
Dr. Ntanios disagrees with my comment that “most studies have shown the greater potency of plant stanols in lowering serum cholesterol” and cites one published study (Westrate and Meijer 1998) and one abstract. Westrate and Meijer compared the efficacy of the “esterified” forms of sterol and stanol and not the “free” forms, which may be an important distinction in terms of cholesterol-lowering efficacy. These data are interesting but did not provide a “clean” comparison because the amount of sterols in the sterol ester margarine differed from the amount of stanols in the stanol ester margarine. There were also differences in the fat composition of the margarines. Dr. Ntanios’ current position seems to contradict his conclusions in two previous reviews he coauthored (Jones et al. 1997, Jones and Ntanios 1998), where he stated that the result in the Westrate and Meijer study “is in contrast to the results of other studies that have compared the relative efficacy of the action of plant sterols vs. stanols and have concluded that the latter is more than twofold more potent as a cholesterol-lowering agent.” No new evidence has surfaced to change the validity of this conclusion.

Dr. Ntanios disagrees that plant sterols can be atherogenic even in phytosterolemic patients. However, the atherogenic potential of plant sterols is supported (Salen et al. 1999) by the fact that (i) monocytes, the precursors of foam cells, can accumulate plant sterols; (ii) in a phytosterolemia patient who died of cardiac sudden death, increased plant sterols amounts were found in the heart-replacing cholesterol; and (iii) treatment with bile acid malabsorption lowers plasma sterol levels, improving cardiac symptoms.

In phytosterolemic patients with accelerated atherosclerosis reported that plasma siterol and campesterol levels can be as low as 7.5 and 2.1 mg/dl, respectively, while plasma cholesterol levels are frequently not elevated. Dr. Ntanios agrees with the concern that even a small increase in blood levels of plant sterols may be atherogenic in hypercholesterolemic patients, based on the findings by Glaeck et al. (1991). However, Dr. Ntanios’ current position again contradicts his previous conclusion (Jones et al. 1997), that “elevated absorption of phytosterol in hypercholesterolemic individuals may identify those individuals . . . at increased risk for premature CHD” and that “intervention for these individuals may also include reduced sources of phytosterol-enriched foods.” The implication by Dr. Ntanios that long-term safety is not an issue because plant sterols “have been part of the human diet for millennia” is misleading. The typical Western intake of plant sterols is 200–300 mg/d, significantly less than the 2000–3000 mg/d required to lower serum cholesterol. Westrate and Meijer (1998) showed that consumption of 3000 mg/d of plant sterol esters increases plasma plant sterol levels, and especially campesterol by an average of 70%. No actual range of absolute plasma sterol levels attained was given. Thus, additional stud-

LITERATURE CITED


