup	349	27
Milk, 1%	246	1.0 cup	349	14
Ricotta, part skim milk	124	0.5 cup	337	6
Yogurt, plain, low fat	170	6.0 oz	311	12
Milk, whole	244	1.0 cup	290	11
Chocolate milk	250	1.0 cup	280	24
Soybeans	180	1.0 cup	261	12
Ricotta, whole milk	124	0.5 cup	255	9
Turnip greens	164	1.0 cup	249	3
Pink salmon	85	3.0 oz	241	0
Cowpeas (blackeyes)	165	1.0 cup	211	25
Yogurt, plain, whole milk	170	6.0 oz	206	8
Taro, tahitian	137	1.0 cup slices	204	9
Pork frankfurter	76	1.0 link	203	0
Whey protein powder isolate	30	1.0 scoop	200	8

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 2: Low-carb foods rich in fiber

DESCRIPTION	GRAMS	SERVING	FIBER (mg)	NET CARBS
Passion-fruit (granadilla), purple	236	1.0 cup	25	31
Feijoa	243	1.0 cup, pureed	16	21
Black turtle beans	185	1.0 cup	15	30
Elderberries	145	1.0 cup	10	16
Avocados	150	1.0 cup, cubes	10	3
Winter hubbard squash	205	1.0 cup, cubes	10	12
Lima beans	170	1.0 cup	9	31
Guavas	165	1.0 cup	9	15
Cowpeas (blackeyes)	165	1.0 cup	8	25
Raspberries	123	1.0 cup	8	7
Loganberries	147	1.0 cup	8	11
Blackberries	144	1.0 cup	8	6
Rowal	114	0.5 cup	7	20
Boysenberries	132	1.0 cup	7	9
Winter butternut squash	205	1.0 cup, cubes	7	15
Gooseberries	150	1.0 cup	7	9
Winter acron squash	245	1.0 cup, mashed	6	15
Blueberries	140	1.0 cup, frozen	6	13
Japanese persimmons	168	1.0 fruit	6	25
Abiyuch	114	0.5 cup	6	14
Edamame	118	1.0 cup	6	3
Broccoli	184	1.0 cup	6	4
Figs	248	1.0 cup	6	29
Kiwifruit	180	1.0 cup, sliced	5	21
Turnip greens	144	1.0 cup, chopped	5	1
Currants, red and white	112	1.0 cup	5	11
Tomatoes	240	1.0 cup	5	4
Coconut meat, dried and unsweetened	28	1.0 oz	5	2

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Valencias oranges	180	1.0 cup sections	5	17
Pears	140	1.0 cup, slices	4	17
Green snap beans	135	1.0 cup	4	5
Sweet yellow corn	165	1.0 cup	4	28
Asparagus	242	1.0 cup	4	2
Sunflower seed kernels	34	1/4 cup	4	3
Beets	136	1.0 cup	4	9
Almonds	35	1/4 cup	4	3
Carambola (starfruit)	132	1.0 cup, cubes	4	5
Spinach	234	1.0 cup	4	3
Grapefruit, all varieties	230	1.0 cup sections, with juice	4	21
Sweet cherries	248	1.0 cup, pitted	4	25
Carrots	128	1.0 cup chopped	4	9
Tangerines (mandarin oranges)	195	1.0 cup, sections	4	23
Peaches	244	1.0 cup	3	12
Peanuts	37	1/4 cup	3	5
Tofu	126	0.5 cup	3	1
Cardoon	178	1.0 cup, shredded	3	4
Hazelnuts or filberts	29	1/4 cup	3	2
Portabella mushrooms	121	1.0 cup sliced	3	3
Summer squash	192	1.0 cup slices	3	8
Onions	160	1.0 cup, chopped	3	12
Mangos	165	1.0 cup pieces	3	22
Papayas	145	1.0 cup	3	13
Stewed tomatoes	255	1.0 cup	3	13
Sour cherries	155	1.0 cup	3	15
Litchis	190	1.0 cup	3	29
Celery	150	1.0 cup, diced	2	4
Dark Chocolate (60–69%)	28	1.0 oz	2	13
Apples	173	1.0 cup slices	2	19
Burdock root	125	1.0 cup	2	24

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Chanterelle mushrooms	54	1.0 cup	2	2
Pumpkin and squash seed kernels	30	1/4 cup	2	2
Butternuts	30	1/4 cup	1	2

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 3: Low-carb foods rich in folate (B₉)

DESCRIPTION	GRAMS	SERVING	FOLATE (mg)	NET CARBS
Cowpeas (blackeyes)	165	1.0 cup	210	25
Soybeans	180	1.0 cup	200	12
Whey protein powder isolate	86	3.0 scoop	200	25
Black beans	257	1.0 cup (8 fl oz)	170	22
Turnip greens	144	1.0 cup, chopped	170	1
Black turtle beans	185	1.0 cup	159	30
Beets, raw	136	1.0 cup	148	9
Spinach	234	1.0 cup	136	3
Protein powder soy based	45	1.0 scoop	130	10
Cardoon	178	1.0 cup, shredded	121	4
Broccoli	156	1.0 cup	105	3
Asparagus	122	0.5 cup	104	2
Green peas	145	1.0 cup	94	13
Okra	92	0.5 cup slices	92	4
Jute	87	1.0 cup	90	5
Boysenberries	132	1.0 cup, unthawed	83	9
Sunflower seed kernels	34	1/4 cup	80	3
Peanuts	36	1/4 cup	45	3
Hazelnuts or filberts	29	1/4 cup	32	4

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 4: Low-carb foods rich in iodine

DESCRIPTION	GRAMS	SERVING	IODINE (mcg)	NET CARBS
Seaweed, whole or sheet	1	1.0 g	16 to 2,984	0
Cod, baked	85	3.0 ounces	99	0
Iodized salt	1.5	1/4 teaspoon	71	0
Shrimp	85	3.0 oz	35	0
Tuna, canned in oil	85	3.0 oz	17	0
Egg, whole	50	1 large	24	1
Cheddar cheese	84	3.0 oz	36	2
Green peas	85	1/2 cup	3	9
Milk, 2%	246	1.0 cup	56	14
Lima beans	85	1/2 cup	8	15
Yogurt, plain, low-fat	226	1.0 cup	75	16

References: Pennington et al. *J Food Compos Anal.* 1995. DOI:[10.1006/jfca.1995.1014](https://doi.org/10.1006/jfca.1995.1014) • Teas et al. *Thyroid.* 2004. PMID:[15588380](https://pubmed.ncbi.nlm.nih.gov/15588380/) • Dasgupta et al. *Environ Sci Technol.* 2008. PMID:[18351111](https://pubmed.ncbi.nlm.nih.gov/18351111/)

Table 5: Low-carb foods rich in iron

DESCRIPTION	GRAMS	SERVING	IRON (mg)	NET CARBS
Oysters	85	3.0 oz	6	4
Blue mussels	150	1.0 cup	6	6
Soy based protein powder	45	1.0 scoop	5	10
Liverwurst spread	55	1/4 cup	5	2
Pumpkin and squash seed kernels	59	1/2 cup	5	5
Sunflower seed kernels	67	1/2 cup	5	6
Black turtle seeds	240	1.0 cup	5	23
Octopus	85	3.0 oz	5	2
Soybeans	180	1.0 cup	5	12
Asparagus	242	1.0 cup	4	2
Deer, top round	102	1.0 steak	4	0
Potatoes, with skin	58	1.0 skin	4	22
Passion-fruit (granadilla)	236	1.0 cup	4	31

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Spinach	234	1.0 cup	4	3
Cowpeas (blackeyes)	170	1.0 cup	4	29
Lima beans	180	1.0 cup	4	26
Veal	113	4.0 oz	3	0
Canned tomatoes	255	1.0 cup	3	13
Tofu	126	0.5 cup	3	1
Beef, shank crosscuts	85	3.0 oz	3	0
Elk, ground	95	1.0 patty	3	0
Turkey	140	1.0 cup, chopped or diced	3	0
Beef, top loin petite roast/filet	113	4.0 oz	3	0
Bison, top round	85	3.0 oz	3	0
Beef, ribeye cap steak	113	4.0 oz	3	2
Peas	144	1.0 cup	3	6
Turnip greens and turnips	163	1.0 cup	3	3
Pinto	94	10.0 oz	3	25
Frankfurter	76	1.0 link	3	0
Beef, round	85	3.0 oz	3	0
Beef, short loin	85	3.0 oz	3	0
Beef chuck, short ribs	85	3.0 oz	3	0
Hazelnuts or filberts	58	1/2 cup	3	4
Almonds	69	1/2 cup	3	7
Butternuts	60	1/2 cup	2	4

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 6: Low-carb foods rich in magnesium

DESCRIPTION	GRAMS	SERVING	MAGNESIUM (mg)	NET CARBS
Pumpkin and squash seed kernels	59	1/2 cup	325	5
Almonds	69	1/2 cup	193	7
Butternuts	60	1/2 cup	142	4
Whey protein powder isolate	56	2.0 scoop	132	17
Spinach	234	1.0 cup	131	3

Appendix B

Peanuts	73	1/2 cup	130	9
Lima beans	170	1.0 cup	126	31
Soybeans	180	1.0 cup	108	12
Hazelnuts or filberts	58	1/2 cup	94	4
Sunflower seed kernels	67	1/2 cup	87	6
Cowpeas (blackeyes)	165	1.0 cup	86	25
Black turtle beans	240	1.0 cup	84	23
Purslane	115	1.0 cup	77	4
Cardoon	178	1.0 cup, shredded	75	4
Amaranth leaves, cooked	132	1.0 cup	73	5
Tofu	126	0.5 cup	73	1
Edamame	118	1.0 cup	72	3
Taro	137	1.0 cup slices	70	9
Passion-fruit (granadilla)	236	1.0 cup	68	31
Winter squash (acorn)	245	1.0 cup, mashed	64	15
Lotus seeds	16	1/2 cup	34	10

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 7: Low-carb foods rich in potassium

DESCRIPTION	GRAMS	SERVING	POTASSIUM (mg)	NET CARBS
Pork and beef salami	100	3.0 oz	1372	15
Canned tomato puree	250	1.0 cup	1098	18
Soybeans	180	1.0 cup	970	12
Potatoes, with skin	245	1.0 cup	926	22
Black turtle beans	185	1.0 cup	801	30
Lima beans	180	1.0 cup	740	26
Winter squash (hubbard)	205	1.0 cup, cubes	734	12
Cowpeas (blackeyes)	165	1.0 cup	690	25
Winter squash (acorn)	245	1.0 cup, mashed	644	15
Chinese cabbage (pak-choi)	170	1.0 cup, shredded	631	1
Winter squash (butternut)	205	1.0 cup, cubes	582	15

Appendix B

Kiwifruit	180	1.0 cup, sliced	562	21
Whey protein isolate powder	29	3.0 scoop	250	8
Almonds	25	1/4 cup	246	4
Pumpkin and squash seed kernels	30	1/4 cup	233	2
Peanuts	37	1/4 cup	232	5
Hazelnuts or filberts	29	1/4 cup	196	2
Sunflower seed kernels	34	1/4 cup	165	3

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 8: Low-carb foods rich in sodium

DESCRIPTION	GRAMS	SERVING	SODIUM (mg)	NET CARBS
Parmesan, grated	100	1.0 cup	1804	14
Feta cheese	150	1.0 cup, crumbled	1708	6
Tomato sauce with onions	245	1.0 cup	1350	20
Pickled atlantic herring	140	1.0 cup	1218	14
Provolone cheese	132	1.0 cup, diced	960	3
Black turtle beans	240	1.0 cup	922	23
Tomato sauce with onions, green peppers, and celery	250	1.0 cup	920	18
Mozzarella, low moisture, part-skim	132	1.0 cup, diced	879	7
Cheddar cheese	132	1.0 cup, diced	862	4
Muenster cheese	132	1.0 cup, diced	829	1
Mexican, queso chihuahua	132	1.0 cup, diced	814	7
Frankfurter, pork	76	1.0 link	620	0
Canned tomatos, stewed	255	1.0 cup	564	13
Mozzarella, whole milk	112	1.0 cup, shredded	544	3
Cottage, nonfat	145	1.0 cup	539	10
Cottage, lowfat, 1% milkfat	113	4.0 oz	459	3
Blue mussels	150	1.0 cup	429	6
Cottage, lowfat, 2% milkfat	113	4.0 oz	348	5
Tuna, white	85	3.0 oz	337	0

Appendix B

Protein powder soy based	45	1.0 scoop	330	10
Blue cheese	28	1.0 oz	325	1
Salmon, pink	85	3.0 oz	324	0
Cardoon, raw	178	1.0 cup, shredded	303	4
Canned tomatos packed in tomato juice	240	1.0 cup	276	4
Pork loin	113	4.0 oz	275	0
Swiss cheese	132	1.0 cup, diced	247	2
Camembert	28	1.0 oz	239	0
Whey protein powder isolate	57	2.0 scoop	211	17

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 9: Low-carb foods rich in thiamine (B₁)

DESCRIPTION	GRAMS	SERVING	THIAMIN (mg)	NET CARBS
Kidney beans	184	1.0 cup	0.7	8
Peas and carrots	278	10.0 oz	0.6	20
Pork loin	85	3.0 oz	0.5	0
Peanuts	67	1/2 cup	0.5	6
Whey protein powder isolate	57	2.0 scoop	0.5	17
Soybeans	180	1.0 cup	0.5	12
Frankfurter, pork	76	1.0 link	0.5	0
Cowpeas (blackeyes)	170	1.0 cup	0.4	29
Pork, ground (96% lean, 4% fat)	85	3.0 oz	0.4	0
Sunflower seed kernels	135	1.0 cup	0.4	17
Burbot fish	90	1.0 fillet	0.4	0
Hazelnuts or filberts	58	1/2 cup	0.4	4
Pork shoulder	85	3.0 oz	0.4	0
Black turtle beans	240	1.0 cup	0.3	23
Yellowtail fish	146	0.5 fillet	0.3	0
Winter squash (acorn)	245	1.0 cup, mashed	0.3	15
Blue mussels	150	1.0 cup	0.2	6
Lima beans	170	1.0 cup	0.2	31

Appendix B

Tomato juice	243	1.0 cup	0.2	8
Butternuts	60	1/2 cup	0.2	4
Feta cheese	150	1.0 cup, crumbled	0.2	6

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 10: Low-carb foods rich in vitamin A

DESCRIPTION	GRAMS	SERVING	VITAMIN A (mcg RAE)	NET CARBS
Liverwurst spread	55	0.25 cup	2250	2
Peas and carrots, mixed	278	10 oz	1323	20
Winter squash (butternut)	205	1.0 cup, cubes	1144	15
Carrots, raw	128	1.0 cup chopped	1069	9
Spinach	234	1.0 cup	945	3
Winter squash (hubbard)	205	1.0 cup, cubes	687	12
Turnip greens	144	1.0 cup, chopped	549	1
Whey protein powder isolate	57	2.0 scoop	495	17
Cheddar cheese	132	1.0 cup, diced	445	4
Collards	95	10 oz	436	3
Muenster cheese	132	1.0 cup, diced	393	1
Swiss cheese	132	1.0 cup, diced	380	2
Pickled atlantic herring	140	1.0 cup	361	14
Chinese cabbage (pak-choi)	170	1.0 cup, shredded	360	1
Provolone cheese	132	1.0 cup, diced	312	3
Mozzarella, low moisture, part-skim	132	1.0 cup, diced	300	7
Parmesan, grated	100	1.0 cup	262	14
Heavy whipping cream	60	1/2 cup	247	2
Mozzarella, whole milk	112	1.0 cup, shredded	200	3
Feta cheese	150	1.0 cup, crumbled	188	6
Bluefish	150	1.0 fillet	180	0
Light whipping cream	60	1/2 cup	168	2

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>

Table 11: Low-carb foods rich in vitamin C

DESCRIPTION	GRAMS	SERVING	VITAMIN C (mg)	NET CARBS
Currants, european black	112	1.0 cup	203	17
Tomato juice	243	1.0 cup	170	8
Kiwifruit	180	1.0 cup, sliced	167	21
Litchis	190	1.0 cup	136	29
Papayas	145	1.0 cup 1" pieces	88	13
Oranges	180	1.0 cup sections	87	17
Brussels sprouts, raw	88	1.0 cup	75	5
Broccoli	184	1.0 cup	74	4
Grapefruit, raw, pink, and red	230	1.0 cup sections, with juice	72	21
Passion-fruit (granadilla)	236	1.0 cup	71	31
Mangos	165	1.0 cup pieces	60	22
Carissa (natal-plum)	150	1.0 cup slices	57	20
Cauliflower	64	1.0 cup	56	2
Tangerines (mandarin oranges)	195	1.0 cup, sections	52	23
Lemon juice	122	1/2 cup	47	8
Currants, red and white	112	1.0 cup	46	11
Carambola (starfruit)	132	1.0 cup, cubes	45	5
Asparagu	242	1.0 cup	45	2
Chinese cabbage (pak-choi)	170	1.0 cup, shredded	44	1
Gooseberries	150	1.0 cup	42	9
Turnip greens	144	1.0 cup, chopped	40	1
Collards	95	10.0 oz	38	3
Lime juice	121	1/2 cup	36	10
Garden cress	50	1.0 cup	35	2
Peas	144	1.0 cup	32	6
Spinach	234	1.0 cup	32	3
Winter squash (butternut)	205	1.0 cup, cubes	31	15
Soybeans	180	1.0 cup	31	12

Appendix B

Rowal	114	0.5 cup	29	20
Blackberry juice	250	1.0 cup	28	19
Canned tomatos	250	1.0 cup	27	18
Summer squash (zucchini)	180	1.0 cup, sliced	23	3
Loganberries	147	1.0 cup, unthawed	23	11
Peas and carrots	278	10.0 oz	23	20
Sweet potato	114	1.0 medium	22	20
Whey protein powder isolate	57	2.0 scoop	20	17
Kale	21	1.0 cup	20	0
Lima beans	170	1.0 cup	17	31
Radishes	116	1.0 cup slices	17	2
Yardlong beans	104	1.0 cup slices	17	10
Winter squash (acorn)	245	1.0 cup, mashed	16	15
Groundcherries (cape-gooseberries or poha)	140	1.0 cup	15	16
Winter squash (hubbard)	236	1.0 cup, mashed	15	8

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>.

Table 12: Low-carb foods rich in vitamin D

DESCRIPTION	GRAMS	SERVING	VITAMIN D (IU)	NET CARBS
Swordfish	85	3.0 oz	566	0
Rainbow trout	71	1.0 fillet	539	0
Salmon, pink	85	3.0 oz	493	0
Flatfish	127	1.0 fillet	177	0
Pickled atlantic herring	140	1.0 cup	158	14
Tilapia	116	1.0 fillet	144	0
Chanterelle mushrooms	54	1.0 cup	114	2
Milk, 1% with added vitamin A and vitamin D	246	1.0 cup	98	14
Milk, 2% with added vitamin A and vitamin D	246	1.0 cup	98	14
Mackerel	28	1.0 oz	83	0
Orange juice, with added calcium and vitamin D	125	1/2 cup	50	14

Appendix B

Pork leg	135	1.0 cup, diced	49	0
Atlantic herring	28	1.0 oz	47	0
Pork shoulder	85	3.0 oz	46	0
Egg, whole	50	1.0 large	41	0
Atlantic cod	85	3.0 oz	40	0
Pork loin	85	3.0 oz	40	0

Reference: USDA Food Composition Databases. Accessed July 30, 2019. <https://ndb.nal.usda.gov/ndb/>.

References

Chapter 1: The Basics of Keto

1. Westman et al. *Am J Clin Nutr.* 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17684196>
2. Wylie-Rosett et al. *Curr Diab Rep.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23266565>
3. Payne et al. *Epilepsia.* 2011. <https://www.ncbi.nlm.nih.gov/pubmed/22004525>
4. Gibson et al. *Obes Rev.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25402637>
5. Johnstone. “7 - Protein and Satiety” in *Satiation, Satiety and the Control of Food Intake.* (eds. Blundell & Bellisle) 2013. <https://doi.org/10.1533/9780857098719.3.128>
6. Stubbs et al. *Obesity.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29105987>
7. Bueno et al. *Br J Nutr.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23651522>
8. Hall et al. *Lancet.* 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21872751>
9. Robinson & Williamson. *Physiol Rev.* 1980. <https://www.ncbi.nlm.nih.gov/pubmed/6986618>
10. Capling et al. *Nutrients.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29207495>
11. Anderson. *Obesity.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26524104>
12. Neal. *Dietary Treatment of Epilepsy: Practical Implementation of Ketogenic Therapy.* 2012. <https://doi.org/10.1002/9781118702772>
13. Urbain & Bertz. *Nutr Metab.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27822291>

Chapter 2: Eating Keto

1. Dreher & Davenport. *Crit Rev Food Sci Nutr.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23638933>
2. Institute of Medicine et al. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids.* 2005. <https://doi.org/10.17226/10490>
3. Athinarayanan et al. *Front Endocrinol.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31231311>
4. Kosinski & Jornayvaz. *Nutrients.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28534852>
5. World Health Organization & Brouwer. *Effect of Trans-Fatty Acid Intake on Blood Lipids and Lipoproteins: A Systematic Review and Meta-Regression Analysis.* 2016. <https://apps.who.int/iris/handle/10665/246109>
6. Gayet-Boyer et al. *Br J Nutr.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/25345440>
7. Wang et al. *J Am Heart Assoc.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26790695>
8. Allen et al. *Food Chem Toxicol.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27751858>
9. Center for Food Safety & Nutrition. *Final Determination Regarding Partially Hydrogenated Oils.* U.S. Food and Drug Administration. <https://www.fda.gov/food/food-additives-petitions/final-determination-regarding-partially-hydrogenated-oils-removing-trans-fat>
10. Humayun et al. *Am J Clin Nutr.* 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17921376>
11. Rafii et al. *J Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26962173>

References

12. Rafii et al. *J Nutr*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25320185>
13. Tang et al. *Am J Clin Nutr*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24429540>
14. Phillips & Van Loon. *J Sports Sci*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/22150425>
15. Helms et al. *Int J Sport Nutr Exerc Metab*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24092765>
16. Aragon et al. *J Int Soc Sports Nutr*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28630601>
17. Helms et al. *J Int Soc Sports Nutr*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24864135>
18. Jungas et al. *Physiol Rev*. 1992. <https://www.ncbi.nlm.nih.gov/pubmed/1557428>
19. Fleming et al. *Int J Sport Nutr Exerc Metab*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/14967870>
20. Volek et al. *Metabolism*. 2002. <https://doi.org/10.1053/meta.2002.32037>
21. Burke et al. *J Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28012184>
22. Phinney et al. *Metabolism*. 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6865776>
23. Wilson et al. *J Strength Cond Res*. 2017. <https://doi.org/10.1519/JSC.0000000000001935>
24. Heatherly et al. *Med Sci Sports Exercise*. 2018. <https://doi.org/10.1249/MSS.0000000000001477>
25. Cipryan et al. *J Sports Sci Med*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29769827>
26. McSwiney et al. *Metabolism*. 2018. <https://doi.org/10.1016/j.metabol.2017.10.010>
27. Greene et al. *J Strength Cond Res*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30335720>
28. Brinkworth et al. *Obesity*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19373224>
29. Noakes et al. *Nutr Metab*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16403234>
30. Johnston et al. *Am J Clin Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16685046>
31. Johnstone et al. *Am J Clin Nutr*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18175736>
32. Meckling et al. *J Clin Endocrinol Metab*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15181047>
33. Kephart et al. *Sport Science*. 2018. <https://doi.org/10.3390/sports6010001>
34. Brehm et al. *J Clin Endocrinol Metab*. 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15598683>
35. Choi et al. *Nutrients*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30513970>
36. Hall et al. *Am J Clin Nutr*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27385608>
37. Robinson & Williamson. *Physiol Rev*. 1980. <https://www.ncbi.nlm.nih.gov/pubmed/6986618>
38. Carnauba et al. *Nutrients*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28587067>
39. Remer. *Eur J Nutr*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11842946>
40. Barzel & Massey. *J Nutr*. 1998. <https://www.ncbi.nlm.nih.gov/pubmed/9614169>
41. Schwingshackl & Hoffmann. *PLoS One*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24852037>
42. Hunt et al. *Am J Clin Nutr*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19279077>
43. Shams-White et al. *Am J Clin Nutr*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28404575>
44. Calvez et al. *Eur J Clin Nutr*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22127335>
45. Fenton et al. *J Bone Miner Res*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19419322>

References

46. von Herrath et al. *Blood Purif.* 1988. <https://www.ncbi.nlm.nih.gov/pubmed/3207474>
47. Brenner et al. *N Engl J Med.* 1982. <https://www.ncbi.nlm.nih.gov/pubmed/7050706>
48. Devries et al. *J Nutr.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30383278>
49. Levey et al. *Am J Kidney Dis.* 1996. <https://www.ncbi.nlm.nih.gov/pubmed/8629624>
50. Levey. *J Am Soc Nephrol.* 1996. <https://www.ncbi.nlm.nih.gov/pubmed/8989740>
51. Knight et al. *Ann Intern Med.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12639078>
52. Jenkins et al. *BMJ Open.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24500611>
53. Babayan. *J Am Oil Chem Soc.* 1981. <https://doi.org/10.1007/BF02666072>
54. DiNicolantonio & O'Keefe. *Mo Med.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/30228616>
55. Wallace. *J Am Coll Nutr.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30395784>
56. Sigalet et al. *JPEN J Parenter Enteral Nutr.* 1997. <https://www.ncbi.nlm.nih.gov/pubmed/9323689>
57. Sigalet & Martin. *J Pediatr Surg.* 1999. <https://www.ncbi.nlm.nih.gov/pubmed/10022140>
58. Tsuji et al. *J Nutr.* 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11694608>
59. Van Wymelbeke et al. *Am J Clin Nutr.* 1998. <https://www.ncbi.nlm.nih.gov/pubmed/9701177>
60. Eyres et al. *Nutr Rev.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26946252>
61. Vincent et al. *Am J Clin Nutr.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30596814>
62. Shin et al. *Am J Clin Nutr.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23676423>
63. Chen et al. *Eur J Clin Nutr.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23169473>
64. Abete et al. *Br J Nutr.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24932617>
65. Micha et al. *Circulation.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20479151>
66. Micha et al. *Curr Atheroscler Rep.* 2012. <https://www.ncbi.nlm.nih.gov/pubmed/23001745>
67. Pham et al. *Jpn J Clin Oncol.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24842864>
68. Durko & Malecka-Panas. *Curr Colorectal Cancer Rep.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24659930>
69. Institute of Medicine (US) Panel on Micronutrients. Iron. 2001. <https://www.ncbi.nlm.nih.gov/books/NBK222309/>
70. Kim et al. *Metabolism.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25838035>
71. Vlassara et al. *Diabetologia.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27468708>
72. Uribarri et al. *J Am Diet Assoc.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20497781>

Chapter 3: Starting Keto

1. Kossoff & Dorward. *Epilepsia.* 2008. <https://www.ncbi.nlm.nih.gov/pubmed/19049584>
2. Hall et al. *Am J Clin Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27385608>

Chapter 4: Maintaining Keto

1. Gibson et al. *Obes Rev.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25402637>
2. Adhering to the Ketogenic Diet - Is It Easy or Hard? (Research Review). Sci-Fit. <https://sci-fit.net/adhere-ketogenic-diet/>
3. Gardner et al. *JAMA.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29466592>
4. Ebbeling et al. *BMJ.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30429127>
5. Ye et al. *J Clin Neurol.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25628734>
6. Athinarayanan et al. *Front Endocrinol.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31231311>

Chapter 5: Troubleshooting Keto

1. Stallings et al. Dietary Reference Intakes for Sodium and Potassium. 2019. <https://doi.org/10.17226/25353>
2. Tankeu et al. *J Clin Hypertens.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28466573>
3. Khan et al. *Ann Intern Med.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31284304>
4. Churuangsuk et al. *Obes Rev.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31006978>
5. Cox et al. *Cell Metab.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27475046>

Chapter 6: Health Concerns

1. Paoli & Bosco. "Chapter 25 - The Ketogenic Mediterranean Diet" in *The Mediterranean Diet.* (eds. Preedy & Watson) 2015. <https://doi.org/10.1016/B978-0-12-407849-9.00025-7>
2. Kossoff et al. *Epilepsia Open.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29881797>
3. Neal. Dietary Treatment of Epilepsy: Practical Implementation of Ketogenic Therapy. 2012. <https://doi.org/10.1002/9781118702772>

Chapter 7: Can Keto Treat ... ?

1. Włodarek. *Nutrients.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30650523>
2. Evangelidou et al. *J Child Neurol.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12693778>
3. Neumeyer et al. *J Autism Dev Disord.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23124396>
4. Klement et al. *PLoS One.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27159218>
5. Khodadadi et al. *Int J Prev Med.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28584617>
6. Martin-McGill et al. *CNS Oncol.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29658772>
7. Sremanakova et al. *J Hum Nutr Diet.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30062812>
8. Erickson et al. *Med Oncol.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28353094>
9. Wheless. *Epilepsia.* 2008. <https://www.ncbi.nlm.nih.gov/pubmed/19049574>
10. Kossoff et al. *Epilepsia Open.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29881797>

11. Martin-McGill et al. *Cochrane Database Syst Rev.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30403286>
12. Safety and Tolerability of the Ketogenic Diet in Amyotrophic Lateral Sclerosis (ALS) - Full Text View - *ClinicalTrials.gov.* <https://clinicaltrials.gov/ct2/show/study/NCT01016522>
13. McDonald & Cervenka. *Neurotherapeutics.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30225789>
14. Di Lorenzo et al. *Eur J Neurol.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25156013>
15. Di Lorenzo et al. *J Headache Pain.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27245682>
16. Vanitallie et al. *Neurology.* 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15728303>
17. Phillips et al. *Mov Disord.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30098269>

Chapter 8: Body Composition

1. Gomez-Arbelaes et al. *J Clin Endocrinol Metab.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/27754807>
2. Johnstone et al. *Am J Clin Nutr.* 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18175736>
3. Sawyer et al. *J Strength Cond Res.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23774282>
4. Colica et al. *Eur Rev Med Pharmacol Sci.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28537652>
5. Nilsson. *Scand J Clin Lab Invest.* 1973. <https://www.ncbi.nlm.nih.gov/pubmed/4771101>
6. Molina & DiMaio. *Am J Forensic Med Pathol.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26108038>
7. Molina & DiMaio. *Am J Forensic Med Pathol.* 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22182984>
8. Cahill. *Annu Rev Nutr.* 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16848698>
9. Hultman. *Scand J Clin Lab Invest.* 1967. <https://www.ncbi.nlm.nih.gov/pubmed/6057997>
10. Later et al. *Eur J Clin Nutr.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20664617>
11. Fernández-Elías et al. *Eur J Appl Physiol.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25911631>
12. Acheson et al. *Am J Clin Nutr.* 1988. <https://www.ncbi.nlm.nih.gov/pubmed/3165600>
13. Knuiman et al. *Nutr Metab.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26697098>
14. Murray & Rosenbloom. *Nutr Rev.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29444266>
15. Hall et al. *Am J Clin Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27385608>
16. Sherrier & Li. *Am J Clin Nutr.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31347659>
17. White & Venkatesh. *Crit Care.* 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21489321>
18. Fournier et al. *J Sports Sci Med.* 2004. <https://www.ncbi.nlm.nih.gov/pubmed/24482591>
19. Maehlum & Hermansen. *Scand J Clin Lab Invest.* 1978. <https://www.ncbi.nlm.nih.gov/pubmed/705238>
20. Owen et al. *J Clin Invest.* 1969. <https://www.ncbi.nlm.nih.gov/pubmed/5773093>
21. Rakova et al. *J Clin Invest.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28414302>
22. Kitada et al. *J Clin Invest.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28414295>
23. Kolanowski. *Diabete Metab.* 1977. <https://www.ncbi.nlm.nih.gov/pubmed/330274>
24. Stallings et al. Dietary Reference Intakes for Sodium and Potassium. 2019. <https://doi.org/10.17226/25353>

References

25. McDermott et al. *J Athl Train*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28985128>
26. Fine & Feinman. *Nutr Metab*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15588283>
27. Ebbeling et al. *JAMA*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22735432>
28. Ervin & Ogden. *NCHS Data Brief*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23742909>
29. WWEIA Data Tables : USDA ARS. <https://www.ars.usda.gov/northeast-area/beltsville-md-bhnrc/beltsville-human-nutrition-research-center/food-surveys-research-group/docs/wweia-data-tables/>
30. Miller et al. *Circulation*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21502576>
31. World Health Organization. Guideline: Sugars Intake for Adults and Children. 2015. <https://market.android.com/details?id=book-jVkJ0DgAAQBAJ>
32. 2015-2020 Dietary Guidelines - Health.gov. <https://health.gov/dietaryguidelines/2015/guidelines/>
33. 2018 Canadian Diabetes Association Guidelines. <http://guidelines.diabetes.ca/fullguidelines>
34. Hall et al. *Cell Metab*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26278052>
35. Hall & Guo. *Gastroenterology*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28193517>
36. Krugh & Langaker. “Dual Energy Xray Absorptiometry (DEXA)” in *StatPearls*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30085584>
37. Eckhardt et al. *Obes Res*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/14694221>
38. Westerterp. *Eur J Appl Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28508113>
39. Williams et al. *Am J Clin Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16685045>
40. Atherton et al. *PLoS One*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23690932>
41. Gardner et al. *JAMA*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17341711>
42. Guo et al. *Obesity*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30672127>
43. Bueno et al. *Br J Nutr*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23651522>
44. Bazzano et al. *Ann Intern Med*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/25178568>
45. Magkos et al. *Cell Metab*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26916363>
46. Wing et al. *Diabetes Care*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21593294>
47. Johnston et al. *JAMA*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/25182101>
48. Nordmann et al. *Arch Intern Med*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16476868>
49. Shai et al. *N Engl J Med*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18635428>
50. Gardner et al. *JAMA*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29466592>
51. Athinarayanan et al. *Front Endocrinol*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31231311>
52. Gardner. *JAMA*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29998334>
53. Qi et al. *JAMA*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29998330>
54. Huang et al. *Diabetes Obes Metab*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29424957>
55. Qi et al. *J Lipid Res*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25548261>
56. Johnstone. “7 - Protein and Satiety” in *Satiation, Satiety and the Control of Food Intake*. (eds. Blundell & Bellisle) 2013. <https://doi.org/10.1533/9780857098719.3.128>

References

57. Stubbs et al. *Obesity*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29105987>
58. Heymsfield et al. *J Cachexia Sarcopenia Muscle*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24532493>
59. Bell et al. *Skeletal Muscle*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27054028>
60. Volpi et al. *Am J Clin Nutr*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12885705>
61. Kimball & Jefferson. *J Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16365087>
62. Jackman et al. *Front Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28638350>
63. Wilkinson et al. *J Physiol*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23551944>
64. Volek et al. *Metabolism*. 2002. <https://doi.org/10.1053/meta.2002.32037>
65. Jabekk et al. *Nutr Metab*. 2010. <https://doi.org/10.1186/1743-7075-7-17>
66. Wood et al. *Metab Syndr Relat Disord*. 2012. <https://doi.org/10.1089/met.2011.0104>
67. Wilson et al. *J Strength Cond Res*. 2017. <https://doi.org/10.1519/JSC.0000000000001935>
68. Gregory. *Int J Sports Exerc Med*. 2017. <https://doi.org/10.23937/2469-5718/1510054>
69. McSwiney et al. *Metabolism*. 2018. <https://doi.org/10.1016/j.metabol.2017.10.010>
70. Kephart et al. *Sport Science*. 2018. <https://doi.org/10.3390/sports6010001>
71. Vargas et al. *J Int Soc Sports Nutr*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29986720>
72. Greene et al. *J Strength Cond Res*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30335720>
73. LaFountain et al. *Mil Med*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30877806>
74. Volek et al. *Metabolism*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12077732>
75. Jabekk et al. *Nutr Metab*. 2010. <https://doi.org/10.1186/1743-7075-7-17>
76. Wilson et al. *J Strength Cond Res*. 2017. <https://doi.org/10.1519/JSC.0000000000001935>
77. Gregory. *Int J Sports Exerc Med*. 2017. <https://doi.org/10.23937/2469-5718/1510054>
78. McSwiney et al. *Metabolism*. 2018. <https://doi.org/10.1016/j.metabol.2017.10.010>
79. Roumelioti et al. *World J Nephrol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29359117>
80. Tinsley & Willoughby. *Int J Sport Nutr Exerc Metab*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26284291>
81. Cicioglu et al. *Biomed Res*. 2017. <http://www.alliedacademies.org/articles/the-effects-of-dehydration-before-competition-upon-body-compositions-leptin-hormone-and-ghrelin-hormone-among-elite-wrestlers.html>
82. Saunders et al. *Med Sci Sports Exercise*. 1998. <https://doi.org/10.1097/00005768-199806000-00017>
83. Prior et al. *J Appl Physiol*. 1997. <https://doi.org/10.1152/jappl.1997.83.2.623>
84. Paul et al. *J Am Coll Radiol*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25841864>
85. Ramsey. *Business Insider*. 2017. <https://www.businessinsider.com/how-much-an-mri-costs-by-state-2017-3>
86. Brehm et al. *J Clin Endocrinol Metab*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12679447>
87. Brehm et al. *J Clin Endocrinol Metab*. 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15598683>
88. Meckling et al. *J Clin Endocrinol Metab*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15181047>
89. Volek et al. *Nutr Metab*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15533250>
90. Noakes et al. *Nutr Metab*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16403234>

References

91. Johnston et al. *Am J Clin Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16685046>
92. Brinkworth et al. *Obesity*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19373224>
93. Choi et al. *Nutrients*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30513970>
94. Urbain et al. *Nutr Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28239404>
95. Bone et al. *Med Sci Sports Exercise*. 2017. <https://doi.org/10.1249/MSS.0000000000001174>
96. Institute of Medicine. "Protein and Amino Acids" in *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)*. 2005. <https://doi.org/10.17226/10490>
97. Humayun et al. *Am J Clin Nutr*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17921376>
98. Rafii et al. *J Nutr*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26962173>
99. Rafii et al. *J Nutr*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25320185>
100. Tang et al. *Am J Clin Nutr*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24429540>
101. Veldhorst et al. *Am J Clin Nutr*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19640952>
102. Robinson & Williamson. *Physiol Rev*. 1980. <https://www.ncbi.nlm.nih.gov/pubmed/6986618>
103. Fleming et al. *Int J Sport Nutr Exerc Metab*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/14967870>
104. Burke et al. *J Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28012184>
105. Phinney et al. *Metabolism*. 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6865776>
106. Heatherly et al. *Med Sci Sports Exercise*. 2018. <https://doi.org/10.1249/MSS.0000000000001477>
107. Cipryan et al. *J Sports Sci Med*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29769827>
108. Garthe et al. *EJSS*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23679146>
109. Roe et al. *Appetite*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22008705>
110. Hall et al. *Lancet*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21872751>
111. Schoenfeld et al. *Sports Med*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27102172>
112. Wilson et al. *J Strength Cond Res*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22002517>
113. Fisher et al. *Medicina Sportiva*. 2011. <https://doi.org/10.2478/v10036-011-0025-x>
114. McMaster et al. *Sports Med*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23529287>
115. Janssen et al. *J Am Geriatr Soc*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12028177>
116. Landi et al. *Clin Geriatr Med*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26195096>
117. Kojima. *Bone*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27321894>
118. Cheng & Chang. *J Nurs Scholarsh*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28755453>
119. Kojima. *J Epidemiol Community Health*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26933121>
120. Kojima. *Disabil Rehabil*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/27558741>
121. Kojima. *J Geriatr Phys Ther*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/27341327>
122. Harris et al. *Res Vet Sci*. 1997. <https://www.ncbi.nlm.nih.gov/pubmed/9160426>
123. Dahl. *J Sci Food Agric*. 1965. <https://www.ncbi.nlm.nih.gov/pubmed/5841078>
124. Harris et al. *Clin Sci*. 1992. <https://www.ncbi.nlm.nih.gov/pubmed/1327657>

References

125. Thomas et al. *J Acad Nutr Diet*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26920240>
126. Hirshkowitz et al. *Sleep Health*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/29073412>
127. Mah et al. *Sleep*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21731144>
128. VanHelder & Radomski. *Sports Med*. 1989. <https://www.ncbi.nlm.nih.gov/pubmed/2657963>
129. Mullington et al. *Best Pract Res Clin Endocrinol Metab*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/21112025>
130. Cote et al. *Biol Psychol*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23046906>
131. Leproult & Van Cauter. *JAMA*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21632481>
132. Penev. *Sleep*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17520786>
133. González-Santos et al. *Arch Androl*. 1989. <https://www.ncbi.nlm.nih.gov/pubmed/2757458>
134. Cortés-Gallegos et al. *Arch Androl*. 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6405703>
135. Nedeltcheva et al. *Ann Intern Med*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20921542>
136. Kelley & Kelley. *J Evid Based Med*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28276627>

Chapter 9: Strength

1. Slater & Phillips. *J Sports Sci*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21660839>
2. Gastin. *Sports Med*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11547894>
3. Smith & Hill. *Br J Sports Med*. 1991. <https://www.ncbi.nlm.nih.gov/pubmed/1839780>
4. Baker et al. *J Nutr Metab*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/21188163>
5. Garnacho-Castaño et al. *Int J Sports Med*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25680073>
6. Garnacho-Castaño et al. *Front Physiol*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31019469>
7. Albesa-Albiol et al. *PLoS One*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31112561>
8. de Sousa et al. *Int J Sports Med*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22127560>
9. Gorostiaga et al. *PLoS One*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22808209>
10. Gorostiaga et al. *PLoS One*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20976067>
11. Macdougall et al. *Can J Appl Physiol*. 1999. <https://doi.org/10.1139/h99-017>
12. Tesch et al. *Eur J Appl Physiol Occup Physiol*. 1986. <https://www.ncbi.nlm.nih.gov/pubmed/3758035>
13. Koopman et al. *Eur J Appl Physiol*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16369816>
14. Ørtenblad et al. *J Physiol*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21135051>
15. Jacobs et al. *Eur J Appl Physiol Occup Physiol*. 1981. <https://www.ncbi.nlm.nih.gov/pubmed/7194784>
16. Lambert & Flynn. *Sports Med*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12076177>
17. Capling et al. *Nutrients*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29207495>
18. LaFountain et al. *Mil Med*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30877806>
19. Wilson et al. *J Strength Cond Res*. 2017. <https://doi.org/10.1519/JSC.0000000000001935>
20. Greene et al. *J Strength Cond Res*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30335720>

21. Kephart et al. *Sport Science*. 2018. <https://doi.org/10.3390/sports6010001>
22. Gregory. *Int J Sports Exerc Med*. 2017. <https://doi.org/10.23937/2469-5718/1510054>
23. Wood et al. *Metab Syndr Relat Disord*. 2012. <https://doi.org/10.1089/met.2011.0104>
24. Paoli et al. *J Int Soc Sports Nutr*. 2012. <https://doi.org/10.1186/1550-2783-9-34>
25. Sawyer et al. *J Strength Cond Res*. 2013. <https://doi.org/10.1519/JSC.0b013e31827da314>
26. Fournier et al. *J Sports Sci Med*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/24482591>
27. Cahill. *Annu Rev Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16848698>
28. Owen et al. *J Clin Invest*. 1969. <https://www.ncbi.nlm.nih.gov/pubmed/5773093>
29. Volek et al. *Metabolism*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26892521>
30. Fournier et al. *Comp Biochem Physiol A Mol Integr Physiol*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12443931>

Chapter 10: Endurance

1. Volek et al. *EJSS*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25275931>
2. Phinney et al. *Metabolism*. 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6865776>
3. Venables et al. *J Appl Physiol*. 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15333616>
4. Volek et al. *Metabolism*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26892521>
5. Lucía et al. *Med Sci Sports Exerc*. 2000. <https://www.ncbi.nlm.nih.gov/pubmed/11039652>
6. Fernández-García et al. *Med Sci Sports Exerc*. 2000. <https://www.ncbi.nlm.nih.gov/pubmed/10795793>
7. Billat et al. *Med Sci Sports Exerc*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11740304>
8. Burke et al. *J Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28012184>
9. McLellan et al. *Can J Appl Physiol*. 1995. <https://www.ncbi.nlm.nih.gov/pubmed/7742769>
10. Faude et al. *Appl Physiol Nutr Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28128633>
11. Maughan & Leiper. *Eur J Appl Physiol Occup Physiol*. 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6686134>
12. Achten & Jeukendrup. *Nutrition*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15212756>
13. Heatherly et al. *Med Sci Sports Exercise*. 2018. <https://doi.org/10.1249/MSS.0000000000001477>
14. McSwiney et al. *Metabolism*. 2018. <https://doi.org/10.1016/j.metabol.2017.10.010>
15. Cipryan et al. *J Sports Sci Med*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29769827>
16. Dostal et al. *Front Physiol*. 2019. <https://doi.org/10.3389/fphys.2019.00912>
17. Shaw et al. *Med Sci Sports Exerc*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31033901>
18. Fleming et al. *Int J Sport Nutr Exerc Metab*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/14967870>
19. Wroble et al. *J Sports Med Phys Fitness*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/29619799>
20. Zinn et al. *J Int Soc Sports Nutr*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28706467>
21. Klement et al. *Nutrition and Medicine*. 2013. <https://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.976.601&rep=rep1&type=pdf>

References

22. Waldman et al. *J Strength Cond Res*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29076962>
23. Lambert et al. *Eur J Appl Physiol Occup Physiol*. 1994. <https://www.ncbi.nlm.nih.gov/pubmed/7851362>
24. Goedecke et al. *Metabolism*. 1999. <https://www.ncbi.nlm.nih.gov/pubmed/10599981>
25. Zajac et al. *Nutrients*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24979615>
26. Michalczyk et al. *Nutrients*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30987297>
27. O’Keeffe et al. *Nutr Res*. 1989. [https://doi.org/10.1016/S0271-5317\(89\)80027-2](https://doi.org/10.1016/S0271-5317(89)80027-2)
28. Leckey et al. *J Appl Physiol*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26586912>
29. Hawley & Leckey. *Sports Med*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26553495>
30. Krogh & Lindhard. *Biochem J*. 1920. <https://www.ncbi.nlm.nih.gov/pubmed/16742941>
31. Hoppeler. *Int J Obes Relat Metab Disord*. 1999. <https://www.ncbi.nlm.nih.gov/pubmed/10367997>
32. Leverve et al. *Novartis Found Symp*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17380791>
33. Wolfe. *Adv Exp Med Biol*. 1998. <https://www.ncbi.nlm.nih.gov/pubmed/9781322>
34. Frayn. *Acta Physiol*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20353493>
35. Romijn et al. *J Appl Physiol*. 1995. <https://www.ncbi.nlm.nih.gov/pubmed/8847257>
36. Joyner & Coyle. *J Physiol*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/17901124>
37. Daniels & Daniels. *Med Sci Sports Exerc*. 1992. <https://www.ncbi.nlm.nih.gov/pubmed/1560747>
38. LeBlanc et al. *J Physiol*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15020699>
39. Perry et al. *Appl Physiol Nutr Metab*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/19088769>
40. Stellingwerff et al. *Am J Physiol Endocrinol Metab*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16188909>
41. Peters et al. *Am J Physiol Endocrinol Metab*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11701428>

Chapter 11: Ketone and MCT Supplements

1. Cahill. *Annu Rev Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16848698>
2. Owen et al. *J Clin Invest*. 1967. <https://www.ncbi.nlm.nih.gov/pubmed/6061736>
3. Scofield et al. *Arch Biochem Biophys*. 1982. <https://www.ncbi.nlm.nih.gov/pubmed/7082002>
4. Webber & Edmond. *J Biol Chem*. 1977. <https://www.ncbi.nlm.nih.gov/pubmed/885847>
5. Stubbs et al. *Front Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29163194>
6. Senior & Loridan. *Nature*. 1968. <https://www.ncbi.nlm.nih.gov/pubmed/5659630>
7. Balasse & Féry. *Diabetes Metab Rev*. 1989. <https://www.ncbi.nlm.nih.gov/pubmed/2656155>
8. Evans et al. *J Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/27861911>
9. Cox et al. *Cell Metab*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27475046>
10. Rodger et al. 2017. <https://researchcommons.waikato.ac.nz/handle/10289/11199>
11. O’Malley et al. *Appl Physiol Nutr Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28750585>
12. Leckey et al. *Front Physiol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29109686>

References

13. Evans et al. *EJSS*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29338584>
14. Waldman et al. *Appl Physiol Nutr Metab*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29451991>
15. Dearlove et al. *Front Physiol*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30984015>
16. Evans et al. *Med Sci Sports Exercise*. 2019. <https://doi.org/10.1249/MSS.0000000000002065>
17. McDonald & Cervenka. *Brain Sci*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30096755>
18. Kovács et al. *Front Psychiatry*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31178772>
19. Martin et al. *Cochrane Database Syst Rev*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26859528>
20. Lima et al. *Clinics*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/25518023>
21. Masino & Rho. “Mechanisms of Ketogenic Diet Action” in *Jasper’s Basic Mechanisms of the Epilepsies*. (eds. Noebels, et al.) 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22787591>
22. Groleau et al. *Dev Med Child Neurol*. 2014. <https://www.ncbi.nlm.nih.gov/pubmed/24749520>
23. Bergqvist et al. *Am J Clin Nutr*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/19064531>
24. Si et al. *Exp Ther Med*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28672997>
25. Gross et al. *Trials*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30654835>
26. Di Lorenzo et al. *Eur J Neurol*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25156013>
27. Seyfried et al. *Biochim Biophys Acta*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/20804725>
28. Schwartz et al. *Front Nutr*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29536011>
29. Winter et al. *Crit Rev Oncol Hematol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28325264>
30. Schwartz et al. *Cancer Metab*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25806103>
31. Gorelick et al. *Biochim Biophys Acta*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26704177>
32. Dichgans & Leys. *Circ Res*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28154105>
33. Benedict & Grillo. *Front Neurosci*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29743868>
34. Włodarek. *Nutrients*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30650523>
35. Cunnane et al. *Front Mol Neurosci*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27458340>
36. Evans & Egan. *Med Sci Sports Exerc*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29944604>
37. Babayan. *J Am Oil Chem Soc*. 1981. <https://doi.org/10.1007/BF02666072>
38. Sigalet & Martin. *J Pediatr Surg*. 1999. <https://www.ncbi.nlm.nih.gov/pubmed/10022140>
39. Sigalet et al. *JPEN J Parenter Enteral Nutr*. 1997. <https://www.ncbi.nlm.nih.gov/pubmed/9323689>
40. Krotkiewski. *Int J Obes Relat Metab Disord*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11571605>
41. D C Harvey et al. *J Nutr Metab*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29951312>
42. Huttenlocher. *Pediatr Res*. 1976. <https://www.ncbi.nlm.nih.gov/pubmed/934725>
43. Payne et al. *Epilepsia*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/22004525>
44. Neal et al. *Epilepsia*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19054400>
45. St-Onge & Jones. *J Nutr*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/11880549>
46. Noguchi et al. *J Nutr Sci Vitaminol*. 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12775120>

References

47. Geliebter et al. *Am J Clin Nutr.* 1983. <https://www.ncbi.nlm.nih.gov/pubmed/6849272>
48. Dulloo et al. *Metabolism.* 1995. <https://www.ncbi.nlm.nih.gov/pubmed/7869927>
49. Crozier et al. *Metabolism.* 1987. <https://www.ncbi.nlm.nih.gov/pubmed/3298941>
50. Lavau & Hashim. *J Nutr.* 1978. <https://www.ncbi.nlm.nih.gov/pubmed/24679>
51. Simón et al. *J Physiol Biochem.* 2000. <https://www.ncbi.nlm.nih.gov/pubmed/11321528>
52. Baba et al. *Am J Clin Nutr.* 1982. <https://www.ncbi.nlm.nih.gov/pubmed/7072620>
53. St-Onge & Jones. *Int J Obes Relat Metab Disord.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12975635>
54. St-Onge et al. *Obes Res.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12634436>
55. St-Onge et al. *Int J Obes Relat Metab Disord.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12532160>
56. Kasai et al. *J Nutr Sci Vitaminol.* 2002. <https://www.ncbi.nlm.nih.gov/pubmed/12775122>
57. Bendixen et al. *Am J Clin Nutr.* 2002. <https://www.ncbi.nlm.nih.gov/pubmed/11756059>
58. Flatt et al. *J Clin Invest.* 1985. <https://www.ncbi.nlm.nih.gov/pubmed/3900133>
59. Hill et al. *Metabolism.* 1989. <https://www.ncbi.nlm.nih.gov/pubmed/2739575>
60. Van Wymelbeke et al. *Am J Clin Nutr.* 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11684530>
61. Alexandrou et al. *Can J Physiol Pharmacol.* 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17632585>
62. Roynette et al. *Nutr Metab Cardiovasc Dis.* 2008. <https://www.ncbi.nlm.nih.gov/pubmed/17368874>
63. Tsuji et al. *J Nutr.* 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11694608>
64. Rudkowska et al. *Metabolism.* 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16483884>
65. Bourque et al. *Metabolism.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12800105>
66. Van Wymelbeke et al. *Am J Clin Nutr.* 1998. <https://www.ncbi.nlm.nih.gov/pubmed/9701177>
67. Coleman et al. *Nutr Res.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27188898>
68. St-Onge et al. *Eur J Clin Nutr.* 2014. <https://www.ncbi.nlm.nih.gov/pubmed/25074387>
69. Maher et al. *Nutrients.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31319633>
70. Kinsella et al. *Physiol Behav.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28689741>
71. Clegg et al. *Eur J Nutr.* 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23179202>

Chapter 12: Blood Sugar

1. CDC. Diabetes Statistic Report. Centers for Disease Control and Prevention. <https://www.cdc.gov/features/diabetes-statistic-report/index.html>
2. Roglic & Others. *International Journal of Noncommunicable Diseases.* 2016. <https://www.who.int/diabetes/global-report/en/>
3. Huang et al. *Biomed Res Int.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29238721>
4. Yuan et al. *Redox Biol.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30384259>
5. Lee & Chan. *Chin Med J.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26021514>
6. Hamed. *Expert Rev Clin Pharmacol.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28276776>

References

7. Einarson et al. *Cardiovasc Diabetol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29884191>
8. Zhang et al. *Diabetes Res Clin Pract*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28088029>
9. Yue et al. *Medicine*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27149468>
10. Tsilidis et al. *BMJ*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25555821>
11. Gummesson et al. *Diabetes Obes Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28417575>
12. Franz et al. *J Acad Nutr Diet*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25935570>
13. Galaviz et al. *Diabetes Care*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29934481>
14. Zinman et al. *Diabetes Care*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28931706>
15. Westman et al. *Expert Rev Endocrinol Metab*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30289048>
16. Myette-Côté et al. *J Physiol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29446830>
17. Ye et al. *J Endocr Soc*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30834357>
18. Yazıcı & Sezer. *Adv Exp Med Biol*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28585204>
19. Kim et al. *Heart Fail Clin*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22999242>
20. Hernandez et al. *Am J Clin Nutr*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20107198>
21. Rosenbaum et al. *Obesity*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31067015>
22. Vazquez & Kazi. *Metabolism*. 1994. <https://www.ncbi.nlm.nih.gov/pubmed/7934983>
23. Barbosa-Yañez et al. *Nutrients*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30513768>
24. Schwingshackl & Hoffmann. *Br J Nutr*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23829973>
25. Jovanovski et al. *Clin Nutr Res*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25954727>
26. Flammer et al. *Circulation*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/22869857>
27. Ras et al. *Int J Cardiol*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23041097>
28. Gimbrone & García-Cardena. *Circ Res*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26892962>
29. Gardner et al. *Am J Clin Nutr*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20573800>
30. Calton. *J Int Soc Sports Nutr*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20537171>
31. Gommers et al. *Diabetes*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26696633>
32. Veronese et al. *Eur J Clin Nutr*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27530471>
33. Verma & Garg. *J Hum Nutr Diet*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28150351>
34. Simental-Mendía et al. *Pharmacol Res*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27329332>
35. Dreher. "Fiber in Type 2 Diabetes Prevention and Management" in *Dietary Fiber in Health and Disease*. (ed. Dreher) 2018. https://doi.org/10.1007/978-3-319-50557-2_11
36. Reynolds et al. *Lancet*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30638909>
37. McRae. *J Chiropr Med*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29628808>
38. Chen et al. *Nutrients*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29278406>
39. Jovanovski et al. *Diabetes Care*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30617143>
40. Myette-Côté et al. *Am J Physiol Regul Integr Comp Physiol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30303707>

References

41. Monnier et al. *JAMA*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16609090>
42. Ceriello & Ihnat. *Diabet Med*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20653741>
43. Brownlee. *Diabetes*. 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15919781>
44. Francois et al. *Am J Physiol Heart Circ Physiol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29030343>
45. Miyashita et al. *Diabetes Res Clin Pract*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15331203>
46. Keogh et al. *Am J Clin Nutr*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18326593>
47. *Diabetes Care*. 2019. http://care.diabetesjournals.org/content/42/Supplement_1
48. Hyde et al. *JCI Insight*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31217353>
49. Kirk et al. *Gastroenterology*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19208352>
50. Noakes et al. *Nutr Metab*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16403234>
51. Phillips et al. *Hypertension*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18195164>
52. Volek et al. *J Am Coll Nutr*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15047685>
53. Varady et al. *Nutr J*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21251283>
54. Johnston et al. *Am J Clin Nutr*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16685046>
55. Athinarayanan et al. *Front Endocrinol*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31231311>
56. Salgado et al. *Arq Gastroenterol*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20721461>
57. Song et al. *Diabetes Care*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17468352>
58. Brehm et al. *J Clin Endocrinol Metab*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12679447>
59. Sharman et al. *J Nutr*. 2004. <https://www.ncbi.nlm.nih.gov/pubmed/15051841>
60. Partsalaki et al. *J Pediatr Endocrinol Metab*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/23155696>
61. Goday et al. *Nutr Diabetes*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27643725>
62. Cohen et al. *J Nutr*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30137481>
63. Urbain et al. *Nutr Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28239404>
64. Yancy et al. *Arch Intern Med*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20101008>
65. Boden et al. *Ann Intern Med*. 2005. <https://www.ncbi.nlm.nih.gov/pubmed/15767618>
66. Foster et al. *N Engl J Med*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12761365>
67. Schofield et al. *Diabetes Ther*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31004282>
68. Ranjan et al. *Diabetes Obes Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28345762>
69. Lennerz et al. *Pediatrics*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29735574>
70. Leow et al. *Diabet Med*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29737587>
71. Padilla et al. *Eur J Appl Physiol*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16896723>
72. Johnson et al. *Appl Physiol Nutr Metab*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21574775>
73. Tucker et al. *Am J Physiol Heart Circ Physiol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29101171>
74. Das et al. *Front Physiol*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29568273>

75. Ghanim et al. *J Clin Endocrinol Metab.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/27906549>
76. Fahs et al. *Appl Physiol Nutr Metab.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20555373>
77. Palomer et al. *Trends Endocrinol Metab.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29290500>
78. Brum et al. *Am J Cardiol.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30078477>
79. Jovanovski et al. *Am J Clin Nutr.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30239559>
80. Ho et al. *Br J Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27724985>
81. Ho et al. *Eur J Clin Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27273067>
82. Hollænder et al. *Am J Clin Nutr.* 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26269373>
83. McNabney & Henagan. *Nutrients.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29231905>
84. Chambers et al. *Curr Nutr Rep.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/30264354>
85. Deopurkar et al. *Diabetes Care.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20067961>
86. Ghanim et al. *Am J Clin Nutr.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20200256>
87. Ghanim et al. *J Clin Endocrinol Metab.* 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21289251>
88. Uribarri et al. *J Am Diet Assoc.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20497781>
89. Baye et al. *Sci Rep.* 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28536448>

Chapter 13: Warnings

1. Kossoff et al. *Epilepsia Open.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29881797>
2. Neal. *Dietary Treatment of Epilepsy: Practical Implementation of Ketogenic Therapy.* 2012. <https://doi.org/10.1002/9781118702772>
3. McGhee & Katyal. *J Am Diet Assoc.* 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11209590>
4. McElhiney et al. *Int J Pharm Compd.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/23965366>
5. Feldstein. *Pediatrics.* 1996. <https://www.ncbi.nlm.nih.gov/pubmed/8632936>
6. Center for Food Safety. *High-Intensity Sweeteners.* <https://www.fda.gov/food/food-additives-petitions/high-intensity-sweeteners>
7. Julian Bakery, Inc. - 07/23/2014. U.S. Food and Drug Administration. <https://www.fda.gov/inspections-compliance-enforcement-and-criminal-investigations/warning-letters/julian-bakery-inc-07232014>
8. Kohmoto et al. *Biosci Biotechnol Biochem.* 1992. <https://www.ncbi.nlm.nih.gov/pubmed/27280817>
9. Oku & Nakamura. *Eur J Clin Nutr.* 2003. <https://www.ncbi.nlm.nih.gov/pubmed/12947435>
10. Gourineni et al. *Nutrients.* 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29510490>
11. Calton. *J Int Soc Sports Nutr.* 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20537171>
12. Vici et al. *Clin Nutr.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27211234>
13. Craig. *Am J Clin Nutr.* 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19279075>
14. Genoni et al. *Nutrients.* 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27223304>
15. Churuangsuk et al. *Obes Rev.* 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31006978>

References

16. Zinn et al. *BMJ Open*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29439004>
17. Institute of Medicine et al. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. 2000. <https://doi.org/10.17226/6015>
18. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium. Dietary Reference Intakes for Calcium and Vitamin D. ((eds. Ross, et al.)). 2011. <https://doi.org/10.17226/13050>
19. Tankeu et al. *J Clin Hypertens*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28466573>
20. Khan et al. *Ann Intern Med*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31284304>
21. Institute of Medicine et al. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids. 2005. <https://doi.org/10.17226/10490>
22. McGill et al. *Nutrients*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25671414>
23. Ma et al. *J Am Diet Assoc*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17904938>
24. Miller et al. *Metab Syndr Relat Disord*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/18370655>
25. Dreher. “Fiber in Type 2 Diabetes Prevention and Management” in *Dietary Fiber in Health and Disease*. (ed. Dreher) 2018. https://doi.org/10.1007/978-3-319-50557-2_11
26. Reynolds et al. *Lancet*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30638909>
27. Evert et al. *Diabetes Care*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31000505>
28. Ho et al. *Am J Clin Nutr*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28356275>
29. Gibb et al. *Am J Clin Nutr*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26561625>
30. Desai et al. *Cell*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27863247>
31. Jovanovski et al. *Diabetes Care*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/30617143>
32. Mardinoglu et al. *Cell Metab*. 2018. <https://www.ncbi.nlm.nih.gov/pubmed/29456073>
33. van der Reijden et al. *Best Pract Res Clin Endocrinol Metab*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/29221567>
34. Institute of Medicine et al. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. 2002. <https://doi.org/10.17226/10026>
35. Kell. *Arch Toxicol*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20967426>
36. Fang et al. *Nutr Metab Cardiovasc Dis*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25439662>
37. Qiao & Feng. *Cancer Causes Control*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23568532>
38. Gommers et al. *Diabetes*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26696633>
39. Veronese et al. *Eur J Clin Nutr*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27530471>
40. Verma & Garg. *J Hum Nutr Diet*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28150351>
41. Simental-Mendía et al. *Pharmacol Res*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27329332>
42. Institute of Medicine et al. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. 1999. <https://doi.org/10.17226/5776>
43. Firoz & Graber. *Magnes Res*. 2001. <https://www.ncbi.nlm.nih.gov/pubmed/11794633>
44. Walker et al. *Magnes Res*. 2003. <https://www.ncbi.nlm.nih.gov/pubmed/14596323>

References

45. Lindberg et al. *J Am Coll Nutr*. 1990. <https://www.ncbi.nlm.nih.gov/pubmed/2407766>
46. Geller et al. *N Engl J Med*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26465986>
47. Food et al. *Current good manufacturing practice for finished pharmaceuticals Part*. 2017. <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=216.24>
48. Stallings et al. Dietary Reference Intakes for Sodium and Potassium. 2019. <https://doi.org/10.17226/25353>
49. Schreiber et al. *Biochim Biophys Acta*. 2012. <https://www.ncbi.nlm.nih.gov/pubmed/21586336>
50. O'Byrne & Blaner. *J Lipid Res*. 2013. <https://www.ncbi.nlm.nih.gov/pubmed/23625372>
51. Grune et al. *J Nutr*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20980645>
52. Institute of Medicine (US) Panel on Dietary Antioxidants and Related Compounds. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. 2014. <https://doi.org/10.17226/9810>
53. A Closer Look at Current Intakes and Recommended Shifts - 2015-2020 Dietary Guidelines - Health.gov. <https://health.gov/dietaryguidelines/2015/guidelines/chapter-2/a-closer-look-at-current-intakes-and-recommended-shifts/>
54. Norman. *Am J Clin Nutr*. 2008. <https://www.ncbi.nlm.nih.gov/pubmed/18689389>
55. Bouillon et al. *Endocr Rev*. 1995. <https://www.ncbi.nlm.nih.gov/pubmed/7781594>
56. Christakos et al. *Physiol Rev*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/26681795>
57. Gardner et al. *Am J Clin Nutr*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20573800>
58. Perticone et al. *Molecules*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31323907>
59. Xiang et al. *Photochem Photobiol Sci*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/26548800>
60. Groesbeck et al. *Dev Med Child Neurol*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/17109786>
61. McNally et al. *Pediatrics*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19596731>
62. Cross et al. *Arch Dis Child*. 2010. <https://www.ncbi.nlm.nih.gov/pubmed/20418339>
63. Tabas et al. *Circulation*. 2007. <https://www.ncbi.nlm.nih.gov/pubmed/17938300>
64. Steinberg. *J Lipid Res*. 2009. <https://www.ncbi.nlm.nih.gov/pubmed/19011257>
65. Feig et al. *Coron Artery Dis*. 2016. <https://www.ncbi.nlm.nih.gov/pubmed/27414247>
66. Athinarayanan et al. *Front Endocrinol*. 2019. <https://www.ncbi.nlm.nih.gov/pubmed/31231311>
67. Kosinski & Jornayvaz. *Nutrients*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28534852>
68. Lusic. *Nature*. 2000. <https://www.ncbi.nlm.nih.gov/pubmed/11001066>
69. Wibisono et al. *J Pediatr*. 2015. <https://www.ncbi.nlm.nih.gov/pubmed/25649120>
70. McDermott et al. *J Athl Train*. 2017. <https://www.ncbi.nlm.nih.gov/pubmed/28985128>
71. Hall et al. *Lancet*. 2011. <https://www.ncbi.nlm.nih.gov/pubmed/21872751>
72. Keene. *Pediatr Neurol*. 2006. <https://www.ncbi.nlm.nih.gov/pubmed/16814077>