Atherogenic Potential of Peanut Oil-Based Monounsaturated Fatty Acids Diets

Dear Editor,

In the dietary management of coronary heart disease (CHD), there is increasing recognition (1,2) that the traditionally recommended high carbohydrate, low-fat diet for hypercholesterolemia (3) may elicit undesirable blood lipid changes, including reductions in high-density lipoproteins and apolipoprotein A-1, while concurrently elevating triglycerides (TG) and very low density lipoproteins (4–6). Because of these untoward blood lipid changes induced by high-carbohydrate, low-fat diets, substitution of monounsaturated fatty acids (MUFA) for saturated fats may be a more effective strategy than substitution of carbohydrate for saturated fats in order to lower total and low density lipoprotein (LDL) serum cholesterol levels without adversely influencing high density lipoprotein, very low density lipoprotein, TG, and apolipoprotein A-1 (7–9). Most experimental diets have employed olive oil or canola oil as the MUFA source; however other MUFA-rich foods such as nuts (10) and avocados (11) have also been demonstrated to improve blood lipid profiles. In a recent report to *Lipids*, O'Byrne *et al.* (12) have shown that a low-fat, high-MUFA (14% energy) diet based upon high-MUFA (76–80%) peanuts improved total and LDL serum cholesterol levels in 12 postmenopausal women. Although diets based upon MUFA-rich peanuts (*Arachis hypogea*) should, in theory, be nonatherogenic because they reduce total and LDL cholesterol, there is substantial evidence to indicate that peanut oil, despite its hypolipidemic effects, is highly atherogenic to animals and possibly to humans as well.

Although the total MUFA content of peanut oil is high and can range from 36 to 59%, dependent upon the specific cultivar (13), it has been shown to be unexpectedly atherogenic when fed to laboratory animals (monkeys, rabbits, and rats) as part of a cholesterol-rich or a cholesterol-free diet (13–16). Because peanut oil can so rapidly produce atherosclerotic lesions which have similar biochemical and pathological characteristics to those in human atherosclerosis, it is routinely used in rabbit models to induce atherosclerosis (17,18). The reason for the high atherogenicity of peanut oil is unclear; however, it has been suggested that it may be due to residual lectins (glycoproteins with high affinity binding to cellular carbohydrate residues) found in the oil, since peanut oil induces fibromuscular arterial lesions in contrast to other vegetable oils which induce fatty lesions (19). Alternatively, the specific TG structure may also be responsible for its atherogenicity (20). In native peanut oil, all of the long-chain polyunsaturated fatty acids are found in the *sn*-3 position of glycerol; however, by utilizing a process to randomize the fatty acids within the TG, the atherogenicity of peanut oil has been shown to be reduced (21). It should be pointed out that the randomization process in some cases may also reduce peanut oil's lectin content (20,21).

When contrasting olive to peanut oil, rabbits which were fed peanut oil showed a higher frequency of arterial lesions, more intimal proliferation, and thicker intimas than did rabbits fed olive oil (15). Peanut oil-containing, atherogenic diets induce a preferential increase in intimal collagen and result in a characteristic fibromuscular lesion in intimal plaques that is attributable to the addition of peanut oil to the atherogenic diet (22,23). It has been suggested that residual peanut lectin (PNA) found in peanut oil, because of its specificity for D-galactose residues, may bind arterial smooth muscle cells expressing these sugar residues and thereby induce its characteristic fibromuscular lesions (19). In support of this concept are data which have shown that PNA stimulates *in vitro* vascular smooth muscle cell proliferation and that added lactose could inhibit the PNA-induced stimulation (24). A similar *in vivo* experiment would be able to distinguish if peanut oil's atherogenicity is more attributable to its PNA content or to its specific TG structure.

Although there are no direct epidemiological studies evaluating the atherogenic potential of peanut oil, there is suggestive information from India that implicates peanut oil with higher mortality rates from CHD. In India, wherein vegetable oils constitute 80% of the per capita fat consumption, there are regional preferences in the choice of oils, and peanut oil is preferred in southern states, whereas northern states use mustard oil (25). In southern India, the mortality rate from CHD 30 yr ago was reported to be seven times higher than that in northern India and similar to that in the United States and England (26,27). A more recent report showed the prevalence of CHD to be 61.6% higher in southern Indians compared to their more northerly counterparts (28). It is possible that one of the factors underlying these regional differences may be different levels of peanut oil consumption. In contrast to peanut oil, which is a fairly recent addition to the human diet, olive oil has been part of the traditional Mediterranean diet for thousands of years (29) and has been shown both epidemiologically (30) and clinically (31) to reduce the risk for CHD in subjects living in industrialized countries. These

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studies suggest that olive oil may be superior to peanut oil in terms of its cardiovascular health benefits in such populations. Until further trials are conducted, it would seem premature to recommend peanut oil as part of high-MUFA diets for the management of CHD in Western populations.

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