Highlight Article

Research advancements in palm oil nutrition*

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Palm oil is the major oil produced, with annual world production in excess of 50 million tonnes. About 85% of global palm oil produced is used in food applications. Over the past three decades, research on nutritional benefits of palm oil have demonstrated the nutritional adequacy of palm oil and its products, and have resulted in transitions in the understanding these attributes. Numerous studies have demonstrated that palm oil was similar to unsaturated oils with regards to effects on blood lipids. Palm oil provides a healthy alternative to trans-fatty acid containing hydrogenated fats that have been demonstrated to have serious deleterious effects on health. The similar effects of palm oil on blood lipids, comparable to other vegetable oils could very well be due to the structure of the major triglycerides in palm oil, which has an unsaturated fatty acid in the stereospecific numbers (*sn*)-2 position of the glycerol backbone. In addition, palm oil is well endowed with a bouquet of phytonutrients beneficial to health, such as tocotrienols, carotenoids, and phytosterols. This review will provide an overview of studies that have established palm oil as a balanced and nutritious oil.

Keywords: Health / Lipid / Nutrition / Palm oil / Palm olein / Phytonutrient

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1 Introduction

Palm oil contributes significantly to the world's oils and fats market. The oil is used globally, with Malaysia being a major exporter, selling to over 150 countries worldwide. This global acceptance of palm oil is due to its competitive price vis-a-vis other oils and its suitability in various food applications, such as frying, specialty fats, margarines, shortenings, vegetable ghee, etc. In addition, there is now increasing awareness of its health attributes. This may be yet another major factor contributing to the growing demand for the oil.

Almost 85% of the world's palm oil is used as food and this has meant that the nutritional properties of palm oil and its fractions must be adequately demonstrated through research. The fatty acid composition of palm oil, of almost 50% saturated fatty acids (SFA), has been the focus of attention in determining its nutritional adequacy in relation to coronary heart disease (CHD) risk. Palmitic acid (44%) is the major SFA in palm oil, counter-balanced by almost 39% monounsaturated oleic acid and 11% polyunsaturated linoleic acid. This composition is very different from palm kernel oil (obtained as a co-product from the seed or kernel) which is almost 80% saturated. In addition, the fractionation of palm oil produces a more liquid form of the oil referred to as palm olein (POo), which contains up to 44% oleic acid and 13% linoleic acid. Besides having a balanced fatty acid composition, palm oil is also rich in a number of phytonutrients—carotenoids, tocopherols, tocotrienols, sterols, squalene, coenzyme Q10, phospholipids, and polyphenols. Although these minor components constitute less than 1% of the oil, they nevertheless play an important role in the stability and quality of the oil. In addition, all these phytonutrients have antioxidant properties and some of them exhibit nutritional and health benefits beyond their antioxidant function.

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Abbreviations: BMI, body mass index; **CHD,** coronary heart disease; **CVD,** cardiovascular disease; **DAG,** diacylglycerols; **E,** energy; **FAO,** Food Agricultural Organisation; **FFA,** free fatty acids; **HDL-C,** high density lipoprotein cholesterol; **hsCRP,** high-sensitivity C-reactive protein; **IPOo,** interesterified palm olein; **LDL,** Clow density lipoprotein cholesterol; **MAG,** monoacylglycerol; **MPOB,** Malaysian Palm Oil Board; **MUFA,** monounsaturated fatty acids; **POo,** palm olein; **PUFA,** polyunsaturated fatty acids; **P/S,** polyunsaturated/saturated; **RBDPO,** refined bleached deodorized palm oil; **RPO,** red palm olein; **SFA,** saturated fatty acid; **SFO,** sunflower oil; ***sn***, stereospecific numbers; **TAG,** triacylglycerols; **TC,** total cholesterol; **VLDL-C,** very low density lipoprotein cholesterol; **WHO,** World Health Organisation

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Using a carefully evolved research strategy, the Malaysian Palm Oil Board (MPOB) has focused on multi-pronged nutrition trials in animals and humans to prove the nutritional worthiness of palm oil and its products. This has resulted in over 200 publications in high impact peer reviewed journals. Collaborative projects have been undertaken at biomedical centres of excellence abroad where palm oil was compared to the indigenous oils used in the various countries. The results have shown that palm oil is as good as the indigenous oils, in some instances even better in its cholesterolemic response to the other oils and fats studied. The studies have yielded results that not only demonstrate the nutritional adequacy of palm oil and its products but also transitions in the science of edible oils and fatty acid effects on CHD.

The results from these studies have helped palm oil gain market share and positioned it as a safe and nutritious oil. The worldwide focus on *trans* fats as unhealthy has also opened the door further for palm oil as the healthy natural substitute. MPOB has developed many *trans* free formulations for margarines and shortenings.

2 *sn*-2 hypothesis

The nutritional properties of palm oil have been demonstrated since the 80's and specifically reported by Hornstra et al. (1987) [1] where it was described that although palm oil contains 50% long chain saturated fats, it has a distinct antithrombotic effect based on observations from an animal study on arterial thrombogenesis in vivo. These observations became more prominent in many more studies carried out subsequently (described elsewhere in this review). Ong and Goh (2002) [2] later hypothesized that palm oil does not increase cardiovascular risk because the oil is highly structured to contain predominantly oleic acid at the stereospecific numbers (*sn*)-2 position in the major triacylglycerols (TAG). More recently, the report of an Expert Consultation on Fats and Fatty Acids in Human Nutrition (2010) [3] has noted that "there is possible evidence to suggest that the TC and low density lipoprotein cholesterol (LDL-C) raising effects of palmitic acid are lower for vegetable than animal sources because it is present predominantly in the *sn*-1 and *sn*-3 position as opposed to *sn*-2 position as in animal fats such as lard". As such, palm oil, like other vegetable oils, including olive oil, has the favourable oleic acid in this position. Therefore, this will lead to an acceptance that palm oil is as good as olive oil. This will also explain why even though 50% saturated, palm oil behaves more like a monounsaturated fat.

2.1 Significance of the *sn*-2 hypothesis

The body uses fats for long-term energy storage because they provide about six times as much energy as an equal weight of hydrated glycogen [4]. Many different fats and oils are sources of TAG in the human diet. These oils originate from fruits (e.g., palm and olive oils) or from seeds (corn, rapeseed, and soybean oils) as well as animal and fish sources. Animal fats like butter and lard are solid at room temperature while vegetable oils like corn, soybean, and peanut oils are liquid. However, their structures are chemically similar.

Fats and oils are made up of a mixture of TAG which consists of a glycerol backbone to which three fatty acids are esterified. The positions of fatty acid attachment on the glycerol backbone are referred to by (*sn*) -1, -2 and -3. The attachment of which fatty acid to which position has an important effect on the fat/oil properties [5]. Figure 1 shows the schematic structure of a TAG with three fatty acids bonded to the glycerol backbone.

The fatty acids in fats and oils are classified as SFA, monounsaturated fatty acids (MUFA), or polyunsaturated fatty acids (PUFA). With this classification, palm kernel oil, which has 80% SFA (lauric acid, C12:0, myristic acid, C14:0 and palmitic acid, C16:0), and very little MUFA and PUFA, is a saturated fat. Olive oil, on the other hand, has 80% oleic acid (C18:1) and 9% PUFA but only 10% SFA, and so is predominantly monounsaturated. Sunflower oil has 70% linoleic acid (C18:2) and only 12% SFA, and hence is polyunsaturated.

In vegetable oils, oleic acid (a MUFA) is predominantly situated at the *sn*-2 position, while in animals fats it is predominantly palmitic acid or stearic acid (C16:0 or C18:0-saturated fat) that is situated there. Even though POo and lard have similar proportions of SFA, MUFA, and PUFA, they differ significantly in their positional distribution on the TAG molecule. POo TAG contains only 7–11% palmitic acid at the *sn*-2 position while about 87% is unsaturated fatty acids (oleic acid and linoleic acid). Lard has the highest amount of palmitic acid in the *sn*-2 position at 70%. In human milk, palmitic acid is predominantly present in *sn*-2 (53–57%) while cow milk fat contains less palmitic acid in the *sn*-2 position (38%) [6]. It is now believed that the distribution of fatty acids in the TAG is more important than the fatty acid composition (FAC) alone in conferring the oils' "saturated" or "unsaturated properties."

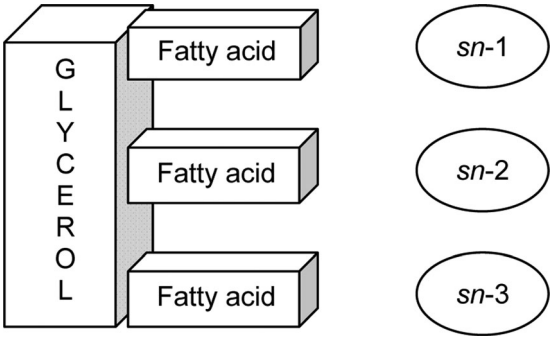

Figure 1. TAG structure showing the stereospecific numbering of sn-1, -2 and -3.

2.2 Lipid digestion and metabolism

The digestion of fat occurs when the enzyme lipase is present. The lipases involved in this process are lingual, gastric, pancreatic, and co-pancreatic, found in the mouth, stomach, and small intestine, respectively.

In the stomach, predigestion of 10–30% of the fat occurs in the presence of lingual and gastric lipases, and bile salts produced by the liver. The lingual and gastric lipases cleave the *sn*-3 fatty acids, resulting in the formation of 1,2-diacylglycerols (1,2-DAG) and *sn*-3 free fatty acids (FFA). The acidic medium in the stomach then facilitates conversion of *sn*-1,2-DAG to *sn*-1,3-DAG. The *sn*-1,3-DAG and *sn*-3 FFA (if <12 carbons) are readily absorbed in the intestine. The schematic diagram below shows the hydrolysis route of TAG at different locations in the human body (Fig. 2).

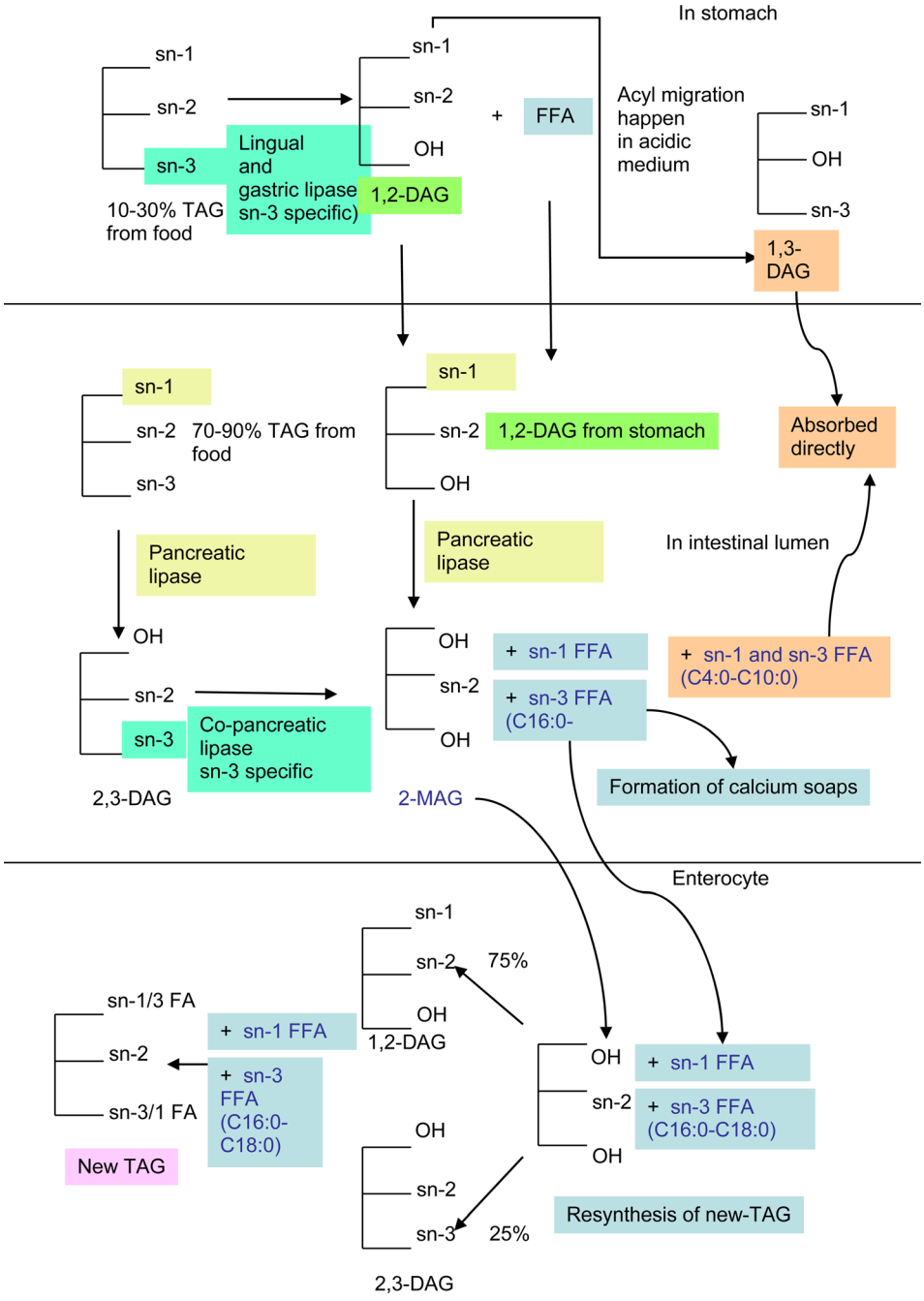

Figure 2. Hydrolysis route of TAG at different locations.

In the small intestine, particularly the duodenum, digestion of 70–90% of the fat occurs in the presence of pancreatic and co-pancreatic lipases. Pancreatic lipase hydrolyses the *sn*-1 fatty acids, while co-pancreatic lipase hydrolyses the *sn*-3 fatty acids. The products from these hydrolyses are 2-monoacylglycerol (2-MAG), and *sn*-1 and *sn*-3 FFA.

The *sn*-2 fatty acid as 2-MAG is transported by chylomicrons which now contain 93% of new TAG (solely from the food source) in its core. These new TAG are a result of resynthesis of 2-MAG and FFA (majority long-chain SFA) in the intestine. Short and medium chain fatty acid absorption is not via chylomicrons. Chylomicrons are then secreted into the blood stream via the lymphatic system. Lipoprotein lipase which lines the blood vessel walls hydrolyzes the new TAG in the chylomicrons. Chylomicron remnants, 2-MAG, and FFA are then produced. The chylomicron remnants carrying the cholesterol ester and TAG are transported back to the liver, while 2-MAG and FFA are used for liver TAG synthesis or energy supply and storage. Eating long-chain SFA and with elaidic acid *(trans isomer of oleic acid)* at *sn*-2 of TAG may slow down hydrolysis of the chylomicron TAG, liver uptake, and clearance of the chylomicron remnants. The presence of a large amount of chylomicron remnants in the blood can lead to increased plasma cholesterol and atherogenesis which can be detrimental to health.

In the intestines, the *sn*-1 and *sn*-3 short- and medium-chain FFA are absorbed directly after hydrolysis. Long-chain SFA will either be absorbed or predominantly react with 2-MAG for resynthesis of new TAG and chylomicron formation. The s*n*-1 and *sn*-3 long-chain free SFA are not absorbed or have delayed absorption as their melting points are above body temperature. Furthermore, in the presence of calcium in the intestines, the long-chain free SFA tend to precipitate as calcium soaps which are excreted (Table 1). Therefore, long chain SFA when situated at *sn*-1 and -3 positions of the glycerol backbone are more difficult to be absorbed.

Surprisingly, pancreatic lipase and its co-lipase have low activity on long-chain PUFA with 20 carbons and more, especially arachidonic acid (C20:4n-6), eicopentaenoic acid (C20:5n-3), and docosahexaenoic acid (C22:6n-3), although they are located at *sn*-3 [7]. These long-chain PUFA occur as 2,3-DAG instead of at *sn*-3. The 2,3-DAG are only hydrolyzed by hepatic lipase in the liver. They are retained in the chylomicron remnants transported to the liver to produce 2-MAG and free long-chain PUFA. The slow hydrolysis of long-chain PUFA at *sn*-3 in TAG reduces the supply of *sn*-2 MAG and delays the resynthesis of new TAG in the intestine. Hence, *sn*-3 positioned long-chain PUFA are not directly absorbed by the body, while *sn*-2 long-chain PUFA are directly absorbed as 2-MAG. This might also lead to lesser excretion of long-chain PUFA from the body during the process of digestion.

Table 1. Summary of absorption for some common fatty acids at the sn-1 and sn-3 positions in tag

Common name	Fate after hydrolysis
Short-chain fatty acids (C4–C6)	Absorbed directly
Medium-chain fatty acids (C8–C10)	Absorbed directly
Long-chain saturated fatty acids	Delayed absorption by minor phosphatidic acid pathway or form calcium soaps and excreted
Long-chain polyunsaturated fatty acids	Delayed formation of TAG and reduced supply of 2-MAG

The absorption and digestion of fat in infants are slightly different from those in adults. At birth, infants have to adapt to the high fat content of breast milk after relying mainly on glucose for energy during foetal development. Pancreatic secretion of lipase is low and the immature liver cannot provide sufficient bile salts to solubilize the digested lipids. Hence, new-born babies digest fats less efficiently than adults. However, breast milk contains lipoprotein lipase and bile-salt-stimulated-lipase that may assist the baby digest milk TAG. In addition, the baby itself secretes a TAG lipase from glands in its stomach and tongue. The milk lipase (only occurs in babies) in the intestinal lumen hydrolyses 2-MAG to glycerol and FFA for direct absorption before resynthesis of new TAG. This milk lipase shortens the route of digestion and absorption of *sn*-2 long-chain SFA in infants. Besides, human milk with palmitic acid (long-chain SFA) predominantly at *sn*-2 forms mixed micelles with bile salts in the milk itself. This again allows good and rapid absorption of TAG by infants [8–10]. If an infant formulation contains long-chain SFA at *sn*-1 and *sn*-3, absorption will be delayed as the milk lipase may only act on 2-MAG formed in the intestine before new TAG is resynthesized. Hence, the tendency to form long-chain calcium soap results in hard stools or constipation, and reduced calcium absorption in infants. The TAG structure of human milk is unique as it optimizes the absorption of palmitic acid. This is why *sn*-2 palmitic acid is preferred in infants over *sn*-1 and *sn*-3 palmitic acid.

Human clinical trials [11, 12, 32] and animal studies [21, 22] have been carried out to determine the effects of stereospecific fats on their lipid profiles. The position of a SFA in TAG exerts two effects on plasma TC. If long-chain SFA occurs at *sn*-1 and *sn*-3, they are either neutral or tend to lower total cholesterol (TC). If at *sn*-2, they generally raise TC.

2.3 *sn*-2 hypothesis and palm oil

In palm oil, the long-chain SFA (palmitic acid) is predominantly at *sn*-1 and *sn*-3, and mainly excreted through the formation of calcium soaps (evidence from human infants and animals). The other main fatty acid in palm oil, oleic acid, is situated at *sn*-2 where it is absorbed into the body and induces beneficial effects similar to olive oil (~80% oleic acid at *sn*-2) [11, 12, 13].

Hence, it is wrong to group palm oil with the traditional saturated fats. In addition, plasma lipid response to a palm oil-rich diet was found to be mild, and appeared more dependent on age, gender, high body mass index (BMI), daily cholesterol ingestion [11, 13, 25] and the synthetic naure of the test oils used [24]. More scientific evidence with adequate and well controlled study designs are required to clear the misconception over palm oil and its nutritional implications, especially in human adults, and is currently the focus of research at MPOB.

3 Effects of palm oil and its fractions on blood lipids and lipoproteins

3.1 Animal studies

In a study to investigate the effects of different dietary fats on arterial thrombosis using the aorta loop technique in rats, it was demonstrated that palm oil exhibited a distinct anti-thrombotic effect comparable to PUFA oils including rapeseed, linseed, and sunflower seed oils [1]. Rand et al. (1988) [14] measured collagen activated platelet aggregation in rats fed 50% energy as palm oil or sunflower oil. They reported greater platelet aggregation in the sunflower oil fed rats compared to the palm oil fed rats. Subsequently, several animal studies compared palm oil with the more unsaturated oils and saturated fats for their effects on blood lipids, lipoproteins and other cardiovascular risk factors.

Oluba et al. (2008) [15] examined the effects of palm oil and soybean oil (both at 5%E level) on serum lipid and some serum enzymes over 6 wk. At the end of the trial, serum TC and TAG were significantly reduced in the rats fed palm oil from those fed soybean oil. This shows that palm oil consumption better ameliorates coronary heart disease risk than soybean oil. In a very recent study, Gouk et al. (2013) [16] fed mice with POo, chemically interesterified palm olein (IPOo), and soy bean oil and found that mice receiving the soy bean oil diet gained significantly higher amounts of subcutaneous fat and total fat compared with the POo group, despite similar body mass gain being recorded. Mice fed with the IPOo diet gained 14.3% more fat per food consumed when compared with the POo group, despite their identical total fatty acid compositions. The authors attributed this observation to the higher content of long chain SFA at the *sn*-1, 3 positions of TAG in POo. In a subsequent paper, Gouk et al. (2014) [17] also reported that SFA of different chain length at *sn*-1 and -3 positions exert profound effects on fat accretion.

In recent years, the hamster, especially the golden Syrian hamster and gerbil have been used extensively as animal models to elucidate sterol synthesis and LDL-C metabolism. Using them, the effects of dietary fatty acids and dietary cholesterol on plasma cholesterol synthesis and LDL-C metabolism in vivo were investigated. In the hamster model, dietary cholesterol feeding induced significant changes in plasma TC and LDL-C, whereas these parameters were relatively unaffected in rats. Several studies with palm oil included as dietary fat have been carried out and are examined below.

Khosla et al. (1997) [18] examined the effects of dietary oleic and palmitic acid on plasma lipids and lipoprotein metabolism in hamsters fed purified diets with low cholesterol but different quantities of fat (low fat 20%E and high fat 40% E, with constant levels of myristic and linoleic acids). Increasing dietary fat from 20%E to 30%E, and 40%E, by increasing oleic or palmitic acid had no effects on plasma lipid or lipoprotein cholesterol. The similarity in plasma and lipoprotein cholesterol levels was further confirmed by kinetics studies using radio-labeled native/methylated LDL-C or HDL-C. Consistent with circulating LDL-C and high density lipoprotein cholesterol (HDL-C) concentrations, there were also no differences in the clearance of LDL-C and HDL-C.

Wilson et al. (2005) [19] compared the effects of different palm oil preparations (RPO, RBDPO, and RBD + RPO-PO) with coconut oil on plasma cholesterol concentrations and aortic cholesterol accumulation in hypercholesterolemic hamsters. The hamsters fed the three palm oil preparations had lower plasma TC and non HDL-C, but higher HDL-C concentrations while accumulating less aortic cholesterol compared with the coconut oil-fed hamsters.

van Jaarsveld et al. (2002) [20] assessed the effect of POo in a moderate fat diet on the plasma lipoprotein profile and aortic atherosclerosis in a non-human primate model after 25.5 months of dietary exposure. The vervet monkeys fed a moderate fat/moderate cholesterol diet (MFD) with 11%E from lard, POo, and sunflower oil (SFO) had a polyunsaturated/saturated (P/S) ratio of 0.4 for 24 months. Plasma lipids were measured at 6-monthly intervals and atherosclerosis was assessed in the aorta and arteries after 25.5 months of dietary exposure. POo, relative to SFO and lard, significantly reduced the risk of developing early lesions in the peripheral arteries. Therefore, in this primate model of atherogenesis, the isocaloric substitution of lard with POo seems beneficial.

Kritchevsky et al. (2000a) [21] compared the atherogenic effects of refined, bleached, and deodorized (RBD) palm oil with those of randomized RBD palm oil. The RBD palm oil contained 41.2% palmitic acid, of which 2.6% was at the *sn*-2 position. In the randomized palm oil, 13.6% palmitic acid was at the *sn*-2 position. The randomized palm oil was significantly more atherogenic for rabbits than was the RBD palm oil.

In a different study, Kritchevsky et al. (2000b) [22] showed that increasing the amount of palmitic acid at the *sn*-2 position of a fat led to an increased atherogenic effect. Rabbits were fed with four synthetic fats with triglyceride structures of SOS, SSO, POP, and PPO (S = stearic acid, P = palmitic acid, O = oleic acid) and were evaluated for their atherogenic potential. The fats were incorporated into semisynthetic diets

containing 15% fat, of which 58% was the synthetic fat, 24% was sunflower oil, and 18% was high-oleic safflower oil. All of the diets contained 0.05% cholesterol and the rabbits were fed for 20 wk. The blood lipid levels (TC, % HDL, and triglycerides) were similar in all four groups. Average atherosclerosis was similar in rabbits fed SOS compared to SSO (2.4 or 28% of stearic acid in the *sn*-2 position, respectively); however, it was much greater in rabbits fed PPO compared to POP. The authors noted that these findings confirmed their earlier observations that randomization of fats affects their atherogenicity but not their lipidemic effects. Specifically, fats bearing palmitic acid (but not stearic acid) in the *sn*-2 position have increased atherogenicity compared to fats with palmitic acid in the *sn*-1 or *sn*-3 positions.

3.2 Human studies

3.2.1 Palm oil versus saturated fats

While there is general perception that SFA (C12:0–16:0) increase TC and LDL-C in comparison with MUFA and PUFA, several studies on normocholesterolemic and hypercholesterolemic subjects have established that C16:0 from palm oil elicits significantly lower plasma cholesterol responses compared to C12:0- and C14:0-rich diets and have similar effects as C18:0 (Table 2). A study by Ng et al. (1991) [23] found that feeding a moderate fat diet (31%E from fat) rich in C12:0 + C14:0 for 4 wk to 31 healthy normocholesterolemic subjects resulted in a significant increase in serum TC, LDL-C, and HDL-C over baseline levels. However, when the subjects were switched to a C16:0-rich diet, these levels were reduced significantly. In addition, Zock et al. (1994) [24] reported that C14:0 is about 1.5 times more cholesterol raising than C16:0. Sundram et al. (1994) [25] demonstrated that by exchanging 5%E of C12:0 + C14:0 to C16:0 resulted in a significant reduction (9%) in serum TC, primarily a 11% reduction in LDL-C and a more moderate decrease in HDL-C compared to the C12:0 + C14:0 rich diet.

Furthermore, studies comparing C16:0 with C18:0 demonstrated similar effects on the lipid profile. Nestel et al. (1998) [26] compared the effects on plasma lipids of a C18:0-rich diet versus C16:0 in hypercholesterolemic subjects and demonstrated that plasma TC concentrations with the low-fat, C18:0-rich, and the C16:0-rich diets were not significantly different but lower than those measured during the habitual diet period. Neither HDL-C nor plasma TAG differed significantly among the three study diets. Kelly et al. (2002) [27] concluded that there is no significant differences between the two diets enriched in C18:0 and C16:0 in plasma lipoprotein concentrations. Based on the food items, the diet enriched in C16:0 was a mixture of fats with triglyceride structures of POP and POo where palmitic acids were esterified at *sn*-1 or *sn*-3 positions which have relatively neutral effects on cholesterol levels.

Saturated fatty acids with chain lengths C12:0–C14:0 produce a detrimental effect on blood lipids compared to C16:0 in healthy normocholesterolemic young men. C16:0 may be similar to C18:0 in its effects on plasma lipids subject to the fatty acids occupied in the *sn*-2 position.

3.2.2 Palm oil versus unsaturated oils

Several studies have shown that in healthy, normocholesterolemic humans, dietary C16:0 can be exchanged for C18:1 without adverse effects on serum lipid or lipoprotein levels. Ng et al. (1992) [11] and Choudhury et al. (1995) [12] evaluated POo against olive oil on serum lipids and lipoproteins (Table 3). A study by Ng et al. (1992) [11] exchanged 7%E between C16:0 and C18:1 (with energy from

Table 2. Summary of human dietary intervention studies: palm oil versus saturated fats

Reference	Subjects (*n*)	Age (*y*)	BMI (kg/m2)	Design	Dietary fatty acids (%E)						Cholesterol (mg)	Lipids (mmol/L)			
					Total fat	12:0	14:0	16:0	18:0	18:1		TC	TAG	LDL-C	HDL-C
Nestel et al. (1998)	12 men, 8 women	51 ± 7	26.2 ± 3.9	RCT, 2 wk run in, 5 wk	41	-	–	13.1	2.5	21.3	0	5.5	1.6	3.7	1.1
					42			3.8	14.3	20.2	0	5.4	1.5	3.7	1.1
Sundram et al. (1994)	17 men	19–21	20.1 ± 1.8	RCT, 3 wk run in, 4 wk	30.7	0.8	1.2	11.1	1.5	10.8	<200	4.0	1.1	2.4	1.1
					30.5	4.6	2.7	6.9	1.1	10.3		4.4*	1.0	2.7*	1.2
Zock et al. (1994)	36 women, 23 men	18–62	17.9–32.4	RTC, 3 wk	39.2	0.3	1.1	14.9	4.1	11.6	<400	5.0	1.0	3.0	1.5
					39.6	0.4	11.3	4.7	4.3	10.9		5.2*	1.0	3.1*	1.7*
Ng et al. (1991)	27 per group	<30	<26	Parallel, 5 wk	30.0	0.3	0.5	11.6	1.5	12.9	<200	4.8	0.9	3.2	1.3
					30.8	11.0	4.8	4.4	1.1	3.3		4.0**	0.9	2.5**	1.1**

*Significantly different from palmitic acid diet ($P < 0.05$).
**Significantly different from entry levels ($P < 0.05$).

Table 3. Summary of human dietary intervention studies: palm olein versus olive oil

Reference	Subjects (*n*)	Age (*y*)	BMI (kg/m2)	Design	Dietary fatty acids (%E)				Cholesterol (mg)	Lipids (mmol/L)			
					Total fat	16:0	18:1	18:2		TC	TAG	LDL-C	HDL-C
Choudhury et al. (1995)	12 males, 12 females	19–44	<25	RCT, 30 days	30	12.1	13.0	3.2	175	4.7*	1.0*	3.3	0.9*
					31	3.3	24.1	2.0	194	4.6*	1.0*	3.4	0.8*
Ng et al. (1992)	20 males, 13 females	22–41	<28	RCT, 6 wk	34	6.3	21.4	2.6	<200	4.9	1.2	3.3	1.1
					34	13.4	13.8	3.5		4.9	1.2	3.4	1.1
Voon et al. (2011)	9 males, 36 females	30.1 ± 8.3	23.1 ± 3.7	RCT, 5 wk	30.6 ± 2.3	9.7	12.3	4.0	<300	4.8	0.85	3.2	1.3
					31 ± 2.8	4.8	19.1	3.5		4.7	0.84	3.1	1.3

*Significantly different from entry levels ($P < 0.05$).

C18:2 maintained at 3%) and resulted in identical serum TC, LDL-C, HDL-C, and TAG levels. Truswell et al. (1992) [28] reported that HDL-C were 8% lower on canola oil as compared to POo. Similarly, a study by Choudhury et al. (1995) [12] reported that substituting 5%E from C18:1 with C16:0 elicited similar plasma TC, LDL-C, and HDL-C levels. Voon et al. (2011) [29] reported that POo and olive oil have similar effects on blood lipid profile. Hence, in a one-to-one exchange basis, POo demonstrated similar TC, LDL-C, HDL-C, and TAG levels with olive oil in healthy normolipidemic subjects. Sundram et al. (1995) [30] reported that exchange of 4%E between C16:0 and C18:1 while maintaining 6%E from C18:2 resulted in similar plasma lipid levels. Oleic acid has been proven neutral in its cholesterolemic effect. However, the optimum requirement for oleic acid to confer beneficial lipoprotein profiles has not yet been ascertained. In this context, POo containing 44–48% oleic acid was equal in its plasma cholesterol and modulating lipoprotein effects to the higher oleic acid-containing oils, such as olive [2]. Table 4 presents the percentage of total and *sn*-2 FAC of palm oil and olive oil.

Scoltz et al. (2004) [31] evaluated the effect of consuming 33–38%E POo with sunflower oil in healthy subjects (Table 5). POo significantly increased serum TC and LDL-C compared to sunflower oil. The relatively high linoleic acid content (24%E) in SFO may have contributed to its beneficial effect. In contrast to this finding, palm oil was comparable to peanut oil in serum lipids profile in a group of mildly hypercholesterolemic Chinese subjects [32] and in a healthy Indian population [33]. In addition, Marzuki et al. (1991) [34] reported that consumption of POo elicited similar effects on TC, LDL-C, and HDL-C in comparison to a soybean oil diet. The soybean oil diet, however, increased TAG compared to a POo diet. Ng et al. (1991) [23] reported a decrease in TC, LDL-C, and an increase in HDL-C in subjects fed a diet enriched with POo and corn oil at 30%E fat in comparison with baseline values.

In comparison with diets enriched with peanut, corn, or soybean oils, POo appeared comparable in its ability to modulate lipids and lipoproteins. Studies that have inferred a hypercholesterolemic effect of C16:0 may be associated with the dietary fat content, cholesterol load, and the metabolic status of the subjects. It has been hypothesized that C16:0 is only hypercholesterolemic in situations in which the LDL receptors are down regulated [35]. In addition, Hayes and Khosla (1992) [35] also concluded that C16:0 has a variable cholesterolemic effect, where in normocholesterolemic subjects (TC < 225 mg/dL) consuming dietary cholesterol <300 mg/day, it has no impact on plasma cholesterol with the recommended intake of 18:2n-6 [36, 37]. Apart from the fatty acid composition, the minor components in POo, especially tocotrienols and other phytonutrients, have been reported to exert their antioxidant properties by modulating cholesterol levels [38]. These findings merit the re-evaluation of palm oil as a cholesterol-raising fat.

3.2.3 Palm oil versus hydrogenated oils

Compelling data have linked dietary *trans* fatty acids to increased risk of cardiovascular heart disease [39–41]. Evidence from large epidemiological studies, involving 667 to 80,082 men and women in different age groups, followed

Table 4. Total and *sn*-2 fatty acid composition (fac) of palm oil and olive oil (%)

Type of oil	Fatty acid	Total FAC (%)	FA in sn-2 position (%)
Palm oil	C16:0	44.3	11
	C18:0	4.6	2
	C18:1	39	65
	C18:2	10.5	22
Olive Oil	C16:0	13.1	1.4
	C18:0	2.6	–
	C18:1	71.8	82.9
	C18:2	9.8	14

Source: A. S. H Ong and S. H. Goh, 2002 [2].

Table 5. Summary of human dietary intervention studies: palm oil versus polyunsaturated oils

Reference	Subjects (*n*)	Age (*y*)	BMI (kg/m2)	Design	Dietary fatty acids (%E)				Cholesterol (mg)	Lipids (mmol/L)			
					Total fat	16:0	18:1	18:2		TC	TAG	LDL-C	HDL-C
'Scholtz et al. (2004)	18–20 males + females	21–59	<30	RCT, 4 wk	33–38	13.5 2.2	13.8 6.9	3.7 24.2	207–224	5.6 5.0**	1.6 1.3	3.6 3.3**	1.1 1.0
Zhang et al. (1997)	Groundnut oil: 15 males, 11 females Red palm oil: 16 males, 9 females, mildly hypercholesterolemic	32–68	–	Parallel, 6 wk	30 30.2	9.8 4.8	11.6 11.9	5.9 9.5	163 166	5.7* 5.9	1.4 1.3	4.1* 4.4	1.3 1.2
Ghafoorunissa et al. (1995)	12 per group	29–52	16–30	RCT, 8 wk, 6 wk washout	32	12.0 6.0	11.7 11.4	3.4 7.1	<100	4.4 4.3	0.8 0.7	2.5 2.5	0.8 0.7
Ng et al. (1991)	Palm oil: 20 males, 7 females Corn oil 19 males, 7 females	20–34	Palm: 19.5 ± 2.0 Corn: 19.4 ± 2.3	Parellel, double-blind, 5 wk	30	11.3 6.7	12.6 9.8	3.3 10.6	~200	4.0* 3.2*	0.9 0.9*	2.5* 1.8*	1.1* 1.0*
Marzuki et al. (1991)	110 healthy males	16–17	–	RCT, 5 wk, 6 wk washout	36 34	13.8 3.6	15.6 7.3	3.9 18.9	343 342	3.9 4.0	0.7 1.1**	2.4 2.4	1.3 1.3

*Significantly lower from entry diet ($P < 0.05$).
**Significantly different from palm oil diet ($P < 0.05$).

over 6 to 20 years, consistently found a positive association between *trans* fatty acid intake and risk of cardiovascular heart disease [42–46]. Controlled feeding dietary intervention studies have demonstrated that *trans* fatty acids intake compared to *cis*-unsaturated fats and saturated fats increases LDL-C concentrations, reduces HDL-C concentrations, which consequently increases the TC:HDL-C ratio, a better predictor of cardiovascular heart risk [47]. The recent interim report from FAO/WHO Expert Consultation also stated that there is convincing evidence that intake of *trans* fatty acid decreases HDL-C and increases the TC:HDL-C ratio in comparison to SFA (C12:0-C16:0), cis MUFA and PUFA. Mozaffarian et al. (2009) [41] also reported that a 2% increase in energy intake from partially hydrogenated fats or *trans* fatty acids was associated with a 23% increase in the incidence of cardiovascular heart disease. Current evidence also suggests that *trans* fatty acids may be implicated in the risk of sudden cardiac death and metabolic syndrome components.

The increasing use of artificially produced *trans* fats in foods therefore drew grave concern from the public, and led to stricter regulatory measures globally. Consequently, the US Food and Drug Administration required mandatory *trans* fatty acids labelling on packaged foods from January 1, 2006, prompting food manufacturers to find alternatives to commercially-hydrogenated vegetable oils for bakery products, margarines, and fried foods. In this context, palm oil provides the best natural replacement for commercially-produced hydrogenated vegetable oils, as it is naturally solid.

Sundram et al. (1997) [48] demonstrated that a 5.5%E intake of *trans* fatty acid (elaidic acid) was more deleterious than a greater intake of palmitic acid (11%E) contributed by POo (Table 6*)*. The finding showed that *trans* fatty acids increased TC, LDL-C, Lp(a) but decreased HDL-C. In contrast with this finding, other researchers have shown that palmitic acid and *trans* fatty acids elicited identical effects on TC and LDL-C, but not on HDL-C [49, 50]. Our laboratory recently conducted a randomized crossover intervention to investigate the effects of a high oleic POo versus partially hydrogenated soybean oil and palm stearin diets [51] in a group of healthy individuals. The *trans* fatty acids-rich diet significantly increased the TC:HDL-C ratio (a robust risk marker for CVD risk) compared to high oleic POo. In addition, *trans* fatty acids were also found to increase serum high-sensitivity C-reactive protein (hsCRP) in comparison with high oleic POo and palm stearin. hsCRP is recognized as a novel inflammatory marker for CVD risk. From our findings, we support the use of vegetable oils, such as palm oil, in their natural state over one that has undergone hydrogenation for modulating blood lipids and inflammation.

4 Minor components in palm oil and their health benefits

Palm oil is a rich source of beneficial phytonutrients, which are present to 1% of its weight. The most prevalent are tocols (600–1000 parts per million (ppm), carotenes (500–700 ppm), phytosterols (300–620 ppm), squalene (250–540 ppm), coenzyme Q10 (10–80 ppm), polyphenols (40–70 ppm), and phospholipids (20–100 ppm) [52, 53].

Seventy percent of the vitamin E in palm oil occurs as tocotrienols and the remainder as tocopherols [54]. The

Table 6. Summary of human dietary intervention studies: palm oil versus hydrogenated oils

Reference	Subjects (*n*)	Age (*y*)	BMI (kg/m2)	Design	Dietary fatty acids (%E)					Cholesterol (mg)	Lipids (mmol/L)			
					Total fat	16:0	18:1t	18:1	18:2		TC	TAG	LDL-C	HDL-C
Teng et al. (2010)	33 female, 8 male completed	28.8 ± 9.1	21.9 ± 3.9	RCT, 5 wk, 1 wk washout	33.5	8.7	nd	15.3	5.8	<300	4.48	0.83	2.69	1.63
					32.3	8.1	9.9	7.2	3.9		4.72*	0.89*	3.11*	1.42*
					31.7	13.9	nd	10.7	3.6		4.66*	0.88*	2.95*	1.55*
Pedersen et al. (2005)	30 female, 27 completed	19–42	26.5 ± 4.1 (20–36)	RCT, 17 days, 1 wk washout	31.0	10.5	Nd	11.7	5.0	86	4.74	0.90	2.90	1.47
					30.1	3.3	6.8	10.6	4.1	56	4.61	0.92	2.88	1.32
Sundram et al. (1997)	20 men, 9 women, 27 completed	19–39	22.7 ± 2.6 (19–30)	RCT, double-blind, 4 wk	31.7	11.4	nd	13.7	3.3	207	4.9	0.9	3.2	1.3
					31.6	4.6	6.9	10.8	5.3	210	5.2*	0.8	3.8*	1.1
Nestel et al. (1992)	27 mildly hypercholesterolemic men	46.8 ± 9.6 (30–63)	80.2 ± 8.9	RCT, 3 wk	37.0	9.8	<1	12.9	5.7	186	226	128	161	42
					37.0	4.9	5.7	11.3	6.6	168	229	142	165	38

Nd, not detectable.
*Significantly different from palm oil.

structure of tocotrienol and tocopherol are shown in Fig. 3. Both tocotrienol and tocopherol have four isomers each and the former has three double bonds in its isoprenoid side chain. Tocotrienols are unique as they are able to penetrate tissues with saturated fatty layers freely thus performing more efficient metabolic function than tocopherols. Thus, tocotrienols are more powerful antioxidant than tocopherols. MPOB has successfully isolated individual tocotrienol isomers from palm oil by using supercritical fluid chromatography [55] which provides high purity products through green and environmentally friendly processes. Each individual tocotrienol isomer (α-, β-, γ-, δ-) has unique beneficial properties. Accumulation of tocotrienols in tissue imparts tremendous health benefits [56]. Tocotrienols could help reduce blood cholesterol [57–59] and arteriosclerotic functions [60–62], encompass possible anti-angiogenic functions [63–66], exhibit efficient antioxidant activity [67–69] as well as anticancer [58, 70–77] anti-inflammatory effects [78], prevention of arthritis [79], prevention of osteoporosis [80, 81], prevention of skin diseases [82–84], anti-diabetic [85, 86], and neuroprotective properties [87–89].

Carotenoids are natural pigments responsible for the brilliant orange-red colour of palm oil. About 600 types of naturally occurring carotenoids are known but only 13 found in palm oil. Among them, the major ones are in the form of β-carotene, α-carotene, lycopene, phytoene, and phytofluene. Crude palm oil is considered one of the world's richest sources of carotenoids and contains 500–700 ppm. Carotenoids from commercial crude palm oil are concentrated during extraction and fractionation [90] as shown in Table 7.

Carotenoids act as precursors of vitamin A which is required to prevent night blindness [91], improve the vitamin A status of lactating women and their infants [92, 93] improve serum retinol concentrations [94] and combat vitamin A deficiency [95, 96]. Carotenoids can also protect against cardiovascular diseases [97] and suppress the growth of various cancer cells such as breast [98–101], lung, and liver as well as colon tumors. Carotenoids have been shown to enhance cell to cell communication in exerting their anticancer properties [100].

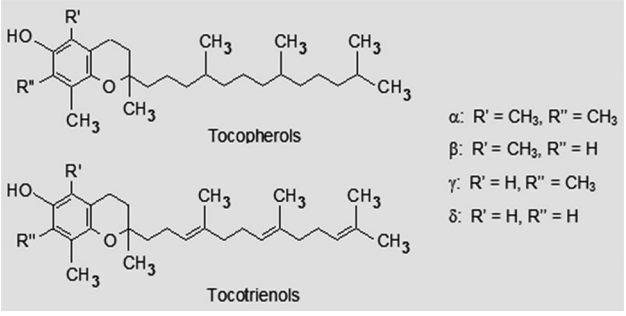

Figure 3. The structure of tocopherol and tocotrienol molecules (adapted from The AOCS Lipid Library).

Table 7. Carotenoid content of various palm oil fractions [89]

	Concentration (ppm)
Crude palm oil	630–700
Crude palm olein	680–760
Crude palm stearin	380–540
Residual oil from fiber	4000–6000
Second-pressed oil	1800–2400

Coenzyme Q_{10}, also known as ubiquinone, is a natural coenzyme in palm oil. It is claimed to possess ten times greater antioxidant property than vitamin E, although the carotenes and vitamin E in their far greater concentrations mask its functionality [102]. Besides being a powerful antioxidant and free radical scavenger [103], Coenzyme Q_{10} also plays a vital role in the mitochondrial electron transport chain and has been shown to exhibit membrane stabilizing properties. It has been used in the treatment of many cardiovascular ailments [104–105] and studies have also demonstrated its anticancer effects [106].

Squalene is a valuable triterpene enormously found in shark liver oil. It is present in trace amounts in palm oil. Squalene is an oxygen transmitter and can aid cardiovascular health [104, 105]. It also has reported antitumor activity in rodents [107], suppresses hyperproliferation of cancer cells [107–110] in addition to exhibiting radioprotective effects [107].

Phytosterols are naturally-occurring substances in all plants and plant-based raw materials in foods. The major phytosterols in crude palm oil are β-sitosterol, campesterol, and stigmasterol. The main interest in palm phytosterols is their cholesterol lowering properties [111–113]. Besides research has also proven that they possess anticancer properties [114] and enhance immune functions [115]. Based on studies carried out at MPOB, palm fruits can serve as an inexpensive source of phenolic antioxidants, the market for which is currently monopolized by grape seed and tea extracts. Although the fat-soluble components have been receiving considerable attention, relatively little importance is given to the water-soluble components of palm oil. Polyphenols are a large family of natural compounds that can be classified as phenolic acids and flavonoids. The major palm phenolics (OPP) include p-hydroxybenzoic, cinnamic, ferulic and coumaric acids, and the flavonoid rutin hydrate [116, 117].

Flavonoids are touted as the most potent free radical scavenger and ion chelator. Phenolics, on the other hand, act as free radical terminators [118]. MPOB has developed and patented a breakthrough process [119] to recover the concentrations of OPP from palm oil mill effluent (POMEI). These compounds are also known to possess anti-carcinogenic [120–122] and cardioprotective properties [114].

Phospholipids form the main building block in all living forms. The main phospholipids in palm oil are

phosphotidylcholine, phosphotidylethanolamine, phosphotidylinositol, and phosphotidylglycerol [123]. Phospholipids are essential components of lipoproteins and biological membranes [124] and they are essential for enhancing brain function [125], energy endurance [124, 126], structural integrity of cells as well as easing digestion and nutrient absorption [126].

5 Conclusions

To date, our extensive nutritional studies have shown that palm oil with high monounsaturation at *sn*-2 position is comparable to monounsaturated oils (e.g., olive, groundnut, and canola oils) in its effect on lipid profile. New concepts related to the cholesterol saturated fat hypothesis have slowly emerged from these studies. The interactive roles of the fatty acids and minor components in palm oil have also contributed significantly to nutritional science. Coupled to this factor is the well-known cost effective attributes of palm oil and its compatibility in various food formulations. All these suggest that palm oil will continue to play a leading role in the world oils and fats market with much greater acceptance among consumers.

CYM is the Director-General of the Malaysian Palm Oil Board and this review article is by invitation for the EuroFedLipid Highlights 2014, October 2014. KN is the Regional Manager for Malaysian Palm Oil Board, Europe Regional Office in Brussels, Belgium.

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