Coconut: In Support of Good Health in the 21st Century

by

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Abstract

Coconuts play a unique role in the diets of mankind because they are the source of important physiologically functional components. These physiologically functional components are found in the fat part of whole coconut, in the fat part of desiccated coconut, and in the extracted coconut oil. Lauric acid, the major fatty acid from the fat of the coconut, has long been recognized for the unique properties that it lends to nonfood uses in the soaps and cosmetics industry. More recently, lauric acid has been recognized for its unique properties in food use, which are related to its antiviral, antibacterial, and antiprotozoal functions. Now, capric acid, another of coconut's fatty acids has been added to the list of coconut's antimicrobial components. These fatty acids are found in the largest amounts only in traditional lauric fats, especially from coconut. Also, recently published research has shown that natural coconut fat in the diet leads to a normalization of body lipids, protects against alcohol damage to the liver, and improves the immune system's anti-inflammatory response. Clearly, there has been increasing recognition of health- supporting functions of the fatty acids found in coconut. Recent reports from the U.S. Food and Drug Administration about required labeling of the *trans* fatty acids will put coconut oil in a more competitive position and may help return to its use by the baking and snack food industry where it has continued to be recognized for its functionality. Now it can be recognized for another kind of functionality: the improvement of the health of mankind.

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I. INTRODUCTION

Mr. Chairman and members of the Asian Pacific Coconut Community, I would like to thank you for inviting me to once again speak to this gathering of delegates on the occasion of your 36th session as you celebrate the 30th anniversary of APCC.

When I addressed the 32nd COCOTECH meeting in Cochin, India, I covered two areas of interest to the coconut community. In the first part, I reviewed the major health challenge facing coconut oil at that time, which was based on a supposed negative role played by saturated fat in heart disease. I hope that my talk was able to dispel any acceptance of that notion. In the second part of my talk I suggested that there were some new positive health benefits from coconut that should be recognized. These benefits stemmed from coconut's use as a food with major functional properties for antimicrobial and anti-cancer effects.

In my presentation today, I will bring you up to date about the new recognition of functional foods as important components in the diet. Additionally, I would like to briefly review the state of the anti-saturated fat situation and bring you up to date on some of the research that compares the beneficial effects of saturated fats with those of omega-6 polyunsaturates, as well as the beneficial effects of the saturated fats relative to the detrimental effects of the partially hydrogenated fats and the *trans* fatty acids. In particular I will review some of the surprising beneficial effects of the special saturates found in coconut oil as they compare with those of the unsaturates found in some of the other food oils. Components of coconut oil are increasingly being shown to be beneficial. Increasingly, lauric acid, and even capric acid, have been the subject of favorable scientific reports on health parameters.

II. FUNCTIONAL PROPERTIES OF LAURIC FATS AS ANTIMICROBIALS

Earlier this year, at a special conference entitled, "Functional Foods For Health Promotion: Physiologic Considerations"; EXPERIMENTAL BIOLOGY '99, Renaissance Washington Hotel, Washington, DC Saturday, April 17, 1999, which was sponsored by the International Life Sciences Institute, ILSI NORTH AMERICA, Technical Committee on Food Components for Health Promotion, the term "functional foods" was defined as "a functional food provides a health benefit over and beyond the basic nutrients."

This is exactly what coconut and its edible products such as desiccated coconut and coconut oil do. As a functional food, coconut has fatty acids that provide both energy (nutrients) and raw material for antimicrobial fatty acids and monoglycerides (functional components) when it is eaten. Desiccated coconut is about 69% coconut fat, as is creamed coconut. Full coconut milk is approximately 24% fat.

Approximately 50% of the fatty acids in coconut fat are lauric acid. Lauric acid is a medium chain fatty acid, which has the additional beneficial function of being formed into monolaurin in the human or animal body. Monolaurin is the antiviral, antibacterial, and antiprotozoal monoglyceride used by the human or animal to destroy lipid-coated viruses such as HIV, herpes, cytomegalovirus,

influenza, various pathogenic bacteria, including listeria monocytogenes and helicobacter pylori, and protozoa such as giardia lamblia. Some studies have also shown some antimicrobial effects of the free lauric acid.

Also, approximately 6-7% of the fatty acids in coconut fat are capric acid. Capric acid is another medium chain fatty acid, which has a similar beneficial function when it is formed into monocaprin in the human or animal body. Monocaprin has also been shown to have antiviral effects against HIV and is being tested for antiviral effects against herpes simplex and antibacterial effects against chlamydia and other sexually transmitted bacteria. (Reuters, London June 29, 1999) See below for details.

The food industry has, of course, long been aware that the functional properties of the lauric oils, and especially coconut oil, are unsurpassed by other available commercial oils. Unfortunately, in the U.S., both during the late 1930s and again during the 1980s and 1990s, the commercial interests of the U.S. domestic fats and oils industry were successful in driving down usage of coconut oil. As a result, in the U.S. and in other countries where the influence from the U.S. is strong, the manufacturer has lost the benefit of the lauric oils in its food products. As we will see from the data I will present in this talk, it is the consumer who has lost the many health benefits that can result from regular consumption of coconut products.

The antiviral, antibacterial, and antiprotozoal properties of lauric acid and monolaurin have been recognized by a small number of researchers for nearly four decades: this knowledge has resulted in more than 20 research papers and several U.S. patents, and this past year it resulted in a comprehensive book chapter, which reviewed the important aspects of lauric oils as antimicrobial agents (Enig 1998). In the past, the larger group of clinicians and food and nutrition scientists has been unaware of the potential benefits of consuming foods containing coconut and coconut oil, but this is now starting to change.

Kabara (1978) and others have reported that certain fatty acids (FAs) (e.g., medium-chain saturates) and their derivatives (e.g., monoglycerides (MGs)) can have adverse effects on various microorganisms: those microorganisms that are inactivated include bacteria, yeast, fungi, and enveloped viruses. Additionally, it is reported that the antimicrobial 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 action of lipids are related to their structure: e.g., monoglycerides, free fatty acids. The monoglycerides are active; diglycerides and triglycerides are inactive. Of the saturated fatty acids, lauric acid has greater antiviral activity than either caprylic acid (C-8), capric acid (C-10), or myristic acid (C-14). In general, it is reported that the fatty acids and monoglycerides produce their killing/inactivating effect by lysing the plasma membrane lipid bilayer. The antiviral action attributed to monolaurin is that of solubilizing the lipids and phospholipids in the envelope of the virus, causing the disintegration of the virus envelope. However, there is evidence from recent studies that one antimicrobial effect in bacteria is related to monolaurin's interference with signal transduction (Projan et al 1994), and another antimicrobial effect in viruses is due to lauric acid's interference with virus assembly and viral maturation (Hornung et al 1994).

Recognition of the antiviral aspects of the antimicrobial activity of the monoglyceride of lauric acid (monolaurin) has been reported since 1966. Some of the early work by Hierholzer and Kabara (1982) that showed virucidal effects of monolaurin on enveloped RNA and DNA viruses was done in conjunction with the Center for Disease Control of the U.S. Public Health Service. These studies were done with selected virus prototypes or recognized representative strains of enveloped human viruses. The envelope of these viruses is a lipid membrane, and the presence of a lipid membrane on viruses makes them especially vulnerable to lauric acid and its derivative monolaurin.

The medium-chain saturated fatty acids and their derivatives act by disrupting the lipid membranes of the viruses (Isaacs and Thormar 1991; Isaacs et al 1992). Research has shown that enveloped viruses are inactivated in both human and bovine milk by added fatty acids and monoglycerides (Isaacs et al 1991), and also by endogenous fatty acids and monoglycerides of the appropriate length (Isaacs et al 1986, 1990, 1991, 1992; Thormar et al 1987).

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 cytomegalovirus (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+ 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 utilize those dietary fats that are sources of known antiviral, antimicrobial, and antiprotozoal monoglycerides and fatty acids such as monolaurin and its precursor lauric acid.

Until now, no one in the mainstream nutrition community seems to have recognized the added potential of antimicrobial lipids in the treatment of HIV-infected or AIDS patients. These antimicrobial fatty acids and their derivatives are essentially nontoxic to man; they are produced *in vivo* by humans when they ingest those commonly available foods that contain adequate levels of medium-chain fatty acids such as lauric acid. According to the published research, lauric acid is one of the best "inactivating" fatty acids, and its monoglyceride is even more effective than the fatty acid alone (Kabara 1978, Sands et al 1978, Fletcher et al 1985, Kabara 1985).

The lipid-coated (envelope) viruses are dependent on host lipids for their lipid constituents. The variability of fatty acids in the foods of individuals as well as the variability from *de novo* synthesis accounts for the variability of fatty acids in the virus envelope and also explains the variability of glycoprotein expression, a variability that makes vaccine development more difficult.

Monolaurin does not appear to have an adverse effect on desirable gut bacteria, but rather on only potentially pathogenic microorganisms. For example, Isaacs et al (1991) reported no inactivation of the common *Escherichia coli* or *Salmonella enteritidis* by monolaurin, but major inactivation of *Hemophilus influenzae*, *Staphylococcus epidermidis* and Group B gram positive *streptococcus*.

The potentially pathogenic bacteria inactivated by monolaurin include L*isteria monocytogenes*, *Staphylococcus aureus*, *Streptococcus agalactiae*, Groups A,F & G *streptococci*, gram-positive organisms, and some gram-negative organisms if pretreated with a chelator (Boddie & Nickerson 1992, Kabara 1978, Kabara 1984, Isaacs et al 1990, Isaacs et al 1992, Isaacs et al 1994,

Isaacs & Schneidman 1991, Isaacs & Thormar 1986, Isaacs & Thormar 1990, Isaacs & Thormar 1991, Thormar et al 1987, Wang & Johnson 1992).

Decreased growth of *Staphylococcus aureus* and decreased production of toxic shock syndrome toxin-1 was shown with 150 mg monolaurin per liter (Holland et al 1994). Monolaurin was 5000 times more inhibitory against *Listeria monocytogenes* than ethanol (Oh & Marshall 1993). Helicobacter pylori is rapidly inactivated by medium-chain monoglycerides and lauric acid, and there appears to be very little development of resistance of the organism to the bactericidal effects (Petschow et al 1996) of these natural antimicrobials.

A number of fungi, yeast, and protozoa are inactivated or killed by lauric acid or monolaurin. The fungi include several species of ringworm (Isaacs et al 1991). The yeast reported is *Candida albicans* (Isaacs et al 1991). The protozoan parasite *Giardia lamblia* is killed by free fatty acids and monoglycerides from hydrolyzed human milk (Hernell et al 1986, Reiner et al 1986, Crouch et al 1991, Isaacs et al 1991). Numerous other protozoa were studied with similar findings; these findings have not yet been published (Jon J. Kabara, private communication, 1997).

Research continues in measuring the effect of the monoglyceride derivative of capric acid monocaprin as well as the effects of lauric acid. Chlamydia trachomatis is inactivated by lauric acid, capric acid, and monocaprin (Bergsson et al 1998), and hydrogels containing monocaprin are potent in vitro inactivators of sexually transmitted viruses such as HSV-2 and HIV-1 and bacteria such as Neisseria gonorrhoeae (Thormar 1999).

III. ORIGINS OF THE ANTI-SATURATED FAT AGENDA

The coconut industry has suffered more than three decades of abusive rhetoric from the consumer activist group Center for Science in the Public Interest (CSPI), from the American Soybean Association (ASA) and other members of the edible oil industry, and from those in the medical and scientific community who learned their misinformation from groups like CSPI and ASA. I would like to review briefly the origins of the anti-saturated fat, anti-tropical oil campaigns and hopefully give you some useful insight into the issues.

When and how did the anti-saturated fat story begin? It really began in part in the late 1950s, when a researcher in Minnesota announced that the heart disease epidemic was being caused by hydrogenated vegetable fats. The edible oil industry's response at that time was to claim it was only the saturated fat in the hydrogenated oils that was causing the problem. The industry then announced that it would be changing to partially hydrogenated fats and that this would solve the problem.

In actual fact, there was no change because the oils were already being partially hydrogenated, and the levels of saturated fatty acids remained similar, as did the levels of the *trans* fatty acids. The only thing that really changed was the term for hydrogenation or hardening listed on the food label.

During this same period, a researcher in Philadelphia reported that consuming polyunsaturated fatty acids lowered serum cholesterol. This researcher, however, neglected to include the information that the lowering was due to the cholesterol going into the tissues, such as the liver and the arteries. As a result of this research report and the acceptance of this new agenda by the domestic edible oils

industries, there was a gradual increase in the emphasis on replacing "saturated fats" in the diet and on the consuming of larger amounts of the "polyunsaturated fats." As many of you probably know, this strong emphasis on consuming polyunsaturates has backfired in many ways: the current adjustments being recommended in the U.S. by groups such as the National Academy of Sciences replace the saturates with monounsaturates instead of with polyunsaturates and replace polyunsaturates with monounsaturates.

Early promoters of the anti-saturated fat ideas included companies such as Corn Products Company (CPC International) through a book written by Jeremiah Stamler in 1963, with the professional edition published in 1966 by CPC. This book took some of the earliest pejorative stabs at the tropical oils. In 1963, the only tropical fat or oil singled out as high in saturated fats was coconut oil. Palm oil had not entered the U.S. food supply to any extent, had not become a commercial threat to the domestic oils, and was not recognized in any of the early texts. An observation by the editorial staff of Consumer Reports noted that

> "...in 1962...one writer observed, the average American now fears fat (saturated fat, that is) 'as he once feared witches.' "

In 1965, a representative of Procter and Gamble told the American Heart Association to change its Diet/Heart statement, removing any reference to the *trans* fatty acids. This altered official document encouraged the consumption of partially hydrogenated fats. In the 1970s, this same Procter and Gamble employee served as nutrition chairman in two controlling positions for the National Heart Lung and Blood Institute's Lipid Research Clinic (LRC) trials and as director of one of the LRC centers. These LRC trials were the basis for the 1984 NIH Cholesterol Consensus Conference, which in turn spawned the National Cholesterol Education Program (NCEP). This program encourages consumption of margarine and partially hydrogenated fats, while admitting that *trans* should not be consumed in excess. The official NCEP document states that "...coconut oil, palm oil, and palm kernel oil...should be avoided..."

In 1966, the U.S. Department of Agriculture documents on fats and oils talked about how unstable the unsaturated fats and oils were. There was no criticism of the saturated fats. That criticism of saturated fat was to come later to this agency when it came under the influence of the domestic edible fats and oils industry, and when it developed the U.S. Dietary Guidelines. These Dietary Guidelines became very anti-saturated fat and remain so to this day. Nevertheless, as we will learn later in my talk, there has started some reversal of the anti-saturated fat stance in the works in this agency in 1998.

In the early 1970s, although a number of researchers were voicing concerns about the *trans* fats, the edible oil industry and the U.S. Food and Drug Administration (FDA) were engaging in a revolving-door exchange that would (i) promote the increasing consumption of partially hydrogenated vegetable oils, (ii) would condemn the saturated fats, and (iii) hide the *trans* issue. As an example of this "oily" exchange, in 1971 the FDA's general counsel became president of the edible oil trade association, and he in turn was replaced at the FDA by a food lawyer who had represented the edible oil industry.

From that point on, the truth about any real effects of the dietary fats had to play catch-up.

The American edible oil industry sponsored "information" to educate the public, and the natural dairy and animal fats industries were inept at countering any of that misinformation. Not being domestically grown in the U.S., coconut oil, palm oil, and palm kernel oil were not around to defend themselves at that time. The government agencies responsible for disseminating information ignored those protesting "lone voices," and by the mid-1980s, American food manufacturers and consumers had made major changes in their fats and oils usage -- away from the safe saturated fats and headlong into the problematic *trans* fats.

Enig and Fallon (1998/1999) have reviewed the above history in "The Oiling of America" published in the Australian magazine Nexus. The magazine has placed this review on the internet and it can be viewed or downloaded from the Nexus website. The internet addresses for the websites are http://www.peg.apc.org/~nexus/OilingAmerica.1.html and http://www.peg.apc.org/~nexus/OilingAmerica.2.html.

IV. THE DAMAGING ROLE OF THE U.S. CONSUMER ACTIVIST GROUP CSPI

Some of the food oil industry (especially those connected with the American Soybean Association (ASA)) and some of the consumer activists (especially the Center for Science in the Public Interest (CSPI) and also the American Heart Savers Association) further eroded the status of natural fats when they sponsored the major anti-saturated fat, anti-tropical oils campaign in the late 1980s.

Actually, an active anti-saturated fat bias started as far back as 1972 in CSPI. But beginning in 1984, this very vocal consumer activist group started its anti-saturated fat campaign in earnest. In particular, at this time, the campaign was against the "saturated" frying fats, especially those being used by fast-food restaurants. Most of these so-called saturated frying fats were tallow based, but also included was palm oil in at least one of the hotel/restaurant chains.

Then in a "News Release" in August 1986, CSPI criticized what it called "Deceptive Vegetable Oil Labeling: Saturated Fat Without The Facts," referring to "palm, coconut, and palm kernel oil" as "rich in artery-clogging saturated fat." CSPI further announced that it had petitioned the Food and Drug Administration to stop allowing labeling of foods as having "100% vegetable shortening"if they contained any of the "tropical oils." CSPI also asked for mandatory addition of the qualifier "a saturated fat" when coconut, palm or palm kernel oils were named on the food label.

In 1988, CSPI published a booklet called "Saturated Fat Attack." This booklet contained lists of processed foods "surveyed" in Washington, DC supermarkets. The lists were used for developing information about the saturated fat in the products. Section III is entitled "Those Troublesome Tropical Oils," and it contains statements encouraging pejorative labeling. There were lots of substantive mistakes in the booklet, including errors in the description of the biochemistry of fats and oils and completely erroneous statements about the fat and oil composition of many of the products.

At the same time CSPI was conducting its campaign in 1986, the American Soybean Association began its anti-tropical oil campaign by sending inflammatory letters, etc., to soybean farmers. The ASA took out advertisements to promote a "[tropical] Fat Fighter Kit." The ASA hired

a Washington DC "nutritionist" to survey supermarkets to detect the presence of tropical oils in foods.

Then early in 1987, the ASA petitioned the FDA to require labeling of "Tropical Fats," and by mid-1987, the Soybean Digest continued an active and increasing anti-tropical oils campaign. At about the same time (June 3, 1987), the New York Times published an editorial, "The Truth About Vegetable Oil," in which it called palm, palm kernel, and coconut oils "the cheaper, artery-clogging oils from Malaysia and Indonesia" and claimed that U.S. federal dietary guidelines opposed tropical oils, although it is not clear that this was so. The "artery-clogging" terminology was right out of CSPI.

Two years later in 1989, the ASA held a press conference with the help of the CSPI in Washington DC in an attempt to counter the palm oil group's press conference of 6 March. The ASA "Media Alert" stated that the National Heart Lung and Blood Institute and National Research Council "recommend consumers avoid palm, palm kernel and coconut oils." Only months before these press conferences, millionaire Phil Sokolof, the head of the National Heart Savers Association (NHSA), purchased the first of a series of anti-saturated fats and anti-tropical fats advertisements in major newspapers. No one has found an overt connection between Sokolof (and his NHSA) and the ASA, but the CSPI bragged about being his advisor.

V. WHAT ABOUT HEART DISEASE AND COCONUT OIL?

The research over four decades concerning coconut oil in the diet and heart disease is quite clear: coconut oil has been shown to be beneficial. This research leads us to ask the question, "should coconut oil be used to both prevent and treat coronary heart disease?"

This statement is based on several reviews of the scientific literature concerning the feeding of coconut oil to humans. Blackburn et al (1988) have reviewed the published literature of "coconut oil's effect on serum cholesterol and atherogenesis" and have concluded that when "...[coconut oil is] fed physiologically with other fats or adequately supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity."

After reviewing this same literature, Kurup and Rajmohan (1995) conducted a study on 64 volunteers and found "...no statistically significant alteration in the serum total cholesterol, HDL cholesterol, LDL cholesterol, HDL cholesterol/total cholesterol ratio and LDL cholesterol/HDL cholesterol ratio of triglycerides from the baseline values..." A beneficial effect of adding the coconut kernel to the diet was noted by these researchers.

Kaunitz and Dayrit (1992) have reviewed some of the epidemiological and experimental data regarding coconut-eating groups and noted that the "available population studies show that dietary coconut oil does not lead to high serum cholesterol nor to high coronary heart disease mortality or morbidity." They noted that 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 very much below the acceptable lower limit of 35 mg/dl) and the LDL/HDL ratio increased 30% from 3.0 to 3.9. These latter two changes are considered quite undesirable. Mendis and Kumarasunderam (1990) also compared the effect of coconut oil and soy oil in normolipidemic young males, and again the coconut oil resulted in an increase in the HDL cholesterol, whereas the soy oil reduced this desirable lipoprotein. As noted above, Kurup and Rajmohan (1995), who studied the addition of coconut oil alone to previously mixed fat diets, had reported no significant difference from baseline.

Previously, Prior et al (1981) had shown that islanders with high intakes 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, however, and lowered their intake of coconut oil, their total cholesterol and LDL cholesterol increased, and their HDL cholesterol decreased. Statements that any saturated fat is a dietary problem is not supported by evidence (Enig 1993).

Studies that allegedly showed a "hypercholesterolemic" effect of coconut oil feeding, usually only showed that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat to which coconut oil was being compared. This appears to be in part because coconut oil does not "drive" cholesterol into the tissues as does the more polyunsaturated fats. The chemical analysis of the atheroma shows that the fatty acids from the cholesterol esters are 74% unsaturated (41% of the total fatty acids 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).

There is another aspect to the coronary heart disease picture. This is related to the initiation of the atheromas that are reported to be blocking arteries. Recent research shows that there is a causative role for the herpes virus and cytomegalovirus in the initial formation of atherosclerotic plaques and the reclogging of arteries after angioplasty. (*New York Times* 1991) What is so interesting is that the herpes virus and cytomegalovirus are both inhibited by the antimicrobial lipid monolaurin, but monolaurin is not formed in the body unless there is a source of lauric acid in the diet. Thus, ironically enough, one could consider the recommendations to avoid coconut and other lauric oils as contributing to the increased incidence of coronary heart disease.

Chlamydia pneumoniae, a gram-negative bacteria, is another of the microorganisms suspected of playing a role in atherosclerosis by provoking an inflammatory process that would result in the oxidation of lipoproteins with induction of cytokines and production of proteolystic enzymes, a typical phenomena in atherosclerosis (Saikku 1997). Some of the pathogenic gram-negative bacteria with an appropriate chelator have been reported to be inactivated or killed by lauric acid and monolaurin as well as capric acid and monocaprin (See above, Bergsson et al 1997 and Thormar et al 1999).

However, the microorganisms most frequently identified as probable causative infecting agents are in the herpes virus family and include cytomegalovirus, type 2 herpes simplex (HSV-2), and Coxsackie B4 virus. The evidence for a causative role for cytomegalovirus is the strongest (Ellis 1997, Visseren et al 1997, Zhou et al 1996, Melnick et al 1996, Epstein et al 1996, Chen & Yang 1995), but a role for HSV-2 is also shown (Raza-Ahmad et al 1995). All members of the herpes virus family are reported to be killed by the fatty acids and monoglycerides from saturated fatty acids ranging from C-6 to C-14 (Isaacs et al 1991), which include approximately 80% of the fatty acids in

coconut oil.

In spite of what has been said over the past four or more decades about the culpability of the saturated fatty acids in heart disease, they are ultimately going to be held blameless. More and more research is showing the problem to be related to oxidized products. One protection man has against oxidized products is the naturally saturated fats such as coconut oil.

VI. THE LATEST ON THE *TRANS* FATTY ACIDS

Both the United States and Canada will soon require labeling of the *trans* fatty acids, which will put coconut oil in a more competitive position than it has been in the past decade. A fear of the vegetable oil manufacturers has always been that they would have to label *trans* fatty acids. The producers of *trans* fatty acids have relied on the anti-saturated fat crusade to protect their markets. However, the latest research on saturated fatty acids and *trans* fatty acids shows the saturated fatty acids coming out ahead in the health race.

It has taken this last decade, from 1988 to 1998, to see changes in perception. During this period, the *trans* fatty acids have taken a deserved drubbing. Research reports from Europe have been emerging since the seminal report by Mensink and Katan in 1990 that the *trans* fatty acids raised the low density lipoprotein (LDL) cholesterol and lowered the high density lipoprotein (HDL) cholesterol in serum. This has been confirmed by studies in the U.S. (Judd et al 1994, Khosla and Hayes 1996, Clevidence 1997).

In 1990, the lipids research group at the University of Maryland published a paper (Enig et al 1990) correcting some of the erroneous data sponsored by the food industry in the 1985 review by the Life Sciences Research Office of Federation of American Societies for Experimental Biology (LSRO-FASEB) (Senti 1985) of the *trans* fatty acids.

Also, in 1993, a group of researchers at Harvard University, led by Professor Walter Willett, reported a positive relationship between the dietary intake of the *trans* fatty acids and coronary heart disease in a greater than 80,000 cohort of nurses who had been followed by the School of Public Health at Harvard University for more than a decade.

Pietinen and colleagues (1997) evaluated the findings from the large cohort of Finnish men who were being studied for a cancer prevention study. After controlling for the appropriate variables including several coronary risk factors, the authors observed a significant positive association between the intake of *trans* fatty acids and the risk of death from coronary disease. There was no association between intakes of saturated fatty acids, or dietary cholesterol and the risk of coronary deaths. This is another example of the differences between the effects of the *trans* fatty acids and the saturated fatty acids and further challenge to the dietary cholesterol hypothesis.

The issue of the *trans* fatty acids as a causative factor in remains underexplored, but recent reports have found a connection. Bakker and colleagues (1997) studied the data for the association between breast-cancer incidence and linoleic acid status across European countries since animal and

ecological studies had suggest a relationship. They found that the mean fatty acid composition of adipose did not show an association with omega-6 linoleic acid and breast, colon or prostate cancer. However, cancers of the breast and colon were positively associated with the *trans* fatty acids. Kohlmeier and colleagues (1997) also reported that data from the EURAMIC study showed adipose tissue concentration of *trans* fatty acids having a positive association with postmenopausal breast cancer in European women.

In 1995 a British documentary on the *trans* fatty acids aired on a major television station in the U.K. This documentary included an expose of the battle between the edible oil industry and some of the major researchers of the *trans* fatty acids. Just this year, this same documentary has been aired on television in France where it was requested by a major television station.

Several of the early researchers into the *trans* problems, Professor Fred Kummerow and Dr. George Mann, have continued their research and/or writing (Mann 1994). The popular media has continued to press the issue of the amounts of *trans* in the foods, for which there are still no comprehensive government data bases, and a recent published paper from a U.S. Department of Agriculture researcher states:

> "Because *trans* fatty acids have no known health benefits and strong presumptive evidence suggests that they contribute markedly to the risk of developing CHD, the results published to date suggest that it would be prudent to lower the intake of *trans* fatty acids in the U.S. diet."(Nelson 1998).

Professor Meir Stampfer from Harvard University refers to *trans* fats as "one of the major nutritional issues of the nation," contending that "they have a large impact" and "...we should completely eliminate hydrogenated fats from the diet" (Gottesman 1998).

Lowering the *trans* fatty acids in the foods in the U.S. can only be done by returning to the use of the natural unhydrogenated and more saturated fats and oils.

Predictions can be made regarding the future of the *trans* fatty acids. Our ability to predict has been pretty good; for example when Enig Associates started producing the marketing newsletter *Market Insights* written by Eric Enig, we predicted that *trans* fatty acids would eventually be swept out of the market. It appears that this prediction may be close to coming true.

Also in the early 1990s, *Market Insights* predicted that CSPI would change its mind about the *trans* fatty acids, which it had spent years defending. CSPI did change its mind, and in fact went on the attack regarding the *trans*, but CSPI never admitted that it had originally been promoting the *trans* or that the high levels of *trans* found in the fried foods in the fast food and other restaurants and in many other foods are directly due to CSPI lobbying. While its change was welcome, CSPI's revisionist version of its own history of support of partially hydrogenated oils and *trans* fatty acids would have fit perfectly into George Orwell's "1984."

VII. COMPARISON OF SATURATED FATS WITH THE *TRANS* FATS

The statement that *trans* fatty acids are like saturated fatty acids is not correct for biological systems. A listing of the biological effects of saturated fatty acids in the diet versus the biological effects of *trans* fatty acids in the diet is in actuality a listing of the **good** (saturated) versus the **bad** (*trans*).

When one compares the saturated fatty acids and the *trans* fatty acids, we see that

(1) saturated fatty acids raise HDL cholesterol, the so-called good cholesterol, whereas the *trans* fatty acids lower HDL cholesterol (Mensink and Katan 1990, Judd et al 1994);

(2) saturated fatty acids lower the blood levels of the atherogenic lipoprotein [a], whereas *trans* fatty acids raise the blood levels of lipoprotein [a] (Khosla and Hayes 1996, Hornstra et al 1991, Clevidence et al 1997);

(3) saturated fatty acids conserve the elongated omega-3 fatty acids (Gerster 1998), whereas *trans* fatty acids cause the tissues to lose these omega-3 fatty acids (Sugano and Ikeda 1996);

(4) saturated fatty acids do not inhibit insulin binding, whereas *trans* fatty acids do inhibit insulin binding;

(5) saturated fatty acids are the normal fatty acids made by the body, and they do not interfere with enzyme functions such as the delta-6-desaturase, whereas *trans* fatty acids are not made by the body, and they interfere with many enzyme functions such as delta-6-desaturase;
and

(6) some saturated fatty acids are used by the body to fight viruses, bacteria, and protozoa, and they support the immune system, whereas *trans* fatty acids interfere with the function of the immune system.

VIII. WHAT ABOUT THE UNSATURATED FATS?

The arteries of the heart are also compromised by the unsaturated fatty acids. When the fatty acid composition of the plaques (atheromas) in the arteries has been analyzed, the level of saturated fatty acids in the cholesterol esters is only 26 percent compared to that in the unsaturated fatty acids, which is 74 percent. When the unsaturated fatty acids in the cholesterol esters in these plaques are analyzed, it is shown that 38 percent are polyunsaturated and 36 percent are monounsaturated. Clearly the problem in not with the saturated fatty acids.

As an aside, you need to understand that the major role of cholesterol in heart disease and in cancer is as the body's repair substance, and that cholesterol is a major support molecule for the immune system, an important antioxidant, and a necessary component of neurotransmitter receptors. Our brains do not work very well without adequate cholesterol. It should be apparent to scientists that the current approach to cholesterol has been wrong.

The pathway to cholesterol synthesis starts with a molecule of acetyl CoA that comes from the metabolism of excess protein forming ketogenic amino acids and from the metabolism of excess carbohydrate, as well as from the oxidation of excess fatty acids. Grundy in 1978 reported that the degree of saturation of the fat in the diet did not affect the rate of synthesis of cholesterol. Research reported in 1997 (Jones 1997), however, showed that the polyunsaturated fatty acids in the diet increase the rate of cholesterol synthesis relative to other fatty acids. Furthermore, research reported

in 1993 (Hodgsons et al 1993) had shown that dietary intake of the omega-6 polyunsaturated fatty acid linoleic acid was positively related to coronary artery disease.

Thus, those statements made by the consumer activists in the United States to the effect that the saturated fatty acids increase cholesterol synthesis is without any foundation. What happens when there is an increase or a decrease of cholesterol in the serum is more like a shift from one compartment to another as the body tries to rectify the potential damage from the excess polyunsaturated fatty acids. Research by Dr. Hans Kaunitz reported in 1978 clearly showed the potential problems with excess polyunsaturated fatty acids.

IX. RESEARCH SHOWING BENEFICIAL EFFECTS OF EATING THE MORE SATURATED FATS

One major concern expressed by the nutrition community is related to whether or not people are getting enough elongated omega-3 fatty acids in their diets. The elongated omega-3 fatty acids of concern are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Some research has shown that (the basic omega-3 fatty acid) α-linolenic acid is not readily converted to the elongated forms in humans or animals, especially when there is ingestion of the trans fatty acids and the consequent inhibition of the delta-6-desaturase enzyme. One recent study (Gerster 1998), which used radioisotope-labeled α-linolenic acid to measure this conversion in adult humans, showed that if the background fat in the diet was high in saturated fat, the conversion was approximately 6% for EPA and 3.8% for DHA, whereas if the background fat in the diet was high in omega-6 polyunsaturated fatty acids (PUFA), the conversion was reduced 40-50%.

Nanji and colleagues (1995) report that a diet enriched in saturated but not unsaturated fatty acids reversed alcoholic liver injury in their animals, which was caused by dietary linoleic acid. These researchers conclude that this effect may be explained by the down-regulation of lipid peroxidation. This is another example of the need for adequate saturated fat in the diet. Cha and Sachan (1994) studied the effects of saturated fatty acid and unsaturated fatty acid diets on ethanol pharmacokinetics. The hepatic enzyme alcohol dehydrogenase and plasma carnitines were also evaluated. The researchers concluded that dietary saturated fatty acids protect the liver from alcohol injury by retarding ethanol metabolism, and that carnitine may be involved.

Hargrove and colleagues (1999) noted the work of Nanji et al and postulated that they would find that diets rich in linoleic acid would also cause acute liver injury after acetaminophen injection. In the first experiment, two levels of fat (15 g/100 g protein and 20 g/100 g protein) were fed using corn oil or beef tallow. Liver enzymes indicating damage were significantly elevated in all the animals except for those animals fed the higher level of beef tallow. These researchers concluded that "diets with high [linoleic acid] may promote acetaminophen-induced liver injury compared to diets with more saturated and monounsaturated fatty acids."

X. RESEARCH SHOWING GENERAL BENEFICIAL EFFECTS FROM FEEDING COCONUT OIL

Research that compares coconut oil feeding with other oils to answer a variety of biological questions is increasingly finding beneficial results from the coconut oil.

Obesity is a major health problem in the United States and the subject of much research. Several lines of research dealing with metabolic effects of high fat diets have been followed. One study used coconut oil to enrich a high fat diet and the results reported were that the "coconut-oil enriched diet is effective in...[producing]...a decrease in white fat stores." (Portillo et al 1998)

Cleary et al (1999) fed genetically obese animals high fat diets of either safflower oil or coconut oil. Safflower oil-fed animals had higher hepatic lipogenic enzyme activities than did coconut oil fed animals. When the number of fat cells were measured, the safflower oil-fed also had more fat cells than the coconut oil-fed.

Many of the feeding studies produce results at variance with the popular conception. High fat diets have been used to study the effects of different types of fatty acids on membrane phospholipid fatty acid profiles. When such a study was performed on mice, the phospholipid profiles were similar for diets high in linoleic acid from high-linoleate sunflower oil relative to diets high in saturated fatty acids from coconut oil. However, those animals fed the diets high in oleic acid (from the high-oleate sunflower oil) or high in elongated omega-3 fatty acids (from menhaden oil) were not only different from the other two diets, but they also resulted in enlarged spleens in the animals. (Huang and Frische 1992)

Oliart-Ros and colleagues (1998), Instituto Technologico de Veracruz, Mexico, reported on effects of different dietary fats on sucrose-induced cardiovascular syndrome in rats. The most significant reduction in parameters of the syndrome was obtained by the n-3 PUFA-rich diet. These researchers reported that the diet thought to be PUFA-deficient presented a tissue lipid pattern similar to the n-3 PUFA-rich diet (fish oil), which surprised and puzzeled them. When questioned, it turned out that the diet was not really PUFA-deficient, but rather just a normal coconut oil (nonhydrogenated), which conserved the elongated omega-3 and normalized the omega-6-to-omega-3 balance.

A recent study measured the effect of high-fat diets, fed for more than three months to the neonatal pig, on the HMG-CoA reductase enzyme's function and gave some surprises. There were two feeding protocols: one with the added cholesterol and one without added cholesterol, but both with coconut oil. The hepatic reductase activity, which was the same in all groups at the beginning of the feeding on the third day and similar on the 42nd day, was increased with and without added cholesterol on the 13th day and then decreased on the 25th day. The data was said to suggest that dietary cholesterol suppressed hepatic reductase activity in the young pigs regardless of their genetic background, that the stage of development was a dominant factor in its regulation, and that both dietary and endogenously synthesized cholesterol was used primarily for tissue building in very young pigs. (McWhinney et al 1996) The feeding of coconut oil did not in any way compromise the normal development of these animals.

When compared with feeding coconut oil, feeding two different soybean oils to young females caused a significant decrease in HDL cholesterol. Both soybean oils, one of which was extracted from a new mutant soybean thought to be more oxidatively stable, were not protective of the HDL

levels (Lu Z et al 1997).

Trautwein et al (1997) studied cholesterol-fed hamsters on different oil supplements for plasma, hepatic, and biliary lipids. The dietary oils included butter, palm stearin, coconut oil, rapeseed oil, olive oil, and sunflowerseed oil. Plasma cholesterol concentrations were higher (9.2 mmol/l) for olive oil than for coconut oil (8.5 mmol/l), hepatic cholesterol was highest in the olive oil group, and none of the diet groups differed for biliary lipids. Even in this cholesterol-sensitive animal model, coconut oil performed better than olive oil.

Smit and colleagues (1994) had also studied the effect of feeding coconut oil compared with feeding corn oil and olive oil in rats and measured the effect on biliary cholesterol. Bile flow was not different between the three diets, but the hepatic plasma membranes showed more cholesterol and less phospholipid from corn and olive oil feeding relative to coconut oil feeding.

Several studies (Kramer et al 1998) have pointed out problems with canola oil feeding in newborn piglets, which result in the reduction in number of platelets and the alteration in their size. There is concern for similar effects in human infants. These undesirable effects can be reversed when coconut oil or other saturated fat is added to the feeding regimen (Kramer et al 1998).

Research has shown that coconut oil is needed for good absorption of fat and calcium from infant formulas. The soy oil (47%) and palm olein (53%) formula gave 90.6% absorption of fat and 39% absorption of calcium, whereas the soy oil (60%) and coconut oil (40%) gave 95.2% absorption of fat and 48.4% absorption of calcium (Nelson et al 1996). Both fat and calcium are needed by the infant for proper growth. These results clearly show the folly of removing or lowering the coconut oil in infant formulas.

XI. RESEARCH SHOWING A ROLE FOR COCONUT IN ENHANCING IMMUNITY AND MODULATING METABOLIC FUNCTIONS

Coconut oil appears to help the immune system response in a beneficial manner. Feeding coconut oil in the diet completely abolished the expected immune factor responses to endotoxin that were seen with corn oil feeding. This inhibitory effect on interleukin-1 production was interpreted by the authors of the study as being largely due to a reduced prostaglandin and leukotriene production (Wan and Grimble 1987). However, the damping may be due to the fact that effects from high omega-6 oils tend to be normalized by coconut oil feeding. Another report from this group (Bibby and Grimble 1990) compared the effects of corn oil and coconut oil diets on tumor necrosis factor-alpha and endotoxin induction of the inflammatory prostaglandin E2 (PGE2) production. The animals fed coconut oil did not produce an increase in PGE2, and the researchers again interpreted this as a modulatory effect that brought about a reduction of phospholipd arachidonic acid content. A study from the same research group (Tappia and Grimble 1994) showed that omega-6 oil enhanced inflammatory stimuli, but that coconut oil, along with fish oil and olive oil, suppressed the production of interleukin-1.

Several recent studies are showing additional helpful effects of consuming coconut oil on a regular basis, thus supplying the body with the lauric acid derivative monolaurin. Monolaurin and the

ether analogue of monolaurin have been shown to have the potential for damping adverse reactions to toxic forms of glutamic acid (Dave et al 1997). Lauric acid and capric acid have been reported to have very potent effects on insulin secretion (Garfinkel et al 1992). Using a model system of murine splenocytes, Witcher et al 1996 showed that monolaurin induced proliferation of T cells and inhibited the toxic shock syndrome toxin-1 mitogenic effects on T cells.

Monserrat and colleagues (1995) showed that a diet rich in coconut oil could protect animals against the renal necrosis and renal failure produced by a diet deficient in choline (a methyl donor group). The animals had less or no mortality and increased survival time as well as decreased incidence or severity of the renal lesions when 20% coconut oil was added to the deficient diet. A mixture of hydrogenated vegetable oil and corn oil did not show the same benefits.

The immune system is complex and has many feedback mechanism to protect it, but the wrong fat and oils can compromise these important mechanisms. The data from the several studies show the helpful effects of coconut fat. Additionally, there are anecdotal reports that consumption of coconut is beneficial for individuals with the chronic fatigue and immune dysfunction syndrome known as CFIDS.

XII. U.S. PATENTS FOR MEDICAL USES OF LAURIC OILS, MEDIUM-CHAIN FATTY ACIDS, AND THEIR DERIVATIVES SUCH AS MONOLAURIN

A number of patents have been granted in the United States for medical uses of lauric oils, lauric acid, and monolaurin. Although one earlier patent was granted to Professor Kabara more than three decades ago, the rest of these patents have been granted within the past decade.

In 1989 a patent was issued to the New England Deaconess Hospital (Bistrian et al 1989) for the invention titled "Kernel Oils and Disease Treatment." This treatment required lauric acid as the primary fatty acid source with lauric oils constituting up to 80% of the diet "using naturally occurring kernel oils."

In 1991 and 1995, two patents were issued to the group of researchers whose work has been reviewed above. The first invention (Isaacs et al 1991) was directed to antiviral and antibacterial activity of both fatty acids and monoglycerides, primarily against enveloped viruses. The claims were for "a method of killing enveloped viruses in a host human...wherein the enveloped viruses are AIDS viruses...[or]...herpes viruses...[and the]...compounds selected from the group consisting of fatty acids having from 6 to 14 carbon atoms and monoglycerides of said fatty acids...[and]...wherein the fatty acids are saturated fatty acids."

The second patent (Isaacs et al 1995) was a further extension of the earlier one. This patent also included discussion of the inactivation of envelop viruses and specifically cited monoglycerides of caproic, caprylic, capric, lauric, and myristic acid. These fatty acids make up more than 80% of coconut oil. Also included in this patent was a listing of susceptible viruses and some bacteria and protozoa.

Although these latter patents may provide the owners of the patents with the ability to extract royalties from commercial manufacturers of monoglycerides and fatty acids, they cannot require

royalties from the human gastrointestinal tract when it is the "factory" that is doing the manufacturing of the monoglycerides and fatty acids. Clearly though, these patents serve to illustrate to us that the health-giving properties of monolaurin and lauric acid are well-recognized by some individuals in the research arena, and they lend credence to our appropriate choice of lauric oils for promoting health and as adjunct treatment of viral diseases.

XIII. HOW CAN WE GET SUFFICIENT COCONUT FAT INTO THE FOOD SUPPLY IN THE U.S. AND OTHER COUNTRIES THAT NEED ITS BENEFITS?

I would like to review for you my perception of the status regarding the coconut and coconut products market in the North American countries such as the United States and Canada at the end of the 20th century and the beginning of the 21st century.

Coconut products are trying to regain their former place in several small markets. The extraction of oil from fresh coconut has been reported in the past decade and my impression is that this is being considered as a desirable source of minimally processed oil, which produces an oil with desirable characteristics for the natural foods market.

There have been some niche markets for coconut products developing during the past half-decade. These are represented primarily by the natural foods and health foods producers. Some examples are the new coconut butters produced in the U.S. and Canada by Omega Nutrition and Carotec, Inc. And, this is no longer as small a market as it has been in past years. Desiccated coconut products, coconut milk, and even coconut oil are appearing on the shelves of many of these markets. After years of packaging coconut oil for skin use only, one of the large suppliers of oils to the natural foods and health foods stores has introduced coconut oil for food use, and it has appeared within the last few months on shelves in the Washington, DC metropolitan area along with other oils. I believe I indirectly had something to do with this turn of events.

XIV. CONCLUSIONS AND RECOMMENDATIONS

As we come close to the end of the year 1999 and set our sights on what could happen in the year 2000 and beyond, there is much to be gained from pursuing the functional properties of coconut for improving the health of humanity.

On the occasion of the 30th anniversary of the Asian Pacific Coconut Community, at this 36th meeting of APCC, I wanted to bring you a message that I hope will encourage you to continue your endeavors on behalf of all parts of the coconut industry. Coconut products for inedible and especially edible uses are of the greatest importance for the health of the entire world.

Some of what I have been telling you, most of you already know. But in saying these things for the record, it is my intention to tell those who did not know all the details until they heard or read this paper about the positive properties of coconut.

Coconut oil is a most important oil because it is a lauric oil. The lauric fats possess unique characteristics for both food industry uses and also for the uses of the soaps and cosmetic industries.

Because of the unique properties of coconut oil, the fats and oils industry has spent untold millions to formulate replacements from those seed oils so widely grown in the world outside the tropics. While it has been impossible to truly duplicate coconut oil for some of its applications, many food manufacturers have been willing to settle for lesser quality in their products. Consumers have also been willing to settle for a lesser quality, in part because they have been fed so much misinformation about fats and oils.

Desiccated coconut, on the other hand, has been impossible to duplicate, and the markets for desiccated coconut have continued. The powdered form of desiccated coconut now being sold in Europe and Asia has yet to find a market in the U.S., but I predict that it will become an indispensable product in the natural foods industry. Creamed coconut, which is desiccated coconut very finely ground, could be used as a nut butter.

APCC needs to promote the edible uses of coconut, and it needs to promote the reeducation of the consumer, the clinician, and the scientist. The researcher H. Thormar (Thormar et al 1999) concluded his abstract with the statement that monocaprin ...is a natural compound found in certain foodstuffs such as milk and is therefore unlikely to cause harmful side effects in the concentrations used. It is not monocaprin that is found in milk, but capric acid. It is likely safe at most any level found in food. However, the levels in milk fat are at most 2 percent whereas the levels in coconut fat are 7 percent.

One last reference for the record. Sircar and Kansra (1998) have reviewed the increasing trend of atherosclerotic disease and type-2 diabetes mellitus in the Indians from both the subcontinent of India and abroad. They note that over the time when there has been an alarming increase in the prevalence of these diseases, there has been a replacement of traditional cooking fats with refined vegetable oils that are promoted as heart-friendly, but which are being found to be detrimental to health. These astute researchers suggest that it is time to return to the traditional cooking fats like ghee, coconut oil, and mustard oil.

There are a number of areas of encouragement. The nutrition community in the United States is slowly starting to recognize the difference between medium chain saturated fatty acids and other saturated fatty acids. We predict now that the qualities of coconut, both for health and food function, will ultimately win out.

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