LIPID PEROXIDATION IN CULINARY OILS SUBJECTED TO THERMAL STRESS

H. Ramachandra Prabhu,

Department of Biochemistry, Kasturba Medical College, Mangalore-575001.

ABSTRACT

The levels of cytotoxic aldehydic products in different culinary oils, with or without thermal stress, (routine domestic or commercial frying) were determined by thiobarbituric acid method. The results showed that (i) thiobarbituric acid reactivity was much higher in edible oils rich in polyunsaturated fatty acids than those rich in saturated fatty acids or monounsaturated fatty acids, even without thermal stress, (ii) the lipid peroxide levels were in proportion to the duration of thermal stress, (iii) nature of the container used (steel, iron or teflon-coated) had no significant effect on the extent of lipid peroxidation under identical conditions of thermal stress and (iv) thermally stressed oils collected from hotels and roadside caterers contained higher levels of cytotoxic aldehydic products, when compared to oils thermally stressed under domestic frying conditions. These results suggest that dietary ingestion of thermally or autooxidatively stressed polyunsaturated fatty acid rich culinary oils is more harmful compared with those similarly treated oils rich in saturated fatty acids and monounsaturated fatty acids.

KEY WORDS: lipid peroxidation, thermal stress, culinary oils.

INTRODUCTION

Lipid peroxidation is oxidative deterioration of polyunsaturated fatty acids (PUFA), i.e. those which contain two or more double bonds. Oxygen-dependent deterioration leading to rancidity has been recognised as a problem in the storage of fats and oils and is more relevant today with the popularity of PUFA rich margarines and cooking oils. The dietary consumption of saturated fats has, in recent years, been regularly cited as a major factor in the pathogenesis of atherosclerosis and its associated sequelae, namely, ischemic heart disease (IHD) and peripheral vascular disease (1). However, it is now generally recognised that the replacement of saturated fats by vegetable oils containing high levels of PUFA may also render the individuals susceptible to cardiovascular diseases (2). PUFA rich groundnut oil has been shown to be pro-atherogenic whereas cocoa butter which contains high levels of saturated

fatty acids (SFA), is much less atherogenic than expected (3). PUFA peroxidise easily with the formation of highly toxic products such as peroxides and aldehydes. Recently, Haywood et. al. (4) have shown that heating of culinary oils to 180° C generated a variety of peroxidation products such as aldehydes (alkanals, trans-2-alkenals and alka-2,4-dienals) and their conjugated hydroperoxy diene precursors. Such aldehydic products are found to be absorbed from the gut into the systemic circulation (1).

In view of the above reports, it was considered essential to determine the lipid peroxide levels in oils having varied PUFA content, with and without thermal stress. The studies were conducted by changing the nature of the vessel and by varying the duration of thermal stress. The study was extended to cover thermally stressed oils collected from hotels and roadside caterers.

MATERIALS AND METHODS

All the chemicals used were commercial products of highest analytical grade available in India. The culinary oils with thermal stress (obtained after deep frying of food items) and without thermal stress were collected from the staff of our college (domestic frying), college canteen, roadside caterers and hotels of Mangalore (commercial frying). Frying temperatures ranged from 170 to 190°C. All culinary oils used for the study, except coconut oil, were of refined variety. Thiobarbituric acid (TBA) reactivity in the culinary oils was determined by following the method of Luotola (5). To 0.2ml oil was added 2ml of 2-thiobarbituric acid (0.67%w/v) followed by 2ml of trichloroacetic acid (10% w/v). After mixing, the tubes were kept in the boiling water bath for 15 min, cooled to 20° C and centrifuged (2000g for 10 min). The TBA chromogens in the aqueous layer were measured at 532nm against appropriate blanks.

(5). It has been shown that the formation of peroxides and carbonyls is markedly inhibited in cod-liver oil when it is supplemented with vitamin E (5).

TBA reactivity of different oils thermally stressed for domestic and commercial purposes (hotels and roadside caterers) is presented in Table 2. Oils thermally stressed domestically at 180-190°C for about 15-20 min. showed TBA reactivity ranging from an absorbancy of 0.04 to 0.26, highest value being for sunflower oil which is richest in PUFA among the oils used and lowest for coconut oil. No significant differences in TBA reactivity were observed between unrefined sesame oil and groundnut oil, and their refined varieties, under domestic frying conditions (data not shown). The lipid peroxide levels of oils collected from hotels and roadside caterers were much higher (124-170 %) than the oils thermally stressed during domestic deep frying. The frying temperatures of commercial frying were in the same range as that

Table 1. Fatty acid composition (%)* and TBA reactivity of edible oils prior to thermal stress.

Fat or Oil	Saturated fatty acids	Monounsaturated fatty acids	Polyunsaturated fatty acids	TBA reactivity, OD at 532 nm
Coconut oil	86	12	2	0.010 ± 0.002
Palm oil	49	42	9	0.025 ± 0.004
Groundnut oil	17	51	32	0.060 ± 0.008
Sesame oil	14	44	42	0.085 ± 0.008
Sunflower oil	10	24	66	0.110 ± 0.010

*Reported values (Ref: 6). OD (optical density) values are the Mean ± SD of 5 separate experiments.

RESULTS AND DISCUSSION

Table 1 summarises the lipid peroxide levels in different edible oils prior to thermal stress. It is significant to note that the levels of lipid peroxidation products increased with the increase in the PUFA content. It is well known that PUFA get peroxidised very readily, which, is dependent on the storage conditions and the amount of antioxidant present

of domestic frying. High lipid peroxide levels under commercial frying appears to be due to the habit of repeated usage of the same oil. The practice of storing the thermally stressed oils for use at a later stage, has also been observed with the roadside caterers. No significant differences were observed with respect to the lipid peroxide content, when the thermal stressing was performed (without food frying) in iron, steel or teflon-coated vessels of similar dimensions

Table 2. TBA reactivity of culinary oils, thermally stressed during domestic and commercial frying of food items.

Edible oils	TBA reactivity, OD at 532nm		% Increase
	Domestic frying	Commercial frying	
Sunflower oil	0.260 ± 0.049 (8)	0.583 ± 0.112 (4)	124
Sesame oil	0.220 ± 0.044 (5)	0.562 ± 0.136 (4)	169
Groundnut oil	0.168 ± 0.042 (6)	0.413 ± 0.112 (3)	146
Palm oil	0.115 ± 0.056 (7)	0.303 ± 0.066 (6)	164
Coconut oil	0.040 ± 0.011 (10)	0.108 ± 0.035 (3)	170

Mean ± SD of (n) observations. Time duration of thermal stress: Domestic-15 to 20 min., Commercial – 2 to 3 hrs.

with identical conditions of heating (Table 3). Iron salts are known to accelerate the extent of lipid peroxidation (5). However, in the present study, no such accelerating effect was observed in iron vessel when compared to other vessels, probably due to the absence of release of iron into the medium.

The toxic effects of PUFA-derived aldehydes are well described in animal models and there is much controversy regarding the role of PUFA-rich diets in promoting, regressing or preventing cardiovascular diseases in humans (1). In 1920, the typical American diet consisted of saturated fat with low level of PUFA and the incidence of deaths due to myocardial infarction was very low. Natural and refined vegetable oils containing high concentrations of PUFA were subsequently introduced and deaths due to myocardial infarction substantially escalated by the year 1960 (7).

The demographic 1978 yearbook of United Nations reports that Sri-Lankan population, of all the

countries surveyed, had the lowest mortality rate from IHD. Their predominant dietary fat is coconut oil (8) which contains only about 2% of PUFA. Subjecting this oil to thermal stress generates little aldehydic products (Table 2). When the normal diets of Sri-Lankan males were changed by substitution of corn oil for their customary coconut oil, their serum HDL cholesterol level decreased from 43 mg% to 25 mg% (9). The people of Kerala state consume coconut kernel and coconut oil as their major dietary fat and the incidence of atherosclerosis is reported to be very low in this state (10).

Kritchvsky and Teppar (11) in their animal feeding studies have shown that diets containing thermally stressed PUFA-laden oils are more atherogenic than those containing unheated oils. The ability of oxidised dietary lipids to accelerate the development of atherosclerosis in New Zealand white rabbits has been reported (12). These observations clearly indicate that dietary consumption of PUFA peroxidation products may represent an important risk factor for

Table 3. TBA reactivity in sunflower oil: Effect of duration of thermal stress in iron, steel and teflon-coated vessels (without frying of food items).

Container used	TBA reactivity at different time durations			
	15 min.	30 min.	45 min.	60 min
Iron	0.176 ± 0.021	0.364 ± 0.044	0.524 ± 0.076	0.690 ± 0.118
Steel	0.170 ± 0.026	0.357 ± 0.051	0.501 ± 0.083	0.674 ± 0.102
Teflon coated	0.166 ± 0.024	0.342 ± 0.057	0.518 ± 0.081	0.686 ± 0.120

Results are the Mean ± SD of 3 experiments.

the induction and development of atherosclerosis in humans.

An approach to solving the dietary problems outlined above may be the commercial fortification of vegetable-derived culinary oils with lipid-soluble, chain-breaking, dietary antioxidant such as α -tocopherol. However, it is indicated that α -tocopherol content of culinary oils subjected to thermal stress is substantially depleted (2). In addition, it is also noted that the concentrations of α -tocopherol naturally present in culinary oils are insufficient to protect PUFA against oxidative deterioration induced by heating (1).

It is concluded from the present study that, for deep frying, it is safer to use culinary oils with low levels of PUFA than those with the higher. However, in order to meet the requirements for essential fatty acids, oils rich in PUFA may be consumed, either unheated or with shallow frying. The dietary consumption of unheated oils (mostly other than butter and coconut oil) is not a common practice. Consumption of oil seeds such as groundnut and sesame without deep frying could be considered as an alternative to meet the dietary requirements for essential fatty acids.

REFERENCES

1. Grootveld, M., Atherton, M. D., Sheerin, A.N., Hawkes, J., Blake, D.R., Richens, T.E., Silwood, C. J. L., Lynch, E. and Claxson, A.W.D. (1998) *In vivo* absorption, metabolism and urinary excretion of α , β -unsaturated aldehydes in experimental animals. *J. Clin. Invest.* 101, 1210-1218.
2. Addis, P.B. and Warner, G.J. (1991) The potential health aspects of lipid oxidation products in food. In *free radicals and food additives*. Arouma, O.I. and Halliwell, B., Editors. Taylor and Francis Ltd., London. p. 77-119.
3. Kritchevsky, D. (1991) Dietary fat and experimental atherosclerosis. *Int. J. Tissue React.* 13, 59-65.
4. Haywood, R.M., Claxson, A.W.D., Hawkes, G.E., Richardson, D.P., Naughton, D.P., Coumbarides, G., Lynch, E.J. and Grootveld, M.C. (1995) Detection of aldehydes and their conjugated hydroperoxydiene precursors in thermally stressed culinary oils and fats: Investigations using high resolution proton NMR spectroscopy. *Free Radic. Res.* 22, 441-482.
5. Luotola, M.T. and Luotola, J.E.I. (1985) Effect of α -tocopherol on the peroxidation of cod-liver oil. *Life Chemistry Reports*, 3, 159-163.
6. Briggs, G. M. and Calloway, D.H. (1984) *Nutrition and Physical fitness*. 11th Ed. Holt, Rinehart and Winston. CBS College Publishing, New York p. 46.
7. Martin, W. (1984) The combined role of atheroma, cholesterol, platelets, the endothelium and fibrin in heart attacks and strokes. *Med. Hypothesis*, 15, 305-322.
8. Kaunitz, H. (1986) Medium chain triglycerides in aging arteriosclerosis. *J. Environ. Pathol. Toxicol. Oncol.* 6, 115-121.
9. Mendis, S., Wissler, R. W., Bridenstine, R.T. and Podbielski, F.J. (1989) The effects of replacing coconut oil with corn oil on human serum lipid profiles and platelet derived factors active in atherogenesis. *Nutr. Reports Int.* 40, 4-11.
10. Kurup, P.A. and Rajmohan, T. (1994) Consumption of coconut oil and coconut kernel and the incidence of atherosclerosis: Proceedings of symposium on coconut and coconut oil in human nutrition, p.35-59.

11. Kritchesky, D. and Tepper, S. A. (1967) Cholesterol vehicle in experimental atherosclerosis. Comparison of heated corn oil and heated olive oil. *J. Atherosclerosis. Res.* 7, 647- 651.
12. Staprans, I., Rapp, J.H., Pan, X.M., Hardman, D.A. and Feingold, K.R. (1996) Oxidised lipids in the diet accelerate the development of fatty streaks in cholesterol-fed rabbits. *Arterioscler. Thromb. Vasc. Biol.* 16, 533-538.