



MENNO HENSELMANS

Science to master your physique



DIETARY FAT

➤ Lecture [optional]

Dietary fat

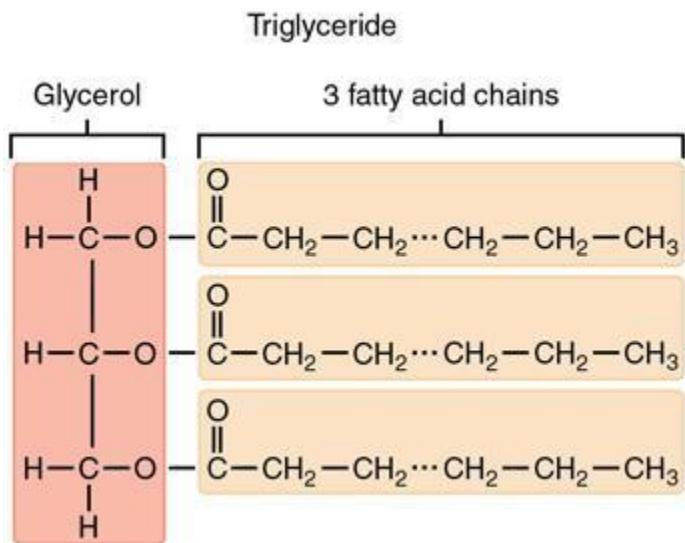
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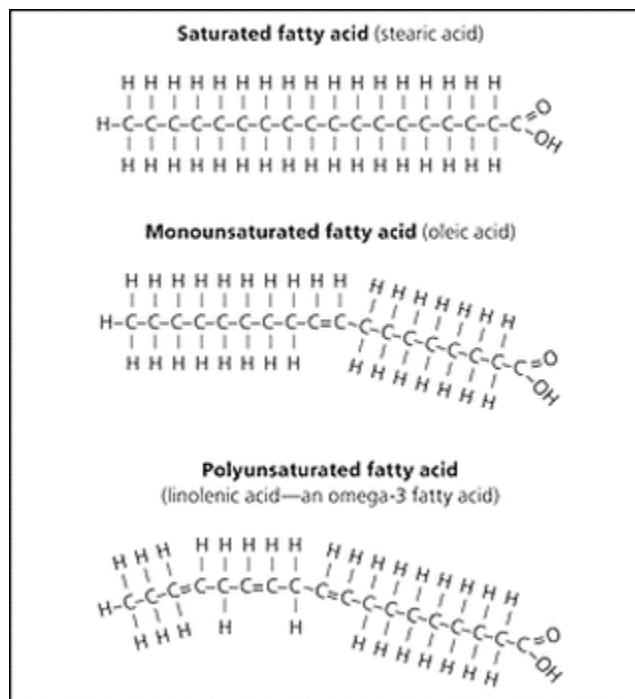
Unless otherwise referenced, all the basic biology and biochemistry comes from the Advanced Nutrition and Human Metabolism textbook by Gropper et al.

As with the other macronutrients, it's important to understand some fundamental theory about fats before we can get to the practical applications. When talking about 'fats', many people confuse lipids, triglycerides, fatty acids and adipose tissue. When we speak of 'fats' in nutrition, we are normally referring to triglycerides and their component fatty acids. Triglycerides consist of a glycerol backbone connected to 3 fatty acids, as depicted below.



The structure of a triglyceride.

The fatty acids may be saturated (SFA), monounsaturated (MUFA, possessing one carbon-carbon double bond), or polyunsaturated (PUFA, having two or more carbon-carbon double bonds).



An example of each fatty acid type.

More generally, dietary fats belong to the class of chemical substances we call lipids. Triglycerides, phospholipids (primarily phosphatidylcholine), and sterols (mainly cholesterol) are the lipids you'll find in a normal human diet. Of these, triglycerides are by far the most common and the only one many people know. Lipids serve not only as dietary sources of energy and constituents of cell and organelle membranes but also as the fat-soluble vitamins, corticosteroid hormones and certain mediators of electron transport, such as co-enzyme Q. Among the many compounds classified as lipids, only a small number are important as dietary energy sources or as functional or structural constituents within the cell.

1. Simple lipids

- a. Fatty acids
- b. Triacylglycerols/triglycerides, diacylglycerols and monoacylglycerols
- c. Waxes (esters of fatty acids with higher alcohols)
 - (1) Sterol esters (cholesterol-fatty acid esters)
 - (2) Nonsterol esters (vitamin A esters etc.)

2. Compound lipids

- a. Phospholipids
 - (1) Phosphatidic acids (i.e., lecithin, cephalins)
 - (2) Plasmalogens
 - (3) Sphingomyelins
- b. Glycolipids (carbohydrate-containing, structural lipids)
- c. Lipoproteins (lipids in association with proteins)

Before we discuss fitness nutrition, it's useful to understand what these different lipids are, because many people that talk about 'fats' literally don't know what exactly they're talking about.

What are the different types of lipids?

1. Fatty acids

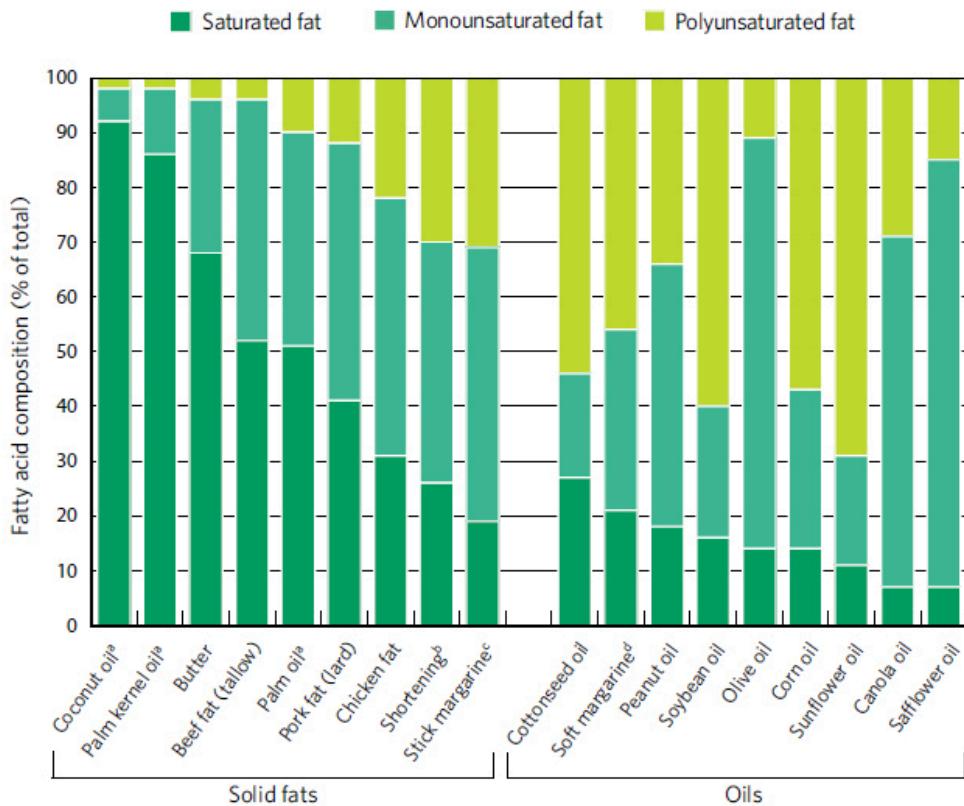
As a class, the fatty acids are the simplest of the lipids. Fatty acids come in 4 forms.

The 3 most common forms of fatty acid are

1. saturated fatty acids (SFA);
2. monounsaturated fatty acids (MUFA, possessing 1 carbon-carbon double bond) and
3. polyunsaturated fatty acids (PUFA, having 2 or more carbon-carbon double bonds).

The table below lists the fatty acid composition of various types of common dietary fat sources. As always, when logging your diet, go by your specific food labels if you have them, not the figure below.

FIGURE 3-3. Fatty Acid Profiles of Common Fats and Oils



a. Coconut oil, palm kernel oil, and palm oil are called oils because they come from plants. However, they are semi-solid at room temperature due to their high content of short-chain saturated fatty acids. They are considered solid fats for nutritional purposes.

b. Partially hydrogenated vegetable oil shortening, which contains *trans* fats.
c. Most stick margarines contain partially hydrogenated vegetable oil, a source of *trans* fats.

d. The primary ingredient in soft margarine with no *trans* fats is liquid vegetable oil.

Source: U.S. Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. USDA National Nutrient Database for Standard Reference, Release 22, 2009. Available at <http://www.ars.usda.gov/bhnrc/ndl>. Accessed July 19, 2010.

Common dietary fat sources and their average fatty acid composition.

Essential fatty acids

Unlike carbohydrates, certain fatty acids are essential for humans, just like certain amino acids are. If you don't consume any fat at all, you're at high risk for retarded

growth, dermatitis, kidney lesions, gallstone formation and eventually an early death. Specifically, [most animals cannot synthesize omega-3 or omega-6 fatty acids](#), because they lack Δ^{12} and Δ^{15} desaturases. These enzymes are found almost exclusively in plants. Therefore, [you normally have to obtain the following 2 essential fatty acids from plants.](#)

1. linoleic acid (LA, 18:2 n-6): a minimum intake of 6% of energy intake or 12 g for women and 17 g for men is generally advised.
2. α -linolenic acid (ALA, 18:3 n-3): a minimum intake of 0.7% of energy intake or 1.1 g for women and 1.6 g for men is generally advised.

From linoleic acid, the body can form other omega-6 fatty acids, notably γ -linolenic (18:3 n-6) and arachidonic acids (20:4 n-6).

From ALA, the body can produce the other omega-3 fatty acids EPA and DHA, although the conversion rate is poor. We'll discuss the other omega-3s in detail later. [ALA also seems to have some health benefits independently of its metabolites, but its precise biological role is still unclear \[2\].](#)

The body can produce non-essential fatty acids itself. The vast majority of fatty acids are non-essential. However, dietary fat is also beneficial for the absorption of fat-soluble vitamins, notably vitamins A and E. To ensure adequate vitamin absorption and cover essential fatty acid requirements, [a minimum dietary fat intake of 10-15% is often recommended.](#) In absolute terms, fat intakes below 20 g a day for women and 30 g a day for men are strongly discouraged.

For optimal health, a fat intake of 20% has often been advised, including by the [European Food Safety Authority, the National Academy of Medicine and the World](#)

[Health Organization](#), although the exact recommendations differ over time and by organization. Based on their standard 2/2.5k kcal intakes for women and men, this would represent a minimum fat intake for optimal health of 44 g for women and 56 g for men.

Take-home message: Fatty acids can be saturated (SFAs), mono-unsaturated (MUFAs), poly-unsaturated (PUFAs) or trans fatty acids. There are 2 essential fatty acids: linoleic acid (LA), an omega-6, and alpha-linolenic acid (ALA; an omega-3). An absolute minimum fat intake of 10% or 20-30 g is strongly recommended with over 20% or 44-56 g being advisable.

Trans fatty acids

Unsaturated fatty acids have a structural property called carbon-carbon double bonds. Where such a bond exists, it can be arranged in 2 manners with radically different effects: either a cis or a trans geometric isomerism. The trans configuration creates the possibility to form a 4th type of fatty acid: the notoriously unhealthy trans fats. Trans fats can [distort cell membranes](#), cause [inflammation](#) [2], [decrease ‘good’ HDL-cholesterol and increase ‘bad’ LDL-cholesterol](#). [Artificial trans fatty acid consumption is associated with various health problems, including diabetes, brain and heart toxicity, infertility, coronary heart disease, depression and Alzheimer’s disease](#) [2, 3, 4, 5, 6].

Artificial trans fats are so toxic that in many countries, including the European Union, food manufacturers are not allowed to produce retail foods with more than 2 g trans fat per 100 g of food. According to [many scientists](#), that's still 2 g too much. The only safe artificial trans fat intake is zero. [A 2% increase in energy intake from trans fat is associated with 23% higher cardiovascular disease risk](#).

Which foods contain trans fat?

Trans fat forms in high-concentrations during partial hydrogenation, a process commonly used to make margarine and frying oils. It makes the fats more solid and stable at room temperature, like most natural saturated fats. Full hydrogenation should not form trans fatty acids, but if the label only says 'hydrogenated' without specifying 'full hydrogenation', beware it may still be partial hydrogenation.

Deep-frying oils also significantly increases their trans-fat content [2], albeit not nearly as much as partial hydrogenation. The total trans fat content is generally no more than a few percent, in comparison to ~50% after partial hydrogenation. Anti-oxidants in oil provide some degree of protection against oxidation. The conversion of PUFAs to trans fatty acids increases with time and temperature, so very briefly deep-fried foods, such as tempura style, are not nearly as harmful as French fries prepared in the same oil that has been fried multiple times already. However, beware that the oil used for deep-frying is often itself already a partially hydrogenated oil and therefore a source of trans fats, irrespective of how much it's subsequently deep-fried.

Most heat-processed vegetable and nut oils also contain a few percent trans fat, as their deodorization forms trans fatty acids: see the table below. This includes otherwise healthy oils like canola/rapeseed oil and olive oil. To ensure you don't consume trans fatty acids, it's best to get cold-pressed, unrefined or virgin oils.

Sample name ^a	Total <i>trans</i> content (%)
Oils high in linoleic acid (9c,12c-18:2)	
Soybean oil A	0.41
Soybean oil B	2.08
Walnut oil A	3.97
Walnut oil B	1.99
Sunflower oil	1.10
Oils high in oleic acid (9c-18:1)	
Triolein	0
Canola oil (high-oleic canola)	2.92
Olive oil	0.52
Canola oil A	3.60
Canola oil B	1.88
Fats high in <i>trans</i> FA	
PH canola oil D ^b	59.49
PH soybean oil D ^b	50.25
PH canola oil C ^b	53.06
PH soybean oil C ^b	43.60
Commercial shortening ^c	20.09

^aAll oils and fats were locally purchased products.

^bPartially hydrogenated (PH) soybean and canola oils were provided by two separate suppliers.

^cThe commercial shortening was purchased locally.

Source

Lastly, many foods produced with partially hydrogenated or other heat-processed oils also contain significant quantities of trans fat, especially in non-retail venues like bakeries, restaurants and shops that aren't required to have food labels: see the table below.

Table 1. Typical Trans Fatty Acid Content of Foods Produced or Prepared with Partially Hydrogenated Vegetable Oils in the United States.

Type of Food	Trans Fatty Acid Content*			% of Daily Energy Intake for 2000-kcal Diet
	g/Typical Serving	g/100 g	% of Total Fatty Acids	
Fast or frozen foods				
French fries†	4.7–6.1	4.2–5.8	28–36	2.1–2.7
Breaded fish burger‡	5.6	3.4	28	2.5
Breaded chicken nuggets‡	5.0	4.9	25	2.3
French fries, frozen†	2.8	2.5	30	1.3
Enchilada‡	2.1	1.1	12	0.9
Burrito‡	1.1	0.9	12	0.5
Pizza†	1.1	0.5	9	0.5
Packaged snacks				
Tortilla (corn) chips†	1.6	5.8	22	0.7
Popcorn, microwave†	1.2	3.0	11	0.5
Granola bar‡	1.0	3.7	18	0.5
Breakfast bar‡	0.6	1.3	15	0.3
Bakery products				
Pie†	3.9	3.1	28	1.8
Danish or sweet roll†	3.3	4.7	25	1.5
Doughnuts†	2.7	5.7	25	1.2
Cookies†	1.8	5.9	26	0.8
Cake†	1.7	2.7	16	0.8
Brownie†	1.0	3.4	21	0.5
Muffin†	0.7	1.3	14	0.3

Source

Even if the label of an at-risk food says 0 g trans fat, it's arguably best to avoid anything that may have trans fats, because manufacturers are often allowed to round down anything under 0.5 g trans fat per serving to 0 g, so they can just make the 'recommended serving size' small enough to get under that limit. This is often done with processed peanut butter, for example. Assuming the worst is often prudent.

Pan- or stir-frying or baking food does not form a significant amount of trans fat, so it's safe even with PUFA-rich oils [2].

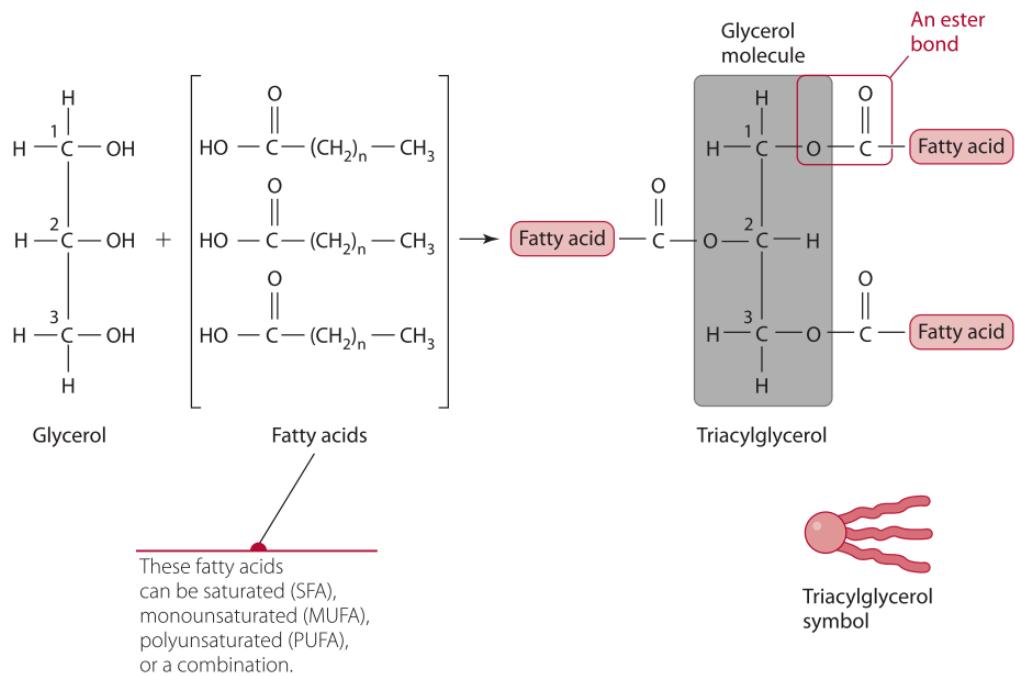
Saturated fatty acids cannot turn into trans fats, as they have no double carbon bonds, so they are safe to process at high heat. Mono-unsaturated fatty acids have only a single double-bond, so they also have minimal risk of forming trans fatty acids. This means that oils low in PUFAs, such as palm oil, maintain a relatively low trans fat content even when extensively deep-fried (~1%).

Most naturally occurring unsaturated fatty acids are of the cis configuration, but ruminant bacteria can produce natural trans fatty acids, so the trans form appears in some natural fats and oils and in dairy products and beef. Conjugated linoleic acid (CLA) is the most well-known natural trans fatty acid. Natural trans fat does not have the adverse health effects that artificial trans fat does.

2. Triglycerides

Most stored body fat is in the form of triglycerides ('3 fatty acids attached to glycerol'), also known as triacylglycerols (TAG), which represent a highly concentrated form of energy. Triacylglycerols is the currently accepted name that has replaced the older name triglycerides [TRIG or TG] in biochemistry circles. However, nutritionists still generally use the name triglycerides.

Triglycerides/triacylglycerols account for nearly 95% of dietary fat. Structurally, they are composed of glycerol with 3 fatty acids attached to it (hence the name), as illustrated below. The fatty acids in triacylglycerols can be all saturated, all monounsaturated, all polyunsaturated, or any combination of the three.



Triglycerides (= triacylglycerols) are composed of glycerol with 3 attached fatty acids.

Glycerol can also be attached to a single fatty acid, forming a monoacylglycerol (MAG) or 2 fatty acids, forming a diacylglycerol (DAG), but MAGs and DAGs are rare in the human body. They also occur in some processed foods, to which they are added as emulsifying agents.

Triglycerides exist as fats (solid) or oils (liquid) at room temperature, depending on the nature of the component fatty acids.

- Triacylglycerols that contain a high proportion of relatively short-chain fatty acids or unsaturated fatty acids tend to be liquid (oils) at room temperature, such as olive oil.
- Triglycerides made up of saturated fatty acids of longer chain length have a higher melting point and thus exist as solids, such as cow butter.

Thus, solid fats such as butter and coconut oil have predominantly saturated fatty acids, whereas liquid oils tend to be predominantly unsaturated fats.

When triglycerides from adipose tissue area need to be catabolized to produce energy, their fatty acids are broken off the glycerol by enzymes: lipases. The free fatty acids, also called non-esterified fatty acids (NEFAs), are then transported by albumin, a protein found in the blood, to various tissues for oxidation ('fat burning').

Take-home message: Body fat is stored in the form of triglycerides, which are composed of a glycerol backbone with 3 fatty acids that can be burned for energy.

3. Sterols and steroids

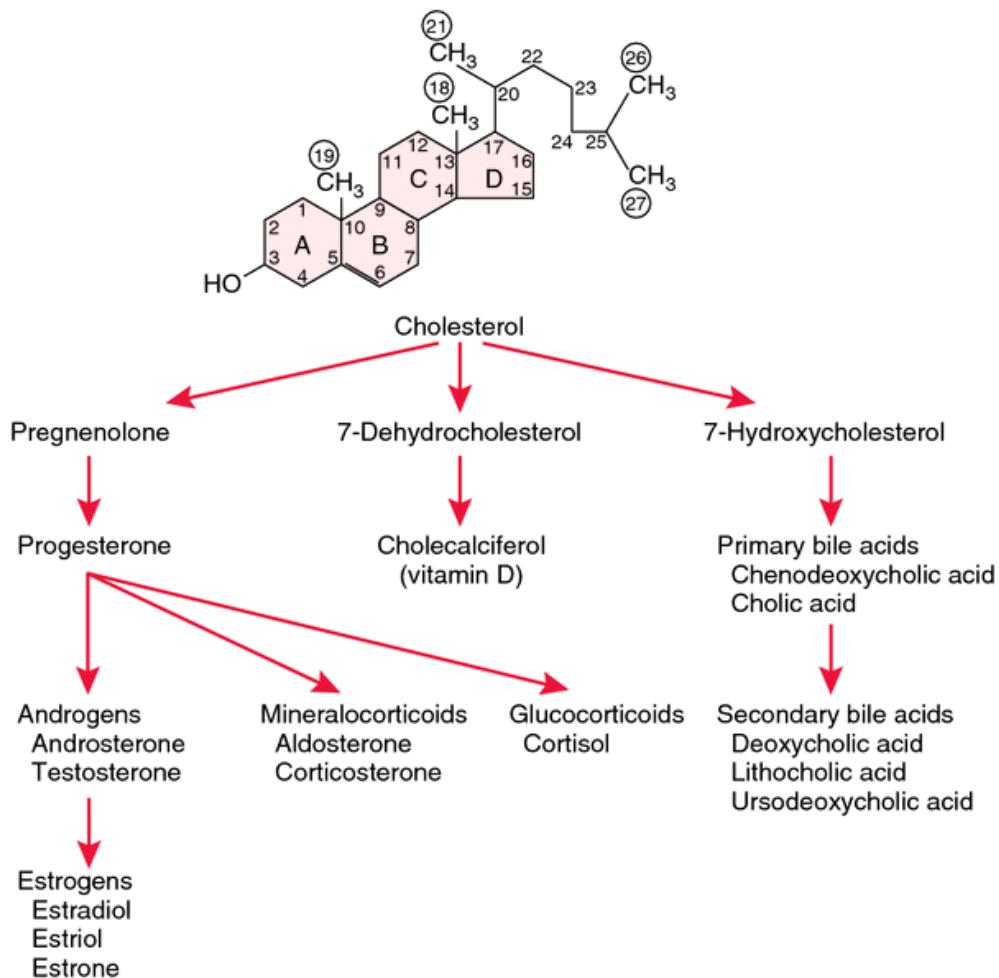
Steroids are a lipid class characterized by a four-ring core structure called the *deep breath* cyclopentanoperhydrophenanthrene nucleus (yes, that's a real word and no, you don't have to remember it), more commonly known as the steroid nucleus.

Androgenic-anabolic steroids are the most well-known steroids in fitness, but it is incorrect to simply call these steroids, as steroids are a much larger category of lipids. Their reputation under the incorrect name 'steroids' exemplifies the ignorance surrounding their use.

Sterols, also called steroid alcohols, are a subgroup of steroids. Cholesterol is the most common sterol in animals and is the precursor for other steroids: your body uses cholesterol to create steroids.

Meats, egg yolk and dairy products are common dietary sources of cholesterol. In the body, this sterol is an essential component of cell membranes, particularly the membranes of nerve tissue. Despite the bad press that cholesterol has garnered over the years because of its implication in cardiovascular disease, it serves as the precursor for many other important steroids in the body, including the following:

- the bile acids
- steroid sex hormones, such as estrogens, androgens, and progesterone
- the adrenocortical hormones, including cortisol and other corticosteroid hormones
- vitamin D (cholecalciferol, the animal form)



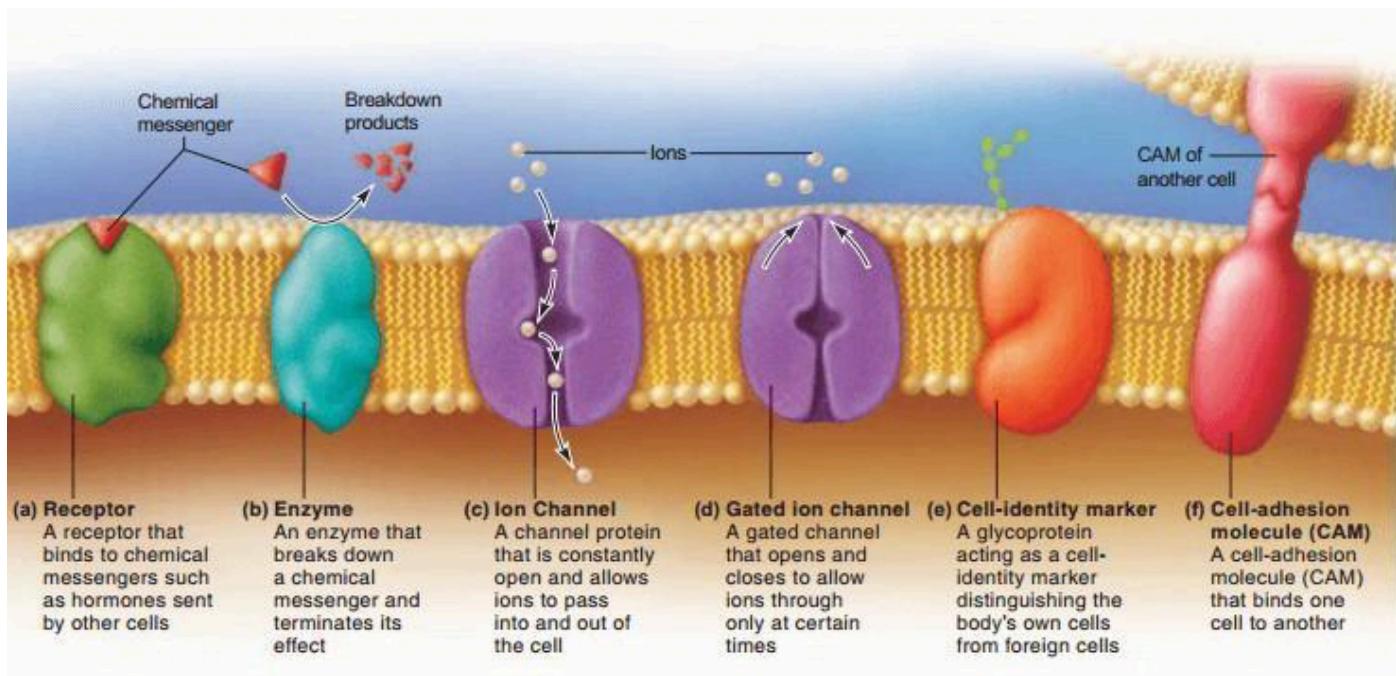
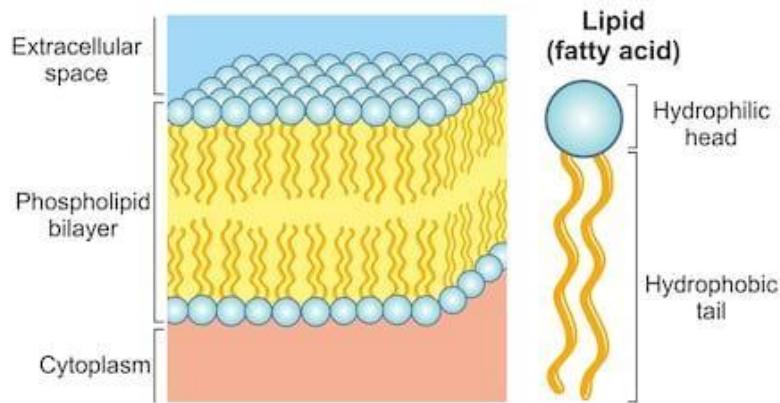
Your body uses cholesterol to create steroid hormones, bile and vitamin D. [Source](#)

Take-home message: Cholesterol is a sterol that your body uses as a building block to produce steroids, including steroid hormones. Anabolic-androgenic steroids (AAS) are but one out of many steroids. Steroids and sterols are all lipids.

4. Phospholipids

Phospholipids, consisting of phosphate, glycerol and fatty acids, are part of your cell membranes. The phospholipid bilayer of a cell influences how the cell functions by determining how the cell reacts to compounds outside the cell. The phospholipid bilayer functions as a 'gatekeeper' of which compounds can enter the cell and which enzymes and hormones can bind to the cell.

Your diet's fatty acid composition influences the fatty acid composition of the phospholipid bilayers in your cell membranes. Which fatty acids you eat thus determines how your cells function. An example we'll discuss in detail later is that your diet's fatty acid composition can influence testosterone production in the Leydig cells of the male testes.



Your body's cells have a membrane with a phospholipid bilayer. This layer's composition is determined by your diet's fatty acid composition. The composition of the layer determines which compounds can enter the cell and which compounds can react with the cell's receptors and enzymes.

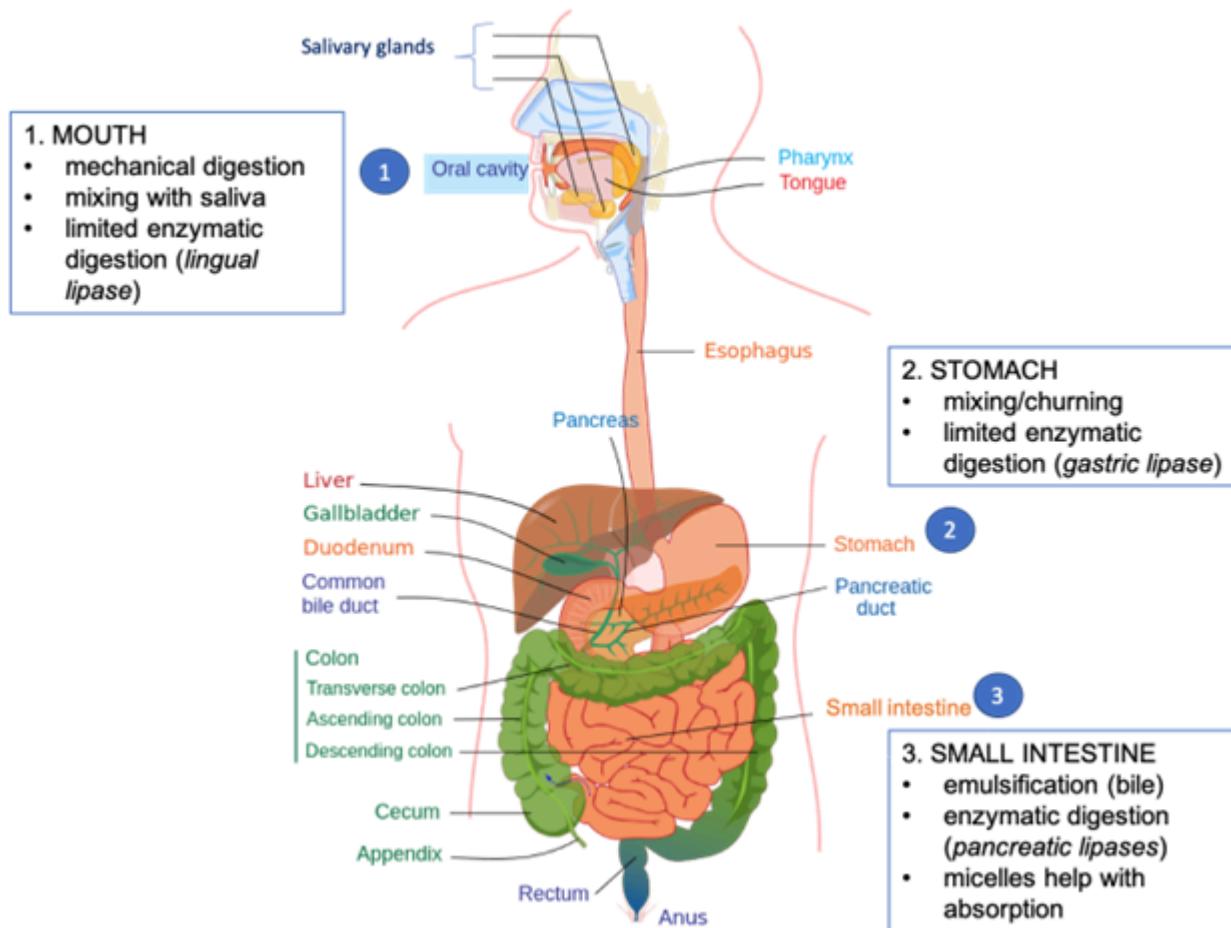
From dietary fat to adipose tissue: fat metabolism

Now that we know what the important lipids in the body are, let's go into how dietary fat can turn into adipose tissue. Your body doesn't just take fats from your diet and put them in storage. Fats are digested and absorbed just like carbs and proteins, but the digestive process is more complicated and this gives fats unique properties.

Fat digestion & absorption

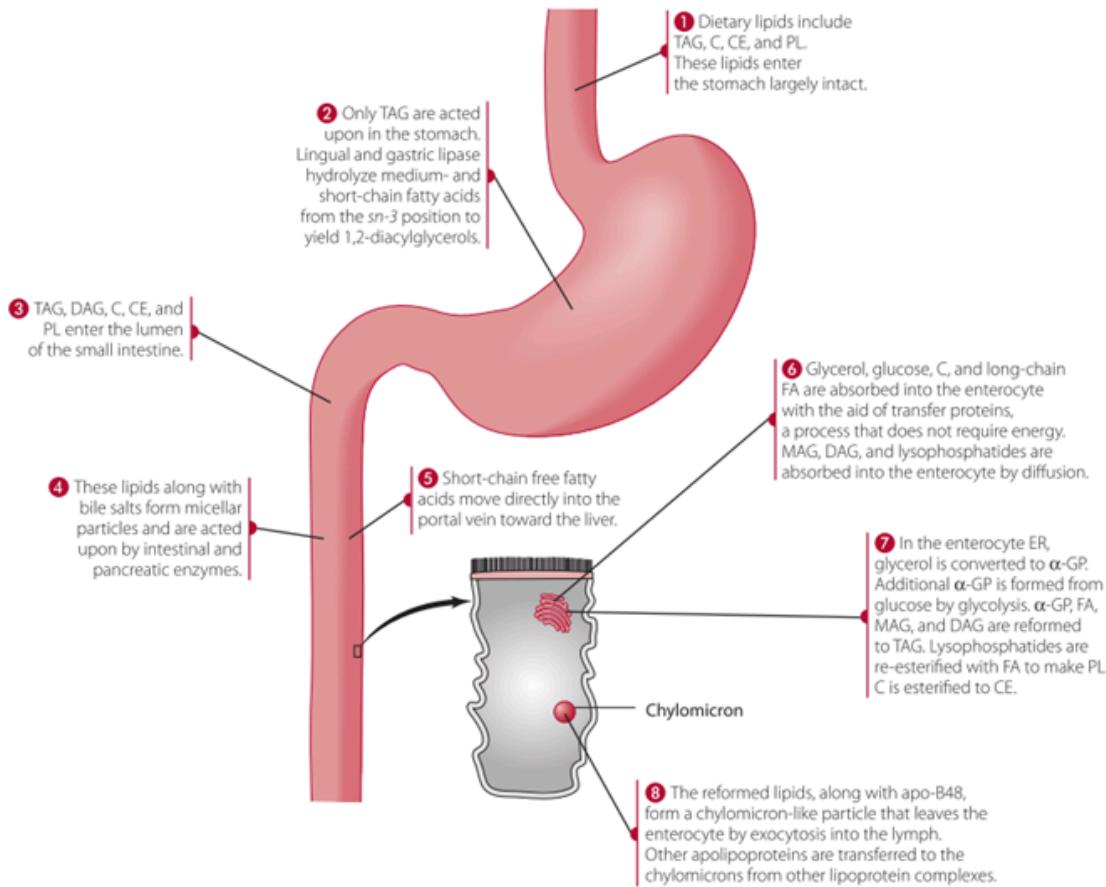
The digestion of fats takes a relatively long time, because triglycerides are hydrophobic ('water fearing'): you've probably seen they often float and don't mix with water. That's a problem for our gut, because the gastrointestinal tract is filled with liquids and digestive enzymes are hydrophilic ('water loving'): they often absorb in water. Our body's solution to this problem is to break down lipids into smaller pieces, a process called emulsification. This is done chemically with bile salts and mechanically with muscle contractions (peristalsis). The smaller the fat globules, the more surface area our digestive enzymes (lipases) have to 'chew' on. Still, typically less than a third of dietary fat is digested before it enters our intestines. Due to their prolonged presence in the stomach, fats should theoretically provide longer-term satiety than carbohydrates, although research findings on this have been mixed, as we'll discuss in the course module on hunger management.

The slow digestion of triglycerides also results in long-term energy provision to the body. [It generally takes over 2 hours after consumption before fat levels peak in the blood and they continue to provide energy for over 6 hours; for large meals, it can be over 12 hours \[2\].](#)



Lipid digestion in our gastrointestinal (GI) tract. [Source](#)

After digestion in the small intestine, we're mostly left with monoglycerides, free fatty acids, glycerol and free cholesterol. These are then either absorbed directly into the blood and transported to the liver or first repackaged into triglycerides and transported to other tissues, mostly our muscles and body fat (adipose tissue).



A summary of the digestion and absorption of dietary lipids. TAG = triglycerides, C = cholesterol, CE = cholesterol ester, PL = phospholipid, DAG = diacylglycerol, MAG = monacylglycerol, FA = fatty acid, α -GP = α -glycerolphosphate.

What are lipids used for?

Once lipids are digested and absorbed into the body, they can be metabolized. The chief uses of triglycerides under normal circumstances are energy production (beta-oxidation in our mitochondria) or storage into adipose tissue (body fat) for later use. However, fats have many more metabolic functions: some essential body fat is literally essential for our survival. Below are the other uses of lipids in the body.

- Phospholipids and cholesterol are structural parts of our cell membranes, providing stability and regulating what can come into the cell.
- Our abdominal organs are surrounded by so-called visceral fat. Too much of this quickly becomes very unhealthy, but a small amount is beneficial to cushion the organs. Similarly, our joints have some fat pads in them to provide shock absorption and to reduce friction.
- Our bones have fat in their bone marrow that's highly metabolically active. It's used to produce blood cells and regulate bone health.
- Our muscles and liver have some fat in them they use to generate energy.
- Brown adipose tissue helps generate heat, which can ironically help you lose fat (white adipose tissue). We'll discuss this in more detail later.
- Some lipids, such as arachidonic acid, are used to create signaling molecules, such as eicosanoids. These are important regulators of inflammation, the immune system and the vascular system. We'll discuss these in more detail in relation to omega-3 fatty acids later on.
- Sphingolipids are structural parts of our nervous system, such as in the sheaths around our neurons, aiding in signal transmission.
- Wax esters are used in our skin and sebum, providing waterproofing and protection.
- Cholesterol is used to produce bile acids that aid our digestion, specifically that of other lipids.
- Female breast tissue has essential fat that's used in part to produce milk.
- Our eyes also have some fat around them, along with some fat in our tears to prevent dehydration and some essential fat in the retina's membranes (DHA).

When our bodies have a surplus of energy, dietary fat is preferentially used to store as body fat, because it is the simplest to convert into this form of the 3 major

macronutrients. Our fat cells, adipocytes, are the body's primary storage site for triglycerides, and around 85% of their volume consists of a globule of fat. Our body fat percentage is primarily determined by how full our fat cells are, as our total fat cell number is relatively constant after adolescence in non-overweight individuals. Fat cells are normally permanent. However, [new fat cells can also be created during significant fat gain](#) (adipogenesis). Fat cells are way too tiny to observe from the outside, so our visual body fatness is purely a function of the total amount of fat stored, not how many fat cells we have.

Our body fat stores are not static. Throughout our body, fat cells are constantly lipolyzing (breaking down) and re-esterifying (rebuilding) their triglycerides based on the fuel requirements of our tissues. Adipocytes can also synthesize fatty acids from glucose, a process strongly influenced by insulin. Insulin accelerates the entry of glucose into the adipose cells, whereas the liver does not respond to this action of insulin. Insulin also stimulates the uptake of fatty acids by adipocytes by stimulating lipoprotein lipase. Adipose cells can combine these fatty acids with glycerol to form triglycerides. The glycerol can be obtained by glycolysis, the breaking down of glucose. Absorbed mono- and diglycerides also provide fatty acids for triglyceride (re)synthesis. Insulin further promotes lipogenesis in adipose tissue by inhibiting hormone-sensitive lipase, which hydrolyzes stored triglycerides, thus favoring triglyceride accumulation: fat gain. In short, insulin stimulates the storage of body fat via various pathways, but remember you can only store what's there, so insulin does not defy energy balance. Net fat storage over time is ultimately decided by energy balance.

Adipose tissue cannot metabolize free glycerol, so glycerol returns to the blood. Plasma glycerol levels can thus be used as an indication of triglyceride turnover in

adipose tissue: high plasma glycerol levels suggest a lot of triglycerides are being metabolized and we're seeing their remaining glycerol backbones as a result.

When energy is scarce, our bodies will burn fat for energy rather than store it. Breaking down lipids is called lipolysis. The lipolysis of a triglyceride results in glycerol and 3 free fatty acids. Lipolysis is stimulated by hormones such as epinephrine and norepinephrine, adrenocorticotrophic hormone (ACTH), thyroid-stimulating hormone (TSH), glucagon, growth hormone and thyroxine (T4).

Both glycerol and free fatty acids either go to our muscles to be burned as fuel or to our liver to be metabolized into something else that's required more. When fatty acids are transported to muscles to burn for energy, they are oxidized in the mitochondria, the energy production centers of the cells. Burning fatty acids, called β -oxidation, produces energy through oxidative phosphorylation. Fatty acid oxidation is a very efficient process, so the body relies on it extensively. However, just like the fire in a furnace, this oxidation requires oxygen. During high-intensity exercise, energy production requirements may be so high that oxygen becomes a limiting factor for energy production via the oxidation of fatty acids. In this case, our muscles will start relying primarily on glucose as fuel, as you learned in the module on carbohydrates.

How carbs, protein and fats interact

In the liver, fatty acids and especially glycerol can be converted into other nutrients that are more needed at the time. The liver is the body's primary repackaging site for nutrients. In the liver, glycerol can serve as a substrate for glucose (gluconeogenesis) or for new triglycerides by combining the glycerol with 3 fatty acids. Glycerol can be created from glucose, which in turn can be obtained from liver glycogen stores or

absorbing glucose from the blood. Fatty acids can be created from glucose via *de novo lipogenesis*, but this generally only occurs in energy surplus with excess carbohydrate availability, so the liver tends to use free fatty acids. After a new triglyceride is formed, it's combined with phospholipid, cholesterol and proteins to form VLDLs and high-density lipoproteins (HDLs). The lipoproteins are then released into the circulation to travel to other tissues via the blood. The liver can also convert free fatty acids into ketones (explained in more detail in the module on ketogenic diets). The liver's capacity to convert nutrients into each other is a key reason why fat loss and gain generally simply comes down to total energy balance instead of which macronutrients you consume.

Under normal circumstances, protein is preferentially used for protein synthesis up until the requirements thereof, after which the excess protein is generally oxidized for energy. Excess protein intake can also be used to create glucose when carbohydrate intakes are low, because all but 2 amino acids can serve as substrate for gluconeogenesis, and thereby also fatty acids via *de novo lipogenesis*.

Carbohydrates are preferentially burned for energy, especially during high-intensity exercise. A supply of around 300-700 g glycogen is generally kept in reserve for this purpose. Excess glucose that is not glycolyzed or stored as glycogen can be converted to fatty acids, but *de novo lipogenesis* typically only occurs in energy surplus on relatively low-fat diets.

Fats are preferentially stored, both in cell membranes and some essential places, as well as in body fat during periods of excess energy availability. Fats are also readily oxidized to produce energy. Whether fats are stored or burned depends primarily on how much carbohydrate we consume and is regulated by our blood sugar levels. The

more carbohydrates we consume, the more of the fat we consume is stored rather than used for energy. Hyperglycemia (high blood sugar) triggers the release of insulin, which promotes glucose transport into our adipose cells and lipogenesis instead of lipolysis. Hypoglycemia (low blood sugar) results in a reduced intracellular supply of glucose and greater reliance on lipolysis for energy.

In short, your body efficiently regulates its choice of substrate use for energy production and fat storage based on what is available to it from the diet. That's why, in the long term, energy balance is the primary determinant of changes in your body fat level, not your diet's macronutrient composition.

Now that you understand the role of fat intake for fat loss and our metabolism, we can discuss the roles fatty acids have specific to muscle growth.

Dietary fat's effect on muscle growth

Essential fatty acids are essential for our survival, but we only need a very low fat intake to consume our essential fatty acids. Earlier, you learned that the body can properly function with just 10-15% of energy coming from fat and optimal health doesn't require more than 20% of fat. There are 2 key theoretical benefits for strength trainees of consuming more fat.

1. Increased anabolic hormone production.
2. Inherent anabolic effects of certain fatty acids.

Let's start with the effects of your fat intake on your hormones. After we've discussed the theory, we'll look at the empirical evidence from studies.

Fat and your hormones

Dietary fat can be beneficial for our sex hormone production. Saturated fatty acids can serve as indirect substrate for steroid hormones, as they are a precursor for cholesterol, which is in turn a precursor for steroid hormones. [Fatty acids can also influence testosterone levels by changing cell membranes](#), particularly in the testes. Fatty acids are part of the phospholipid layer of cell membranes. Different fatty acid compositions make the cell membranes function differently. For example, in rodents we know that the phospholipid composition of the cell membranes of the testes affect their binding to luteinizing hormone (LH), which is the signal from your brain to start producing testosterone. A higher intake of certain fatty acids can thus increase LH binding in testicular cells, which makes them produce more testosterone. The cell membrane also determines its binding to other proteins, enzymes and hormones,

including sex-hormone binding globulin (SHBG), which in turn affects the proportion of free and bioavailable testosterone.

As a result, [higher fat intakes consistently increase anabolic hormone levels](#) in both sexes, namely [testosterone](#), [estrogen](#), growth hormone and IGF-1, by 10-25% when going from fat intakes around 20% to 40% even under isocaloric conditions [3, 4, 5, 6, 7, 8, 9]. [Free testosterone](#) [2, 3] and [growth hormone levels](#) are also affected ~10-15% by dietary fat energy content, independently of energy intake, though in some research low-fat diets decrease sex-hormone binding globulin levels enough to keep free testosterone levels stable. There is probably very little benefit of increasing your fat intake much above 40% of energy intake. [Vidic et al. \(2021\)](#) found that a diet with 65% fat and a ketogenic diet with 75% fat both resulted in a 33-34% increase in free testosterone levels.

We also have a case study by [Shemesh et al. \(2011\)](#), in which a 22-year-old male bodybuilder had erectile dysfunction despite being otherwise very healthy. He was on a bro bodybuilding diet with fewer than 20 grams of fat per day. Increasing this to 30% of energy intake without increasing reported total energy intake increased his bioavailable testosterone level 6-fold and resolved his erectile dysfunction. One confounder is that he also increased in body fat percentage from 4% to 8% according to the BodPod, but his sexual dysfunction had lasted 4 years, so assuming he hadn't been in contest shape for 4 years, that shouldn't have been the sole cause and the higher fat intake was likely at least partly beneficial to his recovery.

Interestingly, [3 studies found a negative correlation between protein intake and testosterone level](#) [2, 3], suggesting a high protein intake is bad for your testosterone. However, two of these studies only measured total testosterone levels. Sex-hormone

binding globulin (SHBG) decreased alongside testosterone in the high protein group in [the one study that measured it](#), meaning estimated bioavailable testosterone levels remained similar in the high and low protein diet groups. Moreover, [a 2023 review](#) found that only high protein intakes above 3.4 g/kg (1.54 g/lbs) per day decreased testosterone levels, and this was largely based on short-term research with multiple confounders. In several other studies, diets higher in fat as well as protein intake produced increases in testosterone level, so any negative effect of protein on testosterone is likely vastly overshadowed by a high fat intake. Furthermore, as you learned in the module on protein, research is also clear that higher protein intakes are better or at worst neutral for muscle growth, with scant evidence for any detrimental effects. As such, it's unlikely high protein diets have a considerable negative effect on your testosterone level in practice.

The following tables show an overview of the literature on diet composition and hormone levels for men and women.

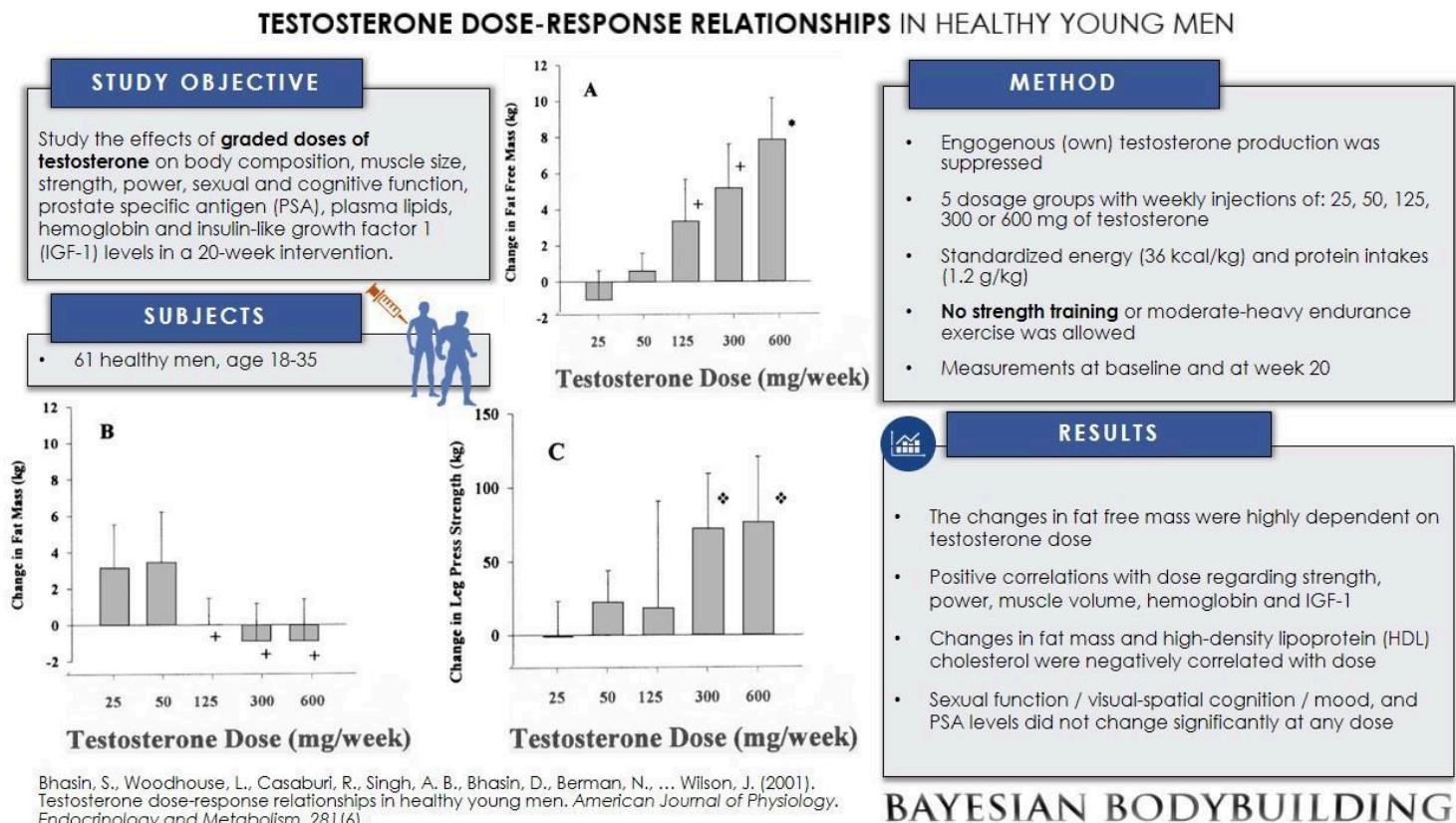
➤ Research overview table

[Effect of macronutrient intake on anabolic hormone levels](#)

The magnitude of increase in anabolic hormone levels you can achieve by increasing your dietary fat intake up to ~40% of total energy intake may be large enough to have body composition effects.

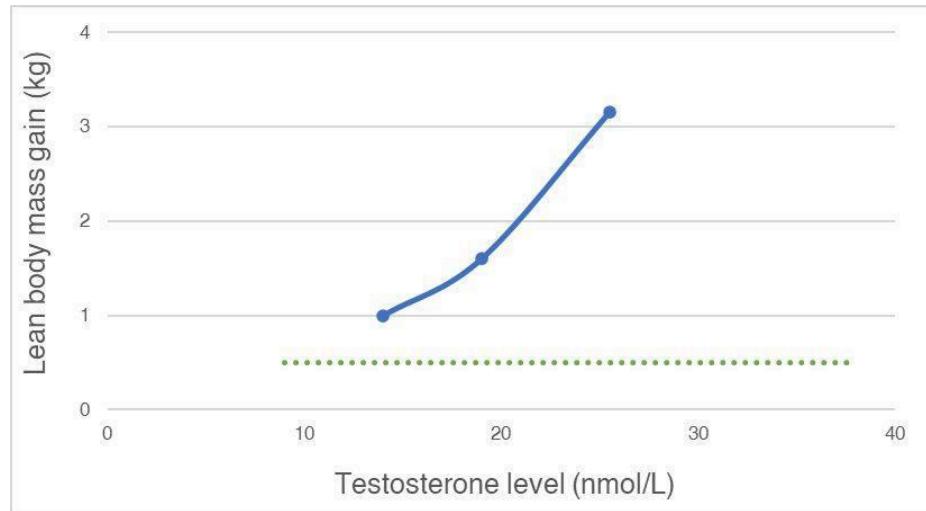
The effect of testosterone on your strength and body composition

Testosterone is also well known for its anabolic properties. Testosterone activates the androgen receptor, which directly signals the genes in your muscle cells to start producing more protein to grow. Consequently, there is a dose-response effect of testosterone on muscle size and strength: the more testosterone you have, the stronger and more muscular you become. [2]: see the infographic below. It doesn't matter if the testosterone comes from an injection or your body: testosterone is testosterone. So large differences in testosterone level will cause large differences and small differences will cause small differences. (For the statistics lovers, it is a logarithmic function.)



Bhasin, S., Woodhouse, L., Casaburi, R., Singh, A. B., Bhasin, D., Berman, N., ... Wilson, J. (2001). Testosterone dose-response relationships in healthy young men. *American Journal of Physiology, Endocrinology and Metabolism*, 281(6).

Based on the above study in which testosterone levels were artificially manipulated, even differences in testosterone level within the physiological (drug-free) range can make a several pound difference in lean body mass (LBM) **without any strength training**. A multitude of studies on testosterone replacement therapy confirm that changes in our testosterone level below, within and above the physiological range affect our body composition in a dose-response manner [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11]. For example, [a study by Wittert et al. \(2003\) investigated elderly men with normal testosterone levels](#) square in the middle of the physiological range (17 nmol/L). Increasing their bioavailable testosterone level by 27% – an increase achievable with a higher fat intake – resulted in significant body recomposition. Over the first 6 months of the study, they gained 1 kg (2.2 lbs) lean body mass and lost 0.2 kg (0.44 lbs) fat. In contrast, the placebo group lost 0.9 kg (1.98 lbs) lean body mass and gained 0.9 kg (1.98 lbs) fat. These body recomposition differences were statistically significant and did not significantly change over the rest of the 12-month study. The ‘more is better’ effect of testosterone is neatly illustrated by a study by [Wang et al. \(2000\)](#). They gave just barely hypogonadal men exogenous testosterone to get their T levels into varying levels of the normal range. The higher their testosterone dosage, the more lean body mass they gained after 180 days, even without exercising: see the graph below. Fat mass also decreased by around a kilo on average and the higher the testosterone level, the greater the decrease in fat mass.



Lean body mass gain in men on testosterone replacement therapy (blue) plotted against their final testosterone level, superimposed over the physiological range of testosterone (green): there was a clear dose-response even in the normal range. [Source](#)

These effects are also found in younger individuals. For example, [Herbst et al. \(2003\)](#) injected healthy men with normal testosterone levels (570 ng/dL) with 100 mg of testosterone per week, making their levels fluctuate between the upper-middle and peak of the reference range (734-1106 ng/dL). They experienced positive body recompensation with a 2.5 kg (5.5 lbs) increase in lean body mass in 8 weeks, which was greater than at 4 weeks.

[A 2020 meta-analysis](#) confirms that testosterone supplementation increases fat-free mass with generally positive but less consistent effects on physical performance. Based on the total literature, every 100 ng/dL increase in testosterone results in roughly 1 pound extra lean body mass.

In contrast to the above research where testosterone levels are artificially manipulated *within* individuals, research correlating testosterone levels with baseline body

composition *between* individuals is less consistent with some [null findings](#). Still, multiple studies have found the expected [positive associations between baseline \(untrained\) fat-free mass and \(free\) testosterone levels](#) [2, 3, 4, 5, 6]. Testosterone also correlates with performance in athletes in some research. For example, [soccer players with more testosterone can run faster and jump higher](#).

For science lovers: how reverse causation can cause paradoxical findings in observational research

Interestingly, [the relationship between androgens and fat mass seems to be stronger than for fat-free mass](#) [2, 3, 4, 5]. Persons with more testosterone seem to primarily be leaner rather than more muscular than persons with lower testosterone. Multiple studies have found that [men with more testosterone are leaner than men with less testosterone, but they do not have more total fat-free mass](#) [2, 3]. We know that testosterone greatly aids muscle growth yet doesn't help much with fat loss, as it doesn't lower your appetite or increase energy expenditure appreciably, so what gives? Reverse causation may explain this paradox. Higher doesn't reduce body fat levels: lower body fat levels cause higher testosterone. Adipose tissue produces aromatase, an enzyme that converts androgens to estrogens, so the less fat you have, the higher your ratio of testosterone to estrogen generally is. Up to a point at least, because as you learned, being too lean is bad for your hormonal health; however, this level of leanness is rare and thus probably not represented in observational research. The lack of clear relationship between testosterone and baseline fat-free mass in several studies is probably because it's inherently a weak relation that's further confounded by the fact leaner individuals normally have less fat-free mass than fatter individuals.

Across individuals, testosterone levels also don't always correlate with the magnitude of gains in strength and size over time from training. In several studies in untrained as well as trained individuals, there was [no relation between testosterone levels and rates of muscle growth between individuals](#) [2, 3, 4]. The within-individual studies with strength training also demonstrate a seemingly additive rather than a synergistic effect of testosterone and strength training [1, 2, 3, 4], although formal interaction effects have not been properly tested. Lack of an interaction effect would mean your testosterone level does not influence your relative rate of gains: you would gain the same percentage increase in muscle from strength training with higher as with low testosterone levels. On high testosterone levels, the same percentage increase would constitute a larger absolute increase though due to the higher baseline muscle mass. Increasing your lean body mass by 20% would constitute a 12 kg (26.4 lbs) gain from a baseline of 60 kg (132.3 lbs) LBM yet a 16 kg (35.2 lbs) gain from a baseline of 80 kg (176.4 lbs) LBM. In contrast to the above studies suggesting testosterone only has additive effects on muscle growth, other studies did find correlations between androgen levels and muscular gains, suggesting testosterone potentiates the results from strength training.

- In [a study by Ahtiainen et al. \(2003\)](#), trained men with higher testosterone levels gained strength faster. Here the correlation between strength gains and testosterone level was not just significant but also strong. In untrained individuals, however, there was no relation between testosterone level and gains from strength training.
- [A study in older women](#) found that testosterone level correlated with the rate of muscle growth when they started strength training.

Also, in [research on elite Olympic weightlifters](#), testosterone levels and power development correlated over the long run.

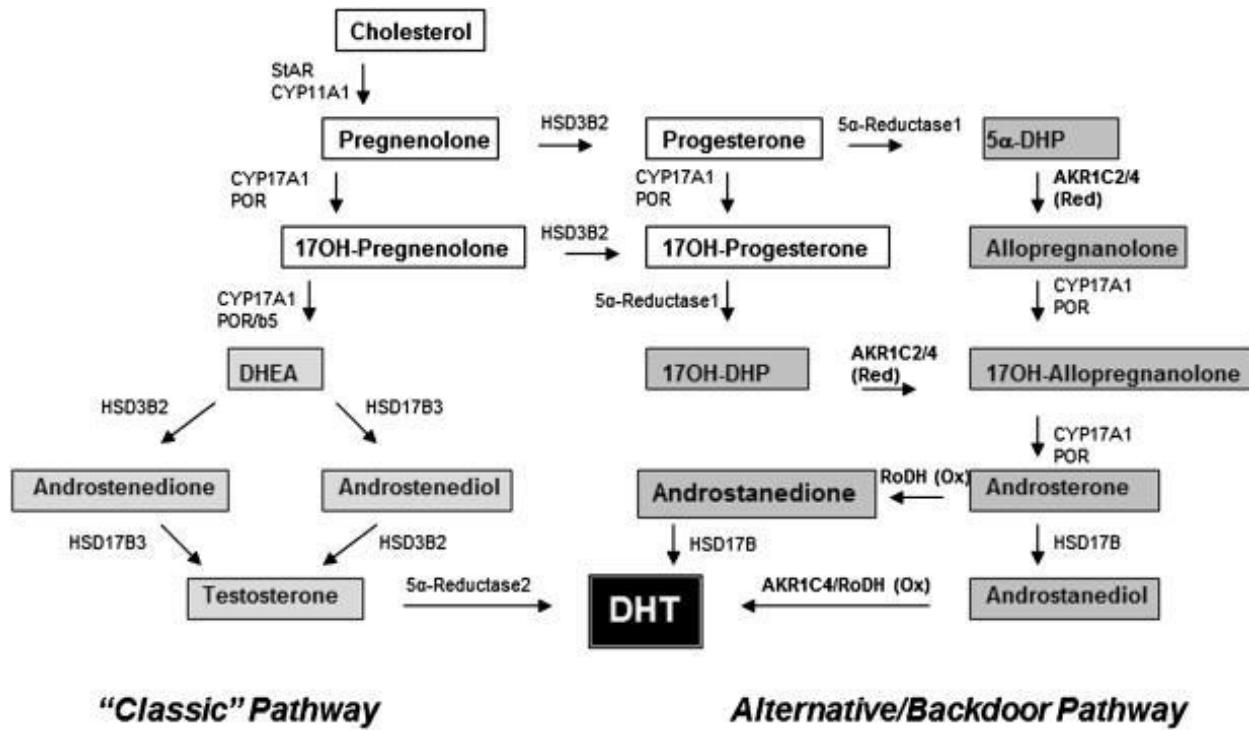
How come there is a contrast between the effect on muscle growth and strength between manipulating someone's testosterone level directly and comparing testosterone levels across different individuals? There are 2 good reasons for this.

First, observational research has lower statistical power than within-subject studies, because there is no control group and the results are confounded by the multitude of other between-individual factors that can change someone's testosterone level or muscle growth (sleep, nutrition, stress, other genetic factors, training efforts, etc.). Sample sizes are also very low with only ~10 subjects in many of the groups in the available studies.

Second, testosterone metabolism varies per individual. That's why 'normal testosterone' can vary 3-fold. Some individuals may get the same effect out of half the testosterone as another person because they may have more androgen receptors, stronger affinity for the receptor, a higher amount of unbound testosterone or less antagonistic activity on the androgen receptor. For example, [Mitchell et al. \(2013\)](#) found that testosterone level did not correlate with muscle growth, but androgen receptor protein content did. Similar results were obtained by [Morton et al. \(2018\)](#) for androgen receptor content. Based on this, you would expect that differences in testosterone level *between* individuals are not very relevant, but changes *within* an individual are, which is exactly what the research shows. The experience of androgenic-anabolic steroid users also aligns with this: some bodybuilders respond far more strongly to a given dose of steroids than others.

Other androgens may be important as well. For example, in [a study on identical twins](#), free testosterone level had only a weak, insignificant relationship with baseline fat-free mass, but androsterone level did significantly correlate with baseline fat-free mass.

DHEA and androstenediol levels also predicted the ratio of fat to fat-free mass gains during 100 days of 1000 kcal overfeeding, i.e. nutrient partitioning, with moderate strength. Since they were identical twins, the difference in androgen production had to be environmental instead of genetic. This shows that optimizing your diet, exercise and overall lifestyle to increase your androgen production can increase your gains.



Testosterone metabolism: testosterone and DHT are the most potent androgens, but there are several other intermediate androgens.

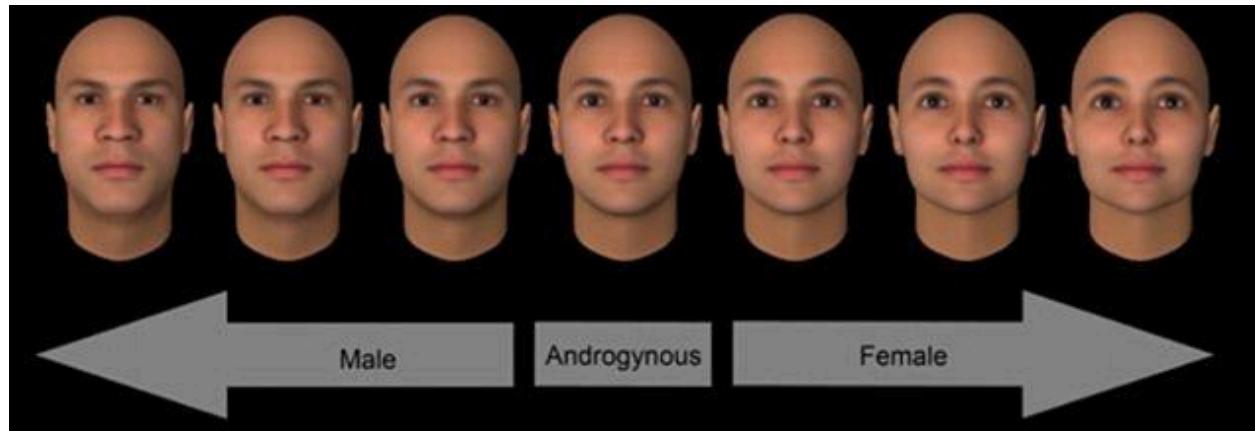
In conclusion, the literature on the relation between testosterone and baseline muscle mass and potential for muscle growth is not as simple as you would think. This appears to be because differences in testosterone level between individuals don't say much, but within an individual it's clear that increasing his testosterone level within the physiological range will increase lean body mass by up to several pounds, possibly more in the case of a hypogonadal male. Hormone therapy and AAS use clearly

potentiate the rate of muscle growth and extend the ceiling level of potential muscle growth. Higher testosterone levels may also increase the rate at which you gain muscle from strength training, but the synergy appears to be relatively small. Most of the muscle growth seems to be from the testosterone itself, whether you train or not.

What about women?

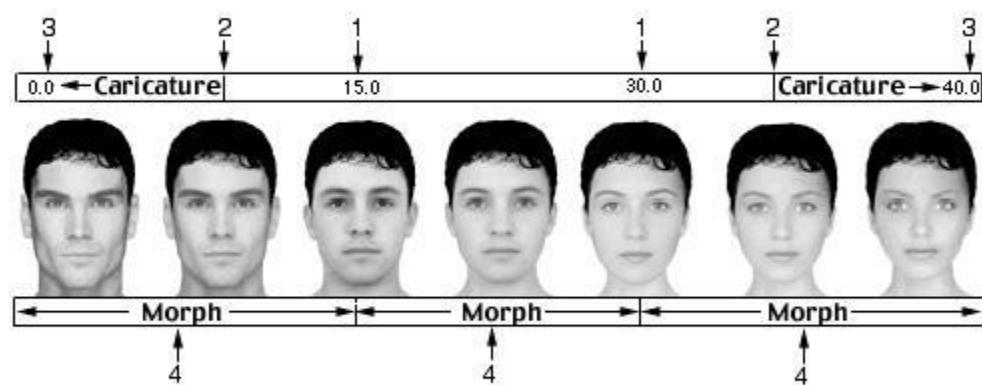
Testosterone seems to have the same neuromuscular effects in women, but since women typically have over 10x lower testosterone levels than men, changes within the physiological range make very little absolute difference for women's body composition. The reference range for women's testosterone levels is around 15-70 ng/dL, so if we go by the 1 pound per 100 ng/dL rule of thumb, it makes sense that the effects of say a 20% change are going to be very small. Nevertheless, higher testosterone levels in women correlate with higher levels of lean body mass, just like in men [2]. We also see some null findings across individuals and sometimes stronger relationships with low body fat than with high muscle mass, just like in men. In fact, in some research the testosterone level of natural female athletes can explain 66% to 90% of how fast they gain strength and power.

Physiologically, many sex differences exist on a continuum. We might think of many things as either masculine or feminine, but most features exist on a spectrum. For example, when you take away cultural cues such as hair style and make-up, people are surprisingly bad at identifying a face as male or female.



The continuum of masculine to feminine facial features.

Here's the same continuum drawn out to caricature levels.





The use of androgenic compounds in women can cause virilization, demonstrating how sex differences exist on a spectrum.

In conclusion, testosterone has the same anabolic and androgenic ('man making') effects in women as in men.

Why testosterone likes to take it slow

Despite the research being clear that increasing someone's testosterone level increases their lean body mass, increasing someone's testosterone level with lifestyle modifications, such as a higher fat diet, does not seem to noticeably change their physique. Most studies do not find any benefits of higher fat diets on muscle growth (see the section on empirical evidence). That's likely because testosterone's effects take weeks to months to manifest. [Howard et al. \(2022\)](#) found 200 mg weekly testosterone supplementation did not significantly affect muscle protein synthesis in the first month of the study, only during the second month. Even during a cycle of

steroids where testosterone is elevated well over 10 times the physiological range, it can take a month before the effects noticeably ‘kick in’, as it’s called. Many fast-acting drugs are even used as ‘kickstarters’ during steroid cycles for this reason.

This latency in the effects of testosterone makes sense if you know how testosterone affects strength training adaptations. Testosterone does not have many direct effects like cortisol, for example. Instead, testosterone acts on the androgen receptor, this information is passed on to the cell cores and they give the instruction to synthesize muscle proteins. Genomic effects like these that are mediated by the transcription and synthesis of new proteins are relatively slow. You’re effectively rebuilding your body. Genomic effects are like installing a new lamp in your room, in contrast to more direct effects of, say, cortisol that just flips on the light switch or increases the voltage.

Testosterone also considerably aids the formation of new satellite cells, which in turn help myonuclear addition. Myo-what? ‘Myo’ refers to muscle and nucleus refers to a cell core, so myonuclei are muscle fiber cell cores. Your genes are located within these muscle cell nuclei. They contain the blueprint to create new proteins via a process called myonuclear transcription. You can think of your myonuclei as command centers that support muscle growth in their surrounding part of the muscle fiber. After considerable muscle hypertrophy, nearby satellite cells are activated and fuse to the muscle fibers to enlarge them and aid in the creation of more new muscle proteins. This process of forming new nuclei in a muscle fiber is called myonuclear addition.

When we look at who gains more muscle from strength training, we can see that the genetically lucky among us ('high responders') experience a greater increase in satellite cell number and myonuclei, along with more muscle growth, than others ('low responders' or more informally 'hardgainers'). Without myonuclear addition, muscle

growth is limited, because each nucleus has a limited transcriptional capacity. In other words, each command center can only support a limited area around it: this area is called its myonuclear domain. [When satellite cell activity is suppressed, which limits the formation of new myonuclei, muscle growth initially occurs as normal but plateaus thereafter. Myonuclear addition seems to be required to achieve muscle growth past a certain level,](#) past the myonuclear domain, although the domain size seems to have some flexibility.

Since testosterone stimulates satellite cell activity and accretion, it likely becomes more relevant in more muscular individuals. Indeed, [the correlation between testosterone and strength is far stronger in stronger athletes than in weaker athletes](#) and in one study, [trained but not untrained men with higher testosterone levels gained strength faster.](#)

Estrogen

In the research overview you can also see that [low-fat diets decrease \(free\) estrogen levels in women, independent of energy intake](#) (and likely in men as well at very low intakes). Many people regard estrogen as one of the catabolic ‘bad guy’ (bad woman?) hormones. This is complete nonsense. Estrogen does many favorable things for your physique.

- [Estrogen aids in muscle repair.](#)
- [Estrogen is anti-catabolic](#), i.e. it prevents muscle damage and loss, and even mildly anabolic. [Hundreds of studies have demonstrated the anabolic effects of estrogen.](#)
- [Estrogen decentralizes your body fat distribution](#), thereby improving your metabolic health and causing you to store less fat around your midsection.
- [Estrogen makes your joints, bones and tendons stronger.](#)

- While estrogen is often said to make you fat, [estrogen actually increases your metabolism](#). It does increase water retention, however, which is easily mistaken for fat gain.

Estrogen's bad reputation in bodybuilding can largely be traced back to anabolic steroid users. In men, injecting large amounts of testosterone increase estrogen levels along with it because the testosterone aromatizes to estrogen. This is still often not truly a problem for their muscles, performance or fat level, but it does cause massive water retention to have estrogen levels severalfold those of a natural trainee. This can amount to several kilograms of water weight, enough to make a bodybuilder in contest shape still look puffy. Bro bodybuilders commonly mistake this water retention for fat mass. Combine that with the side-effects of multi-physiological estrogen levels, such as gynecomastia ('gyno'), and estrogen becomes the enemy.

Conclusion

In summary, increasing your testosterone level is most likely beneficial for your muscle and strength gains, so higher fat diets that increase anabolic hormone levels may slightly improve body recomposition and strength development. The effects can take weeks or even months to manifest and they are modest, nothing like those of high dose anabolic-androgenic steroids, but they may amount to a few pounds of muscle over time. To preserve optimal sex hormone functioning, [some reviews have argued that athletes should not consume fewer than 25% of their energy from fat](#). However, the effects of going over a fat intake of 20% of energy intake are small, so if there is good reason to prefer a higher carbohydrate diet, that should take priority. When there is no reason to prefer a higher carbohydrate diet though, fat intakes of up to 40% may favorably alter anabolic hormone production and subsequently body composition.

The anabolic properties of fat

Dietary fat is not just anabolic indirectly by virtue of its positive effect on anabolic hormones or via its association with cholesterol. Nor is it just anabolic in terms of containing energy, like carbohydrates are primarily as you've seen in the course topic on carbohydrates. In contrast to carbohydrates, certain fatty acids have been found to stimulate muscle protein synthesis. Certain fatty acids may also have anabolic effects in lean tissues by way of modulating their interactions with proteins and hormones. Fatty acids can be incorporated into a cell's membrane and this can profoundly change a cell's metabolism.

Omega-3s

Nutritional interest in the omega-3 fatty acids has escalated enormously in the last decades because of their health benefits. An abundance of research finds positive associations between health and a high omega-3 concentration in the body or the diet [2, 3, 4]. Omega-3 fatty acids are particularly hypolipidemic: they lower blood lipids, particularly triglycerides while potentially slightly raising HDL-cholesterol). Omega-3s are also very anti-inflammatory. Less consistent improvements have been found in insulin resistance, endothelial function, depression, thrombosis (blood clots), hypertension and inflammatory joint pain. The benefits are strongest in unhealthy individuals. The latest meta-analyses show omega-3 intake is associated with reduced cardiovascular disease and mortality [2, 3].

The 3 major omega-3 fatty acids are EPA, DHA, both found almost exclusively in fish, and ALA, the essential fatty acid, found mostly in plants: see the following table.

Table 5.2 Dietary Sources and Tissue Distribution of the Major n-3 Polyunsaturated Fatty Acids

Major Members of Series	Tissue Distribution in Mammals	Dietary Sources
α -linolenic acid 18:3 n-3	Minor component of tissues	Some vegetable oils (soy, canola, linseed, rapeseed) and leafy vegetables
Eicosapentaenoic acid 20:5 n-3	Minor component of tissues	Fish and shellfish
Docosahexaenoic acid 22:6 n-3	Major component of membrane phospholipids in retinal photoreceptors, cerebral grey matter, testes, and sperm	Fish and shellfish

Unlike adipose tissue, omega-3s are a desirable form of stored fat. Specifically, the omega-6 AA- (arachidonic acid) and the omega-3s ALA-, EPA-, and DHA-containing phospholipids or triglyceride are incorporated into your cell membranes (illustrated below). The higher the degree of unsaturation among the fatty acids within a membrane, the greater the fluidity of that membrane, which is generally a positive trait for the cell. Fluids membranes can, very roughly speaking, interact more effectively with hormones and proteins.

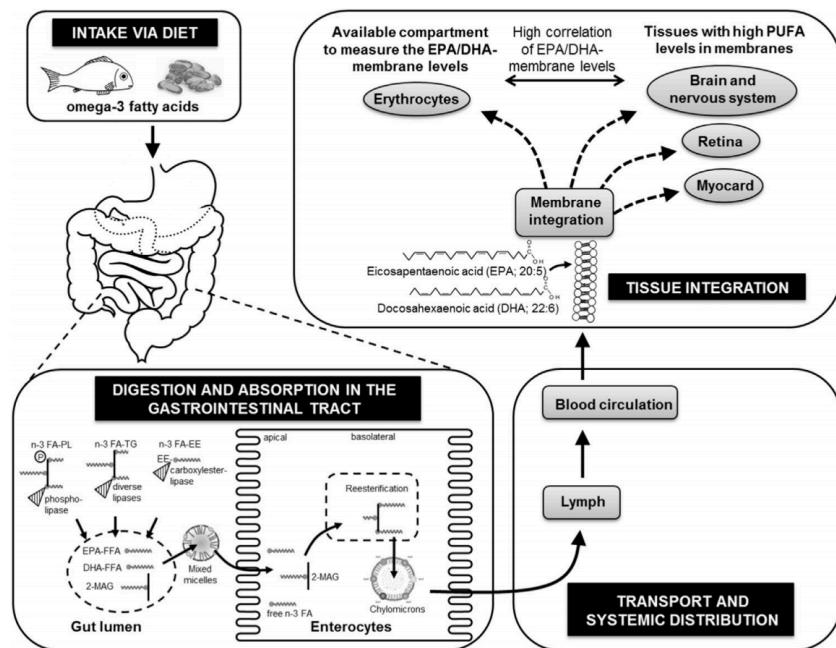
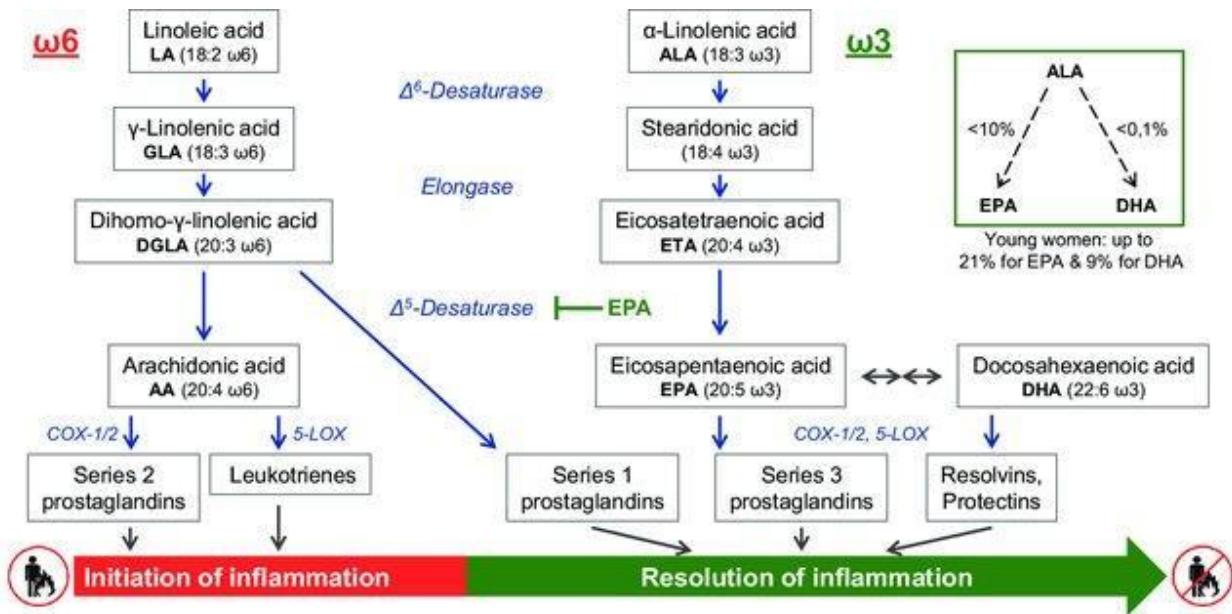


Fig. 1. Route of long-chain omega-3 fatty acids (n-3 FA) from food to tissue. After emulsification of the fats in the stomach, they enter the small intestine where the n-3 FA are cleaved off from their various types of bonds to form free fatty acids and 2-monoacylglyceride (2-MAG). Free n-3 FA and 2-MAG are taken up as mixed micelles. In the enterocytes, n-3 FA are re-esterified to triacylglycerides, which are then incorporated into chylomicrons and transferred via the basolateral membrane to the lymph and thus to systemic circulation. The blood then transports n-3 FA to the target tissues, where they are primarily incorporated in membranes.

Having at the very most 4 times as much omega-6 as omega-3 in your body and preferably closer to 1:1 is commonly recommended, because in isolation, omega-6 fatty acids have opposite effects of omega-3s. However, [in a live human body, the omega 3:6 ratio is probably trivial compared to the importance of just consuming enough omega-3 in the first place.](#) Omega-6 is not the enemy. While omega-3 is anti-inflammatory, [the most common omega-6, linoleic acid \(LA\), is not pro-inflammatory in living humans](#) in most studies, contrary to popular belief and many older textbooks.

Mechanistically, omega-6 fatty acids have the potential to be inflammatory for 2 major reasons. First, all PUFAs oxidize relatively easily, which can trigger oxidative stress and inflammation. However, while isolated omega-6 fatty acids are easily oxidized, [fatty acids in the human body mostly occur in the form of fatty acid esters](#), such as triglycerides, which don't oxidize as easily.

The second major putative pathway of omega-6 intake to inflammation is via arachidonic acid (AA). Linoleic acid can be converted to arachidonic acid, which can in turn be converted into pro-inflammatory eicosanoids, as illustrated below. However, the concentration of AA differs by cell type and is regulated by various processes. Thus, [higher LA intakes do not linearly result in higher AA levels, nor higher systemic inflammation levels.](#) Moreover, [AA can be converted into many eicosanoids, not all of which are inflammatory: some are even anti-inflammatory \[2\].](#)



Mechanistically, omega-3 fatty acids have more direct potential to be anti-inflammatory, whereas omega-6 fatty acids have more direct potential to be inflammatory. However, this does not always turn out to be the case in living humans. [Source](#)

Thus, [in the context of a healthy diet made up of whole foods, omega-6 fats are not inherently bad for you](#). A 2018 Cochrane meta-analysis of 19 RCTs found no significant effect of omega-6 fat intake on all-cause mortality or blood lipid fractions. In some research, [omega-6 fats have in fact been found to improve cardiovascular health](#). As such, most people should emphasize consuming enough omega-3s, not restricting their omega-6 intake of whole foods.

Effects on performance and body composition

Omega-3 fatty acids are [anti-catabolic and even directly anabolic in several ways](#).

- [Omega-3 fatty acids can lower chronic inflammation levels \[2\]](#) and thereby increase the inflammatory signal for cell repair and lower protein breakdown rates, as per the course section on inflammation's signal-to-noise relation with muscle growth.
- [Omega-3s protect you against excessive muscle damage](#) and [improve neuromuscular recovery \[2\]](#).
- [Omega-3 fatty acids can lower cortisol levels, reducing catabolic activity and improving nutrient partitioning \[2\]](#).
- [Omega-3 fatty acids can increase testosterone production](#), according to some research.
- [Omega-3 fatty acids can increase muscle anabolic signaling and protein synthesis rates after meals](#), although [a 2024 meta-analysis](#) found that omega-3s don't generally significantly increase *muscle* protein synthesis, only whole-body protein synthesis.

Various studies have found [increases in lean body mass and muscle growth as a result of increased omega-3 intake \[2, 3, 4, 5, 6, 7\]](#), [as well as increased muscle performance](#) (e.g. strength) [\[2, 3, 4, 5\]](#) and [improved recovery](#). However, the benefits are inconsistent and often small. In many studies they don't reach statistical significance. [A 2022 meta-analysis on older adults](#) found mixed positive effects on strength but no significant effect on lean body mass. [An earlier 2020 meta-analysis in the elderly](#) did find a significant but small positive effect on muscle mass and again mixed effects on functional performance. The benefits were greater in studies with durations of at least 6 months and dosages of at least 2 g EPA + DHA per day. [A 2023 meta-analysis by](#)

[André et al.](#) found a small positive effect of omega-3 supplementation on muscle mass that was just short of statistical significance ($p = 0.11$) and a similarly small but in this case statistically significant positive effect on muscle strength. Interestingly, this analysis could not find an effect of supplementation dosage or any interaction effect of age or the presence of strength training or not.

[Omega-3 fatty acids can also increase fat oxidation rates \[2, 3\]](#) and [increase your metabolism by increasing protein synthesis rates and lean body mass \[2\]](#). A few studies have even found that [increasing omega-3 intake resulted in a decrease in fat mass \[2, 3, 4, 5\]](#), though it did not always reach statistical significance, which is expected given that in much of the research the omega-3 fatty acids were added on top of the regular diet and thus increased caloric intake. If the omega-3 intake comes from supplements added to the diet, thus increasing total daily energy intake, it will generally not result in meaningful fat loss.

➤ Research overview

[Effect of omega-3 intake on body composition and performance](#)

Conclusion

While most benefits are inconsistent and small, the wide range of potential benefits makes a high omega-3 intake appealing. How much omega-3 do we need for maximal benefits? Most recommendations are based on the prevention of disease. [Most official health recommendations are in the range of 250 – 500 mg combined EPA + DHA per day from fish](#). However, [other scientists recommend 2+ g EPA + DHA per day](#),

especially for people at risk for cardiovascular disease and [recent analyses find benefits up to 4 g per day for cardiovascular health](#). For athletes, [a review by Thieliecke & Blannin \(2020\)](#) showed that studies with more than 2 g EPA/DHA result in more consistent benefits on muscle recovery and performance than lower doses. [Van Dusseldorp et al. \(2020\)](#) is the only true dose-response study we have. It compared neuromuscular recovery with supplementation of 2, 4 or 6 grams of daily fish oil supplementation. The 6 g group with 4.2 g total EPA + DHA showed the fastest recovery, suggesting there are benefits of supplementing more than 2.8 g total EPA + DHA per day. However, given the cost, lack of clear dose-response effects in the literature as a whole and the potential for oxidation of very high dosages of fish oil, we recommend 2 g combined EPA + DHA per day for most strength trainees. From a high-quality and bioavailable source, this come very close to maximizing the benefits of omega-3. Endurance and concurrent athletes may benefit more from higher doses.

[Side-effects from even double this omega-3 intake are rare](#), so they shouldn't be a concern.

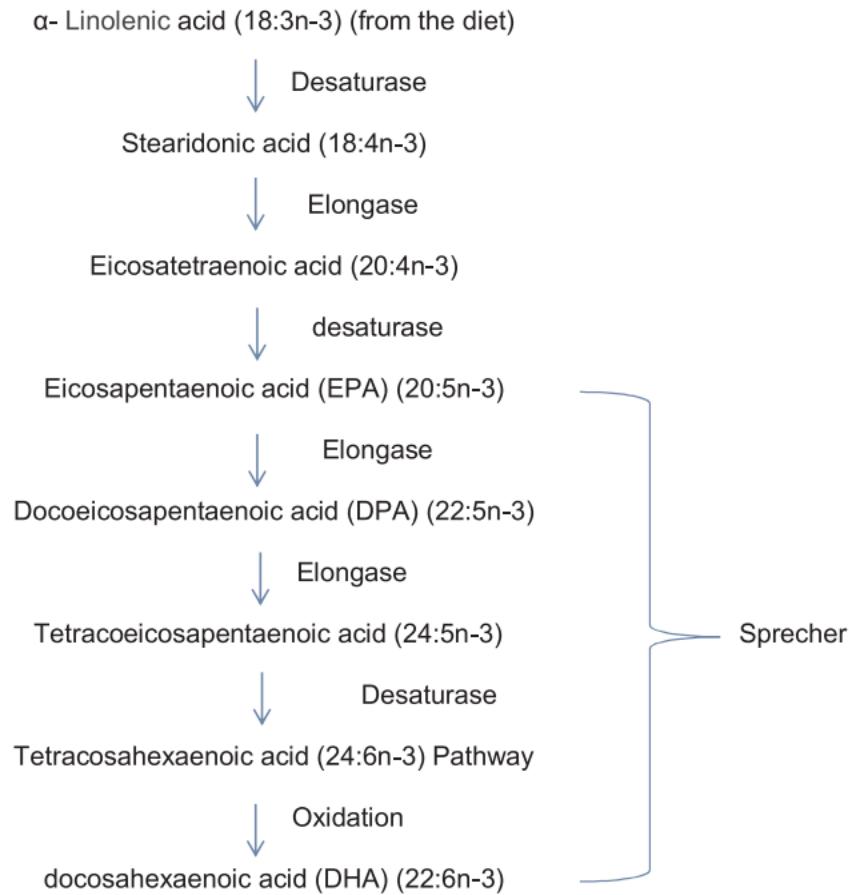
The best way to get at least 2 grams of daily EPA + DHA is to eat at least 700 grams of fatty fish per week. Supplements have lower omega-3 bioavailability than fish (see supplements module), so with fish oil pills, an omega-3 intake of 3 g of combined EPA and DHA per day may be needed for near-maximal benefits.

If budget is a concern, 500 mg EPA plus DHA from food or 750 mg from supplements is the minimum recommended intake for strength trainees.

If you decide to supplement rather than eat fish, a quality supplement is crucial to avoid paying for poison, as we'll discuss in the module on dietary supplements.

You don't need to consume your omega-3 target every day, as the fatty acids are stored and incorporated into your cell membranes. So you can in principle just eat around 700 grams of salmon once a week and you're covered for the whole week.

Alpha-linoleic acid (ALA) is not counted towards omega-3 intake in our recommendations, in contrast to many textbooks. In the past, ALA was considered the primary omega-3 fatty acid, as it's the parent compound for both EPA and DHA: ALA can be converted into either (illustrated below). However, most ALA in the body is typically oxidized. [The conversion rate of ALA to EPA and DHA is only a few percent \[2, 3\]](#), so you cannot realistically fulfill your EPA and DHA requirements by consuming ALA. [The body can quite effectively convert EPA to DHA](#) (but not effectively vice versa) though.



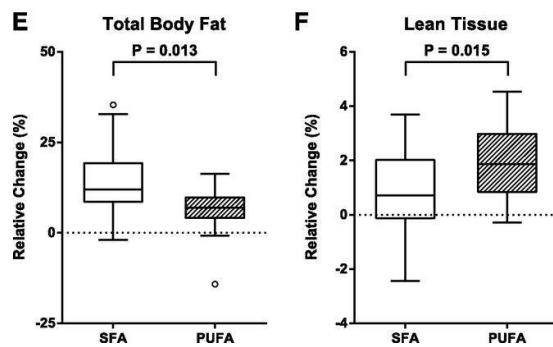
The conversion of ALA to EPA to DHA. [Source](#)

Omega-6 intake from whole foods need not be restricted as long as omega-3 intake is optimized. Overweight individuals may also want to limit total intake, though this tends to naturally occur as a result of their energy restricted diet.

Other polyunsaturated fat

Other polyunsaturated fatty acids, particularly the wrongfully stigmatized omega-6, may also exert anabolic effects, like omega-3 fatty acids.

[The LIPOGAIN project](#) compared subjects overeating on 750 kcal worth of muffins loaded with either palm oil, rich in saturated fat, or sunflower oil, rich in omega-6 polyunsaturated fat. The omega-6 group gained nearly 3 times as much lean body mass and less fat than the saturated fat group: see the data below.



[Norris et al. \(2009\)](#) compared subjects adding either safflower oil, rich in omega-6, or CLA, a natural trans-fat, to an otherwise identical diet in terms of macronutrients. The omega-6 group gained a significant amount of lean body mass without a change in fat mass, whereas the CLA group lost a significant amount of fat mass without a change in lean body mass. This supports the anabolic role of omega-6 fatty acids.

[Multiple other cross-sectional studies](#) have found associations with lean body mass and PUFA intake. We also have [data that PUFAs can be anabolic in the lean tissues of animals](#). See the research overview below for study details.

Author/year (Reference #)	Study design	Body composition technique	Main associations PUFA vs. lean mass
Welch et al. 2014 ³⁸	Cross-sectional population-based (UK); 2,689 healthy women (18–79 years); Food-frequency questionnaire	DXA	Higher lean mass associated with higher ratio of dietary PUFA/SFA (but not with long-chain n-3 PUFA)
Reinders et al. 2015 ³⁹	Cross-sectional population-based (Iceland); 836 older adults (66–96 years); Plasma phospholipid fatty acids	CT	Larger muscle size with higher plasma PUFA. Lower intramuscular fat with higher plasma LA (but not with EPA)
Belury et al. 2016 ⁴⁰	Cross-sectional population-based (USA); 139 middle-aged adults (80% white); Erythrocyte fatty acids	DXA	Higher lean mass with higher erythrocyte LA (but not with long-chain n-3 PUFA)
Cardel et al. 2015 ⁴¹	Cross-sectional (USA); 311 children (7–12 years) racially diverse; Two 24-h dietary recalls	DXA; CT	Higher lean mass and lower body fat% and visceral fat with higher dietary PUFA
Norris et al. 2009 ⁴²	RCT cross-over 16-week intervention (USA); 55 premenopausal women (<70 years) with obesity and type 2 diabetes supplemented with 8 g/day safflower oil or conjugated linoleic acid	DXA	Higher lean mass and less trunk fat with LA-rich safflower oil
Rosqvist et al. 2014 ⁴³	RCT overfeeding for 7 weeks (Sweden); 39 young normal weight men overfed (+750 kcal/day) on high sunflower oil or palm oil	MRI; DXA; bod-pod	Same weight gain (1.6 kg) but higher lean mass and less visceral fat and hepatic lipids with LA-rich sunflower oil

Abbreviations: CT, computed tomography; DXA, dual-energy X-ray absorptiometry; EPA: eicosapentaenoic acid; LA, linoleic acid; MRI, magnetic resonance imaging; PUFA, polyunsaturated fatty acids; RCT, randomized controlled trial; SFA: saturated fatty acids.

However, [a 2024 replication study of the LIPOGAIN project in overweight individuals](#) did not find a significant difference between saturated and polyunsaturated fat overfeeding on lean body mass gains. They also could not replicate the association between PUFA consumption and lean body mass in UK Biobank data.

TABLE 2 Effect of isocaloric refeeding high-fat diets varying in oil sources on body composition and plasma insulin during weight regain over 2 weeks (after caloric restriction) in a rat model of semistarvation-refeeding

Dietary fat types vs. lard (SFA rich)	Fat mass	Lean mass	Insulinemia
Olive oil (MUFA rich)	Ns	Ns	↓
Fish (menhaden) oil (EPA rich)	Ns	Ns	↓
Coconut oil (MCT rich)	↓↓	Ns	↓
Grapeseed oil (LA rich)	↓↓	↑↑	?
Sunflower oil (LA rich)	↓↓	↑↑	?
Safflower oil (LA rich)	↓↓↓	↑↑↑	↓↓
Linseed (flaxseed) oil (LA and ALA rich)	↓↓↓	↑↑↑	↓↓
Safflower:linseed mixtures	↓↓↓	↑↑↑	↓↓
ARASCO oil (ARA rich)	↓↓↓↓	↑	Ns
DHASCO oil (DHA rich)	↓↓↓↓	↑	Ns

Note: The results are presented schematically, with changes (increases or decreases using arrow symbols) in body composition and insulinemia relative to those of animals refed a high SFA (lard) diet, namely, ↑ marginal increase; ↑↑ modest increase; ↑↑↑ marked increase; ↓ marginal decrease; ↓↓ modest decrease; ↓↓↓ marked decrease; ↓↓↓↓ very marked decrease; Ns, no significant difference; ?, unknown (no data). Insulinemia refers of area under the curve of plasma insulin over 2 h during a test of glucose tolerance. Data are derived from Dulloo et al.¹⁴⁹ and Yepuri et al.⁵⁸

Abbreviations: ALA, α-linolenic acid; ARA, arachidonic acid; DHA: docosahexaenoic acid; EPA, eicosapentaenoic acid; LA, linoleic acid; MUFA, monounsaturated fatty acids; SFAs, saturated fatty acids.

Mono-unsaturated fat

We have even less research on the anabolic properties of mono-unsaturated fat.

However, the research we have is promising. An Italian pilot study found that [having people eat 12 green olives a day for a month significantly reduced fat mass and increased muscle growth](#). These results could be attributed to the found decrease in chronic inflammation levels. Alternatively, the body recomposition effect may be specific to olive oil. Previous [animal research has found promising anabolic and anti-catabolic effects of oleuropein, a phenolic compound in olives](#). A major limitation of this study was that macronutrient intakes were not controlled.

[Noakes et al. \(2006\)](#) performed a randomized controlled trial of the effectiveness of 3 different weight loss diets. See the table below for the diet compositions. The primary finding was that the high unsaturated fat (HUF) diet resulted in significantly less lean

body mass loss than the other 2 diets. This is quite striking, since the low carb diet had significantly more protein. The low-fat diet had a similar amount of protein and saturated fat but fewer unsaturated fats. Unfortunately, since both MUFA and PUFA intake were higher, we can't say which was responsible for the HUF diet's better muscle retention in this study. Based on the other research, PUFA is the likely candidate, but it's possible MUFAs have anabolic effects too.

Table 3

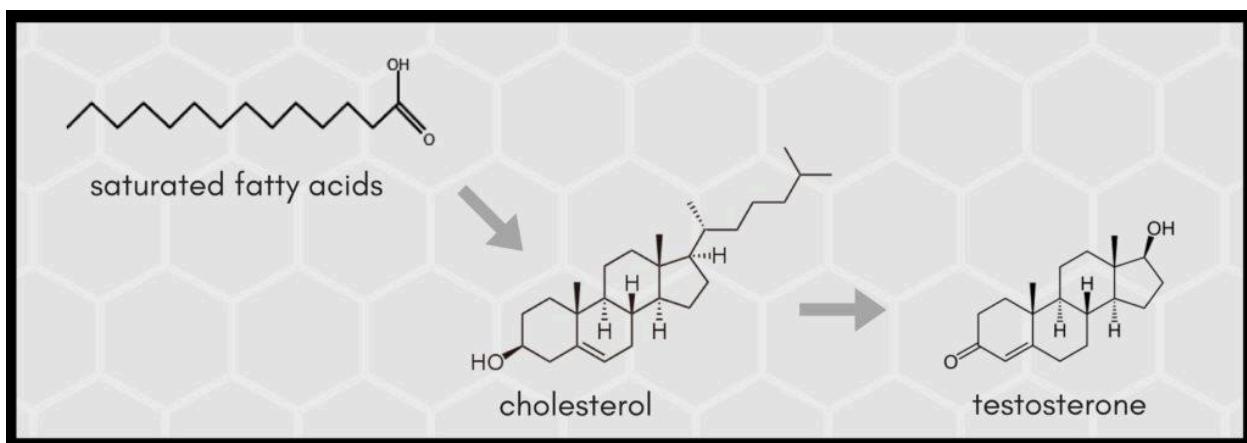
Nutrient intake by dietary treatment during weight loss and weight maintenance assessed using weighed food records¹²

Nutrient	VLCARB		VLF		HUF	
	Weight loss	Maintenance	Weight loss	Maintenance	Weight loss	Maintenance
Energy (kJ)	6193 (\pm 82)	7706 (\pm 167)	6061 (\pm 168)	7000 (\pm 333)	5996 (\pm 88)	7659 (\pm 201)
% energy protein³	33.1 (\pm 0.85)	30.5 (\pm 0.91)	19.9 (\pm 0.33)	20.3 (\pm 0.55)	22.6 (\pm 0.46)	21.4 (\pm 0.51)
% energy fat³	55.1 (\pm 1.96)	54.3 (\pm 2.53)	11.7 (\pm 0.32)	12.5 (\pm 0.59)	27.4 (\pm 0.84)	28.0 (\pm 0.88)
% energy carbohydrate³	8.8 (\pm 2.71)	12.4 (\pm 3.38)	67.7 (\pm 0.60)	66.0 (\pm 0.92)	47.9 (\pm 0.83)	48.7 (\pm 1.07)
% energy saturated fat⁴	17.6 (\pm 0.77)	17.7 (\pm 1.01)	4.5 (\pm 0.16)	5.1 (\pm 0.30)	5.4 (\pm 0.18)	6.0 (\pm 0.32)
% energy MUFA³	27.0 (\pm 1.16)	26.2 (\pm 1.41)	3.3 (\pm 0.11)	3.6 (\pm 0.21)	12.0 (\pm 0.48)	12.3 (\pm 0.51)
% energy PUFA³	6.3 (\pm 0.13)	6.5 (\pm 0.28)	1.7 (\pm 0.03)	1.8 (\pm 0.16)	7.6 (\pm 0.29)	7.2 (\pm 0.30)
Calcium (mg)⁵	959 \pm 14	1297 \pm 58	867 \pm 32	1079 \pm 55	969 \pm 19	1169 \pm 43

Saturated fat

There is no known mechanism by which saturated fat could directly increase muscle growth, nor any data to speak of that it does, although [1 cross-sectional study in strength-training elderly individuals found a correlation between saturated fat intake and lean body mass growth](#). On the contrary, [adding milk fat to casein does not increase myofibrillar protein synthesis](#).

Indirectly though, saturated fat is potentially beneficial for muscle growth in the long run because it can increase anabolic hormone levels. Saturated fats can serve as a building block for cholesterol, which in turn can be converted to testosterone, as illustrated below. A low saturated fat intake is associated with reduced testosterone production. For example, [men going from a 40% fat diet with a high saturated fat intake to a 25% fat diet with a low saturated fat intake experienced a decrease in total and free testosterone levels; going back to their original diet caused testosterone levels to increase again. Several other studies have also found that diets low in saturated fat reduce circulating testosterone levels.](#)



If (saturated) fat is only beneficial to increase our sex hormone levels, this may explain why fat has fallen out of favor among bodybuilders. Since most higher-level bodybuilders are on androgenic-anabolic steroids (AAS), they have no need for saturated fat and they can get away with far lower fat intakes. Before drugs became so widespread in bodybuilding, the old-school bodybuilders weren't afraid of fat at all. Even during the Golden Age of Bodybuilding (Schwarzenegger, Gironda, etc.), whole milk, eggs and meat were all regarded as muscle building foods that were associated with virility and strength.

Isn't saturated fat unhealthy though?

Saturated fat is commonly said to clog your arteries and slowly kill you. Saturated fat's bad reputation traces back to Ancel Benjamin Keys' 'diet-heart hypothesis' from 1952. This hypothesis states that saturated fat increases blood cholesterol levels, cholesterol clogs up your arteries and therefore saturated fat causes heart attacks. This was supported by his subsequent Seven Countries Study in 1972, but [Keys's research was all incredibly shaky epidemiological and animal research without any controlled studies in humans](#). [Many scientists obviously critiqued the diet-heart hypothesis](#), but the simple and fearful message that saturated fat was the cause of the ongoing heart disease epidemic proved more powerful than scientific skepticism.

Research since then has provided only very limited support for the diet-heart hypothesis. While high cholesterol levels are almost certainly a risk factor for cardiovascular health (see section on interpreting cholesterol bloodwork), saturated fat intake does not consistently affect our cardiovascular health. As far back as [1963](#), [Morris et al.](#) reported no relation between people's saturated fat intake and their blood cholesterol levels. Since then, [many scientific reviews have concluded there is little effect of saturated fat consumption on cardiovascular mortality or disease \[2, 3, 4\]](#).

- [A 2010 meta-analysis of prospective cohort studies found no relation between saturated fat intake and coronary heart disease \(CHD\), stroke or cardiovascular disease \(CVD\)](#).
- [A 2015 meta-analysis of observational studies found no relation between saturated fat intake and CVD, CHD, ischemic stroke, type 2 diabetes or, the most important measure of all, all-cause mortality](#).
- [A 2014 meta-analysis on both RCTs and observational research found no clear relation between any fatty acid intake and coronary risk](#).

- Some research actually finds that a higher [saturated fat intake is associated with a reduced risk of heart disease, stroke and mortality](#).

The best evidence we have is arguably [a 2020 Cochrane review of high-quality, long-term RCTs](#). It found that reducing saturated fat intake had “little or no effect” on all-cause mortality or cardiovascular mortality, cancer mortality, diabetes diagnosis, HDL cholesterol, serum triglycerides or blood pressure, but it did result in small reductions in weight, serum total cholesterol, LDL cholesterol and BMI and a substantial reduction in cardiovascular disease risk. The reduction in cardiovascular disease risk was mediated by the decrease in total cholesterol level, supporting that our cholesterol levels are a good predictor of our future cardiovascular health. Unfortunately, the observed fat loss majorly confounds the interpretation of this analysis, because we cannot say if the improved cardiovascular health was the result of the fat loss or the reduction in saturated fat intake.

However, we do [have strong evidence from a meta-analysis of RCTs](#) that saturated fat intake increases cholesterol levels. So if we know higher saturated fat intakes can increase cholesterol levels and higher cholesterol levels are generally associated with cardiovascular risk, why does saturated fat intake often not relate to cardiovascular risk?

There are several reasons for this. First, [what you replace the saturated fat with is the key determinant of how the reduction in saturated fat affects your health \[2, 3\]](#), though contested [here](#). The type of nutrient or food we’re comparing a saturated fat source to seems to matter greatly. Compared to carbs or protein, saturated fat appears to have a largely neutral effect with often no change in cholesterol levels or at worst an increase in both ‘bad’ LDL and ‘good’ HDL cholesterol. However, compared to unsaturated fat,

saturated fat tends to worsen the cholesterol profile. While saturated fat intake per se is quite neutral in effect for our blood lipids, [higher intakes of mono- and poly-unsaturated fat intake typically improve our cholesterol profile](#), unless they're processed trans fats [2, 3, 4, 5, 6, 7].

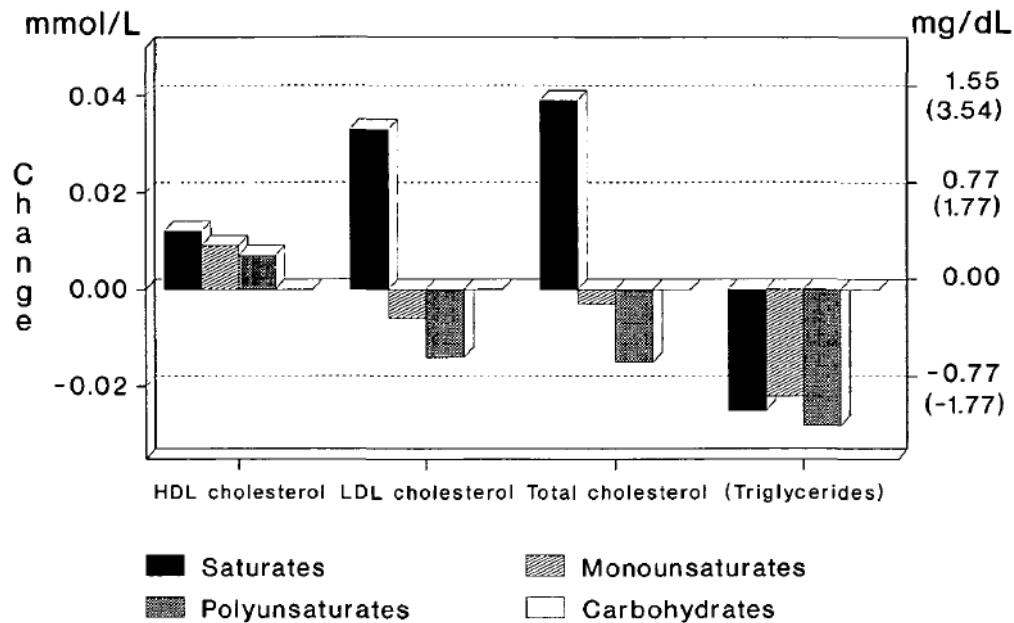


FIGURE 1. Bar graph showing predicted changes in serum lipids and lipoproteins when 1% of energy as carbohydrate is replaced by fatty acids of a particular class under isocaloric, metabolic-ward or similar conditions. Coefficients are valid both ways; thus, replacement of 1% of energy from saturated fat by carbohydrates will cause a fall in cholesterol or rise in triglycerides equal to the length of the black column. Values between brackets refer to predicted changes in triglycerides, expressed in milligrams per deciliter.

Other research supports that [unsaturated fat is better for your cardiovascular health than saturated fat](#) [2, 3, 4, 5], in line with its effects on our cholesterol profile, although [a 2017 meta-analysis by Hamley found no positive effects of replacing saturated fats with omega-6 fats](#).

[The food source of saturated fat also greatly influences cardiovascular risk](#). If you up your saturated fat intake significantly by increasing your consumption of barbequed red meat and this replaces your intake of fish and olives, that's likely not going to be good

for your health. But if you replace hydrogenated vegetable oils with whole milk or cheese, that's likely going to improve your health. Some research even finds positive effects of higher saturated fat intakes from certain sources on blood lipids. [Engel et al. \(2017\)](#) found that the consumption of whole instead of skimmed milk slightly improved HDL cholesterol levels without any detrimental effects on LDL cholesterol, triglycerides, blood glucose or insulin. [Other research finds favorable effects of cheese and meat compared to carbs for the cholesterol profile.](#)

Even if you're a case where saturated fat does increase your cholesterol level, [saturated fat tends to increase LDL particle size in controlled human research \[2, 3\]](#), making it more buoyant and less likely to get stuck and clog up your arteries, which might confer protection against atherosclerosis, if it's not offset by higher cholesterol content of the lipoproteins.

As for fats vs. carbs, [a 2016 meta-analysis of controlled experiments found no beneficial effects on mortality of reducing dietary fat intakes](#). Death rates were similar in studies where the participants did or did not reduce their dietary fat intake.

In summary, saturated fat is beneficial for anabolic hormone and cholesterol production, both of which may be slightly beneficial for muscle growth. Health wise, we could say saturated fat is largely neutral, in comparison to unsaturated fat, which is generally health promoting. As long as the saturated fat comes from whole foods and it does not dominate your diet's fatty acid profile, there is little risk associated with it. As a concrete guideline, strength trainees should not avoid saturated fats from whole foods but also not exceed a saturated fat intake of 33% of total fat intake. The majority of fat intake should be unsaturated. This closely aligns with the official [Dietary Guidelines for Americans](#), which recommends limiting saturated fat to no more than

10% of total energy intake. Your cholesterol profile is a good indicator of your cardiovascular risk. As long as your cholesterol levels are good and you adhere to the above guidelines, there is no strong reason to limit your saturated fat intake further. If your cholesterol levels are suboptimal, it's worth experimenting with a reduction in saturated fat to see if your cholesterol levels improve.

Total fat intake and other fats

As you learned in the course topic on carbohydrates, [a scarce minority of research found that nitrogen balance is greater on high-fat diets than on low-fat diets. The high fat group also lost significantly less lean body mass than the high carb group.](#) The higher fat group diet had a higher ratio of polyunsaturated to saturated fat, so it is not clear if it was the polyunsaturated fat specifically or (also) the higher total fat intake that preserved muscle mass during the diet.

[Several studies have found that total fat intake is significantly correlated with injury risk \[2, 3\].](#) “The odds ratios revealed that runners consuming less than the commonly recommended 30% of total calories from fat were 2.5 times as likely to sustain an injury compared with runners consuming 30% or more. [...] sports nutritionists may want to consider ~36% as a conservative minimum fat intake for avoiding injuries, as long as carbohydrate and protein needs are also met.” This research is mostly in female runners though, because this is an often-studied topic, injury risk is high during running and women benefit greatly from the injury-protective effect of increased estrogen and growth hormone levels. (We'll go into more detail on sex differences in the separate course topic.) Since the mechanisms are likely hormonal and anti-inflammatory, a high fat intake should also be protective against strength training injuries in men.

Unpublished cross-sectional research found positive correlations between total fat intake, bench press strength and the increase in metabolism after strength training.

Women that ate more fat also tended to be leaner than women that ate less fat.

Cross-sectional research is limited in its ability to show causation, but since we know the anabolic effects of fat only manifest in the long-term, these findings make sense and cross-sectional research is more likely to find these benefits than studies lasting only a few weeks.

On a final note, the natural trans-fatty acid CLA (conjugated linoleic acid) and the omega-6 fatty acid AA (arachidonic acid) have been shown to have anabolic effects, but all of the positive research on these is sponsored by the supplement industry, so these are not included here. Same story for the lipid phosphatidic acid.

Empirical evidence

Theory is interesting, but as you learned in the course module on carbohydrates, the theoretical benefits of higher fat intakes don't show up in most studies. The reason we don't see any evidence of the theoretical benefits of higher fat intakes is likely that most studies are only ~8 weeks long, which is too short to observe the benefits of slight increases in sex hormone levels. Moreover, these studies did not specify which fats the subjects should consume, which generally means they did not have a high omega-3 intake. The typical sample of college strength trainees tends to consume a lot of saturated and processed fats, not fish and non-oxidized PUFAs. Thus, while higher fat intakes do not seem to confer significant benefits for strength development or muscle growth within a matter of weeks, they may confer a slight benefit over the course of months.

For reference, the following table – the same from the course module on carbohydrates – summarizes the literature on muscle growth in studies comparing diets with different ratios of fats-to-carbs but the same total energy intake.

➤ Literature overview

[The effect of fat intake on muscle growth and strength development](#)

Dietary fat intake recommendations

Fat intake does not have major effects on our gains. However, based on the inherently anabolic and hormonal effects of dietary fat, a fat intake of at least 20% is recommended for good hormonal health and performance. Anecdotally, much lower fat intakes also result in major increases in appetite. For maximum benefits, a fat intake of 40% of energy intake may be advisable when this doesn't decrease carbohydrate intake to unsustainably low levels.

These guidelines correspond well with the 20-35% [Acceptable Macronutrient Distribution Range \(AMDR\)](#) for fat intake recommended by many health organizations [2]. So proportional to total energy intake, strength trainees should eat a similar fat intake to sedentary individuals.

For maximum benefits from omega-3 fatty acids, consume 700+ grams of fatty fish per week or supplement 3 grams of combined EPA + DHA in the form of fish oil pills.

All other fatty acids should also be present in the diet. The exact ratios are probably not worth calculating, as long as the diet has a good source of saturated fat (e.g. dairy, meat, coconut or chocolate), mono-unsaturated fat (e.g. olives, avocado, nuts, eggs) and poly-unsaturated fat. PUFAs are present in almost all foods, so a sufficient total fat intake combined with specific attention to omega-3 should naturally result in a sufficient PUFA intake, but it's good to keep in mind PUFAs are most beneficial and SFAs are least important.

Fat intake for women

Astute students will have noticed the recommended fat intake for women is slightly higher than for men. Women can benefit from higher fat intakes for several reasons.

For one, women burn more fat and less carbohydrate (and less protein) than men at the same exercise intensity. Since they rely less on carbohydrate as fuel, they also don't store as much glycogen post-exercise.

Both differences in the nervous system and the hormonal system, including estrogen, are responsible for women's lesser reliance on glycogen. For example, the fight-or-flight hormone adrenalin burns more fat in women than men. A more obvious explanation is that women normally have a considerably higher fat percentage than men of the same weight, not only on their body but also within their muscles, so it makes sense to use this as the primary energy source.

Basically, women have a glycogen and protein sparing metabolism. This means women don't need as much carbohydrate or protein in their diet as men to fuel their exercise sessions.

The lesser need for carbohydrates frees up calories to consume as fat. Fats have very positive effects on the hormonal and cardiovascular health of women. In general, the more fat women eat, the more estrogen and testosterone they produce. As you learned, testosterone and estrogen are both anabolic hormones, despite the broscience fear of estrogen.

[Low-fat diets may even reduce breast size](#), which may not just be a result of fat loss but of low anabolic hormone levels, since [estradiol and IGF-1 levels are significantly correlated with breast size](#) in women that aren't on the pill for birth control.

A high-fat diet may also be easier to adhere to for women than men. [Dietary fat is 15% more satiating in women than in men](#).

Women also have less to fear from potential negative effects of a low carb, high-fat diet (which are already rare). [Fats don't decrease insulin sensitivity as much in women as in men](#). Estrogen plays a large role here. It helps to keep inflammation in check, burn fat and preserve insulin sensitivity. Lower inflammation means polyunsaturated fats are less susceptible to being oxidized, so they can exert their anabolic effects. Women in general have much better metabolic health than men and have [a healthier body fat distribution](#).

By the way, if you're worried about breast cancer, the relation between fat intake and breast cancer risk is found in poor epidemiological studies of inactive, overweight women eating [processed junk fats, like processed red meats](#). Even then the relation is weak and controversial. [Fats like olive oil in fact seem to protect you from cancer](#). If you're lean, you don't smoke, don't go binge drinking too often, eat a healthy diet and you exercise, research has found time and [again](#) and [again](#) and [again](#) that you have nothing to fear from a high-fat diet.

Back on topic, several studies have found that [women with polycystic ovary syndrome lose more fat and less muscle on a low carb diet compared to a low-fat diet](#), even when protein and energy intake are tightly controlled. [Several studies by Jeff Volek et al.](#) have found similar results in overweight and healthy women, but these studies were

confounded by more protein in the low carb diets. [Unpublished research](#) found that women with more fat in their diet burn more calories during exercise, have higher bench press strength and are leaner.

Considering the added benefits and risk of low estrogen levels on low-fat diets, women are advised to err on the higher side of the recommended 20-40% fat intake as a proportion of total energy intake; 40% of REE is generally a good guideline.

AAS

For androgenic-anabolic steroid users, the requirement of saturated fat effectively drops to zero, whereas the health risks related to cholesterol increase. This might explain in part why higher fat diets were the norm in natural bodybuilding, but high-carbohydrate diets have replaced them since steroid use became commonplace in bodybuilding. There's only no requirement for saturated fat if someone consumes enough cholesterol though, which brings us to the next topic related to dietary fat.

Cholesterol

➤ Lecture [optional]

Cholesterol

Cholesterol is another lipid that is important for our health, sex hormone levels and possibly our results in the gym. Unlike triglyceride, cholesterol is not an energy-producing nutrient. We don't have the enzymes to catabolize its four-ring structure. Instead, cholesterol has 2 main uses in our bodies.

1. As a structural component of our cell membranes, including the myelin sheath of our nerves. Cholesterol is also used to create the lipid rafts in our cell membranes that have important signaling functions in our immune system.
2. As a building block to create steroid hormones, bile acids and vitamin D.

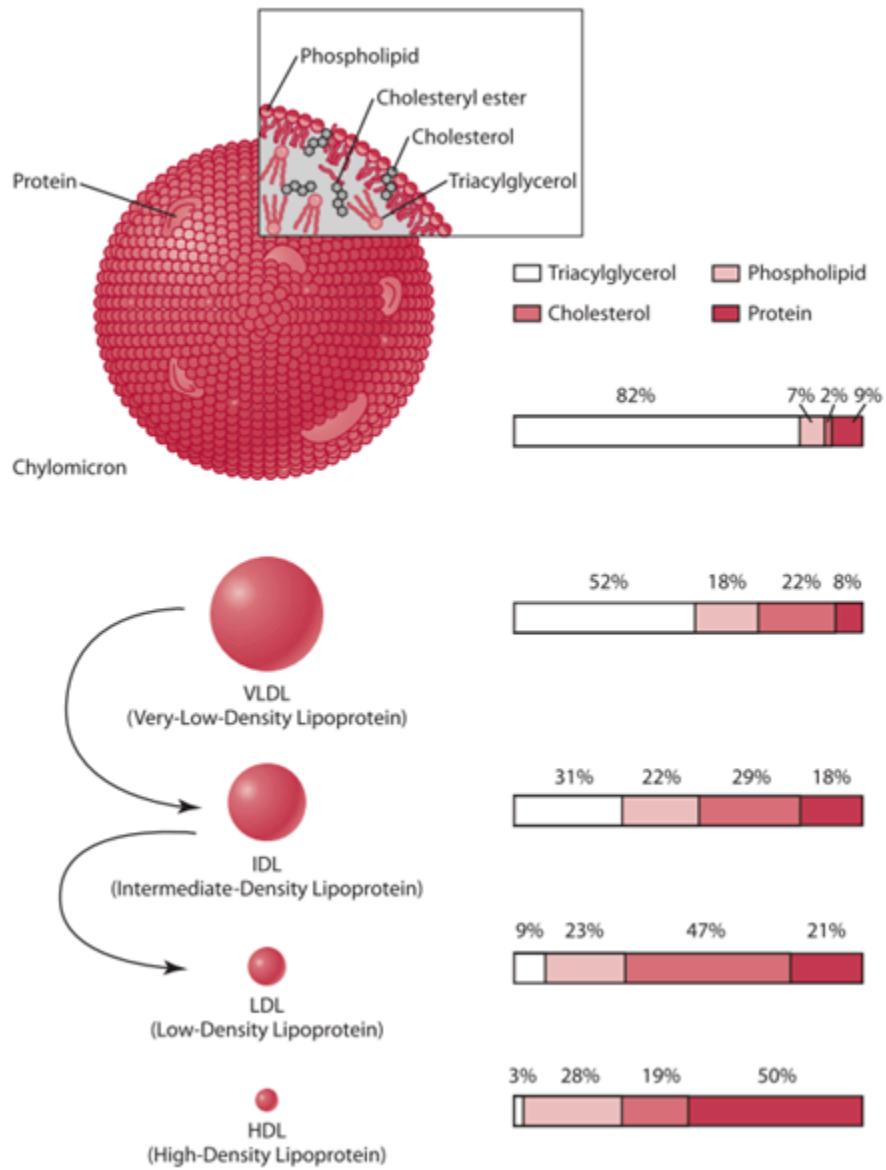
Our bodies obtain cholesterol in 2 ways.

1. [Cholesterol from our diet is absorbed via chylomicrons and high-density lipoproteins \(HDLs\) from the intestines.](#)
2. Cholesterol is created by many of our tissues. The liver is by far the biggest producer. The liver also produces the lipoproteins that transport cholesterol.

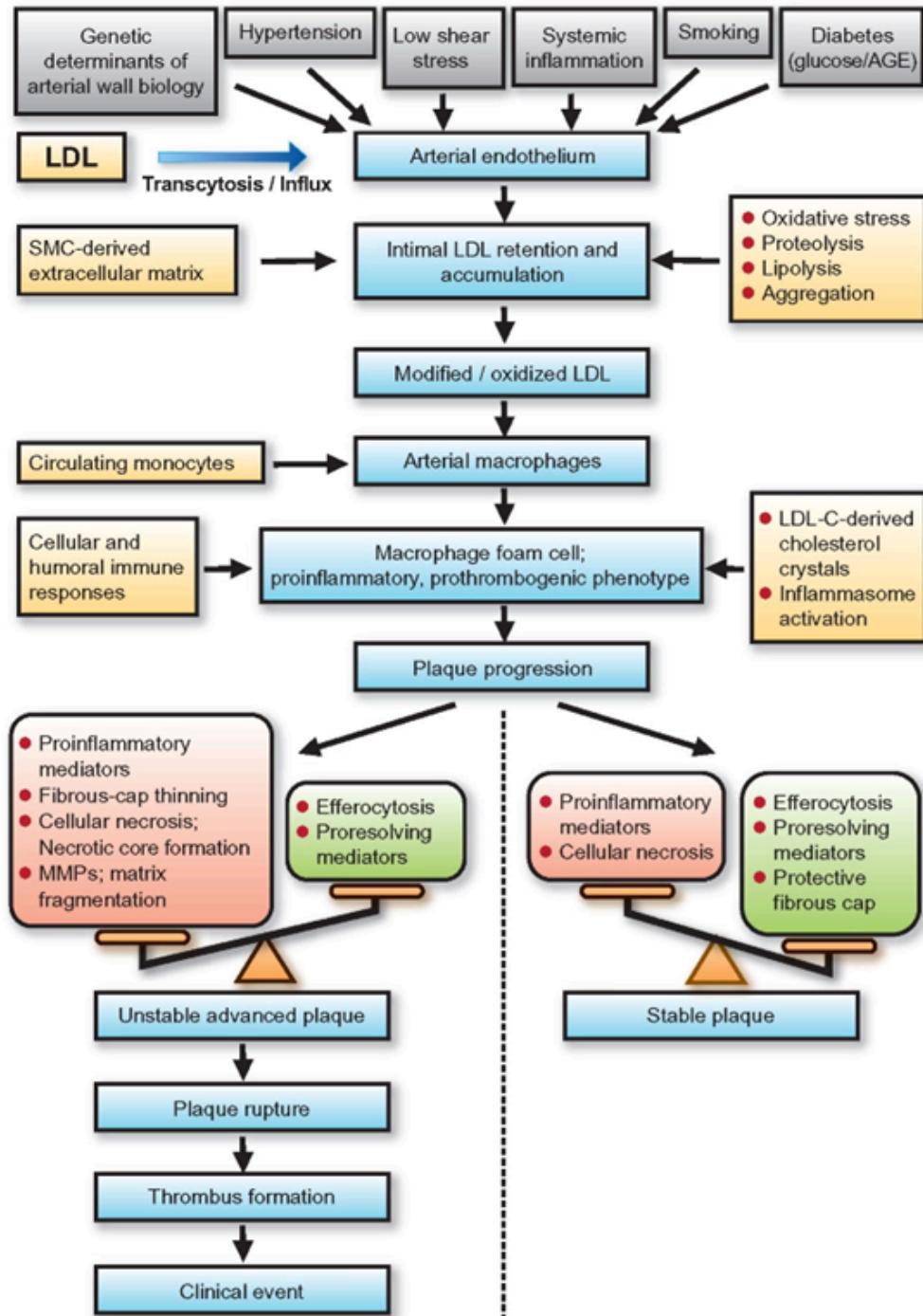
Despite its essential functions, cholesterol is primarily known as 'something that clogs your arteries' by the general population. To understand why, you need to understand cholesterol transport in the body. Cholesterol is not supposed to float freely in the blood. It's carried by lipoproteins, regardless of whether it came from our diet or any other tissue. You've already heard of 2 lipoproteins, but there are 3 more:

1. Chylomicrons transport lipids, including cholesterol, that were absorbed from our diet, to their target tissues.
2. Very low density lipoproteins (VLDLs) transport lipids, including cholesterol, that the liver created, to their target tissues.
3. Lipids have a very low density compared to water and other compounds in our body (which is why lipids generally float on water). Therefore, the more fat is in a lipoprotein, the lower its density. As VLDL deposits some of its lipid shipments, it turns into intermediate-density lipoprotein (IDL)...
4. ...and then into low-density lipoprotein (LDL).
5. There's also high-density lipoprotein (HDL), which the liver creates to collect cholesterol from other tissues to transport back to the liver. HDL has a low density, because it starts off without any lipid cargo.

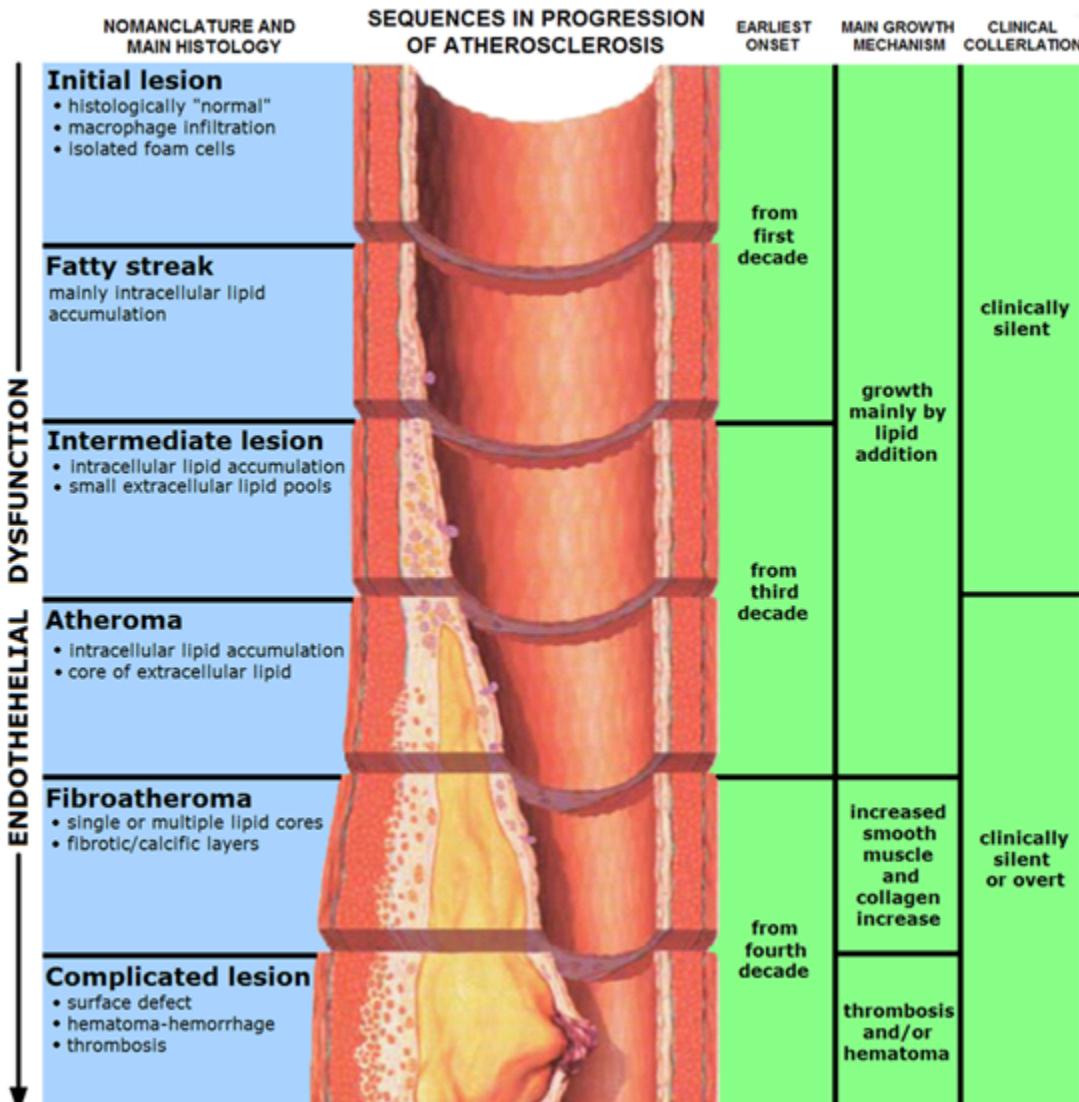
LDL-cholesterol is generally known as 'bad cholesterol' due to the capacity of its load of cholesterol and other lipids to clog up your blood vessels. LDL is singled out as 'the bad guy', because if you haven't eaten recently, there won't be many chylomicrons in the blood anymore. Moreover, VLDL and IDL are short-lived, so most cholesterol floating around in our blood is transported by LDL. However, LDL is not a bad thing. LDL is just a lipid transporter. The problem is that LDL sometimes makes a mess. It can break or get stuck in our blood vessels, causing its cholesterol load to clog up the vessel. Over time, excessive cholesterol accumulation like this can cause plaque that narrows the vessel and obstructs blood flow. Cholesterol getting stuck in our arteries is a common cause of cardiovascular disease. This process is called atherosclerosis and illustrated below.



The lipoproteins in the human body. Chylomicrons transport lipids absorbed from our intestines. VLDLs transport lipids produced by our own body from the liver to their target tissues. As VLDL deposits more of its triglycerides, its density increases and it turns into LDL.



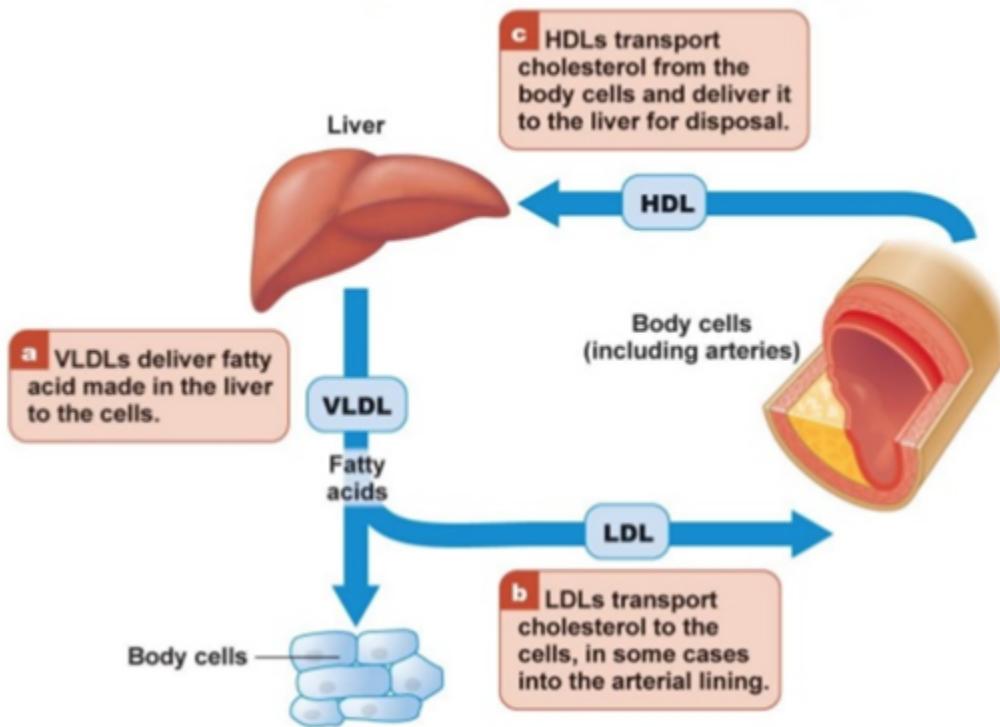
How LDL-cholesterol can lead to plaque build-up in arteries and consequently cardiovascular disease. [Source](#)



The progression of atherosclerosis: plaque accumulates against the wall of an artery, thereby obstructing blood flow and posing the risk of a stroke and other cardiovascular problems.

HDL's primary function is reverse cholesterol transport. Whereas LDL brings cholesterol to our tissues, HDL comes to collect it from them and remove it. Along the way, HDL also collects cholesterol from our blood. Excess cholesterol can be transported to the liver, converted to bile and then excreted. Since LDL is a main cholesterol carrier and

HDL helps to clean up cholesterol in the blood that otherwise clogs your blood vessels, HDL is often called 'good cholesterol' and LDL is called 'bad cholesterol'. HDL is basically the cholesterol clean-up crew.



The roles of the VLDL, LDL and HDL lipoproteins.

Interpreting cholesterol bloodwork

The 'good vs bad' cholesterol categorization is useful to diagnose cardiovascular health, as long as you understand that it is a major simplification. First, both LDL and HDL are important for your body to function and HDL and LDL are lipoproteins that carry more than just cholesterol.

Second, more HDL-cholesterol is not always better. Most research suggests more 'good' HDL-cholesterol is only beneficial up to ~40 mg/dL and at very high levels may even become detrimental for your cardiovascular health [2]. This usually only occurs when the HDL becomes dysfunctional though. At the other end of the spectrum, findings are mixed about whether isolated low HDL-cholesterol without other blood lipid problems is a risk factor for cardiovascular disease [2, 3]: the association with all-cause mortality is inconsistent, but the total body of research does suggest increased cardiovascular risk. Often, low HDL-cholesterol presents along with other risk markers, so it's unclear if low HDL-cholesterol per se constitutes a significant risk or if it's the problems that usually come along with it.

Third, while about 75% of at-risk-for-clogging cholesterol is in LDLs, there's also cholesterol in the other lipoproteins, mostly VLDL (~25%). As such, total non-HDL cholesterol is a better risk marker than simply LDL-cholesterol.

To further improve our risk assessment, we need to look beyond just cholesterol. Cholesterol itself alone cannot enter our artery walls. It can only get stuck in our arterial walls when in apolipoprotein B (ApoB). Apolipoproteins are the proteins that bind our lipoprotein particles together and ApoB is the primary apolipoprotein in chylomicrons, VLDL, IDL, and LDL. Each of those lipoproteins has one ApoB particle. The denser the particle, the smaller it is and thereby the greater the risk of entrapment in the arterial wall but the less cholesterol it has, so the risk of entrapment and the damage of entrapment seem to average out and each ApoB particle carries similar net average risk. Thus, the total ApoB particle number is a great unified measure of the total non-HDL cholesterol as well as the actual cause of plaque formation. Of all the cholesterol numbers, the total ApoB particle number is the best measure of cardiovascular risk [2].

Theoretically, the lower your ApoB, the better. However, [multiple cross-sectional studies have found that low total cholesterol levels are also associated with higher all-cause mortality](#), especially in the elderly [2, 3]. Given the absence of evidence for any negative causal effect of low cholesterol levels, this relationship is probably a confounding effect of certain disease states that lower cholesterol levels and increase our mortality risk. Still, it's not clear that there are benefits of lowering your ApoB if it's already in the healthy range.

Bloodwork should be done fasted, thus ideally in the morning, to avoid confounding the result with the effect on meals, especially fatty meals, on blood lipid and lipoprotein levels.

Considering the major health implications of atherosclerosis, having bloodwork done to check your ApoB particle number is advisable for everyone. If your lab cannot measure ApoB, total non-HDL cholesterol is the best alternative measure.

Older and overweight individuals are at higher risk of cardiovascular disease, but even seemingly healthy, exercising individuals can have high cholesterol levels and this is a health concern. It doesn't appear to matter what the cause of the high non-LDL cholesterol levels is. In addition to the dietary causes, [genetic conditions that cause high LDL-cholesterol levels, such as familial hypercholesterolaemia \(FH\), are also consistently associated with atherosclerosis and cardiovascular disease](#). Conversely, [genetic conditions that cause low LDL-cholesterol levels are associated with reduced cardiovascular risk](#). Some individuals thus have to pay extra attention to their diet to keep their cholesterol levels in check, as they are genetically burdened with increased risk. Intervening earlier in life significantly reduces lifelong exposure to high

atherosclerogenic cholesterol levels and thereby promotes longevity much more effectively than intervening later.

Triglyceride levels in the blood may also be a measure of cardiovascular risk, but any mechanism of action independent of cholesterol is still unknown. As such, it's unclear if triglyceride levels provide information about cardiovascular risk independent of the ApoB particle number. Triglyceride levels strongly correlate with several lipoprotein levels, especially VLDL, and interventions designed to lower triglyceride levels have often failed to improve cardiovascular risk. Similarly, genetic conditions that cause elevated triglyceride levels, such as familial chylomicronaemia (FC), are not consistently associated with increased atherosclerosis. Thus, it's plausible that triglyceride levels are merely a correlate of cardiovascular risk and the ApoB particle number is the true risk indicator. After correcting for cholesterol levels, triglyceride levels are no longer consistently associated with cardiovascular heart disease risk, although some studies still find an independent effect in lower-risk populations. Triglyceride levels naturally increase after eating, especially after high fat meals, seemingly without negative cardiovascular impact, and the definition of hypertriglyceridemia, high levels of triglyceride in the blood, varies substantially with recommendations as low as 100 mg/dL (1.1 mmol/L) to tolerance up to 2 mmol/L. If you have low triglyceride levels and high HDL-cholesterol levels though, some data suggest that high LDL-cholesterol levels are not as harmful, in part because the older but still popular Friedewald estimation formula of LDL-cholesterol levels overestimates LDL-cholesterol levels in people with low triglyceride levels [2]. Individuals with low triglycerides and high estimated LDL-cholesterol should thus retest their LDL-cholesterol levels using a direct measurement.

In conclusion, the total ApoB particle number is the most informative measure for the risk of atherosclerosis and thereby total cardiovascular health. There is strong scientific evidence that a high ApoB reading is a very serious health concern that needs to be addressed. In the health science module, we'll go into more detail on which food choices are best for our (cardiovascular) health.

Cholesterol's effect on muscle growth

The main-stream media demonize cholesterol based on its potential effects in your blood. If you've been basing your perception of cholesterol around that message, you may be surprised to find that [cholesterol has several potential mechanisms of action to increase muscle growth.](#)

- Cholesterol increases membrane viscosity, which may influence membrane stability. This may have an influence on the extent to which muscle cells are damaged during exercise and the magnitude of the inflammatory response.
- Cholesterol seems to play a role in the muscle repair process by controlling inflammation. Muscle damage creates inflammation, which leads to the recruitment of immune cells to assist with the recovery process.
- Cholesterol is essential for lipid raft formation. Lipid rafts assemble the components for signaling pathways and enhance signaling between pathways that play an important role for muscle hypertrophy, such as the growth factors IGF-I and mTOR. Cholesterol depletion can lead to protein missorting, which reduces the signal transduction.

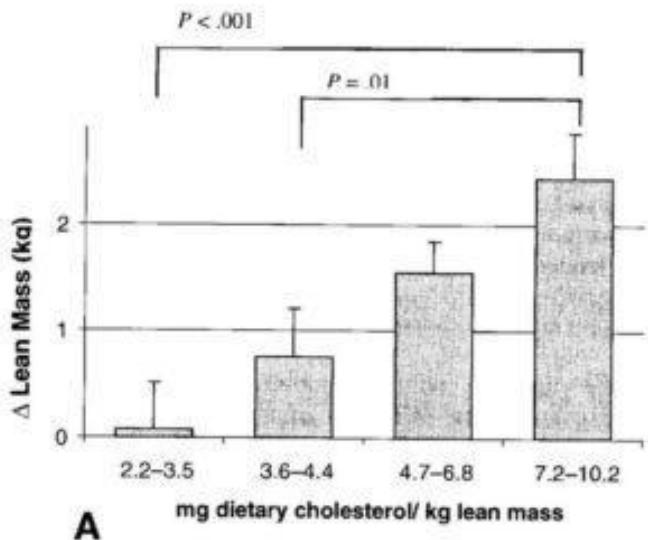
Simply put, cholesterol can help your muscle cells resist damage and can improve their ability to repair themselves after your workouts, which is crucial for muscle growth.

Cholesterol can also improve your gains indirectly.

- Cholesterol is the precursor for anabolic hormones and is crucial for their production.

However, simply having a high serum cholesterol level or eating a ton of dietary cholesterol doesn't necessarily lead to increased testosterone or more lean body mass gain by itself. [The limiting factor of anabolic hormone production is often the transport of cholesterol into mitochondria](#), where its turnover takes place, not necessarily the amount of cholesterol available in the blood stream. So [increased dietary cholesterol intake doesn't consistently lead to increases in testosterone in research](#).

So does a higher cholesterol intake actually help us get more jacked? The research we have is conflicting but promising. [Riechman et al. \(2007\)](#) found that cholesterol may be good for your gains. In their study, 49 elderly individuals completed a 12-week strength training program with nutritional guidelines. Retrospective analysis of participants' nutritional logs showed a linear dose-response relationship between dietary cholesterol intake and lean body mass increases (per DEXA). The more cholesterol they consumed, the more muscle they gained. This relationship held up when protein and fat intake were controlled for. See the figure below.



[Lee et al. \(2011\)](#) compared a high (~800 mg per day) and a low (< 200 mg per day) cholesterol diet in young, healthy adults. The high cholesterol group had a nearly 3 times higher myofibrillar protein synthesis rate 22 hours after intense resistance exercise than the low cholesterol group. Myofibrillar protein synthesis is a measure of muscle growth, specifically how quickly your muscles are creating new proteins, so these findings again suggest a high cholesterol diet is beneficial for muscle growth.

Research has also found that [whole eggs stimulate greater anabolic signaling and subsequent myofibrillar protein synthesis than egg whites with the same amount of protein \[2\]](#). The yolk is very rich in cholesterol, so knowing that cholesterol stimulates myofibrillar protein synthesis, it's likely that it's the cholesterol in whole eggs that makes them great muscle building food.

Most other research on the relationship between cholesterol and muscle growth hasn't been published yet. Below are the data we currently have.

- In a similar design as the first study from Riechman et al., [Riechman & Gasier \(2007\)](#) again found beneficial effects of a high cholesterol intake for muscle growth and strength development, though the effects were more modest than in their previous study and it's unclear if protein intake was controlled for.
- [Riechman et al. \(2008\)](#) performed another replication study and found a dose-response relationship between cholesterol intake and strength development but not lean body mass. Again, it's unclear if protein intake was controlled for.
- In yet another replication study, [Iglay et al. \(2009\)](#) found no relationship between cholesterol intake and muscle growth or strength development. However, they also did not find any effect of 0.9 g/kg (0.41 g/lbs) vs. 1.2 g/kg (0.55 g/lbs) per day of protein, which suggests their study was statistically underpowered to research this topic.
- [Bagheri et al. \(2020\)](#) performed a 12-week RCT in strength training individuals consuming either 6 egg whites or 3 whole eggs post-workout. This resulted in similar total macronutrient intakes but with ~800 mg vs. ~270 mg cholesterol in the diets. Muscle growth and bench press strength gains did not significantly differ between groups but were in absolute terms greater in the whole egg group. The whole egg group lost significantly more fat (group x time significant but between-group was not) and tended to gain more squat strength ($p = 0.06$, again group x time significant but between-group was not). Statistical power was considerably limited by the use of bioelectrical impedance analysis (BIA) to measure body composition and by self-reported dietary intakes from students living in dorms, though the researchers did check up on them daily.

Research on statins also hints at a beneficial role of cholesterol for your muscles.

Statins are a type of medicine used in the treatment of various cardiovascular

(heart-related) problems. [Statins lower your cholesterol and a common side-effect is myopathy.](#) Statin therapy can reduce muscle strength and functionality, induce inflammation in your muscles (myositis) and even complete muscle fiber death (rhabdomyolysis). In some research the use of statins seems to result in compensatory muscle growth. This is probably undesirable for a strength trainee, however, given the functional impairments.

A low cholesterol intake might also be a reason why [lacto-ovo-vegetarian diets tend to result in less muscle growth than omnivorous diets in strength trainees, even with a similar protein intake.](#) [Cholesterol content in plant lipids is about 100-times lower than in animals.](#) Of course, many other factors, like protein quality, could also explain the lesser muscle mass of vegetarians.

All in all, the available research suggests a high-cholesterol diet may improve muscle growth and strength development.

The cholesterol conspiracy theory

Before everyone starts eating 10 eggs a day, we feel the research is a bit suspect: only about 1 in 3 studies on the relation between cholesterol and muscle growth have been published, even though the research was conducted several years ago already. And most of the published research mentioned above was performed by the same principle investigator, Dr. Riechman, who received research grants totaling roughly \$2.7 million (that we could find) from sources including the U.S. Poultry & Egg Association.

However, it's just a fact that scientific research is expensive, so people willing to put a lot money into it, often have something to gain from it. Individuals like Bret Contreras

and Menno Henselmans that pay for scientific research out of their own pocket are very rare. The scientific industry is set up so that the integrity of the researchers should prevent the sponsors' conflict of interest from deceiving the public. And this generally works. [Food industry funded nutrition research does not have significantly different outcomes than research with other funding](#). Plus, Dr. Riechman also received funding from the American Heart Association and the US Army. Not to mention, it's farfetched to pour millions of dollars and risk your career and reputation to promote – of all things you could sell – cholesterol specifically for – of all possible markets – individuals interested in muscle growth. Us meatheads are a tiny part of the population. Us meatheads interested in scientific research tinier still.

Ok, let's take the tinfoil helmets off and assume there's no major conspiracy theory going on. That begs the question...

But isn't cholesterol bad for your heart?

"Over the past 2 decades we have been feeding our severely burned patients with between 30 and 40 eggs per day.

[...]

25 Eggs were given during the day in a variety of forms only limited by the ingenuity of the nursing staff, some of the most popular ways were fried, raw in soups or wine, omelettes and egg-enriched ice cream. During the night 10 raw eggs mixed with half a liter of milk plus ample sugar were fed through a nasogastric tube.

[...]

All of our patients over the years have tolerated this regime surprisingly well.

[...]

Contrary to expectations, serum cholesterol and lipoprotein levels remained normal throughout the period of study."

So reported a group of Israeli researchers [in 1975 in the British Journal of Plastic Surgery.](#)

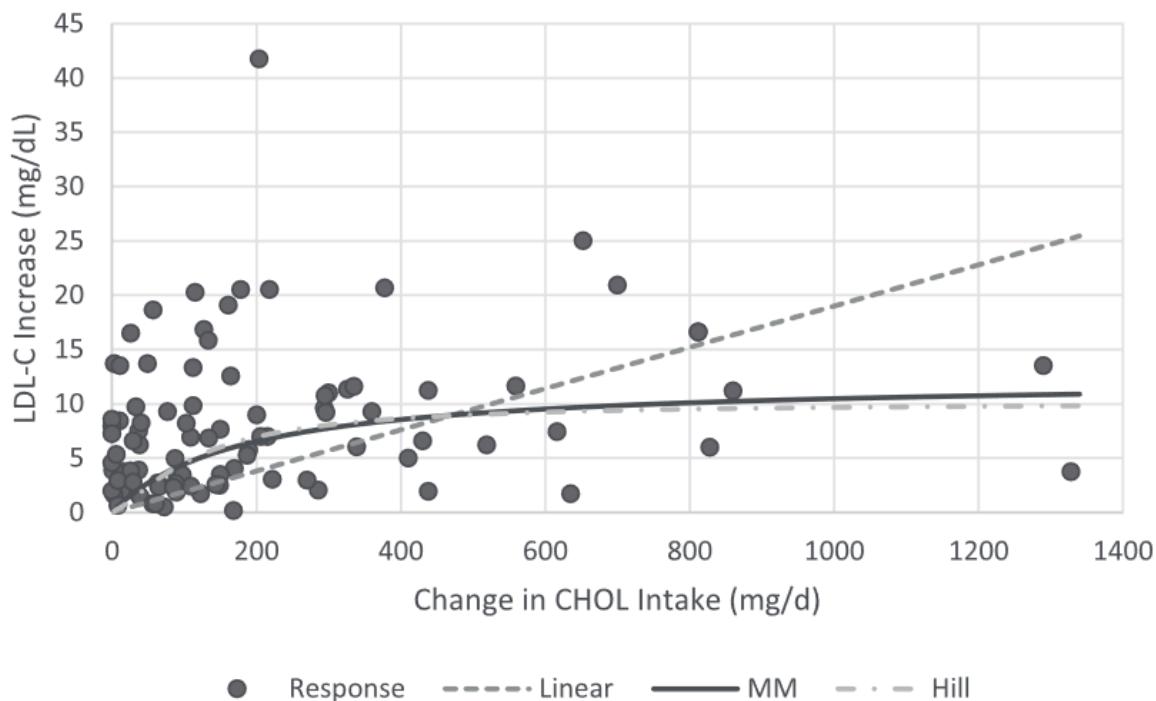
The media will have you believe all that cholesterol you consume ends up as LDL-cholesterol that clogs your arteries and will kill you faster than smoking. As you learned, higher cholesterol levels, particularly [LDL-cholesterol levels, are a risk marker for cardiovascular disease](#) and all-cause mortality. LDL-cholesterol is atherogenic, meaning it can form fatty plaques in our arteries. The theory that dietary cholesterol 1) increases blood cholesterol levels and 2) therefore the risk of cardiovascular disease is thus plausible, yet both parts of this theory have been empirically questioned.

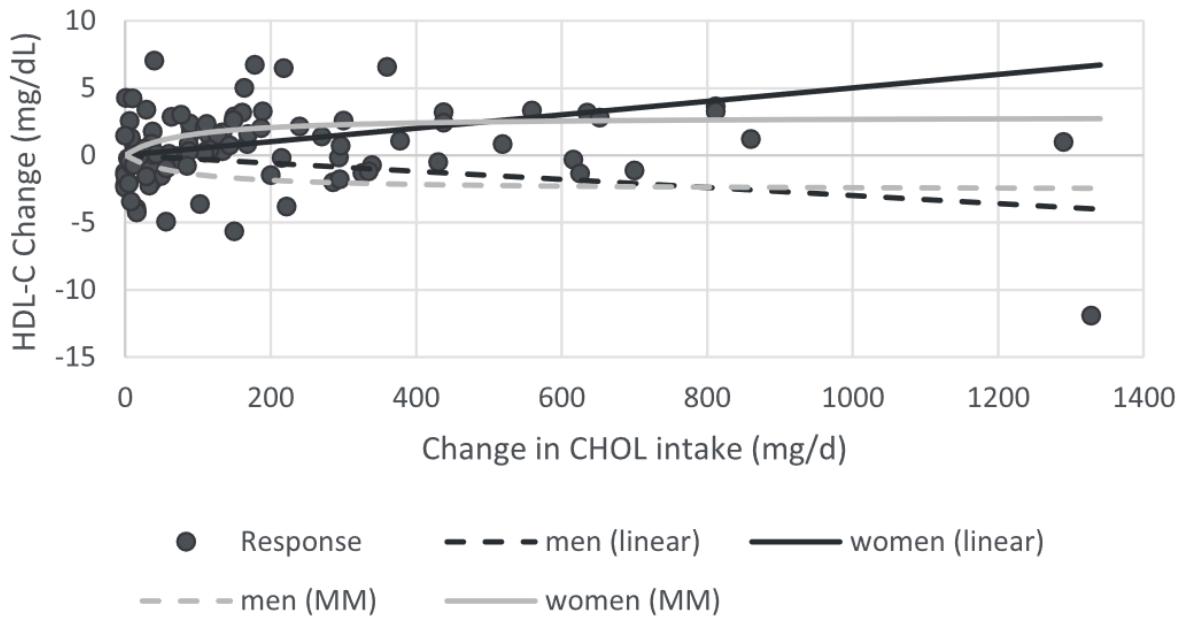
The effect of dietary cholesterol on blood cholesterol

Counterintuitively, [dietary cholesterol intake does not significantly influence blood cholesterol levels in most people \[2, 3\]. The body's blood cholesterol levels are highly regulated](#). If your diet does not contain much cholesterol, your intestines will increase their absorption to compensate. If that's not enough, your body will produce its own cholesterol. Nearly all tissues in the body are capable of synthesizing cholesterol from the general metabolite acetyl-CoA. The liver accounts for only about 20% of endogenous cholesterol synthesis. Among the extra-hepatic tissues, which are responsible for the remaining 80% of cholesterol synthesis, the intestine is probably the most active. [The body synthesizes 0.3 to 3 grams of cholesterol per day](#), which is more than what many people consume in their diet. If your diet contains excessive amounts of cholesterol, your body will reduce its absorption, the liver will decrease its synthesis and the cholesterol excretion rate increases. Thus, for most people, how

much cholesterol you eat does not have much affect on how much cholesterol is in your blood.

However, the latest meta-analysis by [Vincent et al. \(2019\)](#) finds that on average in the entire population, dietary cholesterol intake does influence blood cholesterol levels slightly. They found a non-linear relationship with blood LDL-cholesterol that virtually plateaus at 600 mg per day with a 10 mg/dL increase in LDL cholesterol at that point: see the graph below. [For individuals without diabetes or cardiovascular disease, the optimal LDL-cholesterol level is up to 100-130 mg/dL \[2\]](#), so the maximum effect of dietary cholesterol would be approximately 10% of that range. The effect on HDL-cholesterol was less clear, with a seemingly protective increase in women and a minor decrease in men. The maximum difference between virtually zero cholesterol diets and extremely high cholesterol intakes was about 2.5 mg/dL. That's 4% of [the minimum of 60 mg/dL HDL-cholesterol for optimal health](#).





The effect of dietary cholesterol intake on LDL- and HDL-cholesterol. [Source](#)

The findings of this meta-analysis conflict with another meta-analysis shortly before from [Rouhani et al. \(2018\)](#), who found that increased cholesterol intake from eggs slightly increased HDL and LDL cholesterol (even) more modestly without affecting the ratio between them. Since this meta-analysis focused exclusively on eggs, it's possible that other cholesterol sources, such as processed red meat, are the primary culprit of increased cholesterol, while eggs are somehow safer.

[Dietary cholesterol intake seems to primarily increase blood cholesterol levels in diets rich in animal saturated fats and low in unsaturated fats, with significantly smaller effects in diets with a more balanced fatty acid profile \[2\]](#). So overall diet quality is generally much more important than cholesterol intake specifically.

However, for some people, dietary cholesterol intake does significantly and considerably influence blood cholesterol levels. An estimated [20-33% of people have a](#)

genetic variation that reduces the efficiency of blood cholesterol regulation so that their dietary cholesterol intake does influence their blood cholesterol level. The effect is slight and variable and even in these hyper-responders, a high cholesterol diet often does not negatively influence their total cholesterol profile that much. If total blood cholesterol increases during a high cholesterol diet, both ‘good’ HDL and ‘bad’ LDL cholesterol often increase in a similar proportion [2, 3]. Still, this increase in LDL-cholesterol is a theoretical risk factor that is likely not fully countered by the increased HDL-cholesterol.

Most of the effect of dietary cholesterol on serum lipids occur in the range of 0-200 mg per day. Since the average population consumption in most countries and the world is in the range of 200-300 mg per day [2] and many high-protein animal foods are relatively high in cholesterol, it's difficult in practice to get cholesterol intake below 200 mg per day for non-vegans. That begs the question: is this worth doing?

The effect of dietary cholesterol on CVD

Is the modest and variable effect of dietary cholesterol intake on our blood cholesterol levels a concern? Probably not for most people. Randomized controlled trials quite consistently show no effect of cholesterol intake on cardiovascular health, even in type II (pre)diabetics [2, 3, 4]. In cross-sectional studies, the relationship between cholesterol levels and disease is highly inconsistent, ranging from negative to a J-curve to neutral and in some datasets even an inverse relationship, especially in the elderly, which would suggest more cholesterol intake was somehow protective [2, 3, 4].

Dietary cholesterol may also increase HDL-cholesterol particle size and functionality.

Since HDL-cholesterol is anti-atherogenic, it can counteract the artery clogging effect

of LDL-cholesterol and [the minor increase in total blood cholesterol from diets rich in cholesterol is predicted to have little effect on cardiovascular disease risk](#). Many review papers and other scientific health authorities agree [dietary cholesterol intake is not significantly associated with cardiovascular disease risk in the context of an overall healthy diet](#) [2, 3, 4, 5, 6, 7, 8].

[The public health recommendation in the US to limit cholesterol intake to 300 mg per day was mostly the result of the media scare about saturated fat in the 1960s](#). Canada and most European and Asian countries didn't adopt a limit on cholesterol intake in their public health recommendations. In 2015, after incessant critique from the scientific community, the US Departments of Agriculture and Health and Human Services also dropped the advice to limit dietary cholesterol intake from their [Dietary Guidelines for Americans](#).

Nevertheless, not all scientists agree dietary cholesterol is safe. [A 2019 meta-analysis of 6 cohorts in the US, by Zhong et al.](#), found a significant association between cholesterol intake and all-cause mortality. Their analysis conflicts with [a larger meta-analysis by Xu et al. in China from the same time, a much larger 2015 meta-analysis of 40 prospective studies by Berger et al.](#), [a 2022 meta-analysis by Mousavi et al. of prospective cohort studies on egg consumption](#), and another [2022 meta-analysis by Mofrad et al. of prospective cohort studies on egg consumption](#), all 4 of which found no significant relation between cholesterol intake and any cardiovascular disease. While it is easiest to study very long-term outcomes in observational studies, observational studies have many confounding variables that make it difficult to determine which variable caused what effect. For example, in the 2019 meta-analysis by Zhong et al. with negative health associations of cholesterol, cholesterol intake correlated positively with red meat intake and negatively with fiber

intake. While these were adjusted for in the analysis, [potential for confounding always remains](#). Confounding was particularly likely in the 2019 meta-analysis because the relationship between cholesterol intake and all-cause mortality remained when adjusted for serum cholesterol levels. This indicates the correlation between cholesterol intake and mortality was not because cholesterol intake increased blood cholesterol levels. Since that's the primary mechanism cholesterol is thought to be harmful, these results strongly suggest that another factor, namely a crappy diet, was associated with both high cholesterol intake and disease, while cholesterol and disease themselves were not actually causally related.

All in all, if your overall diet is very healthy and you do not have diabetes or a cardiovascular disease, how much cholesterol is in your diet is probably not a major concern. However, it's advisable for everyone to have bloodwork done at least once every few years. If your cholesterol profile is not in the ideal range, you may want to experiment with a low cholesterol diet to see if that improves your cholesterol profile. If so, you should weigh the potential physique benefits against the potential risks of high cholesterol intakes.

Take-home messages

- The available research indicates a high cholesterol diet may be advantageous for muscle growth and strength development by increasing muscle cell integrity and signaling for muscle growth. The optimal cholesterol intake seems to be at least 7.2 mg dietary cholesterol per kg (3.27 mg/lbs) of lean body mass or more than 400 mg in men.
- Since your body autoregulates your blood cholesterol level, a high cholesterol intake often does not increase your serum cholesterol level. Even in hyperresponders, the effect of dietary cholesterol on the blood cholesterol profile is often modest. Randomized controlled trials and the literature as a whole support that any effects generally do not come with increased risk of cardiovascular disease.

Here's how much cholesterol is in various foods according to the USDA.

Food	Serving size	Cholesterol content
Egg yolk	1 egg yolk (18 g)	195 mg
Whole egg	1 large (60 g)	222 mg
Shrimp	100 g	152 mg
Butter	1 tbsp. (14 g)	30 mg
Cheese	1 slice (17 g)	19 mg

	100 g	114 mg
Whole milk	1 cup (230 ml)	24 mg
	100 ml	10 mg
Whole milk yoghurt	100 g	13 mg
Red meat	100 g	72 mg
Poultry	100 g	58 – 84 g (lower end for lean protein)
Giblets (kidney, liver)	100 g	275 – 515 mg
Fish	100 g	43 – 65 g (higher end for fatty fish)

Since only animal foods contain significant amounts of bioavailable cholesterol, vegans may want to compensate for their low cholesterol intake by consuming more (saturated) fat so the body can produce enough of its own cholesterol. However, there is no research that this will have the same anabolic effects as a higher dietary cholesterol intake.

Brown fat

A specific type of fat mass worth discussing as a final topic is brown fat. [Brown adipose tissue](#) obtains its name from its high degree of vascularity and the abundant mitochondria present in its adipocytes. Not only do brown fat cells contain larger numbers of mitochondria than white fat cells do, but the mitochondria also are structurally different and contain uncoupling protein 1 to promote thermogenesis (heat production) at the expense of producing ATP. Accordingly, the main function of brown fat is heat production and it is not a primary storage site of energy. Humans have very little brown fat, especially compared to other animals.

Two types of external stimuli trigger thermogenesis.

1. Ingestion of food.
2. Prolonged exposure to cold temperature (not including the effect of shivering).

Stimulus 2, cold exposure, has garnered an interest in using cold therapy to promote energy expenditure and thereby fat loss. In theory, it works. The more practical question though is: is it worth it and realistic? Most studies find that very uncomfortable temperatures are required to cause moderate increases in energy expenditure.

- [Ouellet et al. \(2012\)](#) found that 6 men experienced a ~250 kcal increase in energy expenditure after wearing a cryo-suit infused with 18°C water for 3 hours (~80% increase in BMR) compared to being at room temperature. 18°C doesn't sound too bad, but if you've ever worn a wetsuit in the sea, you'll know wearing a suit like this feels notably colder than a regular 18°C ambient temperature. This temperature resulted in observable but mild shivering in the participants. [Blondin et al. \(2015\)](#) replicated these findings, showing that going from 25°C, what they

called room temperature but many people would consider warm, to wearing the 18°C cryo-suit led to an ~80% increase in BMR.

- [Yoneshiro et al. \(2012\)](#) found major interindividual variability in the effect of cold exposure on brown fat thermogenesis. 6 out of 13 men experienced a somewhat impressive 410 kcal (28%) increase in energy expenditure after sitting on ice blocks in their underwear for 2 hours. However, the remaining 7 did not have any significant increase at all (+3%, 42 kcal). [Thermogenesis in response to food and cold are closely related](#), so a way to determine if you likely respond to cold therapy is to judge how much your skin temperature increases after eating. If you quickly start sweating after a meal, your brown fat may activate strongly after cold exposure.
- [Acosta et al. \(2018\)](#) studied a full dose-response of cold exposure from cold to shivering. In a 19°C room, participants wore a water-cooling vest that started at 16.6°C and gradually decreased until the participants were significantly shivering. Energy expenditure gradually increased to a peak of +17% when shivering.
- [Chen et al. \(2013\)](#) studied a more moderate design of 24°C vs. 19°C, though the participants only wore hospital scrubs and had to sleep under sheets without a blanket. These conditions increased BMR by 5.3%.

Several hundred Calories extra energy expenditure sounds promising, yet there are several considerations that limit the practical application of cold therapy, not even mentioning *how* you would get yourself cold enough if it's not cold outside.

1. Brown fat thermogenesis is only significantly activated when there is perception of cold beyond the point of comfort. Mere exposure to cold in daily life doesn't cut it. [Zhang et al. \(2022\)](#) found no effect of temperatures between 19-25°C on any component of energy expenditure. Shivering seems to be required to raise

your energy expenditure significantly. When cutting and lean, many people are quite cold intolerant, so suffering freezing temperatures is not exactly a miracle fat loss cure.

2. Our appetite tends to increase when we're exposed to cold. While [mild short-term cold exposure may not increase our appetite](#) and [moderate short-term heat exposure does not decrease our appetite](#) [3], [most research agrees that people and animals in general eat more in the long run when they feel colder](#) [3, 4, 5, 6, 7, 8, 9]. In terms of difficulty in achieving fat loss, there's not much point in increasing your energy expenditure when your appetite rises alongside it.
3. Measurements in the above studies were generally done fasted and completely sedentary. Food and movement both naturally cause thermogenesis and thereby reduce the extent of brown fat thermogenesis that's required.
4. [The body adapts to cold exposure and these adaptations mostly reduce energy expenditure as the body becomes more efficient at dealing with the cold.](#)

As such, instead of a single acute trial, it's more interesting to look at the effects of living in certain temperatures. We have the perfect natural experiment for this: the seasons. Does our BMR increase in the winter compared to the summer? Findings on this are mixed. Several studies find that our BMR does indeed increase in the winter. However, people also typically weigh more in the winter, which would explain the higher BMR. [When you correct for body composition, people's BMRs are virtually identical in the summer and winter](#). The largest scale experiment was conducted in Rochester, Minnesota, in the US, where it's comfortable in the summer (22°C) and freezing cold (-7°C) in the winter. Other research in the Netherlands also finds that while people have a lower sleeping metabolic rate in the summer than the winter, [total daily energy expenditure does not change across seasons](#). Some research even finds that

people that [regardless of season, people that live in warmer settings are leaner than those that live in colder settings](#). These findings are hard to reconcile with the theory that cold exposure increases our metabolism and not our appetite. The main issue is probably that most people in modern societies don't suffer cold temperatures for prolonged periods anymore even in the harshest of winters, as indoor temperatures are generally controlled around a cozy room temperature.

In conclusion, [cold therapy arguably has no practical fat loss application for most individuals](#). It's worth considering for people that fulfill the following criteria.

1. They're likely responders, based on good cold tolerance and high heat production after meals. You must tolerate a temperature below 20°C with minimal clothing for several hours a day to get an appreciable effect.
2. They're willing to endure considerable cold, likely enough to cause shivering.
3. Their appetite is not the main obstacle for the control of their energy balance.
4. They can easily control their ambient temperature or are willing to wear cryo-wear.
5. They can deal with being cold for their work. Fine motor control will suffer in the cold, so for people that rely on precise hand movements for their job, like graphical designers, musicians or professional gamers, the cold is arguably not worth it.

Practical applications

- Fat intake should whenever possible be kept at a minimum of 20% of total energy intake. For maximum anabolic effects an intake of 40% of BMR for men and 40% of REE for women is advisable, assuming no carb hyperresponsiveness or high carbohydrate exercise requirement.
- Since each fatty acid has different effects, the diet should have a regular source of each fatty acid type: saturated, mono-unsaturated and poly-unsaturated fat. Unsaturated fatty acids are particularly important, as they confer direct anabolic effects. Saturated fat's anabolic actions are likely restricted to its indirect effects via increased anabolic hormone production.
- Omega-3 fat is particularly important to get enough of: 2-3 g of combined EPA + DHA is recommended for low chronic inflammation, high muscle protein synthesis and a high thermic effect of food.
- Cholesterol is normally entirely safe for your health and may increase muscle growth, so if there are no clear health contraindications, an intake of at least 7.2 mg dietary cholesterol per kg (3.27 mg/lbs) of lean body mass is recommended.
- Brown fat thermogenesis is not significant enough and often associated with increased hunger so as to make it practically infeasible as a fat loss aid.

Macros mini-summary

Now that you've learned how to optimize each of the 3 main macronutrients of a diet, here's a short recap on them. You can think of this as a general template for non-ketogenic diets. This short version does not overrule the nuances you learned on how to determine the optimal intake of each macronutrient.

➤ Lecture

[Macros implementation mini-summary](#)