

# Coconut oil – scientific facts

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*Coconut oil and its health benefits have been challenged once again by an US-based professor who has labelled it as ‘a pure poison’. The pertinent question we ask is whether her observations are based on scientific facts. We would like to dispel any negative connotation and arrest the spread of mis-information about the oil of Kalpavriksha. Here we discuss and present scientific facts that support the health benefits of coconut oil.*

## **Constituting fatty acids of coconut oil and their health benefits**

The negative reputation of coconut oil stems from the assumption that saturated fatty acids (SFAs) are deleterious for the human heart. Like most tropical oils, coconut oil has high (82%) saturated fatty acids, specifically lauric acid (LA) and myristic acid. Hence, it is natural to consider coconut oil as a ‘poison’. From a common man’s perspective, any oil that is rich in SFAs is considered bad for health, whereas the beneficial effects of poly-unsaturated fatty acids (PUFAs), especially  $\omega$ -3 and  $\omega$ -6 fatty acids containing natural oils, are well recognized. According to the guidelines of American Dietetic Association (ADA) and Dietitians of Canada, individual SFAs differ in their effects on blood lipid levels. Also, the effect of SFAs on cardiovascular ailments is still under debate<sup>1</sup>. As early as 1981, Prior *et al.*<sup>2</sup> proved that populations which rely on coconut as a source of edible oil do not develop any harmful health effects. This was further corroborated by Kaunitz and Dayrit<sup>3</sup>,

who proved that high dietary coconut oil did not evince any biomarkers associated with coronary heart disease (CHD). Even though the contributory role of saturated fats in cardiovascular ailments is uncertain, coconut oil chiefly comprises SFAs in the form of medium-chain fatty acids (MCFAs) like caproic acid (C6), caprylic acid (C8), capric acid (C10), LA (C12), etc. Medium-chain triglycerides (MCTs) are easily absorbed in the intestine because of their greater solubility and are transported through portal vein to liver to produce ketones and hence energy. On the contrary, long-chain fatty acids (LCFAs) enter the lymphatic portal system, and are deposited in the body and stored as fat. Also, clinical studies have proven that MCFAs have cardiovascular benefits and play a crucial role in the reduction of cardiovascular diseases (CVDs)/CHD-associated risks<sup>4</sup>. A study by Assunção *et al.*<sup>5</sup> reported that 40 women showed a decrease in abdominal fat when they consumed coconut oil and followed a physical activity routine. Another study<sup>6</sup> categorically substantiated that coconut oil provides a satiated feeling though it

did not affect resting energy expenditure. The effects of coconut oil supplementation on body composition and lipid profile of rats that had undergone physical exercise revealed that such supplementation did not interfere with body mass<sup>7</sup>. Coconut oil is nature’s richest source of LA at about 50% of its composition; and human breast milk comes at a distant second with around 6% LA. MCTs, especially LA in its pure form, have antiviral<sup>8</sup> and antibacterial properties, and studies prove that they may help balance gut bacteria and combat pathogenic bacteria<sup>9</sup>. They also help the digestive system because they are easily utilized by the body. When used with a healthy diet and other ways to support gut bacteria, MCFAs may help improve gut health over time.

## **Nutrient richness of virgin coconut oil**

Virgin coconut oil (VCO) obtained from fresh coconut endosperm without any chemical process is rich in polyphenols

and vitamin-E. Hence it has antioxidant and anti-inflammatory properties, thereby possessing cardio-protective effects, and helps inhibit adjuvant-induced arthritis<sup>4,10,11</sup>. It was also observed that VCO consumption during chemotherapy helps improve the functional status and quality of life of patients diagnosed with breast cancer. Additionally, it reduced the symptoms related to side effects of chemotherapy<sup>12</sup>. Antioxidant and hepatoprotective effects of VCO supplementation against hepatotoxicity and oxidative damage via improving antioxidant defence system in rats have also been reported<sup>13</sup>.

The neuroprotective effects of coconut oil has been recognized and hence it is being administered to reduce the cognitive deficits associated with Alzheimer's disease (AD)<sup>14-16</sup>. Recently, the molecular and biochemical basis of neuroprotective effects of coconut oil has started emerging. Improved neuronal survival was observed in a study with coconut oil supplementation, as it rescued the cell cultures treated with amyloid beta ( $A\beta$ ) compared to cultures treated only with  $A\beta$  (ref. 15). VCO has been shown to neutralize the effects of inflammasomes, macromolecular structures that are critical for the development of neurological disorders, in rats induced with AD. This effect has been attributed to its constituent ferulic acid that has the ability to bind fibrillar  $A\beta$  and inhibit its prolongation<sup>17</sup>. Another study examined the effects of LA, the most predominant MCT found in coconut oil, on activated microglia in mice<sup>18</sup>. This study proved

that LA can reverse neuronal damage in patients with AD who consumed coconut oil. LA may attenuate glial activation in a G-protein-coupled receptor (GPR)-dependent pathway and hence reduce the subsequent neuronal damage in patients with AD, who consume coconut oil<sup>17</sup>. In conclusion, consumers should be aware of the scientific background regarding the health benefits of coconut oil, so that it is consumed without any known deleterious effects.

1. Zelman, K., *J. Am. Diet. Assoc.*, 2011, **111**(5), 655–658.
2. Prior, I. A., Davidson, F., Salmond, C. E. and Czochanska, Z., *Am. J. Clin. Nutr.*, 1981, **34**, 1552–1561.
3. Kaunitz, H. and Dayrit, C. S., *Philipp. J. Coconut Stud.*, 1992, **17**, 165–171.
4. Babu, A. S., Veluswamy, S. K., Arena, R., Guazzi, M. and Lavie, C. J., *Postgrad. Med.*, 2014, **126**(7), 76–83.
5. Assunção, M. L., Ferreira, H. S., dos Santos, A. F., Cabral, C. R. and Florêncio, T. M., *Lipids*, 2009, **44**(7), 593–601.
6. Valente, F. X., Cândido, F. G., Lopes, L. L., Dias, D. M., Carvalho, S. D. L., Pereira, P. F. and Bressan, J., *Eur. J. Nutr.*, 2018, **57**(4), 1627–1637.
7. Resende, N. M., Felix, H. R., Sore, M. R., Neto, A. M. M., Campos, K. E. and Volpato, G. T., *An. Acad. Bras. Ciênc.*, 2016, **88**(2), 933–940.
8. Enig, M. G., In Proceedings of AVOC Lauric Oil Symposium, Manila, Philippines, 1997.
9. Nitbania, F. O., Siswanta, D. and Solikhah, E. N., *Procedia Chem.*, 2016, **18**, 132–140.
10. Nevin, K. G. and Rajamohan, T., *Clin. Biochem.*, 2004, **37**, 830–835.
11. Vysakh, A., Ratheesh, M., Rajmohan, T. P., Pramod, C., Premlal, S., Girish Kumar, B. and Sibi, P. I., *Int. Immunopharmacol.*, 2014, **20**, 124–130.
12. Law, K. S., Azman, N., Omar, E. A., Musa, M. Y., Yusoff, N. M., Sulaiman, S. A. and Hussain, N. H. N., *Lipids Health Dis.*, 2014, **13**(1), 139.
13. Famurewa, A. C., Aja, P. M., Maduagwuna, E. K., Ekeleme-Egedigwe, C. A., Ufebe, O. G. and Azubuiké-Osu, S. O., *Biomed. Pharmacother.*, 2017, **96**, 905–911.
14. Fernando, W. M. A. D. B., Martins, I. J., Goozee, K. G., Brennan, C. S., Jayasena, V. and Martins, R. N., *Br. J. Nutr.*, 2015, **114**(1), 1–14.
15. Nafar, F., Clarke, J. P. and Mearow, K. M., *Neurochem. Int.*, 2017, **105**, 64–79.
16. Rahim, N. S., Lim, S. M., Mani, V., Abdul Majeed, A. B. and Ramasamy, K., *Pharm. Biol.*, 2017, **55**(1), 825–832.
17. Mirzaei, F., Khazaei, M., Komaki, A., Amiri, I. and Jalili, C., *Food Chem. Toxicol.*, 2018, **118**, 68–83.
18. Nishimura, Y., Moriyama, M., Kawabe, K., Satoh, H., Takano, K., Azuma, Y. T. and Nakamura, Y., *Neurochem. Res.*, 2018, **43**(9), 1723–1735.

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