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Dodecanoic-Acid in Extra Virgin Coconut Oil, May Reduce the Incidence of Heart Disease and Cancer in Humans

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Abstract: ***Lauric-acid is a 12-carbon, medium-length, long-chain fatty acid that makes up around 50 percent of the fatty acids within coconut oil. It is a powerful compound that is sometimes extracted from the coconut for use in developing monolaurin. Monolaurin is an anti-microbial agent that is able to fight bacteria, viruses, yeasts, and other pathogens. Since one cannot ingest lauric-acid alone because it is irritating and not found alone in nature, one is most likely to consume it in the form of extra virgin coconut oil or from fresh coconuts. This prospective research is about the effects of lauric-acid in prevention of cancer and heart disease by reviewing applied studies on animals and humans. The viral, fungal and microbial effects in the cause of some types of cancer and heart disease is well established, therefore, the anti-pathogenic and anti-parasitic effects of lauric-acid is the other way of importance of this compound.***

Keywords: Coconut oil, Lauric-acid, Cancer, Heart disease, Monolaurin

1. Introduction

Coconut oil is one of the richest sources of saturated fat, with around 90% of calories as saturated fatty acids. Coconut oil is very different from most other cooking oils and contains a unique composition of fatty acids. Additionally, coconut oil consists almost entirely of Medium Chain Triglycerides.[47]

These fatty acids go straight from the digestive tract to the liver, where they are likely to be turned into ketone bodies and provide a quick source of energy. The most abundant fatty acid in coconut oil is the 12-carbon Lauric-Acid, which is broken down into a compound called monolaurin in the body. Lauric-acid and monolaurin are both very interesting due to the fact that they can kill microbes like bacteria, fungi and viruses. For this reason, coconut oil can be protective against various infections.[48, 49, 50] The physical properties of coconut oil are listed below:

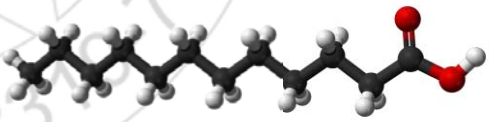
a) Color: Coconut oil is colorless at or above 30° C. It is white when in its solid form.
b) Odor: The typical smell of Coconut oil is that of coconuts only if not refined, bleached, and deodorized.
c) Melting Point: Coconut oil melts at 25° C which is about 76° Fahrenheit. It is solid below this temperature.
d) Smoking Point: 1770° C that is 3500° Fahrenheit.
e) Solubility in Water: Coconut oil forms a white homogenous mixture when beaten well in a little water. Otherwise, it is insoluble in water at room temperature.

Coconut Oil is predominantly composed of saturated fatty acids which is about 94%, with a good percentage which is above 62% of Medium Chain Fatty Acids among them.

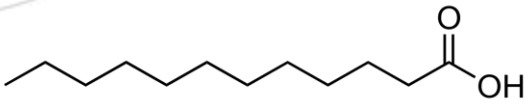
2. Chemical Composition

The amount of moisture present in Coconut oil varies greatly depending upon the moisture content of the source, such as Copra or Fresh Coconut, including the method of extraction and also on the processing done on it. But obviously, coconut derived from well-dried copra will have less moisture than that from less dried copra or from fresh coconuts which is called virgin coconut oil. Similarly, the oil subjected to heat will have less moisture than that which was not exposed to heat. Fatty Acids compounds of coconut oil: [46]

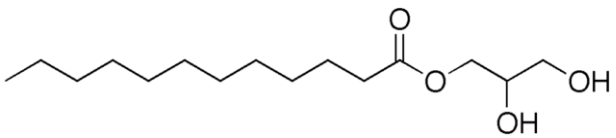
Lauric-acid or dodecanoic-acid, is a saturated fatty acid with a 12-carbon atom chain, thus falling into the medium chain fatty acids, is a white, powdery solid with a faint odor of bay oil or soap. Lauric acid, as a component of triglycerides, comprises about half of the fatty acid content in coconut oil.[44,45] It is also found in human breast milk which is 6.2% of total fat, fresh cow's milk that is2.9%, and fresh goat's milk up to 3.1%.[44]



Three dimensional molecule of Lauric-acid



Lauric-acid



Monolaurin, also known as glycerol mono-laurate, glyceryl laurate or 1-Lauroyl-glycerol, is a mono-glyceride. It is the mono-ester formed from glycerol and lauric-acid. Its chemical formula is $C_{15}H_{30}O_4$.

3. Materials and Methods

By reviewing several published studies, and collecting the information and conclusions, we put them into two groups.

The first group is studies which had done on animal models and the second one is on humans. We hope our perspective research which is based on evidences by important studies leads to the true and rational conclusion of the protective effects of lauric-acid on heart disease and cancer prevention.

a) Animal Studies of Unprocessed Coconut Oil

In 1980, Hoostmark compared how 10% coconut fat and 10% sunflower oil affected the lipoprotein distribution in male Wistar rats. Rats had fed coconut oil, produced significantly lower levels (p=<0.05) of VLDL and significantly higher (p=<0.01) alpha-lipoproteins (HDL) relative to sunflower oil feeding.[16]

In 1981, Awad compared the effects of diets containing 14% coconut oil, 14% safflower oil or a 5% mostly soybean oil on accumulation of cholesterol in tissues in male Wistar rats. The synthetic diets had 2% added corn oil with a total fat of 16% Total tissue cholesterol accumulation for animals on the safflower diet was 6 times greater than for animals fed the coconut oil, and twice that of the animals fed the control oil.[1]

b) Human Studies

In 1992, Dayrit and Kaunitz have reviewed some of the epidemiological and experimental studies regarding coconut-eating groups and concluded that the dietary coconut oil, does not lead to high serum cholesterol nor to high coronary heart disease mortality or morbidity. In 1989 Mendis et al, reported undesirable lipid changes when young adult Sri Lankan males were changed from their normal diets by the substitution of corn oil for their customary coconut oil. Although the total serum cholesterol decreased 18.7% from 179.6 to 146.0 mg/dl and the LDL cholesterol decreased 23.8% from 131.6 to 100.3 mg/dl, the HDL cholesterol decreased 41.4% from 43.4 to 25.4 mg/dl (putting the HDL values below the acceptable lower limit) and the LDL/HDL ratio increased 30% from 3.0 to 3.9. These latter two changes would be considered quite undesirable. As noted above, Kurup and Rajmohan (1995) studied the addition of coconut oil alone to previously mixed fat diets and report no significant difference.[25, 27]

In 1981, one study had shown that islanders with high intake of coconut oil showed no evidence of the high saturated fat intake having a harmful effect in these populations. When these groups migrated to New Zealand, and lowered their intake of coconut oil, their total cholesterol and LDL cholesterol raised, and their HDL cholesterol decreased.[34]

In 1961, Frantz and Carey fed an additional 810 kcal/day fat supplement for a month to males with high normal serum cholesterol levels; there was no significant difference from the original levels, although the fat supplement was hydrogenated coconut oil type.[12]

In 1961, Halden and Lieb showed similar results in a group of hyper-cholesterolemics when coconut oil was included in their diets. Original serum cholesterol levels were reported as 170- 370 mg/dl. Straight coconut oil produced a range from 170 to 270 mg/dl. Coconut oil combined with 5% sunflower oil and 5% olive oil produced a range of 140 to 240 mg/dl.[12]

In 1959, Hashim and colleagues had shown quite clearly that feeding a fat supplement to hyper-cholesterolemics, where half of the supplement, 21% of energy, was coconut oil and the other half was safflower oil, resulted in significant reductions in total serum cholesterol. The reductions averaged 29% and ranged from 6.8 to 41.2%.[13]

In 1957, Ahrens and colleagues had shown that adding coconut oil to the diet of hyper-cholesterolemics lowers serum cholesterol from 450 mg/dl to 367 mg/dl. [26]

In 1967, Bierenbaum followed 100 young men with documented myocardial infarction for 5 years on diets with fat restricted to 28% of energy. There was no significant difference between the two different fat mixtures. That means, 50/50 corn and safflower oils or 50/50 coconut and peanut oils, which were fed as half of the total fat allowance, both diets reduced serum cholesterol. This study clearly showed that 7% of energy as coconut oil was as beneficial to the 50 men who consumed it as for the 50 men who consumed 7% of energy as other oils such as corn oil or safflower. Both groups were far better than the untreated controls.[2]

In 1994, Sundram fed whole food-diet to healthy normo-cholesterolemic males, where approximately 30% of energy was fat. Lauric-acid and myristic acid from coconut oil, supplied nearly 5% of energy. Relative to the baseline measurements of the subjects prior to the experimental diet, this lauric and myristic acid-rich diet showed an increase in total serum cholesterol from 166.7 to 170.0 mg/dl (+1.9%), a decrease in low density lipoprotein cholesterol (LDL-C) from 105.2 to 104.4 mg/dl (-0.1%), an increase in high density lipoprotein cholesterol (HDL-C) from 42.9 to 45.6 mg/dl (+6.3%). There was a 2.4% decrease in the LDL-C/HDL-C ratio from 2.45 to 2.39. These findings indicate a favorable alteration in serum lipoprotein balance that was achieved when coconut oil was included in a whole food diet at 5% of energy.[40]

In 1994, Tholstrup reported similar conclusions with whole-food diets high in lauric and myristic acids from palm kernel oil. The HDL cholesterol levels increased significantly from 37.5 to 46.0 mg/dl, P<0.01, and the LDL-C/HDL-C ratios decreased from 3.08 to 2.69. The increase in total cholesterol was from 154.7 to 170.9 mg/dl on the experimental diet.[41]

In 1991, Ng fed 75% of the fat ration as coconut oil (24% of energy) to 83 adult normo-cholesterolemics, which were 61 males and 22 females. The highest value on the experimental diet for total cholesterol was increased 17%. Which means from 169.6 to 198.4 mg/dl, HDL cholesterol was increased 21.4% that is from 44.3 to 53.8 mg/dl, and the LDL-C/HDL-C ratio was gone down 3.6% that is from 2.51 to 2.42.[33]

Ginsberg, provided an average American diet with 2-3 times more myristic-acid (C14:0), 4.5 times more lauric-acid (C12:0), and 1.2 times more palmitic and stearic-acid (C16:0 and C18:0) than their mono-unsaturated fat-diet and the National Cholesterol Education Program (NCEP) Step 1-diet, there was no increase in serum cholesterol. As a matter of fact, serum cholesterol levels for this diet group,

decreased nearly 3% from 177.1 mg% to 171.8 mg% in the 22 week feeding trial duration. (Ginsberg, B.H., et al. 1982)

When unprocessed coconut oil is added to an otherwise normal diet, there is frequently no change in the serum cholesterol although some studies have shown a decrease in total cholesterol.[8] It appears from many of the research reports the effect that coconut oil has on serum cholesterol is the opposite in individuals with low serum cholesterol values and those with high serum values. We see that there may be an increase of serum total cholesterol, LDL cholesterol and especially HDL cholesterol in individuals with low serum cholesterol. On the other hand there is a lowering of total cholesterol and LDL cholesterol in hyper-cholesterolemics as noted above.

Studies that supposedly showed a hyper-cholesterolemic effect of coconut oil feeding, in fact, usually only showed that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat being compared. This appears to be in part because coconut oil does not *drive* cholesterol into the tissues as does the more poly-unsaturated fats. The chemical analysis of the atheroma shows that the fatty acids from the cholesterol esters are 74% unsaturated that is 41% is polyunsaturated and only 24% are saturated. None of the saturated fatty acids were reported to be lauric-acid or myristic-acid (Felton et al 1994).

c) Coconut Oil and Cancer

In 1987, Lim Sylianco has reviewed 50 years of studies, showing anti-carcinogenic effects of dietary coconut oil. These animal studies showed quite clearly the non-promotional effect of feeding coconut oil.[28]

In 1984, one study by Reddy, straight coconut oil was more inhibitory than MCT(Medium Chain Trygliceride) oil to induction of colon tumors by azoxymethane. Chemically induced adeno-carcinomas differed 10-fold between corn oil (32%) and coconut oil (3%) in the colon. Both olive oil and coconut oil developed the low levels, approximately 3%, of the adeno-carcinomas in the colon, but in the small intestine, animals fed coconut oil did not develop any tumors while 7% of animals fed olive oil did.[37]

In 1986, Cohen concluded that the non-promotional effects of coconut oil were also seen in chemically induced breast cancer. In this study, the slight elevation of serum cholesterol in the animals fed coconut oil was protective as the animals fed the more poly-unsaturated oil had reduced serum cholesterol and more tumors. This means, an overall inverse effect was observed between total serum lipids and tumor incidence for the four high-fat groups.[5]

4. Discussions

a) Anti-microbial effect of coconut oil

The cancer causing effect of parasitic micro-organisms are well established. Therefore the anti-viral, anti-bacterial and anti-fungal effect of lauric-acid in extra virgin coconut oil is important. The lauric-acid in coconut oil is used by human body to make the same disease-fighting fatty acid derivative mono-laurin that babies make from the lauric-acid they get from their mothers' milk. The mono-glyceride-mono-laurin is the compound that keeps infants from getting viral, bacterial or protozoal infections.[15]

Recognition of the antimicrobial activity of the mono-glyceride of lauric-acid, that is mono-laurin has been reported since 1966. The seminal work can be credited to Jon Kabara. This early research was directed at the virucidal effects, due to possible problems related to food preservation. Some of the early work by Hierholzer and Kabara in 1982, that showed virucidal effects of mono-laurin on enveloped RNA and DNA viruses was done in conjunction with the Center for Disease Control of the US Public Health Service with selected prototypes or recognized representative strains of enveloped human viruses. The envelope of these viruses is a lipid membrane. In 1978, Kabara and others have reported that certain fatty acids that is medium-chain –saturates, and their derivatives which is mono-glycerides, can have adverse effects on various micro-organisms. Those micro-organisms that are inactivated include bacteria, yeast, fungi, and enveloped viruses.[15]

The medium-chain saturated fatty acids and their derivatives act by disrupting the lipid membranes of the organisms (Isaacs and Thormar 1991and Isaacs et al 1992). In particular, enveloped viruses are inactivated in both human and bovine milk by added fatty acids and mono-glycerides.(Isaacs et al 1991) as well as by endogenous FAs and MGs (Isaacs et al 1986, 1990, 1991, 1992 and Thormar et al 1987).

All three mono-esters of lauric-acid are shown to be active anti-microbials, that is alpha- and beta-MG. Also, it is mentioned that the anti-microbial effects of the FAs and MGs are additive and total concentration is critical for inactivating viruses (Isaacs and Thormar 1990).

The properties that determine the anti-infective effects of lipids are related to their structure which is mono-glycerides, free fatty-acids. The mono-glycerides are active. However, di-glycerides and tri-glycerides are inactive. In saturated fatty acids, lauric-acid has greater anti-viral properties than either caprylic-acid (C-10) or myristic-acid (C-14).[16]

The action attributed to mono-laurin is that of solubilizing the lipids and phospholipids in the envelope of the virus causing the disintegration of the virus envelope. In effect, it is reported that the fatty acids and mono-glycerides, produce their killing-inactivating effect by lysing the lipid bilayer plasma membrane. However, there is evidence from recent studies that one antimicrobial effect is related to its interference with signal transduction (Projan et al 1994).

Some of the viruses inactivated by these lipids, in addition to HIV, are the measles-virus, herpes simplex virus-1 (HSV-1), vesicular stomatitis virus (VSV), visna virus, and cytomegalo-virus (CMV). Many of the pathogenic organisms reported to be inactivated by these antimicrobial lipids are those known to be responsible for opportunistic infections in HIV-positive individuals. For example, concurrent infection with cytomegalovirus is recognized as a serious complication for HIV-positive individuals (Macallan et al 1993). Thus, it would appear to be important to investigate the practical aspects and the potential benefit of an adjunct nutritional

support regimen for HIV-infected individuals, which will use those dietary fats that are sources of known anti-viral, anti-microbial, and anti-protozoal mono-glycerides and fatty acids such as monolaurin and its precursor lauric-acid.[18, 19]

These anti-microbial fatty acids and their derivatives are essentially non-toxic to man.[20, 21] They are produced in vivo by humans when they ingest commonly available foods that contain adequate levels of medium-chain fatty acids such as lauric-acid. Based on the published research, lauric-acid is one of the best inactivating fatty acids, and its mono-glyceride is even more effective than the fatty acid alone (Kabara 1978, Sands et al 1978, Fletcher et al 1985, Kabara 1985).

The lipid coated viruses are dependent on host lipids for their lipid constituents. The variability of fatty acids in the foods of individuals accounts for the variability of fatty acids in the virus envelop and also explains the variability of glycolprotein expression.[22]

b) The Coconut-producing Countries

Whole coconut as well as extracted coconut oil has been a mainstay in the food supply in many countries in parts of Asia and the Pacific Rim throughout the centuries. Recently though, there has been some replacement of coconut oil by other seed oils. This is unfortunate since the benefits gained from consuming an adequate amount of coconut oil are being lost.

Based on the per capita intake of coconut oil in 1985, as reported by Kaunitz in1992, the per capita daily intake of lauric-acid can be approximated. For those major producing countries such as the Philippines, Indonesia, and Sri Lanka, and consuming countries such as Singapore, the daily intakes of lauric-acid were approximately 7.3 grams in Philippines, 4.9 grams in Sri Lanka, 4.7 grams in Indonesia, and 2.8 grams in Singapore. In India, intake of lauric-acid from coconut oil in the coconut growing areas that is Kerala, range from nearly 12-20 grams per day (Early 1995), whereas the average for the rest of the country is less than half a gram. An average high of approximately 68 grams of lauric-acid is calculated from the coconut oil intake previously reported by Kaunitz in1981, for the Tokelau Islands. Other coconut producing countries may also have intakes of lauric-acid in the same range (Kaunitz et al 1992 and 1981).

c) Lauric-Acid Dosage

It is not well established exactly how much food made with lauric-acid oils is needed to have a protective effect of lauric-acid in the diet. Infants approximately consume 0.3 to1 gram per kilogram of body weight, if they are fed human milk or an enriched infant-formula which contains coconut oil. This amount appears to have been protective. Adults could probably benefit from the consumption of 10 to 20 grams of lauric-acid per day. Growing children probably need about the same amounts as adults. Although further studies are needed to mention the exact dosage of lauric-acid in a diet to be protective.

5. Acknowledgements

We would like to thank Professor Thomas Seyfried PhD. In diology department of Boston college for inspiring us in cancer research and his hypothesis of the metabolical aspect of the cause of cancer, which is very helpful in our research about cancer prevention.

6. Conclusion

Almost 50% of the fatty acids in coconut oil is the 12-carbon Lauric-Acid. When lauric-acid is digested, it forms a substance called monolaurin. Both lauric-acid and monolaurin can kill harmful pathogens like bacteria, viruses and fungi. As we see from our research, lauric-acid in extra virgin coconut oil has the protective effect against cancer and heart disease. This important and beneficial compound can be found in human milk that is beneficial to infants in reducing the cancer risk and future heart disease. However additional research is needed for extra benefits of lauric-acid and consumption of extra virgin coconut oil.

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