Potential Supportive Mechanisms of Coconut Oil and Cancer Development

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As a whole, high-fat diets have been correlated with increased risk of skin, liver, breast, colon, and prostate cancers (1,2,3,4,5,6,7,8,9), but the health effects may be stratified differently depending the type and source of fat.

Medium Chain Triglycerides (MCTs) have unique structural and physiological properties that may offer differential effects on development of certain tumors than other fats (10,11). It is generally agreed that trans fatty acids and polyunsaturated fats (PUFAs) such as linoleic acid favor the development of tumors, with omega 3 PUFAs exerting a neutral or protective effect.

Coconut oil and its high percentage of MCTs has been linked to fewer incidences of cancer for liver (12,13,14), colon and small intestine (11,15), breast (4,17,18,20), and pancreas (21) when compared to PUFAs like corn oil. PUFAs can also be sub-categorized into omega 3, 6 and 9 fatty acids which also may also have further differences on their effects on cancer incidence and progression.

Omega 6 PUFAs promote inflammation, alter signal reception at the cell membrane, decrease immunity, and oxidize more readily - supporting cancer development and growth (22). Omega 3 PUFAs (EPA and DHA) may be associated with fewer cancers when compared to studies using omega-6-PUFAs (23-33), but the associations are still inconclusive (34-47).

Possible Mechanisms of Action:

Coconut oil has the potential to modulate cancer risk in a number of ways.

Cell Membrane Structure and Stability:

Because of its unique composition of fats, coconut oil promotes healthy cell membrane structure which invites healthy signal transmission and adds cell stability from oxidative stress. It also has a neutral effect on hormones unlike linoleic and linolenic acid that have anti-inflammatory and pro-inflammatory effects.

Coconut oil may also help promote healthy levels of cholesterol in the body. Cholesterol helps support cell membrane health and also acts as a precursor to sex and steroid hormones. Endogenous cholesterol production accounts for the majority of cholesterol levels in the body as it can be made from fatty acids, amino acids, and glucose.

While much focus on cholesterol has centered on heart disease, its associations with cancer are
often overlooked. Low levels of cholesterol have been associated with an increased risk of various types of cancer (48, 49).

Despite its high concentration of saturated fatty acid, Coconut oil may have a neutral to supportive effect on cholesterol levels in the body. While the fats in coconut oil may raise total cholesterol and LDL, it is believed to have a more profound effect on HDL levels and other important risk markers of heart disease (50-67).

**Ketone Metabolism**

The MCTs that comprise two-thirds of coconut oil's composition are metabolized uniquely into a non-glucose source of energy known as ketones that may be cancer protective (68). MCTs are quickly oxidized into ketone bodies which supply a quick source of energy to the liver and the rest of the body.

MCTs may be efficacious in conditions where energy needs are increased such as following surgery and general malnourishment. Additionally, MCT's may also offer a dietary source of ketone bodies in a modified ketogenic diet as there is often poor compliance with a traditional ketogenic diet (69).

In a mouse model of glioma, a ketogenic diet improved survival rate in the mice. The results were linked with a reduction of reactive oxygen species produced by tumor cells, but also with a modulation of gene expression responsible for tumor growth and oxidative stress. The changes in gene expression indicated protective mechanisms that may go beyond simply reducing glucose (70).

An additional study using a ketogenic diet in mice using MCTs and omega 3 fatty acids reported delayed gastric adenocarcinoma growth (71). Another study with mice suggested that a reduction in tumor growth may be due to general modulation of the immune system (72).

A contraindication to consumption of MCTs would include states of ketoacidosis, where diabetics are at most risk. Additionally, poor clearance of MCTs by the liver may occur in patients with cirrhosis and they should be closely monitored (73).

**Immune support:**

MCT's may also provide antitumor benefits while maintaining normal immune system function (74).

Patients with gastrointestinal tract cancer requiring total parenteral nutrition received a mix of MCT and LCT or just LCT. Those receiving MCT/LCT had significantly improved nutritional status measured by complements C3 and C4, total lymphocyte count, and immunoglobulins, as well as higher prealbumin levels (75).
Certain viral and infectious processes may promote cancer incidence and growth. Coconut oil's MCTs and their corresponding monoglycerides may also possess anti-microbial properties that may help modulate inflammation indirectly and modulate cancer risk.

References:

2.) Carroll KK, Khor HT. Effects of dietary fat and dose level of 7, 12-dimethylbenz(alpha)-anthracene on mammary tumor incidence in rats. m1975; 30: 226
7.) Baumann CA, Rusch HP. Effect of diet on tumors induced by ultraviolet light. Am J Cancer. 1939; 35: 213-221
8.) Black HS. Influence of dietary factors on actinically-induced skin cancer. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis 1998 Nov; 422(1): 185-190
12.) Opie EL 1944, the influence of diet in the production of tumors of the livery by butter yellow. J Exper, ed 80.:219-230
13.) Klline BE et al 1946. The carcinogenicity of dimethylaminoazobenzene in diets containing fatty acids of coconut oil or of corn oil
15.) Broitman SA et al. Polyunsaturated fat, cholesterol, and large bowel carcinogenesis. 1977;Cancer 40:245
16.)Gammal EB, Carroll KK, Plunkett ER. Effect of dietary fat on mammary carcinogenesis by 7, 12-dimethylbenz(a)anthracene. 1967; Cancer Res. 27: 1737-1742
17.)Carroll KK, Khor MT. Effects of dietary fat and dose level of 7, 12-dimethylbenz(a)anthracene on mammary tumor incidence in rats. 1970; Cancer Res 30:2260
31.) Ip C 1997 Review of the effects of trans fatty acids, oleic acid, n-3 polyunsaturated fatty acids, and conjugated linoleic acid on mammary carcinogenesis in animals. Am J Clin Nutr 66 (suppl) 1523 S01529 S
35.) Erickson KL 1998. is there a relation between dietary linoleic acid and cancer of the breast, colon, or prostate? Am J Clin Nutr. 68:5-7


69.) (Otto C 2008)


