

# **C15:0 – Found in Dairy – May Be an Essential Fat**

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## **STORY AT-A-GLANCE**

- › C15:0, an odd-chain saturated fat, may be an essential nutrient. Research suggests it plays a crucial role in cellular health and disease prevention
- › The discovery of C15:0's importance began with studies on Navy dolphins. Higher C15:0 levels were associated with healthier aging in these marine mammals
- › C15:0 deficiency may lead to "Cellular Fragility Syndrome," characterized by fragile red blood cells, anemia, and increased risk of chronic diseases like diabetes and cardiovascular issues
- › Modern diets have seen a decline in C15:0 intake due to reduced consumption of whole-fat dairy and changes in cattle feeding practices
- › Increasing C15:0 intake through diet or supplementation may offer significant health benefits, challenging long-held beliefs about saturated fats and nutrition

**C15:0, also known as pentadecanoic acid, is an odd-chain saturated fat primarily found in dairy products, some fish, and certain plants. The story of C15:0's importance begins in an unlikely place – with dolphins.**

**Dr. Stephanie Venn-Watson, a veterinary epidemiologist, was brought on board the Navy's Marine Mammal program about 20 years ago to help understand aging in dolphins and protect their health. The Navy has been caring for a population of dolphins for over 60 years, and these dolphins are living much longer in captivity (40 to 50+ years) compared to their wild counterparts (around 20 years).**

As these dolphins aged, researchers noticed they developed conditions similar to humans, including chronic inflammation, high cholesterol, and fatty liver disease. Intriguingly, some dolphins aged healthier than others, despite receiving the same care. This observation led to a deep dive into the dolphins' diet and metabolomics.

Using advanced techniques, researchers looked at which small molecules predicted healthier aging in dolphins. This is where C15:0 first emerged as a significant factor. Dolphins with higher levels of C15:0 were found to be healthier as they aged. This discovery in dolphins opened a whole new avenue of research into C15:0's potential benefits for human health.

The simplicity of the dolphin diet, consisting solely of fish, allowed researchers to eliminate many confounding factors present in human diets. By studying the fatty acid content of different fish species, they found significant variations in C15:0 levels. This insight led to targeted dietary modifications for the dolphins, which resulted in improved health outcomes.

The dolphin studies provided the first clues about C15:0's potential as an essential fat and its role in healthy aging. This unexpected path from marine biology to human health underscores the interconnectedness of species and the potential for cross-species research to yield valuable insights for human well-being.

## **The Cellular Stability Hypothesis and the Vital Role of C15:0**

**C15:0 has emerged as a crucial player in maintaining your cellular health.** Venn-Watson's recent comprehensive study,<sup>1</sup> has proposed what she calls the "Cellular Stability Hypothesis." This hypothesis suggests that C15:0 plays a vital role in stabilizing your cell membranes, protecting them from a newly discovered form of cell death called ferroptosis.

Ferroptosis is a process involving the peroxidation of fragile fatty acids in your cell membranes, combined with abnormal intracellular iron levels. This deadly combination leads to the production of harmful reactive oxygen species, disabling your mitochondria

– the powerhouses of your cells – and ultimately causing cell death. It's a silent killer that may be at the root of many chronic diseases plaguing our society today.

Dr. Venn-Watson's research suggests that when your C15:0 levels drop below 0.2% of total fatty acids in your cell membranes, you enter a state she calls "Cellular Fragility Syndrome." This syndrome is characterized by fragile red blood cells, anemia, iron overload in the liver, and increased risk of conditions like Type 2 diabetes, cardiovascular disease and fatty liver disease. It's a domino effect that starts at the cellular level and cascades into systemic health issues.

## **Is C15:0 an Essential Fatty Acid?**

For decades, you've known about two essential fats: alpha-linolenic acid or ALA (an omega-3) and linoleic acid (LA, an omega-6). However, recent studies have provided compelling evidence that C15:0 should be added to this exclusive list.

What makes a fat "essential" is that your body cannot produce enough of it on its own, meaning you must obtain it through your diet to maintain optimal health. This discovery is reshaping our understanding of nutrition and could have far-reaching implications for public health policies and dietary recommendations.

C15:0 meets the criteria for an essential fat in several ways. For starters, your dietary intake directly correlates with circulating levels in your body, indicating that it's primarily obtained through diet. Low levels are also consistently associated with increased risk of chronic diseases, suggesting its crucial role in maintaining health.

Supplementation can raise your circulating levels and improve health outcomes in animal models and humans, demonstrating its potential as a therapeutic agent. It also has dose-dependent mechanisms of action that target key physiological processes, further solidifying its importance in your body's functioning.

## **The Far-Reaching Impact of C15:0 Deficiency**

The impact of C15:0 deficiency is far-reaching and multifaceted. The "Cellular Fragility Syndrome" resulting from C15:0 deficiency is characterized by a cascade of health issues. It starts with fragile red blood cells susceptible to lipid peroxidation, leading to anemia and dysmetabolic iron overload syndrome (DIOS). This iron overload can trigger ferroptosis in the liver, potentially leading to advanced nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH).

The syndrome also encompasses insulin resistance, metabolic syndrome, Type 2 diabetes, and cardiovascular disease. Perhaps most alarmingly, it includes systemic iron overload and ferroptosis, which can accelerate aging and tissue damage throughout your body. It's a complex web of interconnected health issues, all potentially stemming from a deficiency in this one crucial fat.

## **The Mounting Evidence for C15:0's Importance**

The evidence supporting the importance of C15:0 is mounting. Numerous epidemiological studies have shown that people with lower circulating C15:0 levels have a higher risk of developing chronic diseases. For example, a meta-analysis<sup>2</sup> of 33 prospective cohort studies found that people with higher C15:0 levels had a lower risk of developing Type 2 diabetes.<sup>3</sup>

This is particularly significant given the global diabetes epidemic we're currently facing. Lower C15:0 levels were also associated with an increased risk of cardiovascular disease in multiple studies,<sup>4</sup> adding another layer to our understanding of heart health. Additionally, people with NAFLD tend to have lower plasma C15:0 concentrations,<sup>5</sup> suggesting a potential role for this fatty acid in liver health.

## **The Decline of C15:0 in Modern Diets**

So, why have we seen a decline in C15:0 intake over the past few decades? Several factors have contributed to this trend. There's been a widespread reduction in the consumption of whole-fat dairy products due to health recommendations to limit

saturated fat intake. These recommendations, while well-intentioned, may have had unintended consequences by reducing our intake of beneficial fats like C15:0.

Changes in cattle feeding practices have also affected C15:0 content in dairy products, with grass fed animals producing milk with higher C15:0 content than those fed corn silage. This shift towards grain-fed cattle in industrial farming may have inadvertently reduced the C15:0 content in our dairy supply.

Seasonal variations in C15:0 content of milk, with lower levels in winter, also play a role. This seasonal fluctuation suggests that our ancestors may have had naturally varying levels of C15:0 intake throughout the year, a pattern that's been disrupted by modern food production and distribution methods.

There's also been a decline in fish consumption in some populations, and certain fish species are good sources of C15:0. Global changes in fish populations, with smaller fish containing less fat and potentially less C15:0, have further contributed to this decline. These factors combine to create a perfect storm of C15:0 deficiency in modern diets.

## **Practical Ways to Increase Your C15:0 Intake**

Based on the available research, your cell membranes require more than 0.2% C15:0 to ensure cellular stability. Optimal circulating C15:0 concentrations should be between 0.4% to 0.64% of total fatty acids. C15:0 deficiency is defined as 0.21% or less of total circulating fatty acids.<sup>6</sup>

To achieve adequate circulating levels, a daily dietary C15:0 intake of around 100 to 200 mg may be necessary. These numbers provide a concrete target for addressing C15:0 deficiency and optimizing your cellular health.

Given the importance of C15:0 for your cellular health and disease prevention, it's crucial to ensure adequate intake. Here are some practical ways to increase C15:0 in your diet:

Embrace full-fat dairy products, especially from grass fed cows. A cup of whole milk contains about 100 mg of C15:0.<sup>7</sup>

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Include grass fed beef in your diet. It contains more C15:0 than grain-fed beef.

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Eat certain types of fish, particularly mullet and catfish, which have C15:0 content similar to milkfat.

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Consider lamb, especially Australian lamb, which is high in C15:0.

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Choose butter over margarine, preferably from grass fed cows.

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Incorporate full-fat **cheese made with animal rennet** into your diet.

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Opt for organic pasture-raised eggs.

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Explore traditional fermented dairy products like kefir or cultured butter.

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## Embracing the C15:0 Revolution

While more research is needed to fully understand the optimal intake and long-term effects of C15:0 supplementation, the current evidence suggests that increasing your dietary intake of this fatty acid could have significant health benefits.

By incorporating C15:0-rich foods into your diet and being mindful of factors that affect C15:0 content in foods, you can take proactive steps towards better cellular health and potentially reduce your risk of chronic conditions like Type 2 diabetes, cardiovascular disease and NAFLD.

As we continue to learn more about C15:0 and its effects on health, it's likely that dietary recommendations will evolve to include this important nutrient. In the meantime, focusing on whole, nutrient-dense foods, particularly those from grass fed animals and certain fish species, can help ensure you're getting adequate amounts of this newly recognized essential fat.

That said, while increasing C15:0 intake may offer health benefits, it's just one piece of the puzzle. A balanced diet, regular exercise, stress management, and other healthy lifestyle factors all play crucial roles in maintaining your overall health and preventing chronic diseases.

The story of C15:0 is a testament to the complexity of human nutrition and the ongoing nature of scientific discovery. It reminds us that there's always more to learn about how our bodies function and what they need to thrive. By staying informed about these discoveries and being willing to adjust our habits accordingly, we can continually optimize our health and well-being.

Stay curious, stay informed, and above all, stay healthy. Your journey to optimal health is a personal one, and discoveries like C15:0 remind us that there's always more to learn. By staying open to new information and being willing to challenge long-held beliefs, you can continue to refine and improve your approach to health and wellness.

## Sources and References

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- <sup>1,7</sup> [Metabolites 2024, 14\(7\), 355; doi: 10.3390/metabo14070355](#)
- <sup>2</sup> [Crit Rev Food Sci Nutr. 2021;61\(16\):2705-2718](#)
- <sup>3</sup> [Circulation. 2016 Apr 26;133\(17\):1645-54](#)
- <sup>4</sup> [PLoS Med. 2021 Sep 21;18\(9\):e1003763](#)
- <sup>5</sup> [PLoS One. 2017 Dec 15;12\(12\):e0189965](#)
- <sup>6</sup> [Clin Chem. 1987 Oct;33\(10\):1869-73](#)