Palm oil – widely consumed, sadly misunderstood

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The oil palm, ***Elaeis guineensis***, has come a long way since it was introduced by the British in 1870[1] as an ornamental plant in Malaya. The fruit is unique in that it produces two types of oil – palm oil from the outer orangy mesocarp and palm kernel oil from the kernel of the fruit. Both these two types of oils have very different physico-chemical properties but it is palm oil, or rather its liquid fraction- palm olein, which is consumed in much greater amounts and making a significant contribution to meeting the growing world's demand for fats and oils. This had attracted the envious attention of producers of other major edible oils from different nations, as well as featuring palm oil in human interventions to investigate the impact of the oil on the cardiovascular risk profile by scientists worldwide for the past three decades.

First the good news! Palm oil was once described as "Nature's gift to the world" for the oil is very versatile and has a myriad of food uses including cooking oils, margarines, shortenings, cocoa butter equivalents, etc. Interestingly, palm oil possesses two unique properties not found in other common edible oils or saturated fats. Firstly, about 90% of its 16:0 is located at the outer fatty acid arms (sn-1 and sn-3 positions) of the fat molecule with only 10% at the middle or sn-2 position. Secondly, its vitamin E content comprises two-thirds of tocotrienols – tocols with funny looking unsaturated tails which confer beneficial effects including strong biological antioxidant action, anti-cancer effects and neuroprotection!

Now the bad news! The general perception about saturated fats by most scientists and laymen for more than a generation is that they are bad for health, they cause heart disease. Palm oil is traditionally regarded as a saturated fat because it contains about 40% of the saturated palmitic acid (16:0) but only 10-12% of the omega-6 essential polyunsaturated linoleic acid (LA, 18:2) and 0.4% of the omega-3 alpha-linolenic acid (ALA, 18:3). It was for this reason and others of low scientific merit that palm oil was lumped together with the infamous coconut oil in the mid-1980s, given the tag "tropical oils" and dubiously named the main villains in causing blockage to arteries and subsequently heart attack and stroke in western populations which by the way, hardly consumed palm oil at the time. Matters did not help after Professor Ancel Keys published his Seven Countries Study[2] which showed a positive association between the consumption of saturated fats and cardiovascular heart disease (CHD) in different national populations. This caused a saturated fats and cholesterol phobia across the globe which lasted till the present day.

Are all saturated fats the same? The author would say "Definitely not". Their physiological/biochemical effects differ depending mainly on five major factors namely, i) dietary level, ii) dietary source (vegetable or animal), iii) relative content of the blood cholesterol-raising fatty acids – lauric (12:0), myristic (14:0) and palmitic (16:0) and *trans* fatty acids, iv) stereospecificity of 16:0 on the fat molecule, and v) content of minor components with health benefits. Professor Gerald Hornstra from Maastricht University, Netherlands was the first to notice from laboratory experiments in 1973 that palm oil did not behave like a saturated fat when he plotted sum of saturated fatty acids (SFA) versus Obstruction Time (OT) in his intriguing "arterial loop" model in the rat.[3] He further highlighted in 1989[4] that when palm oil replaced the major dietary fat in several western studies,[5-9] palm oil did not raise blood cholesterol but in fact lowered it by 7% to 38%. Similarly, Heber *et al*.[10] observed that when palm oil was added at 17.5% kcal or some 20-fold the usual palm oil content in the typical American diet, blood cholesterol levels were not elevated in the adult subjects. However, these hypocholesterolemic effects observed for palm oil were obtained by comparing with "at entry levels" which strictly could not be considered as co-variates. Fine, let us look at other evidences available.

Soon afterwards during 1991-1992 while working at the Institute for Medical Research (IMR) in Kuala Lumpur, the author had published in prestigious American Journals that palm oil had normocholesterolemic effects in healthy Malaysian adults[11] and had comparable non-hypercholesterolemic effects as virgin olive oil – the

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main dietary oil in the Mediterranean diet.[12] Similar normocholesterolemic findings for palm oil versus olive oil were later reported by scientists at the Human Nutrition Unit of the University of Sydney.[13]

How do observed cholesterolemic responses compare with responses predicted by the Hegsted regression in palm oil intervention studies? To answer this question, Professor Hayes and co-workers[14] from Brandeis University rotated 24 monkeys from 3 species through 5 purified diets containing 31% energy as various palm-fat blends for 12 weeks. They plotted the observed plasma cholesterol changes against the predicted responses by the Hegsted Equation[15] and obtained a significant Pearson correlation of r=0.85. However, when palm palmitic acid was omitted from the Hegsted Equation (i.e. palm 16:0 was regarded as neutral), a near-perfect correlation fit was obtained with r=0.98. Clearly, the classic regression equation fails to recognise the non-linear blood cholesterol response associated with dietary 18:2 intake. Hayes and Khosla (1994)[16] proposed the 'linoleic acid threshold" hypothesis to help explain the above findings. What this means is that when the polyunsaturated LA (18:2) is consumed at about 6% kcal, there is a maximum LDL-receptor activity and therefor maximum LDL cholesterol-lowering effect; at this dietary level of LA, it does not really matter what type of SFA are in the diet.

By the 1990's, it was apparent that human feeding trials conducted in Malaysia, China, The Netherlands and the United States of America (USA) had indicated that dietary palm oil did not behave like a saturated fat in that it did not obey well the Hegsted Equation[15] nor the Keys Equation (1957)[17] often used to predict changes in plasma cholesterol brought about by changes in dietary fats. However, getting the consensus of the global scientific community on this matter is another thing! This was because some palm oil feeding studies conducted on Caucasians raised blood cholesterol! How could you explain this- getting different results with the same test fat in different populations? Well, the experts had these to say. Firstly, overweight/obese subjects or those habitually consuming high-cholesterol diets (>400 mg cholesterol/day) have down-regulated LDL-receptor status and therefore impaired clearance of circulating LDL-cholesterol. Thus individuals with different "anthropomorphic" characteristics or LDL-receptor activity status/set-point would respond differently to the same test fat challenge.[18] Secondly, individuals with different apolipoprotein E phenotype have different cholesterolemic responses. People carrying the e-4 allele are at greater risk to CHD, as well as exhibiting a greater response to a LDL-lowering diet compared to subjects with the e-3 or e-2 allele.[19]

In the December 2013, International Palm Oil Congress (PIPOC 2013) in Kuala Lumpur, the author had reported a joint meta-analysis by University of Malaya-Malaysian Palm Oil Board (MPOB)- International Medical University (IMU) which showed that palm oil had neutral cholesterolemic effects. However, an Italian research team beat us to the Press and published a meta-analysis in the American Journal of Clinical Nutrition in 2014[20] which reported similar normocholesterolemic effects for palm oil. Any more skeptics?

A meta-analysis of 21 prospective cohort trials by Siri-Tarino *et al.*[21] from the Harvard School of Public Health reported that saturated fats (SATs) do not pose a risk to ischaemic heart disease (relative risk, rr =1.07) and surprise, surprise – SATs were found to be protective against stroke (rr = 0.81)! This means that scientists worldwide should now get their perspectives for saturated fats, including palm oil, right.

The author is of the opinion that if we were to remove all saturated fatty acids from our diet today, and consume a diet containing only polyunsaturated and monounsaturated fatty acids, proteins and carbohydrates, we will become sick in a matter of weeks. Without the saturated fatty acids in our diet to "balance things out", we will be subjected to aggressive attack on unsaturated fatty acids within our tissues by reactive

oxygen species (ROS – by-products of our body's aerobic respiration), putting us in great oxidative stress. How long can our bodies withstand this devastating onslaught of ROS before we finally succumb to ROS-induced chronic diseases, we will never really know because no Research and Ethics Committee will ever approve a human clinical trial to investigate this outcome!

Keywords: Palm oil, saturated fats, blood cholesterol, cardiovascular risk

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