



# Amyotrophic Lateral Sclerosis

ISSN: 1748-2968 (Print) 1471-180X (Online) Journal homepage: <https://www.tandfonline.com/loi/iafd19>

## ALSUntangled 15: Coconut Oil

### The ALSUntangled Group

To cite this article: The ALSUntangled Group (2012) ALSUntangled 15: Coconut Oil, Amyotrophic Lateral Sclerosis, 13:3, 328-330, DOI: [10.3109/17482968.2012.671629](https://doi.org/10.3109/17482968.2012.671629)

To link to this article: <https://doi.org/10.3109/17482968.2012.671629>



Published online: 08 May 2012.



Submit your article to this journal [↗](#)



Article views: 19619



View related articles [↗](#)



Citing articles: 2 View citing articles [↗](#)

## ALSUntangled 15: Coconut Oil

*The ALSUntangled Group*

In an online video from the Christian Broadcasting Network, a Neonatologist named Dr. Mary T. Newport recently reported that treatment of her husband's Alzheimer's disease with coconut oil resulted in significant improvements in his memory, reading, ability to draw a clock and ability to run (1). At the end of the this video, it was stated that “coconut oil may also help Parkinson Disease, ALS, epilepsy, dementia, schizophrenia, autism, and viruses such as HIV and herpes. Evidence supporting these claims is not presented. Here, on behalf of patients with ALS (PALS) who are asking about it, ALSUntangled reviews the evidence for coconut oil in ALS.

### What is coconut oil?

Extracted from the meat or kernel of coconuts, coconut oil comprises 2.5% of world vegetable oil production (2). It is available in a basic “virgin” form, and also an “RBD” (refined, bleached and deodorized) form which has no coconut aroma or taste. It can be partially hydrogenated to increase its melting point; in this process, the mono- and polyunsaturated fats in coconut oil are combined with hydrogen to make them more saturated (2). Partial hydrogenation results in the production of trans fats while “fully hydrogenated” coconut oil contains no trans fats. Coconut oil can also be fractionated into different medium-chain fatty acids; fractionated coconut oil is sometimes referred to as MCT (medium chain triglyceride) oil. Various forms of coconut oil are used in flavoring, cooking oil, engine fuel, engine lubricant, skin moisturizer, soap, lighting, herbicide, and even sand flea repellent (2).

### Why might coconut oil work in ALS?

Mitochondrial dysfunction is likely to play an important role in ALS pathophysiology (3). Among the mitochondrial problems identified, cells extracted from patients with ALS show decreased complex 1 activity (4), which contributes to impaired energy

production. MCT in coconut oil are converted *in vivo* into ketone bodies. In cultured neurons treated with drugs impairing complex 1 function, the addition of ketone bodies can restore complex 1 function (5). Thus, ingestion of coconut oil, by raising ketone levels, could theoretically help compensate for mitochondrial dysfunction and impaired energy production in patients with ALS.

Alternatively, there is growing evidence suggesting that nutritional status (6) and lipid metabolism (7) are important prognostic factors in patients with ALS. Coconut oil could slow ALS progression simply by acting as a high calorie supplement that increases circulating lipids.

### What relevant animal data exists in ALS?

ALSUntangled was unable to find any published studies on coconut oil in ALS animal models. However, given the above-hypothesized mechanisms of coconut oil, published studies looking at ketogenic (KD, 8) and high fat (HFD, 9) diets in ALS mice may be relevant.

In KD study, 6 male SOD1-G93A mice were fed a KD beginning at 50 days of age and compared with 5 male SOD1-G93A animals fed a regular lab diet in terms of blood ketone levels, weight, time to failure on a rotarod motor performance assay, survival, and post-mortem spinal cord motor neuron counts. KD fed mice showed a 3.5 fold increase in blood ketone bodies, trended toward better preserved weight (though the results were not statistically significant), maintained rotarod motor function significantly longer and had significantly higher post-mortem spinal cord motor neuron counts. No difference in survival between the groups was noted.

In the HFD study, 13 SOD1-G93A mice were fed a HFD beginning at 4 weeks of age and compared with 12 SOD1-G93A mice fed a regular diet in terms of body mass, adipose tissue accumulation, several biochemical markers of muscle denervation, post-mortem spinal cord motor neuron counts and

survival. HFD fed mice had significantly better higher body mass index, significantly higher adipose tissue accumulation, reduced expression of biochemical markers of denervation, significantly higher post-mortem spinal cord motor neuron counts, and significantly improved survival.

Methodological flaws in both of these animal studies limit their conclusions; these include a very small sample size, lack of randomization, lack of blinding on many of the outcome measures, and initiation of therapy well before ALS symptom onset.

### **What are the efficacy, safety and cost of coconut oil in human ALS?**

ALSUntangled could find no published clinical trials of coconut oil, KD or HFD in PALS. A trial of KD in PALS was previously open (10) but closed due to lack of enrollment (11). A trial of a HFD is currently open and recruiting patients (12). ALSUntangled investigators are unaware of any of our own patients taking coconut oil whose objective outcome data we could review.

On her website, Dr. Mary Newport shared an email reported to be from a person with a nearly 3 year history of familial ALS (FALS) who had been taking coconut oil for 3 months (most recent dose 8 tablespoons per day) as well as magnesium (13). This PALS described improved lower extremity muscle size and strength, improved ability to stand and pivot, improved sensation and bruising in leg since starting these supplements. No side effects were reported. Contact information for the author of the email was not provided; as a result important details such as compliance with treatment, use of other concomitant treatments, and availability of objective outcome measures cannot be requested. The posting date was February 4, 2011 and despite multiple requests for follow up information from readers since then, none had been provided at the time of this writing. Since this website promotes Dr. Newport's book "What if there was a cure for Alzheimer's Disease and No One knew?" there is a potential conflict of interest regarding the information posted on it.

Within the Patients Like Me (PLM) community, 14 PALS reported taking coconut oil of various brands, formulations and dosages, and 5 completed detailed treatment reports. Within these 5, four PALS reported using virgin coconut oil and the other did not specify the form/brand. Dosages ranged from 1 to 4 tablespoons daily (one reports taking "8 other daily"), durations from 1 week to 1 year, and adherence from "usually" to "always." Four out of these 5 reported that they "can't tell" of any efficacy from this treatment. The other patient (a 45 year old with a 19 year history of ALS who has been using virgin coconut oil at 4 tablespoons daily for a year along with coenzyme Q10) reported increased thumb movement, decreased pain, decreased restless legs and improved constipation. Adverse events reported

to be associated with coconut oil include stomach upset, nausea and diarrhea; in spite of these, all 5 of the PLM PALS completing detailed treatment histories remained on coconut oil. Costs reportedly ranged from under \$25 to \$49 monthly.

Part of the reason for the variability in dosing in the above group may be that the optimal dose of coconut oil for PALS remains unclear. One website (14) suggests that 86ml of coconut oil (5.8 tablespoons) would raise blood ketones into the range that was effective in one small Alzheimer's Disease trial (15) and for epilepsy (16). Experts such as the World Health Organization, FDA and American Heart Association have previously warned against "significant consumption" of coconut oil due to its high levels of saturated fat (2,17); also coconut oil contains lauric acid, which can raise total blood cholesterol levels (2,17). However, recent meta-analyses have found no evidence for concluding that consumption of saturated fats cause heart disease or stroke (18) and lauric acid may actually improve the HDL to LDL ratio (2,17). In any event, there may be other more potent methods for raising ketone bodies that do not carry these same warnings (19).

### **Conclusion**

Coconut oil has plausible mechanisms for use in ALS involving raising ketone bodies and lipid levels. Ketogenic and high fat diets may have helped slow motor neuron loss in small ALS animal studies with many flaws. Two online PALS have reported subjective improvements in muscle strength while taking coconut oil, while four others have not. One of these two is anonymous and described on a website promoting a book about coconut oil, and the other apparently has a very atypical slowly progressive form of ALS and takes at least one other supplement. Coconut oil at doses of 1–4 tablespoons per day appears generally well tolerated but it is not entirely clear how well these doses raise blood ketone levels. Although several large respected groups have warned against coconut oil intake in large amounts, the rationale behind these warnings has recently been called into question. Given all this, ALSUntangled supports further careful study of coconut oil or other methods of raising ketone bodies in patients with ALS. A reasonable next step would be a small case series of well-characterized PALS using coconut oil or other methods to raise blood ketone levels into the range found to be effective in epilepsy and possibly Alzheimer's, compared to a well-matched historical control group on objectively verifiable outcome measures.

**Disclosures:** ALSUntangled is sponsored by the Packard Center and the Motor Neurone Disease Association.

**References**

1. [http://www.cbn.com/media/player/index.aspx?s=/mp4/LJO190v1\\_WS](http://www.cbn.com/media/player/index.aspx?s=/mp4/LJO190v1_WS)
2. [http://en.wikipedia.org/wiki/Coconut\\_oil](http://en.wikipedia.org/wiki/Coconut_oil)
3. Martin LJ. Mitochondrial pathobiology in ALS. *J Bioenerg Biomemb* 2011;43:569–579.
4. Swerdlow R, Parks JK, Cassarino DS, Trimmer PA, Miller SW, Maguire DJ, et al. Mitochondria in sporadic amyotrophic lateral sclerosis. *Exp Neurol* 1998;153:135–142.
5. Tieu K, Perier C, Caspersen C, Teismann P, Wu DC, Yan SD, et al. D-beta-hydroxybutyrate rescues mitochondrial respiration and mitigates features of Parkinson disease. *Journal of Clinical Investigation* 2003;112:892–901.
6. Desport J, Compreux PM, Truong TC, Vallat JM, Sautereau D, Couratier P. Nutritional status is a prognostic factor for survival in ALS patients. *Neurology* 1999;22:1059–1063.
7. Dorst J, Kühnlein P, Hendrich C, Kassubek J, Sperfeld AD, Ludolph AC. Patients with elevated triglyceride and cholesterol serum levels have a prolonged survival in amyotrophic lateral sclerosis. *J Neurol*. 2011;258(4):613–7.
8. Zhao Z, Lange D, Voustantiyouk A, MaGrogan D, Ho L, Suh J, et al. A ketogenic diet as a potential novel therapeutic intervention in amyotrophic lateral sclerosis. *BMC Neuroscience* 2006;7:29.
9. Lupuis L, Oudart H, Rene F, Gonzalez de Aguilar J, Loeffler J. Evidence for defective energy homeostasis in amyotrophic lateral sclerosis: benefit of a high energy diet in a transgenic mouse model. *PNAS* 2004;101:11159–11164.
10. <http://www.clinicaltrials.gov/ct2/show/NCT01035710?term=ketogenic+diet+als&rank=1>
11. Personal communication with Dr. Dale Lange
12. <http://www.clinicaltrials.gov/ct2/show/NCT00983983?term=als+fat&rank=1>
13. <http://coconutketones.blogspot.com/search/label/amyotrophic%20lateral%20sclerosis>
14. [http://www.reddit.com/t/skeptic/comments/p689p/coconut\\_oil\\_cures\\_alzheimers\\_parkinsons\\_als/](http://www.reddit.com/t/skeptic/comments/p689p/coconut_oil_cures_alzheimers_parkinsons_als/)
15. Henderson S, Vogel J, Barr L, Garvin F, Jones J, Costantini L. Study of the ketogenic agent AC-1202 in mild to moderate Alzheimer's disease: a randomized, double-blind, placebo-controlled, multi-center trial. *Nutrition & Metabolism* 2009;6:31.
16. Freeman J, Veggiotti P, Lanzi G, Taliabue A, Perucca E. The ketogenic diet: from molecular mechanisms to clinical effects. *Epilepsy Res* 2006;68:145–180.
17. Cunningham E. Is there science to support claims for coconut oil? *J Am Diet Assoc* 2011;111(5):786.
18. Siri-Tarino P, Qi S, Hu F, Krauss R. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *Am J Clin Nutr* 2010;535–546.
19. [http://winningthefight.net/uploads/Ketone\\_bodies\\_as\\_a\\_therapeutic\\_strategy\\_for\\_ALS\\_and\\_other\\_Neurological\\_Diseases-1\\_1\\_.pdf](http://winningthefight.net/uploads/Ketone_bodies_as_a_therapeutic_strategy_for_ALS_and_other_Neurological_Diseases-1_1_.pdf)

The ALSUntangled Group currently consists of the following members: Richard Bedlack, Orla Hardiman, Tulio Bertorini, Tahseen Mozaffar, Peter Andersen, Jeff Dietz, Josep Gamez, Mazen Dimachkie, Yunxia Wang, Paul Wicks, James Heywood, Steven Novella, LP Rowland, Erik Pioro, Lisa Kinsley, Kathy Mitchell, Jonathan Glass, Sith Sathornsumetee, Hubert Kwiecinski, Jon Baker, Nazem Atassi, Dallas Forsheew, John Ravits, Robin Conwit, Carlyne Jackson, Alex Sherman, Kate Dalton, Katherine Tindall, Ginna Gonzalez, Janice Robertson, Larry Phillips, Michael Benatar, Eric Sorenson, Christen Shoemith, Steven Nash, Nicholas Marigakis, Dan Moore, James Caress, Kevin Boylan, Carmel Armon, Megan Grosso, Bonnie Gerecke, Jim Wymer, Bjorn Oskarsson, Robert Bowser, Vivian Drory, Jeremy Shefner, Terry Heiman-Patterson, Noah Lechtzin, Melanie Leitner, Robert Miller, Hiroshi Mitsumoto, Todd Levine, James Russell, Khema Sharma, David Saperstein, Leo McClusky, Daniel MacGowan, Jonathan Licht, Ashok Verma, Michael Strong, Catherine Lomen-Hoerth, Rup Tandan, Michael Rivner, Steve Kolb, Meraida Polak, Stacy Rudnicki, Pamela Kittrell, Muddasir Quereshi, George Sachs, Gary Pattee, Michael Weiss, John Kissel, Jonathan Goldstein, Jeffrey Rothstein, Dan Pastula. Note: this paper represents a consensus of those weighing in. The opinions expressed in this paper are not necessarily shared by every investigator in this group.