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The Amazing Benefits of Dairy Fat

Analysis by Dr. Joseph Mercola | December 26, 2023

STORY AT-A-GLANCE

- Studies have repeatedly failed to find an association between full-fat dairy and cardiovascular events. Instead, full-fat dairy actually reduces your risk of cardiovascular events and deaths thereof. Dairy products are also associated with lower risks of Type 2 diabetes, liver disease and more
- Whole-fat dairy contains the odd-chain saturated fats (OCFAs) pentadecanoic acid (C15:0) and heptadecanoic acid (C17:0), which have significant health benefits
- OCFAs are found only in small amounts in certain foods, primarily dairy fat, and your body only makes C17:0. Researchers now believe C15:0 may be an essential fat, as your body cannot make it
- Higher circulating levels of OCFAs in the blood is associated with lower risks of obesity, chronic inflammation, cardiovascular disease, metabolic syndrome, Type 2 diabetes, NASH, COPD, pancreatic cancer and all-cause mortality
- OCFAs do not have an inhibitory effect on glucose burning because they are not converted to acetyl-CoA; rather, they enter the Krebs Cycle as succinyl-CoA. What this means in practical terms is that you don't need to restrict your consumption of full fat dairy, as it won't affect your ability to burn glucose

Do you avoid whole milk, or better yet, raw milk, because of its saturated fat content? If so, you may be missing out on one of the greatest health foods there is. Studies[1] have repeatedly failed to find an association between full-fat dairy and cardiovascular events.

Instead, they've found the opposite — full-fat dairy reduces your risk of cardiovascular events and deaths thereof.

Dairy products are also associated with lower risks of Type 2 diabetes,[2] liver disease and more. One of the reasons for these health benefits is because whole-fat dairy contains health promoting compounds such as:[3]

Specific amino acids	Unsaturated, medium-chain, and branched-chain fats
Odd-chain saturated fats — pentadecanoic acid (C15:0) and heptadecanoic acid (C17:0)	Phospholipids
Vitamins and minerals	Probiotics

Odd-Chain Saturated Fats From Dairy Are Likely Essential Fats

Of these, the odd-chain saturated fats (OCFAs) are of particular importance. In fact, recent research[4] suggests these are likely one of the most essential fats in the human diet, unlike linoleic acid (LA) that most foods are loaded with. It's virtually impossible to become deficient in LA outside of a laboratory diet.

The same cannot be said for the OCFAs. You need to get them from dairy, because that's the primary source. As noted in the 2020 scientific report, "Efficacy of Dietary Odd-Chain Saturated Fatty Acid Pentadecanoic Acid Parallels Broad Associated Health Benefits in Humans: Could It Be Essential?":[5]

> *"Dietary odd-chain saturated fatty acids (OCFAs) are present in trace levels in dairy fat and some fish and plants. Higher circulating concentrations of OCFAs, pentadecanoic acid (C15:0) and heptadecanoic acid (C17:0), are associated with lower risks of cardiometabolic diseases, and higher dietary intake of OCFAs is associated with lower mortality.*

Population-wide circulating OCFA levels, however, have been declining over recent years. Here, we show C15:0 as an active dietary fatty acid that attenuates inflammation, anemia, dyslipidemia, and fibrosis in vivo, potentially by binding to key metabolic regulators and repairing mitochondrial function.

This is the first demonstration of C15:0's direct role in attenuating multiple comorbidities using relevant physiological mechanisms at established circulating concentrations.

Pairing our findings with evidence that (1) C15:0 is not readily made endogenously, (2) lower C15:0 dietary intake and blood concentrations are associated with higher mortality and a poorer physiological state, and (3) C15:0 has demonstrated activities and efficacy that parallel associated health benefits in humans, we propose C15:0 as a potential essential fatty acid."

Dietary Guidelines Got It Backward

The low-fat recommendation has been around for more than 40 years, and since that time cholesterol levels and heart disease rates have gone in the opposite direction of what was intended.

As noted in the featured paper,[6] in the two decades following that recommendation, average intake of whole fat milk dropped more than fourfold, from 283 grams to 65 grams a day, yet prevalence of obesity, Type 2 diabetes, metabolic syndrome and nonalcoholic fatty liver disease (NAFLD) rose to new heights.

Meanwhile, researchers kept finding that people who consumed whole fat milk had lower risks of obesity, Type 2 diabetes and cardiovascular diseases. Clearly, something was off. At the same time, consumption of LA skyrocketed as saturated fats were replaced with processed seed oils, and we now have robust evidence showing that excessive **LA is a key driver of these chronic diseases**, as it destroys mitochondrial function and metabolism.

In short, the U.S. dietary guidelines discouraged consumption of what now appears to be a TRULY essential fat — odd-chained saturated fats found in milk — while encouraging consumption of what was believed to be an essential fat, but in fact is one of the most destructive ingredients in the modern diet.

Contrary to popular belief, LA is not an essential fat, in part because it's found in most foods, making it near-impossible to become deficient. Odd-chained saturated fats, on the other hand, are found only in small amounts in certain foods, primarily milk, and your body only makes C17:0. It doesn't appear to make any C15:0 endogenously,[7] which means you have to get it from your diet.

Milk Fats 101

As you can see by the list above, whole milk contains several different kinds of fat. About 68% of the fats are even-chain saturated fats (ECSFs), the primary ones being:[8]

- Myristic acid (C14:0)
- Palmitic acid (C16:0)
- Stearic acid (C18:0)

The odd-chain saturated fat (OCFA) pentadecanoic acid (C15:0) represents only 1% of the fat content, and heptadecanoic acid (C17:0) makes up 0.5% of the total.[9]

OCFAs Linked to Lower Disease Risk

Previous studies have shown that higher dietary intake of OCFAs, and subsequently higher circulating levels of OCFAs in the blood, is associated with LOWER risks of:[10]

Obesity	Chronic inflammation
Cardiovascular disease	Metabolic syndrome

Type 2 diabetes	Nonalcoholic steatohepatitis (NASH)
Chronic obstructive pulmonary disease (COPD)	Pancreatic cancer
All-cause mortality	

In the video above, Dr. Paul Saladino reviews studies showing similar benefits for butter. For example, one eight-week-long randomized controlled trial found people who ate about 1.5 tablespoons of butter per day had lower levels of inflammation (based on inflammatory markers) at the end of the trial.

A Closer Look at OCFAs

To get a better idea of how OCFAs affect human health and prevent disease, the featured Scientific Reports paper conducted a series of in vitro and in vivo studies using 99% pure OCFAs. First, OCFAs were tested for peroxisome proliferator-activated receptor (PPAR) agonist activity.

There are three primary PPARs: α (alpha), δ (delta) and γ (gamma). PPARs are transcription factors known to reduce triglyceride levels when activated.[11] They're also involved in the regulation of metabolism and inflammation, and they do that by detecting and responding to the presence of dietary fats.[12]

Agonists are compounds that activate a given receptor, so what they were looking for was whether OCFAs might work by activating PPARs.

They also assessed the impact of OCFAs on mitochondrial function and the production of reactive oxygen species (ROS). Mitochondrial dysfunction is at the heart of all disease, and elevated ROS production, which is indicative of inflammation, is another hallmark of most, if not all, disease states.

OCFAs were also tested across a variety of human cell systems mimicking chronic inflammatory and fibrotic disease states. Next, the effects of oral OCFA supplementation were studied in in vivo models of cardiometabolic, inflammatory, liver, hematologic, and fibrotic diseases. Here's a summary of what they found:[13]

C15:0 is a dual, partial agonist for PPARα (65.8%) and PPARδ (52.8%). Effective concentrations of C15:0 needed to reach half-maximum activities for PPARα and PPARδ were 11.5 and 2.7 micrometer (μM), respectively.

C15:0 repaired mitochondrial function and reduced mitochondrial ROS production in a dose-response u-curve. Mitochondrial function and reduced ROS were found in cells supplemented at doses of 10 μM, 20 μM and 50 μM, but at C15:0 concentrations of 100 μM and 200 μM, there were no differences in ROS production compared to non-supplemented controls.

C15:0 reduced proinflammatory and profibrotic states in the human cell systems tested. C17:0 also had these effects, but to a lesser degree.

According to the authors, "this study ... supports that a relatively minor increase in C15:0 concentrations (e.g. from 2.2 μM to 6.7 μM) can positively impact its anti-inflammatory and antifibrotic activities."

On a side note, μM is a unit of measure, not a dose. To convert μM to milligrams (mg), divide it by 1,000. So, a concentration of 2.2 μM equates to 0.0022 mg and 6.7 μM is 0.0067 mg.

Daily supplementation of C15:0 at a dose of 5 mg per kilo of bodyweight lowered inflammation, glucose and cholesterol levels in obese mice.

Daily supplementation of C15:0 at a dose of 35 mg per kilo of bodyweight improved hemolytic anemia in rabbits with diet-induced hypercholesterolemia, anemia and NASH, decreasing the loss of red blood cells and lowering new red blood cell production.

This dose also resulted in lower cholesterol, triglycerides, globulins, and platelets compared to non-supplemented diseased controls. Liver health indices also improved to the point they matched that of healthy controls. They had less severe liver fibrosis, and unlike the diseased controls, they did not progress from Stage 2 to Stage 3 fibrosis.

C15:0 had no off-target pharmacological activities and was noncytotoxic across the 12 human cell systems tested.

C17:0 is a PARδ agonist, with a maximum activity of 39.8%. To achieve half-maximum PPARδ activity, a concentration of 17.4 μM was required.

The ECSFs myristic acid (C14:0) and palmitic acid (C16:0) had similar activity as C15:0. Both are agonists for PPARα and PPARδ, leading the researchers to hypothesize that "carbon chain length may be a determinant of PPARα/δ binding."

None of the saturated fatty acids had PPARγ agonist activity at concentrations below 100 μM.

What Dose of OCFA Will Achieve These Benefits?

To assess the dose required to achieve these kinds of benefits, they gave an oral dose of C15:0 at 35 mg per kilo of body weight to Sprague Dawley rats. Within 30 minutes, plasma concentrations of C15:0 were increased. Maximum concentration (20 μM) was achieved at one hour post-ingestion, and plasma levels remained above baseline for 24 hours.

> *"Thus, a single oral dose of C15:0 at 35 mg/kg succeeded in achieving our targeted active plasma concentrations in this rodent model, between 2.5 to 5 μg/ml (equivalent to 6.7 to 20 μM), from 1 to 8 hours post-dose,"* the authors write.[14] *"Plasma total C17:0 levels also increased, albeit less so than C15:0, following a single oral dose of C15:0."*

To further evaluate the safety of C15:0, rats were dosed orally once a day for 14 days with increasing doses, up to 350 mg per kilo of body weight. No abnormalities were found, and there were also no significant differences in body weights, organ weight-to-body weight ratios, abnormal chemistry values or histologic observations between those who got the C15:0 and the controls.

C15:0 Is Likely an Essential Fat

Based on the findings from this investigation, the researchers concluded that C15:0 (but not C17:0) is most likely an essential fatty acid:[15]

> *"Essential fatty acids are defined as active dietary fatty acids that: (1) are required to maintain a healthy physiological state, (2) are not made at adequate levels endogenously, and (3) require dietary intake in order to maintain healthy concentrations in the body.*
>
> *Given our demonstration of C15:0 and C17:0 as active dietary fatty acids, we reviewed the literature for evidence supporting or negating C15:0 and C17:0 as potential essential fatty acids.*
>
> *Due to reported direct correlations between dietary C15:0 intake and circulating C15:0 concentrations, indicative of primarily diet-based drivers of circulating C15:0, and evidence of endogenous production of C17:0, only C15:0 had supportive evidence across all three criteria that were consistent with a potential essential fatty acid ...*
>
> *Chronic low-grade inflammation, driven by proinflammatory chemokines and cytokines, contributes to cardiometabolic comorbidities and the aging process.*
>
> *Here, daily oral supplementation with C15:0 and C17:0 lowered proinflammatory states in obese mice with metabolic syndrome, as well as lowered proinflammatory biomarkers in primary human cell systems mimicking chronic inflammation ...*

Dyslipidemia and hyperglycemia are components of metabolic syndrome, a cluster of conditions impacting approximately 1 in 3 people globally. Metabolic syndrome increases the risk of type 2 diabetes, heart disease, and all-cause mortality.

In our studies, daily oral C15:0 supplementation over 12 weeks lowered total cholesterol and glucose in an in vivo model with metabolic syndrome."

Dairy Fat — It Does Your Body Good

The take-home from all of this is that diary fat is a crucial source of an essential fat — pentadecanoic acid or C15:0 — that your body needs and cannot make.

A long list of studies[16,17] through the years have shown that this and other OCSFs improve mitochondrial function and increase ATP production,[18] lower your risk of obesity,[19] diabetes[20,21,22] and cardiovascular disease,[23] including the risk of heart problems in diabetics,[24] promote healthy hair growth,[25,26] lower inflammation and much more.

While you can drink whole fat milk, it has 4% fat. Butter has 20 times the fat concentration of whole fat milk, and ghee 25 times the fat as whole fat milk. It is far easier to get these odd chain saturated fats by eating a healthy butter. Ghee is easer and has 25% more fat than butter.

A reasonable dose for most people is 1 tablespoon of butter a day. You can increase that, but it would be unwise to go over 5 tablespoons a day. Unlike raw milk, high quality butters are far more difficult to purchase commercially. About the only way you can is to purchase directly from the farmer.

If you are looking for an easier commercial solution, you can purchase 1 pound of organic ghee for about $20 a pound, including shipping.

Milk Fats Do Not Inhibit Glucose Metabolism

Interestingly, OCFAs are only partially metabolized via the beta-oxidation pathway that ECFAs use. In this pathway, fats are first converted to acetyl-CoA, which allows them to enter the Krebs Cycle. OCFAs, in contrast, are first converted into succinic acid, then succinyl-CoA, which then enters the Krebs Cycle and helps support electron transfer at complex II in the mitochondria. As explained by Georgi Dinkov, an expert on bioenergetic medicine:[27]

> *"Since rising levels of acetyl-CoA has an inhibitory effects on pyruvate dehydrogenase (PDH), eating a diet high in fat with mostly even-chain fats would result in reduction of glucose metabolism, even if all the fats are of the SFA type, as per the Randle Cycle.*
>
> *However, if those fats are of the odd-chain species and enter the Krebs Cycle as succinic acid (i.e. without effect on the acetyl-CoA/CoA ratio), then virtually no such reduction of glucose metabolism is expected to occur and, in fact, PA [pentadecanoic acid] was described in ... Japanese studies as stimulating mitochondrial function and ATP production, which ultimately resulted in improved hair growth.*
>
> *The Japanese researchers even filed a patent for treating hair-loss with PA and in that patent they opined that other odd-chain fatty acids with similar length, especially the C17:0 fat ... would have similarly beneficial effects on hair-growth through increasing mitochondrial function (OXPHOS)."*

Put another way, as I've described in several previous articles, including "Crucial Facts About Your Metabolism," when your fat intake is too high (likely above 35 grams or so), then your body's ability to metabolize (burn) glucose in your mitochondria is reduced. Instead, it gets shuttled into the glycolysis pathway, which is extremely inefficient and produces far fewer ATP molecules per molecule of glucose. That's what Dinkov is talking about here.

> **"OCFAs do not have the same inhibitory effect as ECFAs on glucose burning, because their terminal**

> **metabolic product is not converted to acetyl-CoA but rather enter the Krebs Cycle as succinyl-CoA. In practical terms, this means you don't need to restrict your consumption of full fat dairy, as it won't affect your ability to burn glucose.**

What's fascinating is that OCFAs do not have this inhibiting effect on glucose burning, because they are not converted to acetyl-CoA but rather enter the Krebs Cycle as succinyl-CoA. What this means in practical terms is that you don't need to restrict your consumption of full fat dairy, as it won't affect your ability to burn glucose.

Equally interesting, excessive consumption of other dietary fats has been shown to lower your plasma level of C15:0 and C17:0.[28] So, there are more reasons than one to make sure your total fat intake isn't too high.

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