



## ABSTRACTS ON VIRGIN COCONUT OIL

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### **Insulinotropic potency of lauric acid: a metabolic rationale for medium chain fatty acids (MCF) in TPN formulation.**

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The need for a better lipid system to satisfy the fuel requirements of patients while avoiding the adverse effects of current systems has led to suggestions that medium chain fatty acids (MCFs) be incorporated into TPN-lipid emulsions. Since clinical situations requiring TPN are associated with metabolic processes mediated by insulin, in the present study we have therefore examined the effects of a variety of medium chain fatty acids on insulin release. Using an isolated perfused mouse islet model, various doses of medium chain fatty acids and the essential fatty acid, linoleic acid, were tested and compared. The possibility of an additive effect of an insulinotropic MCF and linoleate when both are provided together was also examined. Effluent perfusate samples collected on ice during these experiments were assayed for insulin by radioimmunoassay. It was found that the ability of 5 mM of a given MCF to stimulate insulin secretion was dependent upon its chain length. Thus, while adipic acid (C6) had no effect, Caprylic acid (C8) had a minimal effect that was not statistically significant, but capric acid (C10) and lauric acid had very potent effects that were of the same magnitude to the effect of linoleate on insulin secretion. When insulin output was assessed as the mean integrated area under the curve during a 20-min perfusion, 5 mM lauric acid enhanced insulin secretion from a basal 7351 +/- 666 pg to 15,756 +/- 1680 pg (P less than 0.01, n = 5). In the same experiments, 5 mM linoleic acid stimulated insulin release to 11,260 +/- 867 pg (P less than 0.05). When C12 and linoleate were added together, each at a submaximally effective concentration of 2.5 mM, insulin output was 12,712 +/- 1011 pg (P less than 0.05, n = 5), which was not statistically different from the values obtained when the islets were perfused with 5 mM of each fatty acid alone. (ABSTRACT TRUNCATED AT 250 WORDS)

[J Surg Res.](#) 1992 Apr;52(4):328-33.

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### **Beneficial effects of virgin coconut oil on lipid parameters and in vitro LDL oxidation.**

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**OBJECTIVES:** The present study was conducted to investigate the effect of consumption of virgin coconut oil (VCO) on various lipid parameters in comparison with copra oil (CO). In addition, the preventive effect of polyphenol fraction (PF) from test oils on copper induced oxidation of LDL and carbonyl formation was also studied. **DESIGN AND METHODS:** After 45 days of oil feeding to Sprague-Dawley rats, several lipid parameters and lipoprotein levels were determined. PF was isolated from the oils and its effect on in vitro LDL oxidation was assessed. **RESULTS:** VCO obtained by wet process has a beneficial effect in lowering lipid components compared to CO. It reduced total cholesterol, triglycerides, phospholipids, LDL, and VLDL cholesterol levels and increased HDL cholesterol in serum and tissues. The PF of virgin coconut oil was also found to be capable of preventing in vitro LDL oxidation with reduced carbonyl formation. **CONCLUSION:** The results demonstrated the potential beneficiary effect of virgin coconut oil in lowering lipid levels in serum and tissues and LDL oxidation by physiological oxidants. This property of VCO may be attributed to the biologically active polyphenol components present in the oil.

Clin Biochem. 2004 Sep;37(9):830-5.

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**A diet rich in coconut oil reduces diurnal postprandial variations in circulating tissue plasminogen activator antigen and fasting lipoprotein (a) compared with a diet rich in unsaturated fat in women.**

**Muller H, Lindman AS, Blomfeldt A, Seljeflot I, Pedersen JI.**

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The effects of high and low fat diets with identical polyunsaturated/saturated fatty acid (P/S) ratios on plasma postprandial levels of some hemostatic variables and on fasting lipoprotein (a) [Lp(a)] are not known. This controlled crossover study compared the effects of a high fat diet [38.4% of energy (E%) from fat; HSAFA-diet, P/S ratio 0.14], a low fat diet (19.7 E% from fat; LSAFA-diet, P/S ratio 0.17), both based on coconut oil, and a diet with a high content of monounsaturated fatty acids (MUFA) and PUFA (38.2 E% from fat; HUFA-diet, P/S ratio 1.9) on diurnal postprandial levels of some hemostatic variables (n = 11) and fasting levels of Lp(a) (n = 25). The postprandial plasma concentration of tissue plasminogen activator antigen (t-PA antigen) was decreased when the women consumed the HSAFA-diet compared with the HUFA-diet (P = 0.02). Plasma t-PA antigen was correlated with plasminogen activator inhibitor type 1 (PAI-1) activity when the participants consumed all three diets (Rs = 0.78, P < 0.01; Rs = 0.76, P < 0.01; Rs = 0.66, P = 0.03; on the HSAFA-, the LSAFA- and the HUFA-diet, respectively), although the diets did not affect the PAI-1 levels. There were no significant differences in postprandial variations in t-PA activity, factor VII coagulant activity or fibrinogen levels due to the diets. Serum fasting Lp(a) levels were lower when women consumed the HSAFA-diet (13%, P < 0.001) and tended to be lower when they consumed the LSAFA-diet (5.3%, P = 0.052) than when they consumed the HUFA-diet. Serum Lp(a) concentrations did not differ when the women consumed the HSAFA- and LSAFA-diets. In conclusion, our results indicate that a coconut oil-based diet (HSAFA-diet) lowers postprandial t-PA antigen concentration, and this may favorably affect the fibrinolytic system and the Lp(a) concentration compared with the HUFA-diet. The proportions of

dietary saturated fatty acids more than the percentage of saturated fat energy seem to have a beneficial influence on Lp(a) levels.

J Nutr. 2003 Nov;133(11):3422-7.

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### **Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau island studies.**

**Prior IA, Davidson F, Salmond CE, Czochanska Z.**

Two populations of Polynesians living on atolls near the equator provide an opportunity to investigate the relative effects of saturated fat and dietary cholesterol in determining serum cholesterol levels. The habitual diets of the toll dwellers from both Pukapuka and Tokelau are high in saturated fat but low in dietary cholesterol and sucrose. Coconut is the chief source of energy for both groups. Tokelauans obtain a much higher percentage of energy from coconut than the Pukapukans, 63% compared with 34%, so their intake of saturated fat is higher. The serum cholesterol levels are 35 to 40 mg higher in Tokelauans than in Pukapukans. These major differences in serum cholesterol levels are considered to be due to the higher saturated fat intake of the Tokelauans. Analysis of a variety of food samples, and human fat biopsies show a high lauric (12:0) and myristic (14:0) content. Vascular disease is uncommon in both populations and there is no evidence of the high saturated fat intake having a harmful effect in these populations.

Am J Clin Nutr. 1981 Aug;34(8):1552-61.

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### **The role of coconut and coconut oil in coronary heart disease in Kerala, south India.**

**Kumar PD.**

Department of Medicine, Medical College, Kerala, South India.

Coronary heart disease (CHD) is common in India and, recently, an increase in the incidence of CHD was reported from the South Indian state of Kerala. The traditional Indian diet is low in fat content. The high incidence of CHD in Indians is, therefore, in contrast to western studies that have correlated high fat, saturated fat and cholesterol intake to CHD. Consumption of coconut and coconut oil that contain high amounts of saturated fat and are thought to be strongly atherogenic, are believed to be one of the main reasons for the high incidence of CHD in Kerala. To explore this presumed link, we studied 32 CHD patients and 16 age and sex matched healthy controls. Consumption of coconut and coconut oil was found to be similar in both groups. The groups did not differ in the fat, saturated fat and cholesterol consumption. The results imply no specific role for coconut or coconut oil in the causation of CHD in the present set of Indian patients from Kerala. The exact reason for the high and increasing incidence of CHD among Indians is still unknown.

Publication Types:

- Clinical Trial
- Controlled Clinical Trial

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**[Wound management with coconut oil in Indonesian folk medicine]**

[Article in German]

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The medical plants which are used to treat wounds and injuries by the ethnic group of Ngada on Flores, an Eastern Indonesian island, will be presented. Additionally, the coconut oil used to treat wounds and to conserve medicinal plants will be analysed biochemically. The people of Ngada use the following plants for wound treatment: seeds of the betel nut (*Areca catechu* L.), fruits of papaya (*Carica papaya* L.), leaves of the Indian Hydrocotyle (*Centelle asiatica* L.), the rhizome of turmeric (*Curcuma domestica* Val. and *Curcumara xanthorrhiza* Roxb.), leaves of betel (*Piper betel* L.). Coconut oil is particularly useful because of its biochemical structure: unlike olive oil and animal fatty tissue, it consists of short-chained and saturated fatty acids. These qualities in coconut oil prevent it from becoming oxidized and rancid, thus making it suitable for the preservation of medicinal plants and for wound treatment.

Chirurg. 2002 Apr;73(4):387-92.

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**Energy restriction with high-fat diet enriched with coconut oil gives higher UCP1 and lower white fat in rats.**

**Portillo MP, Serra F, Simon E, del Barrio AS, Palou A.**

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**OBJECTIVE:** To investigate the effects of overfeeding on a high fat diet, enriched in coconut oil, and the influence of food restriction on the uncoupling protein (UCP1) expression and on body fat content. **DESIGN AND SUBJECTS:** In experiment I, female Wistar rats were fed ad libitum either a normal-fat diet (control group, C) or a high-fat diet (HF), enriched in coconut oil, for 7 weeks. In experiment II, HF rats after finishing experiment I were fed (for 3 weeks) either the normal-fat diet (group CAHF, Control After High Fat) or food restricted diets which provided 60% of the energy intake of group CAHF: a group fed a low-energy, normal-fat diet (LENF) and another fed a low-energy, high-fat diet (LEHF). **MEASUREMENTS:** Body and fatty depot weights. Food intake. Protein and UCP1 levels of interscapular brown adipose tissue. **RESULTS:** High-fat diet feeding promoted an increase in body fat content, body weight and UCP1 levels. Energy restriction induced similar body weight reduction in groups LENS and LEHF. However, some adipose depots were more strongly reduced in the rats fed the high-fat diet enriched in coconut oil (group LEHF) than in the rats fed the normal-fat diet (Group LENS). Specific UCP1 was 2.0 (group LENS) and 3.4 (group LEHF) times higher than in controls (group CAHF). **CONCLUSION:** The coconut-oil enriched diet is effective in stimulating UCP1 expression during ad libitum feeding and in preventing its down regulation during food restriction, and this goes hand in hand with a decrease of the white fat stores.

**Intestinal adaptation in short bowel syndrome without tube feeding or home parenteral nutrition: report of four consecutive cases.**

**Sales TR, Torres HO, Couto CM, Carvalho EB.**

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Because home total parenteral nutrition (TPN) is not available to most of the Brazilian population, an alternative treatment for short bowel syndrome was evaluated. Four patients ages 40-65 y (mean: 53.75 +/- 10.59), three with mesenteric thrombosis, and one with Crohn's disease were studied. The average length of the remaining small bowel in these patients was 54.5 +/- 6.4 cm; the ileocecal valve was preserved in 3 cases. A progressive step diet was used for intestinal adaptation. Administration of pectin was started at the beginning of the special oral diet (step 1), followed by medium-chain triacylglycerols (MCTs) and complex, nonfermentable sugars (step 2); coconut oil (47% MCTs) and simple sugars (step 3); and long-chain triacylglycerols and lactose (step 4). TPN was interrupted at step 3 or 4 when the energy content of the diet reached 150% of the patient's resting energy expenditure, if serum albumin and weight were stable or increasing, and if the frequency, amount, and consistency of stools remained unchanged. Nutritional follow-up showed that patients responded well to this approach; also, patients returned to their previous professional activities. Thus, enteral formulas were not essential for gastrointestinal adaptation. Home TPN should not be indicated on the basis of strict criteria, but rather when a patient fails to adapt to a progressive, special oral diet.

Publication Types:

- Case Reports

Nutrition. 1998 Jun;14(6):508-12.

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**Results of use of metformin and replacement of starch with saturated fat in diets of patients with type 2 diabetes.**

**Hays JH, Gorman RT, Shakir KM.**

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**OBJECTIVE:** To improve glycemic control by substituting saturated fat for starch, to identify any adverse effect on lipids masked by the extensive use of metformin and lipid-lowering drugs, and to attempt to separate dietary effects from effects of multiple drugs. **METHODS:** We undertook a retrospective review of medical records of patients who completed 1 year of follow-up after dietary prescription. The study subjects included 151 patients in the diet group (whose dietary instructions included high saturated fat but starch avoidance) and 132 historical control subjects (who were allowed unlimited monounsaturated fat but had restriction of starch in their diets). **RESULTS:** Hemoglobin A1c (HbA1c) levels improved in both study groups (-1.4 +/- 0.2% [P<0.001]; 95% confidence interval [CI], -1.9 to -0.9). Use of metformin was associated with a decrease in HbA1c (-0.12 +/- 0.003%/mo [P<0.001]; 95% CI, -0.17 to -0.07). The diet group had

an additional decrease of  $-0.7 \pm 0.2\%$  ( $P < 0.001$ ; 95% CI,  $-1.1$  to  $-0.3$ ). Weight increase was associated with the use of insulin ( $+0.3 \pm 0.07$  kg/mo [ $P < 0.001$ ]; 95% CI,  $0.2$  to  $0.5$ ), sulfonylurea ( $+0.18 \pm 0.06$  kg/mo [ $P < 0.01$ ]; 95% CI,  $0.05$  to  $0.30$ ), and troglitazone ( $+0.7 \pm 0.2$  kg/mo [ $P < 0.005$ ]; 95% CI,  $0.3$  to  $1.2$ ). Although not statistically significant, metformin therapy showed a trend for weight loss ( $-0.14 \pm 0.08$  kg/mo;  $P = 0.07$ ). An additional weight loss was noted in the diet group ( $-2.65 \pm 0.62$  kg [ $P < 0.001$ ]; 95% CI,  $-3.87$  to  $-1.44$ ). Hydroxymethylglutaryl-coenzyme A reductase inhibitor use was associated with reduced total cholesterol level ( $-1.7 \pm 0.6$  mg/dL per month [ $P < 0.005$ ]; 95% CI,  $-2.9$  to  $-0.5$ ). The diet group had an additional decrease of  $-13.0 \pm 4.5$  mg/dL ( $P < 0.001$ ; 95% CI,  $-21.9$  to  $-4.1$ ). No significant effect of the diet on triglyceride, low-density lipoprotein, or high-density lipoprotein levels was detected. CONCLUSION: Addition of saturated fat and removal of starch from a high-monounsaturated fat and starch-restricted diet improved glycemic control and were associated with weight loss without detectable adverse effects on serum lipids.

Endocr Pract. 2002 May-Jun;8(3):177-83.

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## Consumption of a Solid Fat Rich in Lauric Acid Results in a More Favorable Serum Lipid Profile in Healthy Men and Women than Consumption of a Solid Fat Rich in *trans*-Fatty Acids<sup>1</sup>

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Solid fats are used in food manufacturing to provide texture and firmness to foods. Such fats are rich in either saturated or *trans*-fatty acids, both of which increase the risk of coronary heart disease. Epidemiological and experimental studies suggest that *trans*-fatty acids increase risk more than do saturates because they lower serum high density lipoprotein (HDL) cholesterol. However, there appear to be differences between saturates in their effect on HDL cholesterol. We investigated whether the consumption of a solid fat rich in lauric acid (C12:0) would result in a more favorable blood lipid profile than the consumption of a solid fat rich in *trans*-fatty acids. We fed 32 healthy men and women two controlled diets in a 2 x 4-wk randomized crossover design. The diets consisted of a background diet supplemented with margarines. In the *trans*-diet, 9.2% of energy was provided by *trans*-fatty acids and 12.9% by saturated fatty acids. In the Sat-diet, energy intake was 0% from *trans*-fatty acids and 22.9% from saturated fatty acids. Lauric acid composed one third of all saturates in the Sat-diet. Serum HDL cholesterol was 0.36 mmol/L lower at the end of the *trans*-diet than at the end of the Sat-diet (95% confidence interval,  $-0.46$  to  $-0.26$ ), whereas serum low density lipoprotein cholesterol and triglyceride concentrations remained stable. Serum total cholesterol was 0.31 mmol/L (95% confidence interval,  $-0.48$  to  $-0.14$ ) lower at the end of the *trans*-diet than at the end

of the Sat-diet. Consumption of a solid fat rich in lauric acid gives a more favorable serum lipoprotein pattern than consumption of partially hydrogenated soybean oil rich in *trans*-fatty acids. Thus, solid fats rich in lauric acids, such as tropical fats, appear to be preferable to *trans*-fats in food manufacturing, where hard fats are indispensable.

[J Nutr.](#) 2001 Feb;131(2):242-5.

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### **Effect of fatty acids on arenavirus replication: inhibition of virus production by lauric acid.**

**Bartolotta S, Garcia CC, Candurra NA, Damonte EB.**

Laboratorio de Virologia, Departamento de Quimica Biologica, Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires, Ciudad Universitaria, Buenos Aires, Argentina.

To study the functional involvement of cellular membrane properties on arenavirus infection, saturated fatty acids of variable chain length (C10-C18) were evaluated for their inhibitory activity against the multiplication of Junin virus (JUNV). The most active inhibitor was lauric acid (C12), which reduced virus yields of several attenuated and pathogenic strains of JUNV in a dose dependent manner, without affecting cell viability. Fatty acids with shorter or longer chain length had a reduced or negligible anti-JUNV activity. Lauric acid did not inactivate virion infectivity neither interacted with the cell to induce a state refractory to virus infection. From mechanistic studies, it can be concluded that lauric acid inhibited a late maturation stage in the replicative cycle of JUNV. Viral protein synthesis was not affected by the compound, but the expression of glycoproteins in the plasma membrane was diminished. A direct correlation between the inhibition of JUNV production and the stimulation of triacylglycerol cell content was demonstrated, and both lauric-acid induced effects were dependent on the continued presence of the fatty acid. Thus, the decreased insertion of viral glycoproteins into the plasma membrane, apparently due to the increased incorporation of triacylglycerols, seems to cause an inhibition of JUNV maturation and release.

[Arch Virol.](#) 2001;146(4):777-90.

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### **Consumption of an oil composed of medium chain triacylglycerols, phytosterols, and N-3 fatty acids improves cardiovascular risk profile in overweight women.**

**Bourque C, St-Onge MP, Papamandjaris AA, Cohn JS, Jones PJ.**

School of Dietetics and Human Nutrition, McGill University, Ste-Anne-de-Bellevue, Quebec, Canada.

Medium chain triacylglycerols (MCT) have been suggested as efficacious in weight management because they possess greater thermogenic qualities relative to long chain triacylglycerols; however, MCT may also increase circulating lipid concentrations, possibly increasing risk of cardiovascular disease (CVD). The present objective was to examine the effect of a diet supplemented with a functional oil (FctO) composed of energy expenditure-enhancing MCT (50% of fat), cholesterol-lowering phytosterols (22 mg/kg body weight), and triacylglycerol-suppressing n-3 fatty acids (5% of fat), versus a

beef tallow-based diet (BT), on plasma lipid and aminothiols concentrations. In a randomized, single-blind, crossover design, partially-inpatient trial, 17 overweight women consumed each oil as part of a controlled, supervised, targeted energy balance diet for 27 days, with 4 or 8 weeks of washout between phases. Mean plasma total cholesterol concentration was lower ( $P < .0001$ ), by 9.1%, on FctO (4.37  $\pm$  0.20 mmol/L) versus BT (4.80  $\pm$  0.20 mmol/L). Mean plasma low-density lipoprotein (LDL) cholesterol was also lower ( $P < .0001$ ) following FctO (2.39  $\pm$  0.15 mmol/L) versus BT (2.86  $\pm$  0.16 mmol/L), representing a 16.0% difference between diets. High-density lipoprotein (HDL) cholesterol and circulating triacylglycerol concentrations remained unaffected by treatment. Ratios of HDL:LDL and HDL:total cholesterol were higher ( $P < .01$ ) by 22.0% and 11.0%, respectively, on FctO versus BT. Plasma total homocysteine remained unchanged with FctO, but decreased ( $P < .05$ ) with control, hence higher ( $P < .05$ ) end points were observed with FctO (6.95  $\pm$  0.33 micromol/L) versus BT (6.27  $\pm$  0.28 micromol/L). Plasma glutathione increased ( $P < .05$ ) by 0.44 micromol/L with FctO supplementation. In conclusion, despite equivocal effects on homocysteine levels, consumption of a functional oil composed of MCT, phytosterols, and n-3 fatty acids for 27 days improves the overall cardiovascular risk profile of overweight women.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

Metabolism. 2003 Jun;52(6):771-7.

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### **Medium-chain oil reduces fat mass and down-regulates expression of adipogenic genes in rats.**

**Han J, Hamilton JA, Kirkland JL, Corkey BE, Guo W.**

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**OBJECTIVE:** To test the hypothesis that adipose tissue could be one of the primary targets through which medium-chain fatty acids (MCFAs) exert their metabolic influence. **RESEARCH METHODS AND PROCEDURES:** Sprague-Dawley rats were fed a control high-fat diet compared with an isocaloric diet rich in medium-chain triglycerides (MCTs). We determined the effects of MCTs on body fat mass, plasma leptin and lipid levels, acyl chain composition of adipose triglycerides and phospholipids, adipose tissue lipoprotein lipase activity, and the expression of key adipogenic genes. Tissue triglyceride content was measured in heart and gastrocnemius muscle, and whole body insulin sensitivity and glucose tolerance were also measured. The effects of MCFAs on lipoprotein lipase activity and adipogenic gene expression were also assessed in vitro using cultured adipose tissue explants or 3T3-L1 adipocytes. **RESULTS:** MCT-fed animals had smaller fat pads, and they contained a considerable amount of MCFAs in both triglycerides and phospholipids. A number of key adipogenic genes were down-regulated, including peroxisome proliferator activated receptor gamma and CCAAT/enhancer binding protein alpha and their downstream metabolic target genes. We also found reduced adipose tissue lipoprotein lipase activity and improved insulin sensitivity and glucose tolerance in MCT-fed animals. Analogous effects of MCFAs on adipogenic genes were found in cultured rat adipose tissue explants and 3T3-L1

adipocytes. DISCUSSION: These results suggest that direct inhibitory effects of MCFAs on adiposity may play an important role in the regulation of body fat development.

Obes Res. 2003 Jun;11(6):734-44.

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**An enteral therapy containing medium-chain triglycerides and hydrolyzed peptides reduces postprandial pain associated with chronic pancreatitis.**

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**BACKGROUND/AIM:** Pain in patients with chronic pancreatitis is difficult to manage. We examined if an enteral formulation containing medium-chain triglycerides (MCT) and hydrolyzed peptides would (1) minimally stimulate the exocrine pancreas by blunting cholecystokinin release and (2) decrease pain in patients with chronic pancreatitis.

**METHODS:** In the first part of the study, on separate days, 6 healthy controls consumed a standard enteral formulation, an enteral formulation containing MCT and hydrolyzed peptides, and a high-fat meal. Baseline and postprandial plasma cholecystokinin (CCK) concentrations were analyzed. Subsequently, 8 patients with chronic pancreatitis were enrolled and instructed to complete a visual analog pain assessment for a baseline period of 2 weeks followed by three cans per day of the enteral formulation containing MCT and hydrolyzed peptides for 10 weeks. **RESULTS:** Mean CCK levels for our control subjects were 0.46 +/- 0.29 pM at baseline, 10.75 +/- 0.45 pM in response to the high-fat meal, and 7.9 +/- 1.25 pM in response to the standard enteral formulation. Of note, CCK levels were 1.43 +/- 0.72 pM in response to the enteral supplement containing MCT and hydrolyzed peptides. In patients with chronic pancreatitis, the average improvement in pain scores from baseline to the conclusion of the study was 61.8% (p = 0.01). This corresponded to a clinical improvement in 6 of the 8 patients. **CONCLUSIONS:** A complete enteral supplement containing MCT and hydrolyzed peptides minimally increases plasma CCK levels. This therapy may be effective in reducing postprandial pain associated with chronic pancreatitis. Copyright 2003 S. Karger AG, Basel and IAP

Pancreatology. 2003;3(1):36-40.

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**Protective effects of medium-chain triglycerides on the liver and gut in rats administered endotoxin.**

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**OBJECTIVE:** To determine if medium-chain triglycerides (MCTs) prevent organ injuries and mortality in rats administered endotoxin and to investigate effects of MCT on the gut.

**SUMMARY BACKGROUND DATA:** Since dietary MCTs prevent alcohol-induced liver injury by inhibiting activation of Kupffer cells in the enteral feeding model, the authors hypothesized that MCT could prevent deleterious conditions in endotoxemia.

**METHODS:** After a preliminary experiment determined the optimal dose of MCT, rats were given MCT (5 g/kg per day) or the same dose of corn oil by gavage daily for 1 week. Then, lipopolysaccharide (LPS) was administered intravenously and survival was

assessed for the next 24 hours. For analysis of mechanisms, rats were killed 9 hours after LPS injection and serum and liver sections were collected. To investigate effects of MCT on the gut, pathologic change, permeability, and microflora were assessed. Kupffer cells isolated by collagenase digestion and differential centrifugation were used for endotoxin receptor CD14 immunoblotting, phagocytic index, and TNF-alpha production assay. RESULTS: All rats given corn oil died after LPS administration; however, this mortality was prevented by MCT in a dose-dependent manner. Rats given corn oil showed liver injury after LPS administration. In contrast, MCT prevented this pathologic change nearly completely. MCT blunted CD14 expression on the Kupffer cells and TNF-alpha production by isolated Kupffer cells; however, there were no differences in phagocytic index between the two groups. The length of the intestinal epithelium was increased in the MCT group compared to the corn oil group. Further, after LPS administration, increases in gut permeability and injury were prevented by MCT. Importantly, MCT also prevented hepatic energy charge and gut injuries in this condition. CONCLUSIONS: Enteral feeding using MCT could be a practical way of protecting the liver and intestine during endotoxemia.

Publication Types:

- Evaluation Studies

Ann Surg. 2003 Feb;237(2):246-55.

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### **Medium-chain triglycerides increase energy expenditure and decrease adiposity in overweight men.**

**St-Onge MP, Ross R, Parsons WD, Jones PJ.**

School of Dietetics and Human Nutrition, McGill University, Ste-Anne-de-Bellevue, Quebec, Canada.

**OBJECTIVE:** The objectives of this study were to compare the effects of diets rich in medium-chain triglycerides (MCTs) or long-chain triglycerides (LCTs) on body composition, energy expenditure, substrate oxidation, subjective appetite, and ad libitum energy intake in overweight men. **RESEARCH METHODS AND PROCEDURES:** Twenty-four healthy, overweight men with body mass indexes between 25 and 31 kg/m<sup>2</sup> consumed diets rich in MCT or LCT for 28 days each in a crossover randomized controlled trial. At baseline and after 4 weeks of each dietary intervention, energy expenditure was measured using indirect calorimetry, and body composition was analyzed using magnetic resonance imaging. **RESULTS:** Upper body adipose tissue (AT) decreased to a greater extent ( $p < 0.05$ ) with functional oil (FctO) compared with olive oil (OL) consumption (-0.67 +/- 0.26 kg and -0.02 +/- 0.19 kg, respectively). There was a trend toward greater loss of whole-body subcutaneous AT volume ( $p = 0.087$ ) with FctO compared with OL consumption. Average energy expenditure was 0.04 +/- 0.02 kcal/min greater ( $p < 0.05$ ) on day 2 and 0.03 +/- 0.02 kcal/min (not significant) on day 28 with FctO compared with OL consumption. Similarly, average fat oxidation was greater ( $p = 0.052$ ) with FctO compared with OL intake on day 2 but not day 28. **DISCUSSION:** Consumption of a diet rich in MCTs results in greater loss of AT compared with LCTs, perhaps due to increased energy expenditure and fat oxidation observed with MCT intake. Thus, MCTs may be considered as agents that aid in the prevention of obesity or potentially stimulate weight loss.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

Obes Res. 2003 Mar;11(3):395-402.

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**Larger diet-induced thermogenesis and less body fat accumulation in rats fed medium-chain triacylglycerols than in those fed long-chain triacylglycerols.**

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It has been previously shown that a diet containing medium-chain triacylglycerols (MCT) leads to less body fat accumulation as compared to a diet containing long-chain triacylglycerols (LCT). We investigated the involvement of diet-induced thermogenesis in the accumulation of body fat in rats fed a diet containing MCT. Twelve male Wistar rats were administered 1 g of MCT or LCT by gavage, and their oxygen consumption was measured for 6 h (experiment 1). Forty male Wistar rats were fed a diet containing 10% MCT or LCT for 6 wk, and their body composition was determined (experiment 2). In experiment 1, oxygen consumption increased to a greater extent after MCT administration than after LCT administration. Diet-induced thermogenesis was significantly (0.67 +/- 0.14 kcal) larger after the administration of 1 g of MCT. In experiment 2, there were no differences in food intake or carcass protein content between the LCT group and MCT group. However, carcass fat and intra-abdominal fat content were significantly lower in rats fed MCT than in those fed LCT. We calculated that ingestion of 1 g of MCT decreased body fat by 0.94 +/- 0.27 kcal relative to the ingestion of LCT. These results suggest that the larger diet-induced thermogenesis observed in rats fed MCT, compared to that of those fed LCT, is one of the main factors involved in the suppression of body fat accumulation in rats fed MCT.

J Nutr Sci Vitaminol (Tokyo). 2002 Dec;48(6):524-9.

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**Comparison of diet-induced thermogenesis of foods containing medium-versus long-chain triacylglycerols.**

**Kasai M, Nosaka N, Maki H, Suzuki Y, Takeuchi H, Aoyama T, Ohra A, Harada Y, Okazaki M, Kondo K.**

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The purpose of this study was to investigate the effect of 5-10 g of medium-chain triacylglycerols (MCT) on diet-induced thermogenesis in healthy humans. The study compared diet-induced thermogenesis after ingestion of test foods containing MCT and long-chain triacylglycerols (LCT), using a double-blind, crossover design. Eight male and eight female subjects participated in study 1 and study 2, respectively. In both

studies, the LCT was a blend of rapeseed oil and soybean oil. In study 1, the liquid meals contained 10 g MCT (10M), a mixture of 5 g MCT and 5 g LCT (5M5L), and 10 g LCT (10L). In study 2, the subjects were given a meal (sandwich and clear soup) with the mayonnaise or margarine containing 5 g of MCT or LCT. Postprandial energy expenditure was measured by indirect calorimetry before and during the 6 h after ingestion of the test meals. Diet-induced thermogenesis was significantly greater after 5M5L and 10M Ingestion as compared to 10L ingestion. Ingestion of the mayonnaise or margarine containing 5 g MCT caused significantly larger diet-induced thermogenesis as compared to that of LCT. These results suggest that, in healthy humans, the intake of 5-10 g of MCT causes larger diet-induced thermogenesis than that of LCT, irrespective of the form of meal containing the MCT.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

J Nutr Sci Vitaminol (Tokyo). 2002 Dec;48(6):536-40.

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### **Enteral nutrition in Crohn's disease: fat in the formula.**

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Enteral nutrition is effective in inducing remission in active Crohn's disease. Speculation on the underlying mechanism of action has moved away from the presentation of nitrogen and towards the fat content of the various enteral feeds. Evidence is accumulating that additional long-chain triglyceride in such feeds impairs the response rate in active Crohn's disease, whereas no deleterious effects of additional medium-chain triglyceride have been identified. It has been proposed that long-chain triglycerides composed from n-6 fatty acids may be the most harmful, since such fatty acids are substrates for inflammatory eicosanoid production. However, recent studies comparing different enteral feeds are not consistent in identifying which additional fatty acids impair response rates to the greatest extent. Despite meta-analyses concluding that polymeric diets (typically containing large amounts of fat) are as effective as elemental diets, it would seem sensible to use enteral feeds with minimal fat content when treating active Crohn's disease.

Publication Types:

- Comment

Eur J Gastroenterol Hepatol. 2003 Feb;15(2):115-8.

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### **The influence of a preserved colon on the absorption of medium chain fat in patients with small bowel resection.**

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**BACKGROUND:** Medium chain C8-C10 triglycerides (MCTs) improve fat absorption in short bowel patients. Effects on overall energy absorption remain unknown. **AIMS:** To determine whether MCTs and medium chain fatty acids (MCFAs) are absorbed in the colon like the short chain fatty acids (SCFAs) or are lost in faeces similarly to long chain fatty acids (LCFAs). **METHODS:** Nine small bowel resected patients without and 10 with a colon in continuity excreted 2-6 MJ/day and were randomised and crossed over between two high fat diets (10 MJ/day, 50% as fat), based on either long chain triglycerides (LCT) alone or equal quantities of LCT and MCT. **RESULTS:** Patients with a colon absorbed C8-C10 fatty acids considerably better than patients without a colon at similar and extreme levels of LCFA malabsorption; the colonic impact on absorption of C14-18 fatty acids was negligible. MCT redoubled fat (MCT+LCT) absorption from 23% to 58% in patients with a colon, and increased overall bomb calorimetric energy absorption from 46% to 58%. The increase in fat absorption from 37% to 46% in patients without a colon did not improve overall energy absorption because malabsorption of carbohydrate and protein increased. **CONCLUSION:** In small bowel resected patients, the colon seems to serve as a digestive organ for medium chain fat, probably absorbed as MCFAs, perhaps because like the SCFAs, they are water soluble. Only patients with a colon gained from MCT treatment.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

Gut. 1998 Oct;43(4):478-83.

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### **Effects of intravenous supplementation with alpha-tocopherol in patients receiving total parenteral nutrition containing medium- and long-chain triglycerides.**

**Manuel-y-Keenoy B, Nonneman L, De Bosscher H, Vertommen J, Schrans S, Klutsch K, De Leeuw I.**

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**OBJECTIVE:** To compare the effects of a lipid emulsion containing medium-chain triglycerides (MCT) and supplemented with alpha-tocopherol to a conventional long-chain triglyceride (LCT) emulsion. **DESIGN:** Randomised double blind study. **SETTING:** Department of Internal Medicine, Antwerp University Hospital. **SUBJECTS AND INTERVENTIONS:** Twenty-four patients with an indication for total parenteral nutrition for a minimum of 10 days were randomly assigned to two groups: group E received as lipid source MCT/LCT (50/50) supplemented with 100 mg DL-alpha-tocopherol/day and group C received LCT. Blood samples were analysed at inclusion, after 4-6 and after 9-11 days. **RESULTS:** In group E, serum alpha-tocopherol doubled from 11.4+/-6.9 at inclusion to 20.9+/-7.9 and to 23.8+/-8.8 microg/ml after 4 and 9 days, respectively, but did not change in group C (P=0.008). Production of thiobarbituric acid-reacting substances (TBARS) after 120 min incubation with copper decreased from 66+/-34 at inclusion to 29+/-25 nmol MDA/mg LDL and VLDL-cholesterol after 4 and to

42+/-17 after 9 days (P=0.022 when compared to group C, which underwent no significant changes). Velocity of production of fluorescent products decreased in group E but not in group C (P=0.026). CONCLUSIONS: Supplementation of TPN containing MCT/LCT with 100 mg DL-alpha-tocopherol/day leads to a doubling in serum alpha-tocopherol and to a decrease in the susceptibility of LDL and VLDL to peroxidation in vitro. SPONSORSHIP: This study was partly financed by B Braun Medical NVSA, Diegem, Belgium.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

Eur J Clin Nutr. 2002 Feb;56(2):121-8.

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### **Killing of Gram-positive cocci by fatty acids and monoglycerides.**

**Bergsson G, Arnfinnsson J, Steingrimsen O, Thormar H.**

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The susceptibilities of three Gram-positive cocci to medium-chain saturated and long-chain unsaturated fatty acids and their one-monoglycerides were studied. The bacteria were incubated with equal volumes of lipid solutions for 10 min. Lauric acid, palmitoleic acid and monocaprin reduced the number of CFU by 6.0 log<sub>10</sub> or greater at 5 mM concentration for streptococci of group A (GAS) and group B (GBS). When further compared at lower concentrations and after longer incubation time monocaprin proved to be the most active. Capric acid showed the highest activity against *Staphylococcus aureus* at 10 mM. However, at lower concentrations monocaprin was the only lipid that showed significant activity against *S. aureus*. The mode of action of monocaprin against GBS was studied by a novel two-color fluorescent assay of bacterial viability and by electron microscopy. The results indicate that the bacteria are killed by disintegration of the cell membrane by the lipid, leaving the bacterial cell wall intact. The highly lethal effect of monocaprin indicates that this lipid might be useful as a microbicidal agent for prevention and treatment of infections caused by these bacteria.

APMIS. 2001 Oct;109(10):670-8.

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### **Physiological effects of medium-chain triglycerides: potential agents in the prevention of obesity.**

**St-Onge MP, Jones PJ.**

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Medium chain fatty acids (MCFA) are readily oxidized in the liver. Animal and human studies have shown that the fast rate of oxidation of MCFA leads to greater energy expenditure (EE). Most animal studies have also demonstrated that the greater EE with MCFA relative to long-chain fatty acids (LCFA) results in less body weight gain and decreased size of fat depots after several months of consumption. Furthermore, both animal and human trials suggest a greater satiating effect of medium-chain triglycerides

(MCT) compared with long-chain triglycerides (LCT). The aim of this review is to evaluate existing data describing the effects of MCT on EE and satiety and determine their potential efficacy as agents in the treatment of human obesity. Animal studies are summarized and human trials more systematically evaluated because the primary focus of this article is to examine the effects of MCT on human energy metabolism and satiety. Hormones including cholecystokinin, peptide YY, gastric inhibitory peptide, neurotensin and pancreatic polypeptide have been proposed to be involved in the mechanism by which MCT may induce satiety; however, the exact mechanisms have not been established. From the literature reviewed, we conclude that MCT increase energy expenditure, may result in faster satiety and facilitate weight control when included in the diet as a replacement for fats containing LCT.

Publication Types:

- Review
- Review, Tutorial

J Nutr. 2002 Mar;132(3):329-32.

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### **Value of VLCD supplementation with medium chain triglycerides.**

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**BACKGROUND:** Medium chain triglycerides (MCT) are energetically less dense, highly ketogenic, and more easily oxidised than long chain triglycerides (LCT). MCT also differ from LCT in their digestive and metabolic pathways. **OBJECTIVE:** To test the effects of MCT supplementation during a very low calorie diet (VLCD). **SUBJECTS AND METHODS:** Three groups of tightly matched obese women with body mass index (BMI)>30 kg/m<sup>2</sup> received an isoenergetic (578.5 kcal) VLCD (Adinax, Novo Vital, Sweden) enriched with MCT or LCT (8.0 and 9.9 g/100 g Adinax respectively) or a low-fat (3 g/100 g) and high-carbohydrate regimen. The diets were administered over 4 weeks. Body composition was measured with DEXA and appetite/satiety-according to Blundell. Beta hydroxybutyric acid concentration in plasma and nitrogen excretion in urine was measured during consecutive days of VLCD. The study was performed in a randomised double-blind manner. **RESULTS:** The MCT group showed a significantly greater decrease in body weight during the first 2 weeks. The contribution of body fat to the total weight loss was higher while the contribution of fat-free mass (FFM) was lower. The MCT group had a higher concentration of ketone bodies in plasma and a lower nitrogen excretion in urine. Hunger feelings were less intense while satiety was higher. These differences were observed during the first 2 weeks of treatment and gradually declined during the third and fourth weeks. **CONCLUSIONS:** Replacement of LCT by MCT in the VLCD increased the rate of decrease of body fat and body weight and has a sparing effect on FFM. The intensity of hunger feelings was lower and paralleled the higher increase of ketone bodies. These effects gradually declined, indicating subsequent metabolic adaptation. Further studies are required to confirm the protein-sparing and appetite-suppressing effects of MCT supplementation during the first 2 weeks of VLCD treatment.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

Int J Obes Relat Metab Disord. 2001 Sep;25(9):1393-400.

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### **In vitro killing of *Candida albicans* by fatty acids and monoglycerides.**

**Bergsson G, Arnfinnsson J, Steingrímsson O, Thormar H.**

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The susceptibility of *Candida albicans* to several fatty acids and their 1-monoglycerides was tested with a short inactivation time, and ultrathin sections were studied by transmission electron microscopy (TEM) after treatment with capric acid. The results show that capric acid, a 10-carbon saturated fatty acid, causes the fastest and most effective killing of all three strains of *C. albicans* tested, leaving the cytoplasm disorganized and shrunken because of a disrupted or disintegrated plasma membrane. Lauric acid, a 12-carbon saturated fatty acid, was the most active at lower concentrations and after a longer incubation time.

Antimicrob Agents Chemother. 2001 Nov;45(11):3209-12.

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### **Effects of different lipid sources in total parenteral nutrition on whole body protein kinetics and tumor growth.**

**Mendez B, Ling PR, Istfan NW, Babayan VK, Bistrian BR.**

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This study examined the short-term effects of three total parenteral nutrition solutions, each containing a different lipid source, on host and tumor protein metabolism in a rat cancer model. Each diet contained 220 kcal/kg per day, including 2 g of nitrogen/kg per day and 50% of nonprotein calories as either a structured lipid of medium-chain triglycerides and fish oil, a physical mix of medium-chain triglycerides and fish oil, or Liposyn II, a long-chain triglyceride. A 3-day intravenous feeding infusion began on day 7 after tumor implantation. Tumor growth rate, nitrogen balance, energy expenditure, and plasma albumin, glucose, and free fatty acids were measured, and whole body protein kinetics and fractional synthetic rates in liver, muscle, and tumor tissues were assessed using a constant infusion of <sup>14</sup>C-leucine. The results revealed that tumor growth rate was slowed in structured lipid-fed animals ( $p = .06$ , one-way analysis of variance) with significant increases in rates of tumor protein synthesis and tumor protein breakdown ( $p < .001$ , one-way analysis of variance). Although muscle fractional synthetic rates were significantly decreased in tumor-bearing animals ( $p < .05$ , two-way analysis of variance), the rates in structured lipid-fed animals were restored. Nitrogen balance improved significantly in structured lipid-fed animals. The results demonstrate that the source of lipid in total parenteral nutrition solutions can influence tumor and host protein metabolism, and that a structured lipid composed of medium-chain triglycerides and fish oil seems to improve protein metabolism in host tissue without stimulating tumor growth.

**Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium chain triglyceride.**

**Baba N, Bracco EF, Hashim SA.**

The mechanism whereby overfeeding with diet containing medium chain triglyceride (MCT) results in diminished body weight and fat was studied. Fifteen male Sprague-Dawley rats were fitted under anesthesia with gastrostomy tubes and divided into two groups. One group was fed MCT diet, the other an isocaloric diet containing long chain triglyceride (LCT) in excess (150%) of spontaneous calorie intake. Both diets, fed for 6 wk, derived 50% of calories from fat. Basal and norepinephrine (25 micrograms/100 g) stimulated O<sub>2</sub> consumption and CO<sub>2</sub> production, as well as metabolic rate were measured. After the rats were killed, total dissectible fat and fat cell size and number were determined. MCT rats gained 15% less weight than LCT controls (p less than 0.001). Total dissectible fat was significantly lower (p less than 0.001) in MCT group, as was mean adipocyte size (p less than 0.001). Resting and maximal norepinephrine-stimulated O<sub>2</sub> consumptions were 39.7 and 22.1% higher in MCT than in LCT group, respectively. Resting and norepinephrine-stimulated metabolic rates were 38.8 and 22.2% higher in MCT than LCT fed rats, respectively. Overfeeding MCT diet results in decreased body fat related to increased metabolic rate and thermogenesis.

Am J Clin Nutr. 1982 Apr;35(4):678-82.

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**Thermogenesis in humans during overfeeding with medium-chain triglycerides.**

**Hill JO, Peters JC, Yang D, Sharp T, Kaler M, Abumrad NN, Greene HL.**

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To test whether excess dietary energy as medium-chain triglycerides (MCT) affects thermogenesis differently from excess dietary energy as long chain triglycerides (LCT), ten male volunteers (ages 22 to 44) were overfed (150% of estimated energy requirement) liquid formula diets containing 40% of fat as either MCT or LCT. Each patient was studied for one week on each diet in a double-blind, crossover design. Resting metabolic rate (RMR) did not change during either week of overfeeding. The thermic response to food (TEF) was greater on day 1 following a meal (1,000 kcal) containing MCT than following an isocaloric meal containing LCT (8 +/- .8% v 5.8 +/- .8% of ingested energy; P less than .05). Moreover, the TEF observed after a 1,000 kcal meal containing MCT increased significantly to 12% (+/- 1.3%) overfeeding. The TEF of the 1,000 kcal meal containing LCT was unchanged by five days of LCT overfeeding (6.6 +/- 1.0% of ingested energy). Energy expenditure during a 20-hour continuous enteral infusion of the diet on day 7 was also significantly higher with the MCT diet than with the LCT diet (15.7 +/- 1.7% v 7.3 +/- .9% of ingested energy; P less than .05). Our results demonstrate that excess dietary energy as MCT stimulates thermogenesis to a greater degree than does excess energy as LCT. This increased energy expenditure, most likely due to lipogenesis in the liver, provides evidence that excess energy derived from MCT is stored with a lesser efficiency than is excess energy derived from dietary LCT.

**Additional References:**

Anti-viral effects of monolaurin. JAQA 1987;2:4-6 7. Issacs CE et al. Antiviral and antibacterial lipids in human milk and infant formula feeds. Archives of Disease in Childhood 1990;65:861-864.

Aveywardena MY and Charnock JS, dietary lipid modification of myocardial eicosanoids following ischemia and reperfusion in the rat, Lipids 1995;30:1151-1156.

Awad AB. Effect of dietary lipids on composition and glucose utilization by rat adipose tissue. Journal of Nutrition 1981;111:34-39.

Bakker N, Van't Veer P, Zock PL. Adipose fatty acids and cancers of the breast, prostate and colon: an ecological study. EURAMIC Study Group. International Journal of Cancer 1997;72:587-591.

Bergsson G, Arnfinnsson J, Karlsson SM, Steingrimsdottir O, Thormar H. In vitro inactivation of Chlamydia trachomatis by fatty acids and monoglycerides. Antimicrobial Agents and Chemotherapy 1998;42:2290-2294.

Bibby DC, Grimble RF. Tumour necrosis factor-alpha and endotoxin induce less prostaglandin E2 production from hypothalami of rats fed coconut oil than from hypothalami of rats fed maize oil. Clinical Science (Colch) 1990;79:657-62.

Bierenbaum JL, Green DP, Florin A, Fleishman AI, Caldwell AB. Modified-fat dietary management of the young male with coronary disease: a five-year report. Journal of the American Medical Association 1967;202:1119-1123.

Blackburn GL, Kater G, Mascioli EA, Kowalchuk M, Babayan VK, Kibarian BR. A reevaluation of coconut oil's effect on serum cholesterol and atherogenesis. The Journal of the Philippine Medical Association 1989;65:144-152.

Boddie, RL and Nickerson, SC. Evaluation of postmilking teat germicides containing Lauricidin, saturated fatty acids, and lactic acid. Journal of Dairy Science 1992;75:1725-1730.

Castelli WP. Editorial: Concerning the possibility of a nut... Archives of Internal Medicine 1992;152:1371-2.

Cha YS, Sachan DS. Opposite effects of dietary saturated and unsaturated fatty acids on ethanol-pharmacokinetics, triglycerides and carnitines. Journal of the American College of Nutrition 1994;13:338-343.

Chen A, Li W, Yang Y. [Detection of human cytomegalovirus DNA in vascular plaques of atherosclerosis by in situ hybridization] (translation from Chinese). Chung Hua I Hsueh Tsa Chih 1995;10:592-593, 638.

Chipley, J.R., Todd, P.T., Atchley, F. and Kabara, J.J. Effects of Fatty Acid Derivatives on the Release of Extracellular Enzymes from Bacteria. In: The Pharmacological Effect of Lipids 11, Jon J. Kabara, ed. American Oil Chemists' Society: Champaign, Illinois, pp. 97-102 (1985).

Chipley. J R . Story. L.D.. Todd, P,T. and Kabara, J.J. Inhibition of Aspergillus Growth and Extracellular Aflatoxin Accumulation by Sorbic Acid and Derivatives of Fatty acids. *J. Food Safety* 3:109-119 (1981).

Cleary MP, Phillips FC, Morton RA. Genotype and diet effects in lean and obese Zucker rats fed either safflower or coconut oil diets. *Proceedings of the Society for Experimental Biology and Medicine* 1999;220:153-161.

Clevidence BA, Judd JT, Schaefer EJ, Jenner JL, Lichtenstein AH, Muesing RA, Wittes J, Sunkin ME. Plasma lipoprotein (a) levels in men and women consuming diets enriched in saturated, cis-, or trans-monounsaturated fatty acids. *Arterioscler Thromb Vasc Biol* 1997;17:1657-1661.

Cohen LA, Thompson DO, --Choi K, Blank M, Rose DP. Dietary fat and mammary cancer. II. Modulation of serum and tumor lipid composition and tumor prostaglandins by different dietary fats: Association with tumor incidence patterns. *Journal of the National Cancer Institute* 1986;77:43.

Cohen LA, Thompson DO, M--aeura Y, Choi K, Blank M, Rose DP. Dietary fat and mammary cancer. I. Promoting effects of different dietary fats on N-nitrosomethylurea-induced rat mammary tumorigenesis. *Journal of the National Cancer Institute* 1986;77:33.

Conley. A J and Kabara. J.J. Antimicrobial Action of Esters of Polyhydric Alcohols. *Antimicrob. Ag and Chemother* 4:501-506 (1973)

Crouch AA, Seow WK, Whitman LM, Thong YH. Effect of human milk and infant milk formulae on adherence of *Giardia intestinalis*. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 1991;85:617-619.

Dave JR, Koenig ML, Tortella FC, Pieringer RA, Doctor BP, Ved HS. Dodecylglycerol provides partial protection against glutamate toxicity in neuronal cultures derived from different regions of embryonic rat brain. *Molecular Chemistry and Neuropathology* 1997;30:1-13.

Dodge JA and Sagher FA. Antiviral and antibacterial lipids in human milk and infant formula. *Archives of Disease in Childhood* 1991;66:272-273.

Ellis RW. Infection and coronary heart disease. *Journal of Medical Microbiology* 1997;46:535-539.

Enig MG, Atal S, Sampugna J and Keeney M. Isomeric Trans Fatty Acids in the U.S. Diet. *Journal of the American College of Nutrition* 1990;9:471-486.

Enig MG. Diet, serum cholesterol and coronary heart disease, in Mann GV (ed): *Coronary Heart Disease: The Dietary Sense and Nonsense*. Janus Publishing, London, 1993, pp 36-60.

Enig, MG. Lauric oils as antimicrobial agents: theory of effect, scientific rationale, and dietary applications as adjunct nutritional support for HIV-infected individuals. in *Nutrients and Foods in AIDS* (RR Watson, ed) CRC Press, Boca Raton, 1998, pp. 81-97.

Epstein SE, Speir E, Zhou YF, Guetta E, Leon M, Finkel T. The role of infection in restenosis and atherosclerosis: focus on cytomegalovirus. *Lancet* 1996;348 Supplement 1:S13-17.

Eraly MG. IV. Coconut oil and heart attack. Coconut and Coconut Oil in Human Nutrition, Proceedings. Symposium on Coconut and Coconut Oil in Human Nutrition. 27 March 1994. Coconut Development Board, Kochi, India, 1995, pp 63-64.

Felton CV, Crook D, Davies MJ, Oliver MF. Dietary polyunsaturated fatty acids and composition of human aortic plaques. *Lancet*, 1994;344:1195-1196.

Fletcher RD, Albers AC, Albertson JN, Kabara JJ. Effects of monoglycerides on mycoplasma pneumoniae growth, in *The Pharmacological Effect of Lipids II* (JJ Kabara, ed) American Oil Chemists' Society, Champaign IL, 1985, pp.59-63.

Florentino RF, Aquinaldo AR. Diet and cardiovascular disease in the Philippines. *The Philippine Journal of Coconut Studies* 1987;12:56-70.

Flournoy, D.J. and Kabara, J.J. The Role of Lauricidin® as an Antimicrobial Agent. In: *Drugs of Today* 21(8):373-377 (1985).

Garfinkel M, Lee S, Opara EC, Akkwari OE. Insulinotropic potency of lauric acid: a metabolic rationale for medium chain fatty acids (MCF) in TPN formulation. *Journal of Surgical Research* 1992;52:328-333.

Gerster H. Can adults adequately convert alpha-linolenic acid (18:3n-3) to eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3)? *International Journal of Vitamin and Nutrition Research* 1998;68:159-173.

Gottesman S. Making Sense of Shortenings. *Baking Buyer* August 1998, p.45-49.

Grundy SM. Cholesterol metabolism in man, *Western Journal of Medicine* 128:13;1978.  
Halden VW, Lieb H. Influence of biologically improved coconut oil products on the blood cholesterol levels of human volunteers. *Nutr Dieta* 1961;3:75-88.

Hargrove JL, Hwang J, Wickwire K, Liu J. Diets with corn oil or soybean oil increase acute acetaminophen hepatotoxicity compared to diets with beef tallow. *The FASEB Journal* 1999;13:A222, Abstract 204.1.

Hashim SA, Clancy RE, Hegsted DM, Stare FJ. Effect of mixed fat formula feeding on serum cholesterol level in man. *American Journal of Clinical Nutrition*. 1959;7:30-34.

Hegsted DM, McGandy RB, Myer ML, Stare FJ. Quantitative effects of dietary fat on serum cholesterol in man. *American Journal of Clinical Nutrition*. 1965;17:281-295.

Hernell O, Ward H, Blackberg L, Pereira ME. Killing of *Giardia lamblia* by human milk lipases: an effect mediated by lipolysis of milk lipids. *Journal of Infectious Diseases* 1986;153:715-720.

Hierholzer, J.C. and Kabara, J.J. In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses. *Journal of Food Safety* 1982;4:1-12.

Hierholzer, J.C. and Kabara, J.J. In Vitro Effects of Monolaurin Compounds on Enveloped RNA and DNA Viruses. *J. Of Food Safety* 4:1-12 (1982).

Hodgson JM, Wahlqvist ML, Boxall JA, and Balazs ND. Can linoleic acid contribute to coronary artery disease? *American Journal of Clinical Nutrition* 1993;58:228-234.

Holland KT, Taylor D, Farrell AM. The effect of glycerol monolaurate on growth of, and production of toxic shock syndrome toxin-1 and lipase by, *Staphylococcus aureus*. *Journal of Anti-microbial Chemotherapy* 1994;33:41-55.

Homung B, Arntmann E, Sauer G. Lauric acid inhibits the maturation of vesicular stomatitis virus. *Journal of General Virology* 1994;75:353-361.

Hornstra G, van Houwelingen AC, Kester AD, and Sundram K. A palm oil-enriched diet lowers serum lipoprotein(a) in normocholesterolemic volunteers. *Atherosclerosis* 1991;90:91-93.

Hornung B, Arntmann E, Sauer G. Lauric acid inhibits the maturation of vesicular stomatitis virus. *Journal of General Virology* 1994;75:353-361.

Hostmark AT, Spydevold O, Eilertsen E. Plasma lipid concentration and liver output of lipoproteins in rats fed coconut fat or sunflower oil. *Artery* 1980;7:367-383.

Huang SC, Frische KL. Alteration in mouse splenic phospholipid fatty acid composition and lymphoid cell populations by dietary fat. *Lipids* 1992;27:25-32.

Isaacs CE, Kashyap S, Heird WC, Thormar H. Antiviral and antibacterial lipids in human milk and infant formula feeds. *Archives of Disease in Childhood* 1990;65:861-864.

Isaacs CE, Kim KS, Thormar H. Inactivation of enveloped viruses in human bodily fluids by purified lipids. *Annals of the New York Academy of Sciences* 1994;724:457-464.

Isaacs CE, Litov RE, Marie P, Thormar H. Addition of lipases to infant formulas produces antiviral and antibacterial activity. *Journal of Nutritional Biochemistry* 1992;3:304-308.

Isaacs CE, Schneidman K. Enveloped Viruses in Human and Bovine Milk are Inactivated by Added Fatty Acids(FAs) and Monoglycerides(MGs). *FASEB Journal* 1991;5: Abstract 5325, p.A1288.

Isaacs CE, Schneidman K. Enveloped Viruses in Human and Bovine Milk are Inactivated by Added Fatty Acids (FAs) and Monoglycerides (MGs). *FASEB Journal* 1991;5: Abstract 5325, p. A1288.

Isaacs CE, Thormar H. Human milk lipids inactivated enveloped viruses. in *Breastfeeding, Nutrition, Infection and Infant Growth in Developed and Emerging Countries* (Atkinson SA, Hanson LA, Chandra RK, eds) Arts Biomedical Publishers and Distributors, St. John's NF, Canada, 1990.

Isaacs CE, Thormar H. Membrane-disruptive effect of human milk: inactivation of enveloped viruses. *Journal of Infectious Diseases* 1986;154:966-971.

Isaacs CE, Thormar H. The role of milk-derived antimicrobial lipids as antiviral and antibacterial agents in *Immunology of Milk and the Neonate* (Mestecky J, et al, eds) Plenum Press, New York, 1991.

Isaacs, C.E. et al. Inactivation of enveloped viruses in human bodily fluids by purified lipids. *Annals of the New York Academy of Sciences* 1994;724:457-464.

Isaacs, CE et al. Membrane-disruptive effect of human milk: inactivation of enveloped viruses. *Journal of Infectious Diseases* 1986;154:966-971.

Jones PJH. Regulation of cholesterol biosynthesis by diet in humans, *American Journal of Clinical Nutrition* 1997;66:438-446.

Judd JT, Clevidence BA, Muesing RA, Wittes J, Sunkin ME, and Podczasy JJ. Dietary Trans Fatty Acids: Effects on Plasma Lipids and Lipoproteins of Healthy Men and Women. *American Journal of Clinical Nutrition* 1994;59:861-868.

Kabara JJ. Fatty acids and derivatives as antimicrobial agents -- A review, in *The Pharmacological Effect of Lipids* (JJ Kabara, ed) American Oil Chemists' Society, Champaign IL, 1978,

Kabara JJ. Inhibition of staphylococcus aureus in *The Pharmacological Effect of Lipids II* (JJ Kabara, ed) American Oil Chemists' Society, Champaign IL, 1985, pp.71-75.

Kabara, J J and Conley. A J A Non-Caloric Role for MCT and Other Lipids In: *Mittelkettige Triglyceride (MCT) in der Diat. Zur Zeitschrih Fur Ernährungswissenschah Supplenta No 17*. H. Kaunitz K Lang and W Fekl, eds pp 17-26 (1974)

Kabara, J.J. Antimicrobial agents derived from fatty acids. *Journal of the American Oil Chemists Society* 1984;61:397-403.

Kabara, J.J. Antimicrobial Compositions. U.S. Patent Number 4,189,481 February 19 1980.

Kabara, J.J. Medium-Chain Fatty Acids and Esters as Antimicrobial Agents. In: *Cosmetic and Drug Presentation: Principles and Practice*, Jon J. Kabara, ed., New York: Marcel Dekker, Inc., pp. 275-304 (1984).

Kabara, J.J. Monolaurin as an Antimicrobial Agent. U.S. Patent Number 4,002,775. Med-Chem Laboratories, January 1977.

Kabara, J.J. Ohkawa, M., Ikekawa, T., Katori, T. and Nishikawa, Y. Examinations on Antitumor Immunological, and Plant-Growth Inhibitory Effects of Monoglycerides of Caprylic, Capric, and Lauric Acids and Related Compounds. In: *The Pharmacological Effect of Lipids 11*, Jon J. Kabara, ed. American Oil Chemists' Society: Champaign, Illinois, pp. 263-272 (1985). Kabara, J.J., Editor. *The Pharmacological Effects of Lipids II*, American Oil Chemists' Society: Champaign, Illinois (1985).

Kabara, J.J. Synergistic Microbiocidal Composition and Method. U.S. Patent Number 4,067,997. January 10, 1978

Kabara, J.J. Toxicological, Bactericidal and Fungicidal Properties of Fatty Acids and Some Derivatives *JAOCS* 56:760-767

Kabara, J.J., editor *The Pharmacological Effect of Lipids I*. American Oil Chemists' Society: Champaign, Illinois (1979).

Kabara, J.J., Editor. *The Pharmacological Effects of Lipids III*, American Oil Chemists' Society: Champaign, Illinois (1989).

Kabara, JJ Fatty Acids and Derivatives as Antimicrobial Agents-A Review. In: *The Pharmacological Effect of Lipids*. Jon J. Kabara, ed Champaign, Illinois: The American Oil Chemists' Society (1979),pp. 1-14

- Kabara. J.J, Swieczkowski, D M. Conley. A J and Truant, J P Fatty Acids and Derivatives as Antimicrobial Agents Antimicrobial Agents and Chemotherapy 2(1):23-28 (1972) Kabara. J.J.. Conley. A J.- Swieczkowski. D M. Ismail, I.A . Lie Ken Jie and Gunstone, F D Antimicrobial Action of Isomeric Fatty Acids on Group A Streptococcus J. Med Chem 16:1060-1063 (1973).
- Kabara. J.J. Antimicrobial Agents Derived From Fatty Acids. J. American Oil Chemists' Society 61:397- 403 (1984).
- Kabara. J.J. Lipids as Host Resistance Factors of Human Milk Nutrition Reviews 38:65-73 (1980).
- Kabara. J.J. Lipids as Safe and Effective Antimicrobial Agents for Cosmetics and Pharmaceuticals. Cosmetics and Perfumery 90:21-25 (1975).
- Kabara. J.J., Vrable, R. and Lie Ken Jie, M.S.F Antimicrobial Lipids: Natural and Synthetic Fatty Acids and Monoglycerides. Lipids 12:753759 (1977).
- Kaunitz H, Dayrit CS. Coconut oil consumption and coronary heart disease. Philippine Journal of Internal Medicine 1992;30:165-171.
- Kaunitz H. Toxic effects of polyunsaturated vegetable oils, in: Symposium on the Pharmacological Effect of Lipids (JJ Kabara, ed) , American Oil Chemists' Society, Champaign, IL, 1978, pp 203-210.
- Keys A, Anderson JT, Grande F. Prediction of serum-cholesterol responses of man to changes in the diet. Lancet, 959;1957.
- Khosla P and Hayes KC. Dietary trans-monounsaturated fatty acids negatively impact plasma lipids in humans: critical review of the evidence. Journal of the American College of Nutrition 1996;15:325-339.
- Kohlmeier L, Simonsen N, van 't Veer P, Strain JJ, Martin-Moreno JM, Margolin B, Huttunen JK, Fernandez-Crehuet Navajas J, Martin BC, Thamm M, Kardinaal AF, Kok FJ. Adipose tissue trans fatty acids and breast cancer in the European Community Multicenter Study on Antioxidants, Myocardial Infarction, and Breast Cancer. Cancer Epidemiology and Biomarkers Prev 1997;6:705-10.
- Kramer JK, Sauer FD, Farnworth ER, Stevenson D, Rock GA. Hematological and lipid changes in newborn piglets fed milk-replacer diets containing erucic acid. Lipids 1998;33:1-10.
- Kurup PA, Rajmohan T. II. Consumption of coconut oil and coconut kernel and the incidence of atherosclerosis. Coconut and Coconut Oil in Human Nutrition, Proceedings. Symposium on Coconut and Coconut Oil in Human Nutrition. 27 March 1994. Coconut Development Board, Kochi, India, 1995, pp 35-59.
- Li, C.Y. and Kabara. J.J. Effects of Lauricidin-on Fornes Annosus and Phellinus Weirii. In The Pharmacological Effect of Lipids. Jon J. Kabara, ed. Champaign, Illionis: The American Oil Chemists' Society (1979). pp. 45-50.
- Lim-Sylianco CY. Anticarcinogenic effect of coconut oil. The Philippine Journal of Coconut Studies 1987;12:89-102.

- Lu Z, Hendrich S, Shen N, White PJ, Cook LR. Low linolenate and commercial soybean oils diminish serum HDL cholesterol in young free-living adult females. *Journal of the American College of Nutrition* 1997;16:562-569.
- Macallan DC, Noble C, Baldwin C, Foskett M, McManus T, Griffin GE. Prospective analysis of patterns of weight change in stage IV human immunodeficiency virus infection. *American Journal of Clinical Nutrition* 1993;58:417-24.
- Mann GV. A short history of the diet/heart hypothesis, in Mann GV (ed): *Coronary Heart Disease: The Dietary Sense and Nonsense*. Janus Publishing, London, 1993, pp 1-17.
- McWhinney VJ, Pond WG, Mersmann HJ. Ontogeny and dietary modulation of 3-hydroxy-3-methylglutaryl-CoA reductase activities in neonatal pigs. *Journal of Animal Science* 1996;74:2203-10.
- Melnick JL, Adam E, DeBakey ME. Cytomegalovirus and atherosclerosis. *Archivum Immunologiae et Therapiae Experimentalis (Wroclaw)* 1996;44:297-302.
- Mendis S, Kumarasunderam R. The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidaemic men. *British Journal of Nutrition* 1990;63:547-52.
- Mendis S, Wissler RW, Bridenstine RT, Podbielski FJ. The effects of replacing coconut oil with corn oil on human serum lipid profiles and platelet derived factors active in atherogenesis. *Nutrition Reports International* 40:No.4;Oct.1989.
- Mensink RP and Katan MB. Effect of Dietary Trans Fatty Acids on High-Density and Low-Density Lipoprotein Cholesterol Levels in Healthy Subjects. *The New England Journal of Medicine* 1990;323:439-445.
- Monserrat AJ, Romero M, Lago N, Aristi C. Protective effect of coconut oil on renal necrosis occurring in rats fed a methyl-deficient diet. *Renal Failure* 1995;17:525-537.
- Nanji AA, Sadrzadeh SM, Yang EK, Fogt F, Maydani M, Dannenberg AJ. Dietary saturated fatty acids: a novel treatment for alcoholic liver disease. *Gastroenterology* 1995;109:547-554.
- Nelson GJ. Dietary fat, trans fatty acids, and risk of coronary heart disease. *Nutrition Reviews* 1998;56:250-252.
- Nelson SE, Rogers RR, Frantz JA, Ziegler EE. Palm olein in infant formula: absorption of fat and minerals by normal infants. *American Journal of Clinical Nutrition* 1996;64:291-296.
- New York Times, Medical Science, Tuesday, January 29, 1991. Common virus seen as having early role in arteries' clogging (byline Sandra Blakeslee).
- Ng TKW, Hassan K, Lim JB, Lye MS, Ishak R. Nonhypercholesterolemic effects of a palm-oil diet in Malaysian volunteers. *American Journal of Clinical Nutrition*, 1991;53:1015S-1020S.
- Oh DH and Marshall DL. Antimicrobial activity of ethanol, glycerol monolaurate or lactic acid against *Listeria monocytogenes*. *International Journal of Food and Microbiology* 1993;20:239-246.

Oliart-Ros RM, Torres-Marquez ME, Badillo A, Guerrero OA. Effects of dietary polyunsaturated fatty acids on sucrose-induced cardiovascular syndrome in rats. 89th AOCS Annual Meeting Abstracts, H&N 5: General Health and Nutrition II, p. 76, Chicago, IL, May 10-13, 1998.

Petschow BW, Batema RP, Ford LL. Susceptibility of *Helicobacter pylori* to bactericidal properties of medium-chain monoglycerides and free fatty acids. *Antimicrobial Agents and Chemotherapy* 1996;40:302-306.

Pietinen P, Ascherio A, Korhonen P, Hartman AM, Willett WC, Albanes D, Virtamo J. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men. The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *American Journal of Epidemiology* 1997;145:876-887.

Portillo MP, Serra F, Simon E, del Barrio AS, Palou A. Energy restriction with high-fat diet enriched with coconut oil gives higher UCP1 and lower white fat in rats. *International Journal of Obesity and Related Metabolic Disorders* 1998;22:974-9.

Prior IA, Davidson F, Salmond CE, Czochanska Z. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. *American Journal of Clinical Nutrition* 1981;34:1552-1561.

Projan SJ, Brown-Skrobot S, Schlievert PM, Vandenesch F, Novick RP. Glycerol monolaurate inhibits the production of beta-lactamase, toxic shock toxin-1, and other staphylococcal exoproteins by interfering with signal transduction. *Journal of Bacteriology*. 1994;176:4204-4209.

Ravnskov U. Quotation bias in reviews of the diet-heart idea. *Journal of Clinical Epidemiology* 1995;48:713-719.

Raza-Ahmad A, Klassen GA, Murphy DA, Sullivan JA, Kinley CE, Landymore RW, Wood JR. Evidence of type 2 herpes simplex infection in human coronary arteries at the time of coronary artery bypass surgery. *Canadian Journal of Cardiology* 1995;11:1025-1029.

Reddy BS, Maeura Y. Tumor promotion of dietary fat in azoxymethane-induced colon carcinogenesis in female F 344 rats. *Journal of the National Cancer Institute* 1984;72:745- 750.

Reiner DS, Wang CS, Gillin FD. Human milk kills *Giardia lamblia* by generating toxic lipolytic products. *Journal of Infectious Diseases* 1986;154:825-832.

Saikku P. *Chlamydia pneumoniae* and atherosclerosis -- an update. *Scandinavian Journal of Infectious Diseases Supplement* 1997;104:53-56.

Sands JA, Auperin DD, Landin PD, Reinhardt A, Cadden SP. Antiviral effects of fatty acids and derivatives: lipid-containing bacteriophages as a model system in *The Pharmacological Effect of Lipids* (JJ Kabara, ed) American Oil Chemists' Society, Champaign IL, 1978, pp 75-95.

Sircar S, Kansra U. Choice of cooking oils--myths and realities. *Journal of the Indian Medical Association* 1998;96:304-307.

Smit MJ, Wolters H, Temmerman AM, Kuipers F, Beynen AC, Vonk RJ. Effects of dietary corn and olive oil versus coconut fat on biliary cholesterol secretion in rats. *International Journal of Vitamin and Nutrition Research* 1994;64:75-80.

- Smith RL. *The Cholesterol Conspiracy*. Warren H Green Inc. St. Louis, Missouri, 1991.
- Sugano M, Ikeda I. Metabolic interactions between essential and trans-fatty acids. *Current Opinions in Lipidology* 1996;7:38-42.
- Sundram K, Hayes KC, Siru OH. Dietary palmitic acid results in lower serum cholesterol than does a lauric-myristic acid combination in normolipemic humans. *American Journal of Clinical Nutrition* 1994;59:841-846.
- Tappia PS, Grimble RF. Complex modulation of cytokine induction by endotoxin and tumour necrosis factor from peritoneal macrophages of rats by diets containing fats of different saturated, monounsaturated and polyunsaturated fatty acid composition. *Clinical Science (Colch)* 1994;87:173-178.
- Tholstrup T, Marckmann P, Jespersen J, Sandstrom B. Fat high in stearic acid favorably affects blood lipids and factor VII coagulant activity in comparison with fats high in palmitic acid or high in myristic and lauric acids. *American Journal of Clinical Nutrition* 1994;59:371-377.
- Thormar H, Isaacs EC, Brown HR, Barshatzky MR, Pessolano T. Inactivation of enveloped viruses and killing of cells by fatty acids and monoglycerides. *Antimicrobial Agents and Chemotherapy* 1987;31:27-31.
- Trautwein EA, Kunath-Rau A, Dietrich J, Drusch S, Erberdobler HF. Effect of dietary fats rich in lauric, myristic, palmitic, oleic or linoleic acid on plasma, hepatic and biliary lipids in cholesterol-fed hamsters. *British Journal of Nutrition* 1997;77:605-620.
- Visseren FL, Bouter KP, Pon MJ, Hoekstra JB, Erkelens DV, Diepersloot RJ. Patients with diabetes mellitus and atherosclerosis; a role for cytomegalovirus? *Diabetes Research and Clinical Practice (Limerick)* 1997;36:49-55.
- Wan JM, Grimble RF. Effect of dietary linoleate content on the metabolic response of rats to *Escherichia coli* endotoxin. *Clinical Science (Colch)* 1987;72:383-385.
- Wang LL and Johnson EA. Inhibition of *Listeria monocytogenes* by fatty acids and monoglycerides. *Applied and Environmental Microbiology* 1992;58:624-629.
- Wang LL and Johnson EA. Inhibition of *Listeria monocytogenes* by fatty acids and monoglycerides. *Applied and Environmental Microbiology* 1992; 58:624-629.
- Willett W. Editorial: Challenges for public health nutrition in the 1990s. *American Journal of Public Health*. 1990;80:1295-1298.
- Witcher KJ, Novick RP, Schlievert PM. Modulation of immune cell proliferation by glycerol monolaurate. *Clinical and Diagnostic Laboratory Immunology* 1996;3:10-13.
- Zhou YF, Buetta E, Yu ZX, Finkel T, Epstein SE. Human cytomegalovirus increases modified low density lipoprotein uptake and scavenger receptor mRNA expression in vascular smooth muscle cells. *Journal of Clinical Investigation* 1996;98:2129-2138.