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Replacing coconut santan with palm oil santan: impact on dietary C12-16 saturated fatty acids, serum total cholesterol, and cardiovascular risk

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ABSTRACT

The theoretical impact of the use of coconut cream (*santan*) powder and palm oil *santan* powder on the dietary levels of C12-16 saturated fatty acids (SFAs) and linoleic acid (18:2), and on serum total cholesterol (TC), was evaluated holding non-*santan* dietary variables constant. The prediction was based on a 2,300-kcal hypothetical diet, containing one *santan*-based dish or snack in each of the 5 daily meals with fat contributing 30% of total calories, while the *santan* contributed a total of 14% kcal (36g). Replacing coconut *santan* with palm oil *santan* reduced the overall dietary C12-16 SFAs from 10.8% kcal to 4.8% kcal (i.e. –6.0% kcal) and the virtual removal of lauric (12:0) + myristic (14:0) acids, while palmitic acid (16:0) rose by 3.3% kcal, and the polyunsaturated linoleic acid (18:2) increased by 1.13% kcal. Applying the Hegsted equation to these dietary fatty acid (FA) changes, predicted a serum TC reduction of 24 -31 mg/dL (0.62- 0.80 mM/L), with the hypocholesterolemic effect being influenced by the low-density lipoprotein receptor (LDL_r) "set-point" of the individual(s) concerned. Thus, the prediction indicated that replacing coconut *santan* with palm oil *santan* in *santan*-based Malaysian dishes or snacks would have a significant beneficial impact on serum TC and hence, cardiovascular risk.

INTRODUCTION

Coconut *santan* has been regarded as an indispensable ingredient in several traditional popular local foods such as *"nasi lemak", "kuih talam", "kuih dadar"* and numerous *santan*-based curry dishes, which would please even the most discerning Asian palate. Malaysian homemakers still use *santan* extracted from freshly grated coconut for their cooking requirements, although many also use alternative stable forms of coconut cream such as coconut *santan* powder (*"serbuk*

santan kelapa").

Proximate composition analysis showed that *santan* extracted from grated coconut, consists of about 29% fat, 3% protein, 3% carbohydate (CHO), and 65% moisture (Tee et al., 1997). Commercially-available coconut *santan* powder, on the other hand, has about twice the level of fat and protein, and much higher levels of CHO. In the case of a popular brand, one pack of coconut *santan* powder (50g)- the equivalent of one coconut, would need to be stirred into 150 ml of warm water to obtain a thick coconut cream for use in cooking.

The highly saturated nature of coconut fat is contributed mainly by the C12-14 SFAs, viz. 41-49% of 12:0 and 20-24% of 14:0, with 16:0 at 9-14%, and a very low level of 18:2 (<2%) [Ng & Chong, 1979]. The consumption of coconut fat has been associated with increased atherogenicity, i.e. elevated plasma low-density lipoprotein cholesterol (LDLC) levels (Ng et al., 1991 & 1992) and increased arterial thrombosis tendency (Hornstra et al., 1975). Consequently, the consumption of coconut *santan* has been discouraged while the use of skim milk as a *santan* substitute has been advocated in healthy recipes of "*Recipe Sihat"* (Tee et al., 1996*)*- a booklet produced in support of the Healthy Eating Campaign launched by the Ministry of Health Malaysia in 1997.

Recently, the production of palm oil *santan* powder by the Palm Oil Research Institute of Malaysia (PORIM) has attracted much attention from the Malaysian authorities, mainly because of the economic potential and the beneficial health implications when the product replaces coconut *santan* in the Malaysian diet. Palm oil-based and coconut *santan* are reported to have similar proximate compositions, while sensory evaluation studies conducted by PORIM on curries prepared with both types of *santan* have yielded comparable scores in terms of appearance, colour, aroma and taste (Zaida et al., 1997). Palm oil *santan* is, however, not yet commercially available.

This article evaluates the change in the dietary C12-16 SFA profile when palm oil *santan* replaces coconut *santan* in a hypothetical diet comprising 5 *santan*-based meals (viz. breakfast, mid-morning snack, lunch, evening `tea', and dinner), and predicts the serum TC response arising from the change in dietary FA profile by applying the Hegsted equation (1965).

Methodology

Amounts of santan used

The amounts of santan used to prepare the dishes and snacks specified in this article were estimated by the present authors from the menus provided in the cook books published by Tiga Noor Publications (1995) and Her World Cook Book (1995). The hypothetical diet used was designed to contain one *santan*-based dish or snack for each of the 5 daily meals so as to enable the detection of any potential difference in the cholesterolemic effect of the two types of *santan*. It is recognised that the hypothetical diet

was not meant to represent a typical Malaysian diet but was designed solely for the purpose of this evaluation. Some basic assumptions were necessary in the present analysis and these included:

a) Coconut *santan* powder and palm oil *santan* powder have similar proximate composition, i.e. 60% fat, 7% protein, 30% CHO, 1% ash, and 2% moisture (Zaida et al., 1997);

b) One pack of *santan* powder contains 60g of the product, equivalent to one coconut;

c) The amount of *santan* varied with the *santan*-based dish or snack in question. In the present hypothetical diet, the equivalent amounts of *santan* used in the 5 dishes or snacks selected ranged from one-half pack (30g *santan* powder) to 90g *santan* powder ;

d) Each of the 5 meals was shared equally among 5 persons;

e) The diet contained 2,300 kcal, with 30% calories (77g) from fat;

f) Other dietary variables not provided by the *santan* remained constant in the two diets being compared.

Results and Discussion

Dietary 14:0 versus 18:2

The amounts of dietary C12-16 SFAs contributed by coconut *santan*

Table 1: Examples of the *santan*-based dish/snack served during each of the 5 daily meals

Meal (for 5 persons)	Main ingredients of dish/snack
Breakfast	**"Nasi lemak":** 3 cups rice (uncooked), 60g santan powder, 5 fried eggs, 100g fried anchovies, 100g fried groundnuts, 1 cucumber
Mid-morning snack	**"Bengka ubi":** tapioca 600g, 30g *santan* powder, 150g palm sugar ("gula melaka"), 1 egg
Lunch	**Chicken in *santan* curry:** 600g chicken, 60g *santan* powder, 3-4 medium-sized potatoes, curry powder
Evening "tea"	**"Kuih dadar":** Filling- 1 grated coconut, 150g palm sugar; Pancake- 30g santan powder, 150g plain flour, 1 egg, 6 pandan leaves
Dinner	**Beef in *santan* curry:** 600g beef, 60g santan powder curry powder

and palm-based *santan* are shown in Table 2. Both types of *santan* were estimated to provide 36g of fat. For the total diet, coconut *santan* provided 27.6g of the C12-16 SFAs, mainly as lauric and myristic acids. In contrast, the palm oil *santan* provided only 12.1g of the C12-16 SFAs, mainly as palmitic acid (16:0) but virtually no 14:0 or 12:0.

There is compelling evidence to indicate that the impact of dietary FA on serum TC is essentially the result of the action of two "key players", viz. 14:0 (the chief cholesterolemic `villain') versus the PUFA, 18:2, which lowers serum TC (Hayes et al, 1992). We now know that the C12-16 SFAs are not equal in their cholesterol-raising potential; this property for 14:0 has been reported to be four times that of 16:0 (Mensink & Katan, 1992). The cholesterolemic potential of 12:0 is often clouded by the fact that it is

Table 2: C12-16 SFAs from coconut-based vs palm oil-based *santan* used in the preparation of common Malaysian dishes and snacks

Meal	Type of food prepared with *santan* powder	Dietary fat from *santan* (g/person)	C12-16 SFAs from the santan used (g)					
			Coconut *santan*			Palm oil *santan*		
			12:0	14:0	16:0	12:0	14:0	16:0
Breakfast	*"Nasi lemak";* (60g *santan)*	$\frac{60 \times 0.6}{5} = 7.2$	3.6	1.4	0.7	0.01	0.07	3.0
Mid-morning snack	*"Kuih talam"*; (30g *santan)*	$\frac{30 \times 0.6}{5} = 3.6$	1.8	0.7	0.3	trace*	0.04	1.5
Lunch	Chicken curry in *santan* (60g *santan*)	$\frac{60 \times 0.6}{5} = 7.2$	3.6	1.4	0.7	0.01	0.07	3.0
Evening "tea"	*"Kuih dadar"*; 1 grated coconut + 30g santan	$\frac{36 + 18}{5} = 10.8$	4.4	2.2	1.1	trace	0.04	1.5
Dinner	Beef curry in *santan* (60g *santan*)	$\frac{60 \times 0.6}{5} = 7.2$	3.6	1.4	0.7	0.01	0.07	3.0
Total fat from santan (g)		36.0						
C12-14 SFAs from santan (g):			17.0	7.1	3.5	0.03	0.3	12.0
% kcal**			6.6	2.8	1.4	0.01	0.1	4.7

*trace = <0.01g; **Based on a 2300-kcal diet containing 77g (30% kcal) fat

difficult to separate this FA from 14:0, but recent data indicated that 12:0 and palmitic acid (16:0) are interchangeable and comparably neutral in both gerbils and monkeys fed cholesterol-free diets (Khosla et al., 1994).

Neutrality of palm 16:0?

Controversy still surrounds the final verdict on the cholesterolemic impact of 16:0. However, the pool of scientific evidence available suggests that the cholesterolemic potential of 16:0 depends on two important variables, viz. (a) the source of the 16:0; and (b) the low-density lipoprotein receptor (LDL_r) "set-point" of the individual(s) concerned. There is substantial evidence to suggest that palm 16:0 tend to be neutral when the activity of the LDL_r is not compromised, as in the case of a study with lean, active Malaysian adults (Ng et al., 1991), or when dietary cholesterol intakes are low, i.e. <300 mg/day (Hayes et al., 1991). However, 16:0 may have a cholesterol-raising potential when the dietary cholesterol intake is high (>400 mg/day), and in hypercholesterolemic or obese individuals. The conditional aspect of 16:0 cholesterolemia has been reviewed elsewhere (Sundram et al., 1994). The impact of triglyceride structure on lipid metabolism (McGandy et al., 1970; Kritchevsky, 1988) and the contribution of the major triglyceride species in palm oil to its normocholesterolemic effects have also been extensively reviewed (Elson, 1992; Ng, 1994).

14:0 "threshold" and cholesterolemia

From the FA profile reported for typical Malaysian diets (Ng et al., 1991; Ng, 1995), the dietary levels of 14:0 and 12:0 are usually <1.0% kcal, unless coconut oil is used as the cooking oil or coconut *santan* is used in food preparation. Thus, the present hypothetical diet contained much higher levels of 12:0 and 14:0 compared to the "urban" diet reported in an earlier study (Ng, 1995).

In the present *santan*-based diet, the total 2.8% kcal 14:0 from coconut *santan* plus the estimated 0.5-0.9% kcal 14:0 from other non-*santan* dietary components would raise the total dietary 14:0 beyond the 14:0 "threshold" of 3% kcal resulting in an elevation of serum TC (Hayes & Khosla, 1992). This is particularly pertinent in view that the 18:2 content reported for common Malaysian diets averaged only 3.2% kcal (Ng, 1995), while the maximum cholesterol-lowering potential of 18:2 is estimated at about 6% kcal (Hayes & Khosla, 1992).

Predicted serum TC response

In order to predict the serum TC response when palm oil *santan* replaced coconut *santan* in the present diet, the predictive equation of Hegsted (1965) given below is used. The above *santan* switch brought about two important dietary FA changes which were incorporated into the predictive equation, viz. (a) C12-16 SFAs were reduced from 10.8% kcal to 4.8% kcal, i.e. a change of -6.0% kcal (Table 1); and

(b) 18:2 was increased from 0.28% kcal to 1.41% kcal, i.e. a change of +1.13% kcal. Dietary cholesterol remained constant, while monounsaturated fatty acids, which are regarded as essentially neutral (Hegsted, 1965), are excluded in this analysis.

Using the Hegsted equation,

$\Delta TC = 8.45\Delta S_{14} + 2.12\Delta S_{16} - 1.87\Delta P + 5.64\Delta C - 6.24$

where ΔTC = change in total cholesterol (mg/dL)

ΔS = change in C12-16 SFA (% kcal)

ΔP = change in 18:2 (% kcal)

ΔC = change in dietary cholesterol (mg/1000 kcal)

Hence,

$\Delta TC = 8.45(-2.7) + 2.12(3.3) - 1.87(1.13) + 0 - 6.24$

$= -22.81 + 7.0 - 2.11 - 6.24$

$= -24.16$ mg/dL

However, when the effects of 16:0 were omitted in the equation as when palm 16:0 is regarded neutral, as observed in lean, active adults (Ng et al., 1991) and non-human primates (Hayes et al., 1991), the predicted change in serum TC is as follows:

$\Delta TC = 8.45(-2.7) + 0 - 1.87(1.13) + 0 - 6.24$

$= -22.81 + 0 - 2.11 + 0 - 6.24$

$= -31.16$ mg/dL

Therefore, switching from coconut *santan* to palm oil *santan* would result in a reduction of 24 - 31 mg/dL TC, depending on the LDL "set-point" of the individual(s) concerned. This hypocholesterolemic effect is not immediate; instead a period of 1 to 2 weeks are required for the effects to be seen, provided that the dietary fat change is sustained throughout this period. The concomitant increase in 1.13% kcal 18:0, the predominant PUFA and essential fatty acid (EFA) brought about by the switch from coconut *santan* to palm oil *santan*, may be regarded as an added bonus, with an overall beneficial impact on serum TC and hence a reduction on cardiovascular risk.

Since there was no change in dietary cholesterol, the cholesterol factor in the Hegsted equation was given a value of zero in this analysis. It is noteworthy that the cholesterol content in the present hypothetical diet may be regarded as high. There are a total of 7 eggs in the main dishes and snacks selected. If the cholesterol content per egg is taken as 250 mg, this meant that the cholesterol contribution from eggs per head alone in this analysis would be approximately 350 mg/day. If one considers also the daily dietary cholesterol from non-egg sources approximating 100-150 mg (Ng, 1995), the cholesterol intake per head in the present diet would exceed 400 mg. Thus the high content of dietary cholesterol and the influx of dietary 14:0 and cholesterol (from eggs) would lead to a down-regulation of the LDL_r, thus interfering with the clearance of circulating LDL resulting in an expansion of the LDL pool, and therefore raising serum TC and cardiovascular risk.

It is emphasised that the present "findings" were based on a prediction using an established regression equation involving only four of the dietary variables (actually only three in this analysis, since dietary cholesterol was kept constant), and which could explain 90% of the total

variation in serum TC (Hegsted et al., 1965). Although the predicted hypocholesterolemic response is probably valid, it is realised however that a human feeding trial would need to be conducted to prove this point.

CONCLUSION

The present predictive analysis showed that replacing coconut *santan* (a 14:0+12:0-rich fat) with palm oil *santan* (16:0-rich fat) in a 2300-kcal diet lowered the contribution of C12-16 SFAs from *santan* (10.8% kcal to 4.8% kcal); more importantly, the hypercholesterolemic myristic acid (14:0) was reduced to less than 1.0% kcal. The use of the Hegsted equation in the present hypothetical *santan*-based diet predicted that the switch from coconut *santan* to palm oil *santan* could result in a significant reduction in serum TC ranging from 24 to 31 mg/dL (0.62 to 0.80 mM/L) and hence a reduction of cardiovascular risk.

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