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Letter to the editor

Warning: thermally-stressed polyunsaturates are damaging to health

Thermal stressing of polyunsaturated fatty acid-rich culinary oils according to routine frying practices (30–90 min. at 180°C in the presence of atmospheric O_2) generates high levels of cytotoxic aldehydic products (up to 50 mmol kg^{-1}) (Haywood et al., 1995) via autocatalytic, self-propagating peroxidation processes (Fig. 1). Although a significant fraction of such aldehydes may remain bound to the triacylglycerol (TAG) glycerol backbone, ^{1}H NMR analysis of deuterated methanol ($C^2H_3O^2H$) extracts of repeatedly-utilised culinary oils revealed that the free aldehydes accounted for much of those detectable (i.e., the aldehydic and, where appropriate, corresponding vinylic proton resonances of *trans*-2-alkenals, *trans,trans*- and *cis,trans*-alka-2,4-dienals and *n*-alkanals were clearly visible in spectra acquired, whereas the characteristic TAG glycerol backbone signals were undetectable), despite the potential loss of high levels of such species via evaporation during the thermal stressing episodes to which they were subjected.

In addition to being implicated in the development of atherosclerosis (Witzum & Steinberg, 1991), and its associated pathological sequelae such as ischaemic heart disease and peripheral vascular disease, such aldehydic lipid oxidation products have been shown to exert genotoxicological (Esterbauer, 1982) and pro-inflammatory (Benedetti, Ferrali, Casini, Peiri, & Comporti, 1980) properties, and can also give rise to gastropathic conditions (Jayaraj, Rees, Touey, & White, 1986); phenomena that are undoubtedly ascribable to the high reactivity of these agents with critical biomolecules (e.g. the apolipoprotein B component of low-density-lipoprotein, DNA base adducts and thiols such as glutathione) (Esterbauer, 1982). However, with the exception of direct damage to the gastrointestinal epithelium, the toxicological effects exerted by orally-ingested aldehydes are, of course, critically dependent on the rate and extent of their in vivo absorption from the gut into the systemic circulation. Therefore, we recently employed high resolution ^{1}H nuclear magnetic resonance analysis to probe the in vivo absorption, metabolism and urinary excretion of typical aldehydic lipid oxidation products (*trans*-2-nonenal and *trans*-2-pentenal) and found that both of these α,β-unsaturated aldehydes are indeed absorbed from the gut into the systemic circulation in vivo, metabolised (primarily via the addition of glutathione across their electrophilic carbon–carbon double bonds), and excreted in the urine as C-3 mercapturate conjugates (Grootveld et al., 1998). However, it should be noted that aldehydic derivatives remaining bound to the TAG glycerol backbone require prior lipase-mediated hydrolysis in order to liberate them as free aldehydes, and hence in this case in vivo absorption of the latter is likely to be dependent on the bioavailability of such lipases. Moreover, the position of aldehydes on the TAG backbone [1-(3-) or 2-] may also influence the rate and extent of their lipase-catalysed release and therefore ability to penetrate the gastrointestinal epithelium.

The above results indicate that the dietary ingestion of thermally-stressed polyunsaturated fatty acid-rich culinary oils promotes the induction, development and progression of cardiovascular diseases since highly reactive aldehydes, therein, have the ability to covalently modify lysine residues of the apolipoprotein B moiety of low-density-lipoprotein, rendering it susceptible to uptake by macrophages, a critical stage in the generation of foam cells in vivo (Witzum & Steinberg, 1991). Indeed, our data are consistent with recent studies which demonstrated that oxidised lipids in the diet substantially accelerate the development of fatty streaks in vivo (Staprans, Rapp, Pan, Hardman & Feingold, 1996). Moreover, evidence for the pro-atherogenic properties of thermally-stressed polyunsaturated fatty acid-rich culinary oils was provided as early as 1967 by Kritchevsky and Tepper (1967) who demonstrated that heating corn oil [polyunsaturated fatty acid content 57% (w/w)] at 215 ± 15°C for 20 min markedly increased its atherogenicity, whereas the pre-heating of olive oil [polyunsaturated fatty acid content 9% (w/w)] in the same manner, failed to influence its atherogenic properties. Of course, this temperature is somewhat higher than those currently employed in commercial frying practices (ca. 180°C) and, as well as causing excessive decomposition of culinary oil polyunsaturates, such extreme thermal stressing episodes can also give rise to substantial losses of α-tocopherol (vitamin E) therein. The latter phenomenon, also readily demonstrable by ^{1}H NMR spectroscopy (Grootveld et al., 1998), is presumably ascribable to the volatilisation of this

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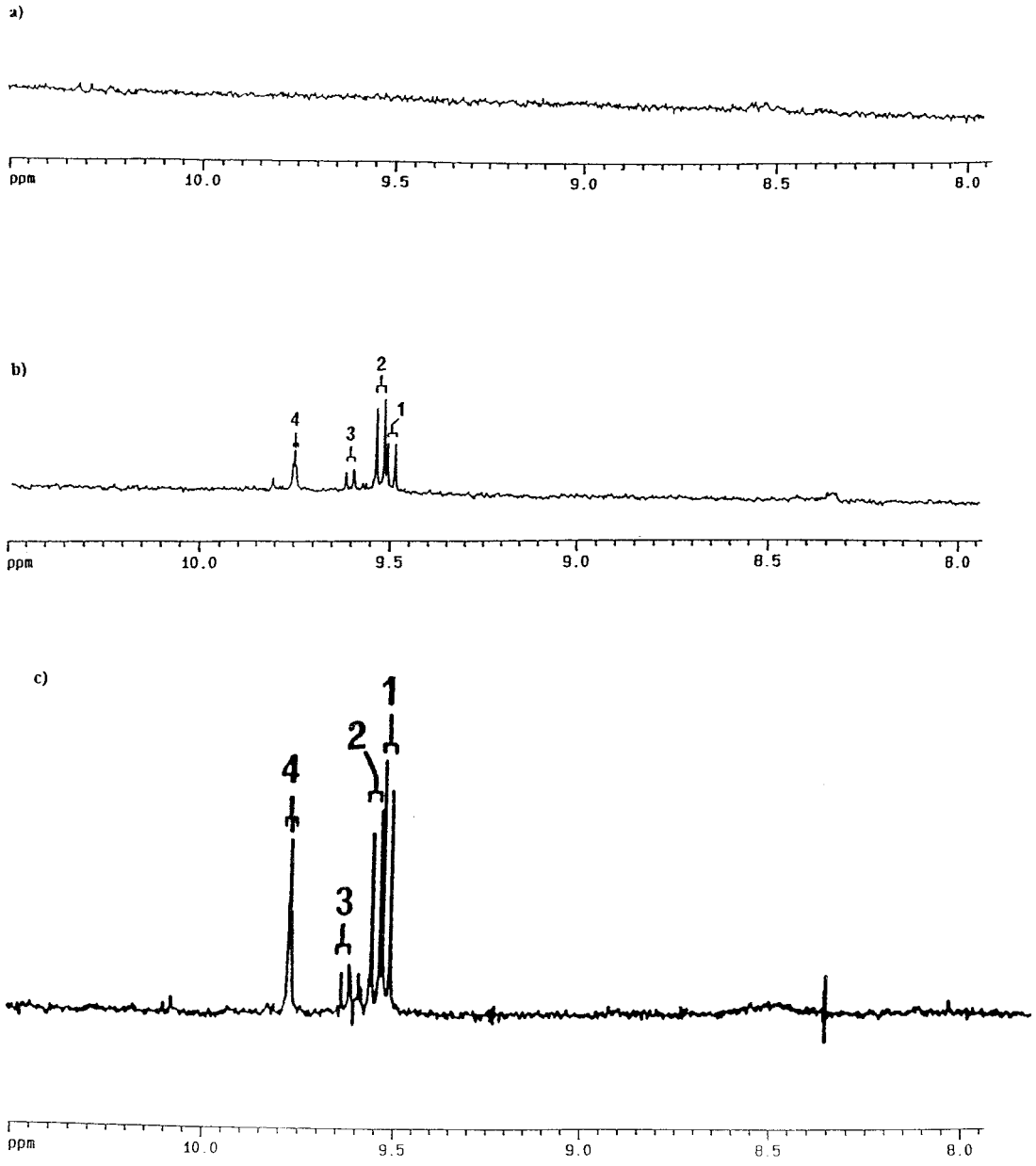

Fig. 1. Expanded aldehydic proton regions of 400 MHz ^{1}H NMR spectra of deuterated chloroform (C^2HCl_3) solutions containing a commercially-available sample of sunflower seed oil (a), before and (b), after heating at a temperature of 180°C for 90 min in the presence of atmospheric O_2. (c), Corresponding spectrum acquired on a C^2HCl_3 solution of a repeatedly-utilised culinary frying oil obtained from a fast-food restaurant. Abbreviations: 1,2,3 and 4, aldehydic group protons (−CHO) of *trans*-2-alkenals, *trans,trans*-alka-2,4-dienals, *cis,trans*-alka-2,4-dienals and *n*-alkanals respectively.

chain-breaking antioxidant (boiling-point 210°C), together with its involvement in peroxidative chain reactions.

Interestingly, experiments conducted in our laboratory have shown that subjection of monounsaturate-rich olive oil to such thermal stressing episodes generates little or no aldehydic products (Haywood et al., 1995), an observation concordant with its "heart-friendly" properties. Of course, monounsaturates are much more resistant to peroxidation than polyunsaturates.

Although we do not dispute the perceived notion that **intact, unperoxidised** dietary polyunsaturated fatty acids are protective against the development of atherosclerosis, the frequent utilisation of culinary oils containing high levels of these agents (predominantly linoleoylglycerols) for frying/cooking processes, and the consequent ingestion of pro-atherogenic aldehydic peroxidation products therein, clearly pose health hazards worthy of increased clinical and public concern. Moreover, the inhalation of vapourised aldehydes by subjects conducting commercial or domestic frying practices, involving polyunsaturate-rich culinary oils, also affords a potential threat to human health.

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