

CARDIOBEATS

Cardio Health Amplified



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

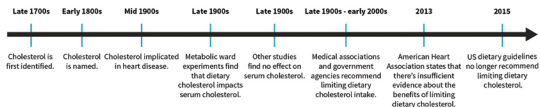
This guide is based on scientific studies, but individual results do vary. If you engage in any activity or take any product mentioned herein, you do so of your own free will, and you knowingly and voluntarily accept the risks. While we mention major known interactions, it is possible for any supplement to interact with other supplements, with foods and pharmaceuticals, and with particular health conditions.

For three decades, the cholesterol issue was clear: too much cholesterol in your blood is bad, and both dietary cholesterol and saturated fat should be kept to a minimum. But in recent years, the media have changed their tune: dietary cholesterol and fat even saturated fat aren't so dangerous, finally; they can even be good for you!

A typical nutrition enthusiast will take sides, either shying away from butter or adding some to everything, even their coffee. Some low carbers tend to downplay cholesterol concerns even celebrate their high numbers^[1] while some vegans brandish their rock bottom numbers^{[2][3]} as proof that eggs and meat are better avoided.

If it's your first time at Cholesterol Club, you have to fight. Fight dogma that is, with scientific evidence as your only weapon. You have to dare consider cardiovascular health as a whole, rather than either magnify or ignore the role of dietary fat and serum cholesterol.

TIMELINE OF CHOLESTEROL RESEARCH AND GUIDELINES



References: Olson. J Nutr. 1998.^[4] Duff. Am J Med. 1951.^[5] Clarke et al. BMJ. 1997.^[6] Brownawell and Falk. Nutr Rev. 2010.^[7] Stone et al. J Am Coll Cardiol. 2014.^[8]

THE DIET-HEART HYPOTHESIS WAS WRONG

First published in 1970, the Seven Country Study led by Ancel Keys suggested that dietary saturated fat raised serum cholesterol levels and therefore increased the risk of cardiovascular disease.^[9] Within the academic and medical communities, this conclusion was widely accepted as fact, and it influences official dietary guidelines even today.

Recent evidence, however, does not clearly support a connection between saturated fat intake and cardiovascular disease.^{[10][11]} Whether serum cholesterol and heart health are affected by saturated fat intake appears to depend on what saturated fat is replacing (or is being replaced by).^[12]

Even the source of the saturated fat matters. Randomized controlled trials have shown that a diet high in saturated fat from butter led to an increase in LDL-C (the “bad cholesterol”), but that a diet equally high in saturated fat from cheese might not.^[13] And to add to the confusion, the most recent evidence suggests that butter has, at worst, a minor effect on cardiovascular health,^[14] despite its increasing LDL-C more than do olive oil and coconut oil, and HDL-C (the “good cholesterol”) less than does coconut oil.^[15]

The evidence on coconut products is equally conflicted. Though purified oil tends to perform worse than coconut meat, the magnitude of its effect on cardiovascular health is uncertain probably because wider dietary patterns have a much greater impact on cardiovascular health.^[16]

DIGGING DEEPER: ARE YOU CUCKOO FOR COCONUTS?

You may love coconuts, or might even guzzle coconut oil for its health benefits, but have you ever eaten an entire coconut in one seating? Probably not. A single coconut of average size contains 400 grams of meat (1,400 kcal). Aside from water, this meat is composed of fat (133 grams, including 18 grams of MCTs medium-chain triglycerides), carbs (35 grams of fiber and 25 grams of sugar), and more protein than you'd expect (13 grams). Combined, coconut's fiber, sorbitol (a sugar alcohol that naturally occurs in some plants), and MCTs can result in a prolonged toilet stay.

Consuming the equivalent amount of fat through a few tablespoons of coconut oil might not cause any digestive issues, since fiber and sorbitol are both subtracted, but the evidence linking coconut oil with a reduction in cardiovascular risk factors is mixed.[17] While there is evidence of good health in island cultures with high coconut intakes,[18] we cannot conclude that these cultures owe their good health to coconuts. And even if coconuts are a healthy component of these cultures' simple diets, it doesn't follow that isolated coconut oil is healthy too especially when added to most modern diets.

Still, when it comes to its effect on your cholesterol, coconut oil compares favorably to both butter and olive oil.[19] Compared to butter, it causes a higher increase in HDL-C (the "good cholesterol") and a lower increase in LDL-C (the "bad cholesterol"). Compared to olive oil, it causes a higher increase in HDL-C and a smaller increase in LDL-C.

TEST RESULTS MATTER, BUT THEY CAN BE MISLEADING

The standard lipid panel is a relatively poor reflection of your cardiovascular health. It estimates how much cholesterol, triglycerides, LDL-C, and HDL-C are in your blood, but it ignores better indicators of cardiovascular health.

LDL particle count is far more important than LDL-C levels. LDL infiltration is a major cause of inflammation, which is a prerequisite to atherosclerosis (a hardening and narrowing of the arteries), [20] and the more particles you have, the more can infiltrate your artery walls. LDL particle size is another potentially important factor, for the smaller and denser LDL particles might find infiltration easier, but its predictive ability is greatly reduced once particle number is accounted for.[21]

So, simply keeping your LDL-C levels in check won't protect you from heart attacks. In fact, one study found that nearly half of hospital patients admitted for cardiovascular disease had ideal levels of LDL-C.[22] Furthermore, some studies have associated low LDL-C levels with cancer, depression, and infectious diseases although whether low LDL-C is a cause or a consequence of these conditions is uncertain.[23]

DIGGING DEEPER: IS LDL-C REALLY “BAD CHOLESTEROL”?

Low-density lipoprotein (LDL) delivers cholesterol to the cells that need it. High-density lipoprotein (HDL) removes excess cholesterol from the bloodstream.

A quick Internet search will probably convince you that LDL cholesterol (LDL-C) is nefarious. It's beyond question at this point that, in certain populations, lowering LDL-C helps reduce the risk of coronary events.^[24]

But cholesterol is also key to, notably, hormone production and the structure of cell membranes, so it certainly isn't inherently bad. LDL particles ferry cholesterol over to cells where it can be used.

If your LDL-C is extremely low, that could even be a sign that something is wonky within your body. One reason relates to infection: both HDL and LDL are needed to fight infection, and severe infection is linked to a reduction in both. Very low LDL-C levels (≤ 70 mg/dL) have been associated with higher risks of both cancer malignancy and sepsis (a life threatening complication of infection),^[25] not to mention an overall higher risk of death;^[26] it is however possible that the diseases cause low LDL-C, rather than the reverse.

Not everyone agrees that very low LDL-C levels are risky,^[27] but it should be noted that some of the reviews concluding that risks are low were funded by companies producing LDL-C-lowering drugs.^[23] The overall takeaway is that the health effects of low LDL-C levels are far from certain, with well-documented cardiovascular benefits on one side and less-well-quantified risks on the other. If your LDL levels are very low, you should probably do a little bit of digging into these topics.

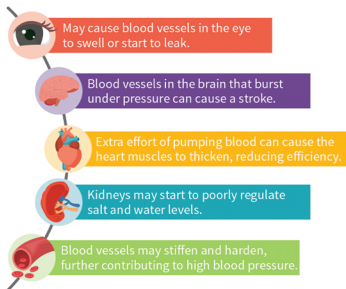
Still, even the standard lipid panel can provide useful information if we look at ratios. On their own, cholesterol, triglycerides, LDL-C, and HDL-C are relatively poor indicators of cardiovascular health, but the ratio of total cholesterol to HDL-C is a strong indicator of heart disease risk,^[28] and the ratio of triglycerides to HDL-C is a strong predictor of heart disease severity,^{[29][30]} insulin resistance,^[31] and LDL particle size.^[32]

BEYOND BLOOD LIPIDS

Even a savvy member of Cholesterol Club a veteran who looks at ratios and LDL particle size needs to realize that there's more to cardiovascular health than blood lipids.

For instance, LDL particles aren't the only factor involved in the inflammation necessary for arterial plaque formation. And of course, blood pressure (BP) also plays a central role in heart health: cardiovascular disease risk is strongly associated with an increase in BP, even when BP is still within normal range.^[33]

BEYOND BLOOD LIPIDS



In sum, blood lipids are but one piece of the cardiovascular health puzzle: they shouldn't be ignored, but neither should they turn into an obsession. Some supplements show evidence for potentially aiding cardiovascular health through a variety of mechanisms — some involving blood lipids, some not. None of these supplements, however, will be able to counteract an unhealthy diet; rather, they should be seen as helpful adjuncts to a healthy diet and lifestyle.

Healthy foods, well-chosen supplements — those may complicate your life a little, but they can prolong it too. Published in 2019, a research letter on the “trends in cardiometabolic mortality in the United States, 1999–2017”^[34] reports that, “while cardiovascular disease (CVD) death rates declined by approximately 36% from 2000 to 2014,^[35] CVD remains the leading cause of mortality among US adults.”

CORE SUPPLEMENTS

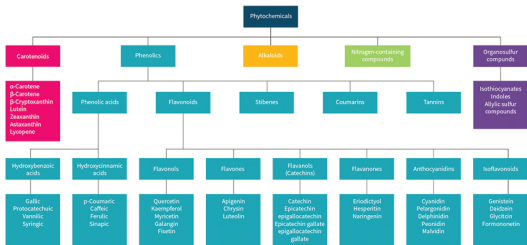
Core supplements have the best safety-efficacy profile. When used responsibly, they are the supplements most likely to help and not cause side effects.

COCOA

WHAT MAKES COCOA A CORE SUPPLEMENT

Low nitric oxide (NO) levels can cause blood vessels to narrow, leading to reduced blood flow. Like the flavonoids in grape seed and Pycnogenol, (-)-epicatechin and other flavonoids in cocoa can help support NO levels, and research shows that cocoa does improve blood flow. Cocoa might also cause a minor decrease in blood pressure in people with hypertension (i.e., high blood pressure), but it has no effect on heart rate.

CLASSIFICATION HIERARCHY OF POLYPHENOLS



Judging from a study on a grape seed extract, the improvement in blood flow from cocoa might be negated by the flavonoid quercetin, whose concurrent supplementation should therefore be avoided.

Taking cocoa with other hypotensive agents could cause low blood pressure. Hypotensive agents can be pharmaceuticals but also supplements: garlic, notably, but also nitrates, grape seed extracts, or pine bark extracts, to mention only the supplements presented in this guide.

HOW TO TAKE COCOA

The standard daily dose for cocoa polyphenols is 1 g, which you can get by eating about 30 g of cocoa powder or 40 g of dark chocolate with a 75% cocoa content. Neither milk chocolate nor white chocolate is a good source of polyphenols.

I GARLIC

WHAT MAKES GARLIC A CORE SUPPLEMENT

Garlic enhances nitric oxide (NO) signaling, but its lowering action on blood pressure is mostly due to its enhancing hydrogen sulfide (H₂S) signaling. Garlic may also fight atherosclerosis (a hardening and narrowing of the arteries). First, garlic can cause a decrease in low-density lipoprotein (LDL-C) and an increase in high-density lipoprotein (HDL-C) and thus help prevent cholesterol from clogging the arteries. Second, garlic can help prevent excess calcium from stiffening the arteries.

Garlic has antiplatelet properties. While this is yet another attribute of garlic that can improve blood flow, it may be a problem for people taking blood thinners, be they antiplatelet agents (such as aspirin) or anticoagulants (such as warfarin/Coumadin and acenocoumarol/Sintrom).

Taking too much garlic, or taking garlic with other hypotensive agents, could cause low blood pressure. Hypotensive agents can be pharmaceuticals but also supplements, such as nitrates, cocoa, grape seed extracts, or pine bark extracts, to mention only the supplements presented in this guide.

Garlic can interact with several pharmaceuticals other than blood thinners and hypotensive agents, notably contraceptives and drugs used to treat tuberculosis and HIV. If you take any medication, talk to your physician before supplementing garlic.

HOW TO TAKE GARLIC

To maximize the benefits of garlic, eat 3–6 cloves daily over several meals. You should first cut or crush them, to activate their bioactive compounds, then cook them or eat them raw.

Supplementation can provide the same benefits. If you dislike the smell or taste of garlic, or if you wish to avoid the bad breath that comes from eating the cloves, take 600–1,200 mg of an aged garlic extract daily.

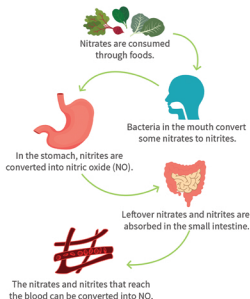
Too much garlic daily (12 cloves) or at once (6 cloves, or 1,200 mg of an aged garlic extract) could cause low blood pressure, especially if taken with other hypotensive agents, and prolong bleeding time. Eating 8 cloves in a day is enough to strongly reduce the efficacy of the anti-HIV drug saquinavir (Fortovase, Invirase).

NITRATES

WHAT MAKES NITRATES A CORE SUPPLEMENT

Nitrates break down into nitrites, which circulate in the body and are turned into nitric oxide (NO) as needed. Elevated NO levels are associated with better blood flow and lower blood pressure.

CLASSIFICATION HIERARCHY OF POLYPHENOLS



Nitrates do not exist as dietary supplements, unfortunately, because of regulations against high quantities of sodium nitrate (a food additive frequently added to meat products). Nitrates can be found in different foods, however, notably beetroot and leafy green vegetables. Beetroot extract capsules will not provide enough nitrates to affect blood flow, but beetroot powder (¼ the weight of raw beetroot) and beetroot juice are valid options.

Taking nitrates with other hypotensive agents could cause low blood pressure. Hypotensive agents can be pharmaceuticals but also supplements garlic, notably, but also cocoa, grape seed extracts, or pine bark extracts, to mention only the supplements presented in this guide.

Leafy greens are often rich in vitamin K1, a fat-soluble vitamin that helps with blood clotting and so might decrease the effectiveness of blood thinners, especially anticoagulants (such as warfarin/Coumadin and acenocoumarol/Sintrom). If you take a blood thinner, you should consult with your physician before consuming a lot of leafy greens.

Due to their goitrogen content, cruciferous vegetables can reduce thyroid hormone production if regularly consumed in high amounts, such as those needed for nitrate supplementation. If you tend to eat a lot of cruciferous vegetables (such as cabbage, collard greens, or kale), make sure to also get enough iodine -- through iodine-rich foods (such as cod, shrimp, milk, yogurt, or cottage cheese), iodine-fortified foods (such as iodized salt), or supplements (75–150 mcg/day).

HOW TO TAKE NITRATES

Aim for 6.4–12.8 mg of nitrates.

CARNITINE

WHAT MAKES CARNITINE A CORE SUPPLEMENT

Carnitine plays a role in cognition,^[36] energy metabolism,^[37] and cardiovascular health.^{[38][39][40]} Though your body can synthesize it out of lysine and methionine, two amino acids, nearly three fourths of the carnitine in omnivorous people comes from the meat, fish, eggs, and dairy products they consume.

Your body's ability to synthesize carnitine decreases as you age. In seniors, carnitine supplementation may reduce muscular fatigue, and preliminary evidence suggests that it may improve muscular control.

Also, people who have suffered a heart attack can supplement carnitine as an add on treatment to possibly lower the risk of both abnormal heartbeats in the lower chambers (i.e., ventricular arrhythmia) and pain in the chest or limbs caused by poor blood circulation (i.e., angina).[41]

HOW TO TAKE CARNITINE

In people at risk but who have not yet suffered cardiovascular complications, 500–2,000 mg of L carnitine per day might offer some protection when taken in conjunction with prescribed medical therapies. People who have already suffered a heart attack, however, would need at least 2,000 mg (i.e., 2 g) and preferably 5,000–9,000 mg (i.e., 5–9 g) to see a reduction in arrhythmia, angina, and all-cause mortality.

L-carnitine can also be consumed as L-carnitine L-tartrate (LCLT) or glycine propionyl L carnitine (GPLC).

You can supplement 500–2,000 mg of L-carnitine through 750–3,000 mg of LCLT or GPLC.

You can supplement 5,000–9,000 mg of L-carnitine through 7,500–13,500 mg of LCLT or GPLC.

However, neither LCLT nor GPLC has proven advantages over regular L-carnitine, both are more expensive, and GPLC also clumps easily in moist environments.

COQ10

WHAT MAKES COQ10 A PRIMARY OPTION

Coenzyme Q10 (CoQ10) is found mostly in mitochondria, the “power plants” in our cells. Our bodies produce it, yet supplementation can provide additional benefits, such as reducing the risk of further heart complications in people who have suffered a heart attack. More research is needed to determine if CoQ10 can also benefit people with less severe cardiac damage.

Statin medications (i.e., cholesterol lowering drugs) can lower CoQ10 levels in the body. If you are taking statins, talk to your physician about supplementing CoQ10.

HOW TO TAKE COQ10

Take 90–150 mg of CoQ10 once a day with a meal containing fat.

Higher doses (200–300 mg) result in higher levels of CoQ10 in the body, but more research is needed to determine if those higher levels translate into greater cardiovascular protection.

I GRAPE SEED

WHAT MAKES GRAPE SEED A PRIMARY OPTION

Low nitric oxide (NO) levels can cause blood vessels to narrow, leading to reduced blood flow. Like the flavonoids in cocoa and pine bark, procyanidins and other flavonoids in grape seeds can help support NO levels.

Studies on grape seed extracts have reported minor reductions in heart rate and, possibly as a consequence, in blood pressure. There was no improvement in blood flow, or only to a small extent in people with vascular risk factors, such as high blood pressure. This possible improvement in blood flow may be negated by the flavonoid quercetin, whose concurrent supplementation should therefore be avoided.

Taking a grape seed extract with other hypotensive agents could cause low blood pressure. Hypotensive agents can be pharmaceuticals but also supplements garlic, notably, but also nitrates, cocoa, or pine bark extracts, to mention only the supplements presented in this guide.

HOW TO TAKE GRAPE SEED

Take 200–400 mg of a grape seed extract once a day with a meal.

I TAURINE

WHAT MAKES TAURINE A PRIMARY OPTION

Taurine (L-*taurine*) is one of the most abundant amino acids in the body, with particularly high concentrations in the heart tissue, where it is thought to help maintain cell membranes and regulate heartbeats. It is not an essential amino acid, since our bodies can make it from vitamin B6, methionine, and cysteine; however, supplementation can modestly but reliably reduce blood pressure in people with congestive heart failure, hypertension (i.e., high blood pressure), or prehypertension. Likewise, in people with congestive heart failure, taurine can modestly but reliably improve cardiac function.

HOW TO TAKE TAURINE

Daily dosage ranges from 1.5 to 6 g, though 3 g is currently considered the upper limit for safe lifetime supplementation. Whichever dosage you go with, split it into 2 or 3 doses a day, with or without food.

VENOTROPICS

WHAT MAKES VENOTROPICS A PRIMARY OPTION

Venotropics can improve the rate at which the blood returns to the heart. They are used to treat chronic venous insufficiency (CVI), which is characterized by blood pooling in extremities. They can also be used to treat leg swelling caused by prolonged sitting or to reduce varicose veins.

Dafalon (90% diosmin, 10% hesperidin) was the first venotropic, but it is slightly less effective than Pycnogenol. Butcher's broom (*Ruscus aculeatus*) and horse chestnut (*Aesculus hippocastanum*) also have venotropic properties.

Pycnogenol is a patented pine bark extract standardized to 65–75% procyanidin. Grape seed extracts, being also rich in procyanidins, might offer similar benefits, but there is currently no study on the subject.

HOW TO TAKE VENOTROPICS

Take 100–200 mg of Pycnogenol with breakfast. Alternatively, take one of the following options twice a day, 12 hours apart.

- 375–750 mg of butcher's broom (i.e., 750–1,500 mg/day)
- 50–75 mg of horse chestnut (i.e., 100–150 mg/day)
- 400 mg of diosmin with 100 mg of hesperidin (i.e., 1,000 mg/day in total).

VITAMIN K

WHAT MAKES VITAMIN K A PRIMARY OPTION

Vitamin K is an umbrella term for a variety of molecules with similar but distinct structures.

- K1 (phylloquinone) is a molecule found in plants.
- K2 (menaquinone) is a group of molecules.
 - K2 MK-4 is mostly found in animal products.
 - K2 MK-7 is mostly found in fermented foods.

In all its forms, vitamin K is fat-soluble and supports blood clotting and calcium regulation; it helps ensure that more calcium gets deposited in bone and less in soft tissues. Hence, vitamin K can both strengthen bones and reduce cardiovascular risk. However, there are notable differences between the different forms.

After being absorbed by your intestines, K1 is taken up by your liver (where vitamin K is used to make clotting proteins, which are then released into your blood) at a higher rate than MK-4, whereas MK-4 is taken up by soft tissues at a higher rate than K1. This should make K1 better at supporting coagulation (i.e., blood clotting), and MK-4 better at preventing calcium from being deposited in the arteries.

Some K1 converts indirectly to MK-4, but how much is unknown. Diets naturally rich in K1 do not seem to reduce cardiovascular risk, but trials supplementing high K1 doses have noted some reduction in coronary artery calcification. The reason may be that, in many plants, K1 is tightly bound to chloroplasts (organelles that contain chlorophyll and conduct photosynthesis), so you could be absorbing very little of what you eat.

Unlike MK-4, MK-7 has been used in trials looking at arterial stiffness and atherosclerosis (a hardening and narrowing of the arteries), and we can say it is likely good at both supporting coagulation and preventing coronary calcification. It is important to note that cardiovascular research has not compared K1 to K2, or MK-4 to MK-7.

Vitamin K is usually safe. Supplementation might cause some nausea or stomach upset, but those effects are uncommon.

K1 is present mostly in leafy green vegetables, many of which are cruciferous. If you plan to increase your K1 intake through plant foods, be aware that cruciferous vegetables contain goitrogens and thus can reduce thyroid hormone production. If you tend to eat a lot of cruciferous vegetables, such as kale, make sure to also get enough iodine through iodine rich foods (such as cod, shrimp, milk, yogurt, or cottage cheese), iodine fortified foods (such as iodized salt), or supplements (75–150 mcg/day).

HOW TO TAKE VITAMIN K

Take 200 mcg (0.2 mg) of MK-7 and, optionally, 500–1,000 mcg (0.5–1 mg) of K1. MK-4 is theoretically better than K1 (for cardiovascular health), but there are not enough data to support a dosage.

SCIENTIFIC LINKS

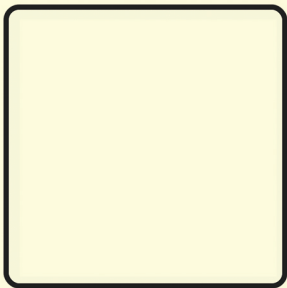
1. Retterstøl K, et al. Effect of low carbohydrate high fat diet on LDL cholesterol and gene expression in normal weight, young adults: A randomized controlled study. *Atherosclerosis*. (2018)
2. Dinu M, et al. Vegetarian, vegan diets and multiple health outcomes: A systematic review with meta-analysis of observational studies. *Crit Rev Food Sci Nutr*. (2017)
3. Craig WJ. Health effects of vegan diets. *Am J Clin Nutr*. (2009)
4. Olson RE. Discovery of the lipoproteins, their role in fat transport and their significance as risk factors. *J Nutr*. (1998)
5. DUFF GL, McMILLAN GC. Pathology of atherosclerosis. *Am J Med*. (1951)
6. Clarke R, et al. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *BMJ*. (1997)
7. Brownawell AM, Falk MC. Cholesterol: where science and public health policy intersect. *Nutr Rev*. (2010)
8. Stone NJ, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. (2014)
9. Coronary heart disease in seven countries. Summary. *Circulation*. (1970)
10. Hamley S. The effect of replacing saturated fat with mostly n-6 polyunsaturated fat on coronary heart disease: a meta-analysis of randomised controlled trials. *Nutr J*. (2017)
11. Chowdhury R, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med*. (2014)
12. Briggs MA, Petersen KS, Kris-Etherton PM. Saturated Fatty Acids and Cardiovascular Disease: Replacements for Saturated Fat to Reduce Cardiovascular Risk. *Healthcare (Basel)*. (2017)
13. Huth PJ, Park KM. Influence of dairy product and milk fat consumption on cardiovascular disease risk: a review of the evidence. *Adv Nutr*. (2012)
14. Pimpin L, et al. Is Butter Back? A Systematic Review and Meta-Analysis of Butter Consumption and Risk of Cardiovascular Disease, Diabetes, and Total Mortality. *PLoS One*. (2016)

15. Khaw KT, et al. Randomised trial of coconut oil, olive oil or butter on blood lipids and other cardiovascular risk factors in healthy men and women. *BMJ Open*. (2018)
16. Siri-Tarino PW, Krauss RM. Diet, lipids, and cardiovascular disease. *Curr Opin Lipidol*. (2016)
17. Eyres I, et al. Coconut oil consumption and cardiovascular risk factors in humans. *Nutr Rev*. (2016)
18. Lindeberg S, et al. Cardiovascular risk factors in a Melanesian population apparently free from stroke and ischaemic heart disease: the Kitava study. *J Intern Med*. (1994)
19. Neelakantan N, Seah JYH, van Dam RM. The Effect of Coconut Oil Consumption on Cardiovascular Risk Factors: A Systematic Review and Meta-Analysis of Clinical Trials. *Circulation*. (2020)
20. Lusis AJ. Atherosclerosis. *Nature*. (2000)
21. Allaire J, et al. LDL particle number and size and cardiovascular risk: anything new under the sun?. *Curr Opin Lipidol*. (2017)
22. Sachdeva A, et al. Lipid levels in patients hospitalized with coronary artery disease: an analysis of 136,905 hospitalizations in Get With The Guidelines. *Am Heart J*. (2009)
23. a b Olsson AG, et al. Can LDL cholesterol be too low? Possible risks of extremely low levels. *J Intern Med*. (2017)
24. Silverman MG, et al. Association Between Lowering LDL-C and Cardiovascular Risk Reduction Among Different Therapeutic Interventions: A Systematic Review and Meta-analysis. *JAMA*. (2016)
25. Shor R, et al. Low serum LDL cholesterol levels and the risk of fever, sepsis, and malignancy. *Ann Clin Lab Sci*. (2007)
26. Brescianini S, et al. Low total cholesterol and increased risk of dying: are low levels clinical warning signs in the elderly? Results from the Italian Longitudinal Study on Aging. *J Am Geriatr Soc*. (2003)
27. Giugliano RP, et al. Clinical efficacy and safety of achieving very low LDL cholesterol concentrations with the PCSK9 inhibitor evolocumab: a prespecified secondary analysis of the FOURIER trial. *Lancet*. (2017)
28. Millán J, et al. Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. *Vasc Health Risk Manag*. (2009)
29. Bampi AB, et al. Comparison of non-invasive methods for the detection of coronary atherosclerosis. *Clinics (Sao Paulo)*. (2009)

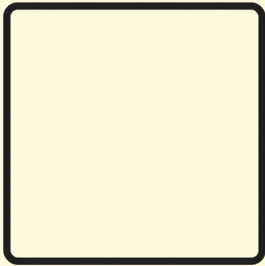
30. da Luz PL, et al. High ratio of triglycerides to HDL-cholesterol predicts extensive coronary disease. *Clinics (Sao Paulo)*. (2008)
31. McLaughlin T, et al. Use of metabolic markers to identify overweight individuals who are insulin resistant. *Ann Intern Med*. (2003)
32. Hanak V, et al. Accuracy of the triglyceride to high-density lipoprotein cholesterol ratio for prediction of the low-density lipoprotein phenotype B. *Am J Cardiol*. (2004)
33. Pei D, et al. Relationship of blood pressure and cardiovascular disease risk factors in normotensive middle-aged men. *Medicine (Baltimore)*. (2011)
34. Shah NS, et al. Trends in Cardiometabolic Mortality in the United States, 1999–2017. *JAMA*. (2019)
35. Sidney S, et al. Recent Trends in Cardiovascular Mortality in the United States and Public Health Goals. *JAMA Cardiol*. (2016)
36. Montgomery SA, Thal LJ, Amrein R. Meta-analysis of double blind randomized controlled clinical trials of acetyl-L-carnitine versus placebo in the treatment of mild cognitive impairment and mild Alzheimer's disease. *Int Clin Psychopharmacol*. (2003)
37. Smeland OB, et al. Chronic acetyl-L-carnitine alters brain energy metabolism and increases noradrenaline and serotonin content in healthy mice. *Neurochem Int*. (2012)
38. Asadi M, et al. The effect of L-carnitine supplementation on lipid profile and glycaemic control in adults with cardiovascular risk factors: A systematic review and meta-analysis of randomized controlled clinical trials. *Clin Nutr*. (2019)
39. Serban MC, et al. Impact of L-carnitine on plasma lipoprotein(a) concentrations: A systematic review and meta-analysis of randomized controlled trials. *Sci Rep*. (2016)
40. Shang R, Sun Z, Li H. Effective dosing of L-carnitine in the secondary prevention of cardiovascular disease: a systematic review and meta-analysis. *BMC Cardiovasc Disord*. (2014)
41. DiNicolantonio JJ, et al. L-carnitine in the secondary prevention of cardiovascular disease: systematic review and meta-analysis. *Mayo Clin Proc*. (2013)

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