REVIEW ARTICLE

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EFFECTS OF SUBSTITUTING PALM OLEIN WITH CARBOHYDRATES ON INSULIN SENSITIVITY: A REVIEW

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ABSTRACT

The role of palm olein on insulin resistance, which predisposes to disease progression of type 2 diabetes, is unclear. This article summarises the effects of substituting palm olein with carbohydrates on insulin sensitivity. Two intervention studies have reported conflicting findings. The RISCK (Reading, Imperial, Surreys, Cambridge and King's) study suggested that saturated fat-enriched diet consisting of mainly palm oil and milk fat did not differ from both high and low glycemic carbohydrates on insulin sensitivity in subjects at risk of developing metabolic syndrome. However, another study reported reduced insulin sensitivity after a diet enriched with palm olein and butter compared with high carbohydrate intake. No epidemiological data exists in this context. More clinical trials using solely palm olein in this area are needed. Further well-controlled large scale studies are needed to furnish the information on palm olein replacement with carbohydrates in diabetes prevention.

Keywords: palm olein, carbohydrates, insulin sensitivity, dietary fats, saturated fats.

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INTRODUCTION

The increased consumption of dietary fats is associated with the worldwide epidemic of obesity and related complications (WHO, 2003). Excess fat deposition would cause adiposopathy (adipose tissue dysfunction) which is linked to abnormal increased production of pro-inflammatory cytokines and decreased release of anti-inflammatory adiponectin (Bays *et al*., 2013). Such phenomenon contributes to the progression of impaired insulin sensitivity, which is one of the clusters of complications leading to the development of type 2 diabetes mellitus (Laakso, 2010). Insulin sensitivity describes the responsiveness of insulin-target cells to insulin. The control of glucose level is initiated with the binding of insulin to insulin receptor which results in tyrosine phosphorylation of insulin receptor (Hirabara *et al*., 2012). Pro-inflammatory mediators accumulated from chronic inflammation due to adiposopathy however suppress insulin signalling through serine phosphorylation, resulting in impaired insulin sensitivity and inhibition of glucose uptake into cells (Olefsky and Glass, 2010).

The rapid growth of fast food restaurants signifies the problems of overconsumption of energy-dense food. Dietary fats have been highlighted in this context due to its higher density of energy content (9 kcal g^{-1}) compared with other macronutrients, *i.e.* carbohydrates (4 kcal g^{-1}). The Food and Agriculture Organisation (FAO) has recommended reduction of total fat intake, in particular saturated fats in replacement with carbohydrates or monounsaturated fats (FAO, 2010). Numerous scientific reports have put saturated fats as the main dietary culprit to negatively impact health and increase the risk of developing chronic diseases including cardiovascular diseases and type 2 diabetes mellitus (Uusitupa *et al*., 1994; Pérez-Jiménez *et al*., 2001; Vessby *et al*., 2001; Summers *et al*., 2002; Shah *et al*., 2007) though there are recent reports suggesting the null effects of saturated fats with the above mentioned diseases (Malhotra,

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2013; Chowdhury *et al*., 2014; de Souza *et al*., 2015). This has alleged concerns on the safety of palm oil consumption linking to saturated fat consumption, given that palm oil is the most consumed vegetable oil in the world (Fry and Fitton, 2010).

Palm olein, which is the liquid fraction of palm oil, is widely used as culinary oil worldwide. Palm olein contains 50% saturated fatty acids (45% palmitic acid and 5% stearic acid), balanced by another 40% monounsaturated and 10% polyunsaturated fatty acids (Yousefi *et al*., 2013). The versatility and stability of palm olein make it suitable for most of the food formulation. In view of the abundant availability in food products, palm olein has attracted much controversy on the suitability in healthier food reformulation. In the 1980s, palm oil had been urged to be removed from the shelves due to the purported detrimental health effects of tropical oils. Food manufacturers had replaced palm olein with partially hydrogenated vegetable oils, such as partially hydrogenated soyabean oil, which was later found to be containing *trans*-fatty acids (McNamara, 2010). Extensive research based on concrete evidence derived from human and epidemiological studies suggest the detrimental effects of *trans*-fatty acids on cardiovascular diseases and related complications. This has lead to regulations set to limit the use of *trans*-fatty acids in food formulations (McNamara, 2010). Meanwhile, the trend for healthier food formulation has then moved towards formulation of low-fat food products. It was claimed that low-fat food products are healthier due to lower energy content; however the energy content derived from dietary fats has mainly been replaced by carbohydrates, in particular refined carbohydrates.

It has been reported that the consumption of refined carbohydrates may lead to development of type 2 diabetes (Maki and Phillips, 2015). We have estimated that load of carbohydrates in a set meal, *i.e*. pasta with a glass of coke, or fried rice with a glass of sweetened drink contributes to >60% carbohydrates in a meal. High intake of carbohydrates in a single meal has been reported to promote hyperglycemic responses (Blaak *et al*., 2012). This is particularly detrimental to individuals with insulin resistance who are susceptible to the development of type 2 diabetes. Predicted upon that, our current review is aimed to examine the effects of substituting palm olein with carbohydrates on insulin sensitivity.

PALM OLEIN *vs*. CARBOHYDRATES ON INSULIN SENSITIVITY

Data from epidemiological studies on this topic are limited. Results showing that higher intake of saturated fats is associated with insulin resistance are confounded by many factors, *i.e*. dietary compounds, total amount of dietary fat, body weight, *etc*. There is no research conducted on the use of solely palm olein, which is a semi-saturated vegetable oil on insulin sensitivity. The information linking saturated fats on this aspect may be limited based on associations between dietary intake and changes in serum or plasma fatty acids with insulin sensitivity or type 2 diabetes (Vessby *et al*., 1994; 2002; Wang *et al*., 2003; Riserus, 2008). The association depicted does not show a causal relationship, hence, vigorously well-controlled intervention studies are warranted for further confirmation. In this respect, we summarise the evidence derived from two intervention studies, comparing palm olein as part of a saturated fat-enriched diet with carbohydrates on insulin sensitivity.

Clinical Evidence

No long-term intervention studies on dietary fats and diabetes are available. Insulin sensitivity, the intermediate marker for disease end-point has been used in dietary assessment. Clinical studies were identified by searching Pubmed, EMBASE, Cochrane databases for the present review. Inclusion criteria were English language articles reporting the measurement of at least one indices of insulin sensitivity and insulin secretion in both postprandial and fasting measurements following the consumption of palm olein-rich meal and carbohydrate-rich meal. Keywords used in the search included 'saturated fat', 'palm', 'carbohydrate', 'low fat', 'insulin sensitivity', 'insulin', 'glucose', 'randomised controlled trial', 'human' and 'clinical trial'. A total of 262 papers were retrieved from the search, only two studies were identified comparing palm olein *vs*. carbohydrates where both sources of dietary fats were blended with butter fat or dairies (*Table 1*). RISCK (Reading, Imperial, Surreys, Cambridge and King's) (Jebb *et al*., 2010), is the largest multi-centre study involving subjects at risk of developing metabolic syndrome. The study was a parallel design, randomised controlled trial comparing high-saturated fat and high glycemic index (HS/HGI), high-monounsaturated fat and high glycemic index (HM/HGI), high-monounsaturated fat and low glycemic index (HM/LGI), low-fat and high glycemic index (LF/HGI), and low-fat and low glycemic index (LF/LGI) diets in 548 subjects completing 24-week intervention each. The study instructed the free-living subjects to consume daily fat intake at 38% en where key sources of fat and carbohydrates were provided. Both palm oil and milk fat were used as dietary fat in place of the control (HS) diet while olive oil and high oleic sunflower oil were used as the fat sources of HM diets. The MUFA-enriched diets were 7% en higher in MUFA content whereas the low-fat

TABLE 1. SUMMARY OF STUDIES COMPARING PALM OIL *vs.* CARBOHYDRATE ON INSULIN SENSITIVITY

Reference	Health status; n (M/W)	Age (yr)	Design	Duration	Treatment	Dietary intervention	Outcomes
Jebb *et al*. (2010)	At risk of metabolic syndrome; 548 (230/318) HS/HGI; 85 HM/HGI; 111 HM/LGI; 116 LF/HGI; 116 LF/LGI; 121	M:52 ± 10 W:51± 9	Randomised, controlled, parallel, non-blinded Intravenous glucose tolerance test (3 hr) after diet intervention	6 months 4 weeks run-in	1. HS/HGI 2. HM/HGI 3. HM/LGI 4. LF/HGI 5. LF/LGI	Isocaloric (2050 kcal per day) diet. *Subjects were provided with key sources of fat* (e.g. *spreads, cooking oils and margarine) and carbohydrates* (e.g. *bread, pasta, rice and cereals)* HS/HGI 37.5% F, 20.5% P, 42% C 16% SAFA, 11.5% MUFA, 5.8% PUFA *Palm olein and milk fat* HM/HGI 35.6% F, 19.5% P, 44.9% C 9.5% SAFA, 16.2% MUFA, 6.6% PUFA *Spread, margarine rich in oleic acid derived from HOS and olive oil, HOS (additional source in Australian centre).* HM/LGI 35.7% F, 19.7% P, 44.6% C 9.6% SAFA, 16.3% MUFA, 6.9% PUFA LF/HGI 27.5% F, 21.4% P, 51.1% C 9.2% SAFA, 9.8% MUFA, 5.2% PUFA LF/LGI 26.1% F, 22.4% P, 51.5%C 8.3% SAFA, 9.7% MUFA, 5.1% PUFA	Insulin sensitivity, Sg, AIR_G, RQUICKI: NS
Perez-Jimenez *et al*. (2001)	Healthy; 59 (30/29)	23.1 ± 1.8	Randomised, controlled, cross-over Modified insulin suppression test on the last day of intervention, where a continuous somatostatin (to inhibit endogenous insulin secretion), insulin and glucose were infused into vein.	4 weeks Bleeding time during insulin suppression on test: 150,160, 170, 180 min	1. HS 2. HM 3. HC Diet sequence: HS → HM → HC or HS → HC → HM	Daily meals were provided to subjects *et al*. HS 2580 kcal; 37.7% F, 18.1% P, 44.2% C 22.6% SAFA, 10.1% MUFA, 5% PUFA *Palm oil and butter* HM 2580 kcal; 38.4% F, 17.5% P, 44.1% C 9.2% SAFA, 24.4% MUFA, 5.2% PUFA *Olive oil* HC 2531 kcal; 27.9 % F, 17.6% P, 54.5% C 9.2% SAFA, 13.5% MUFA, 4.8% PUFA *Biscuits, bread and jam*	Fasting measurements Insulin: HS ↑↑ *vs.* HM and HC Glucose: NS Glucose uptake in monocyte Basal glucose uptake and insulin-stimulated glucose uptake: HS ↓↓ *vs.* HM and HC Modified insulin suppression test Mean glucose: HS ↑↑ *vs*. HM and HC Mean insulin: NS

Note: n - sample size; M - men; W - women; HS - high-saturated fatty acids; HGI - high glycemic index; HM - high-monounsaturated fatty acids; LGI - low glycemic index; LF - low-fat; y - year; F - fat; P - protein; C - carbohydrate; %, % energy; SAFA - saturated fatty acids; MUFA - monounsaturated fatty acids, PUFA - polyunsaturated fatty acids; HOS - high-oleic sunflower oil; Sg - glucose effectiveness; AIR_G - an indicator of endogenous insulin secretion; RQUICKI - revised quantitative insulin sensitivity check index; NS - no significant difference between diets; HC - high-carbohydrate; AUC - area under the curve; ↑↑ - significantly higher; *vs.* - compared to; ↓ - lower compared to baseline; ↓↓- significantly lower; NS - no significant difference between diets.

diets were enriched with 10% en carbohydrates, when compared to HS diets. Intravenous glucose tolerance test (IVGTT) was conducted to examine the changes in insulin sensitivity. The method is highly correlated with euglycemic clamp. The study found that isoenergetic replacement of saturated fat with monounsaturated fat or carbohydrates has similar effect on insulin sensitivity, glucose effectiveness and insulin secretion as measured by IVGTT and revised quantitative insulin sensitivity check index (RQUICKI) calculation.

The cross-over design study conducted by Pérez-Jiménez *et al*. (2001) provided daily meal to 59 young healthy subjects in a four-week randomised controlled trial. The diet intervention started with a four-week saturated fat-enriched diets (38% en total fat; 23% en saturated fats), followed by the randomised allocation of Mediterranean diet (38% en total fat; 10% en saturated fats) and carbohydrate diet (55% en carbohydrates; 10% en saturated fats) for 2 × 4 weeks. The fat source for saturated fat diet was a blend of palm olein and butter while olive oil, nuts and grains were emphasised in Mediterranean diet. The additional carbohydrate content in carbohydrate-enriched diet was mainly biscuits, bread and jam. The isocaloric substitution in this study resulted in an improvement of insulin sensitivity following both Mediterranean and carbohydrate diets, as indicated by a decrease in mean plasma glucose concentrations during the insulin suppression test and an increase in both basal and insulin-stimulated 2-deoxiglucose uptake in peripheral monocytes. Saturated fat-enriched diet, however was found to increase both fasting insulin and non-esterified fatty acid concentrations compared with Mediterranean and carbohydrate diets. However, the clinical applications of this study are limited due to a relatively short duration of study intervention and small sample size. In addition, the dietary effect of saturated fat-enriched diet may be confounded by the habitual diet pattern prior to the commencement of dietary intervention because saturated fat-enriched diet was not included in the randomisation of dietary sequence.

CONCLUSION

No apparent impact by exchanging palm olein with carbohydrates was concluded based on the current limited clinical evidence available. There are no clinical studies using solely palm olein as the dietary fat component for the comparison with carbohydrates, hence, interference from other dietary fat sources, *i.e*. butter and animal fats, hence, exists. The large scale RISCK study provided promising data on the effect of saturated fats on insulin sensitivity, which provides a platform for clinical outcome study using diabetes as study end-point. The influence of carbohydrates and dietary fats should be put into extensive research to examine the long-term impact on human health and disease risk factors.

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