since 1983 (2, 3). We identified 100 commonly consumed foods and used labeling or manufacturer’s data to ascertain the sodium content of each food. In 1994, we examined the sodium content of the same 100 foods, 94 of which were still being marketed. The average sodium content per serving (adjusted for any changes in serving size) decreased by 15% over the 11 y. In 2004, 69 of those foods were still being marketed. Between 1983 and 2004, the average sodium content per serving of those foods decreased by 5% (from 592 to 564 mg), or 0.3%/y. The greatest increase in the sodium content of a food was 82%, and the greatest decrease was 71%. However, from 1994 to 2004, the average sodium content per serving increased by 6% (from 533 to 564 mg).

Modest reductions in the sodium content of certain foods and the introduction of some reduced-sodium products have not been sufficient to reduce sodium consumption. Indeed, according to national food-recall surveys (which underestimate food intake and exclude salt added at the table), daily sodium consumption increased from 2262 mg in 1971–1974 (4) to 2819 mg in 1976–1980 (5) and to 3427 mg in 1988–1994 (6); it decreased slightly, to 3375 mg, in 1999–2000 (7). The increases were due to both the consumption of higher-sodium foods and increased energy intakes. The Institute of Medicine recommends a daily adequate intake of 1500 mg Na (1300 mg for persons 50–70 y old and 1200 mg for those >70 y old) and a tolerable upper level of 2300 mg (2). The Dietary Guidelines for Americans 2005 recommends that Americans consume ≤2300 mg Na/d and that a sodium intake of 1500 mg/d be the target for hypertensive persons, African Americans, and middle-aged and older adults (8).

Clearly, the recommendations by the Institute of Medicine and others have failed to reduce sodium consumption. The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure has recommended reducing sodium consumption by 50% over 10 y, a change that could save an estimated 150 000 lives per year (9, 10). It appears that more effective measures, such as front-label warnings on food packages, restrictions on the sodium content of foods that currently contribute the most sodium to the diet, and well-funded and aggressive education campaigns (such as those sponsored by the British Food Standards Authority), are needed to achieve that goal.

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Michael F Jacobson

Center for Science in the Public Interest
1875 Connecticut Avenue, NW, Suite 300
Washington, DC 20009
E-mail: mjacobson@cspinet.org

REFERENCES

Need to establish threshold soy protein intake for cholesterol reduction

Dear Sir:

The findings by Rosell et al (1) published in a recent issue of the Journal have potentially important public health implications but need to be verified by results from intervention trials. Unfortunately, such trials may not be forthcoming. In their cross-sectional study, Rosell et al found that soy protein intake was inversely related to plasma LDL-cholesterol concentrations among 1033 pre- and postmenopausal British women, about two-thirds of whom were vegetarians or vegans. The finding that soy protein lowers serum cholesterol is obviously not novel. In 1999 the US Food and Drug Administration (FDA) approved a health claim for soy protein concluding that it lowered cholesterol and reduced coronary heart disease risk (2).

The results by Rosell et al are noteworthy because the mean soy protein intake of the women in the highest intake category was only 11.2 g/d. By comparison, the FDA established 25 g/d as the threshold intake needed for cholesterol reduction. Twenty-five grams is approximately twice the soy protein intake of Japanese adults, and the consumption of this amount of soy by non-Asians represents a significant dietary challenge (3). Furthermore, the incentive for doing so is arguably not very compelling, because the LDL-cholesterol-lowering effect (≈5%) of soy protein is, although clinically relevant, relatively modest (4).

Consuming ≈11 g soy protein/d is relatively easy, however, and can be enthusiastically encouraged even on the basis of a modest reduction in cholesterol. There is a need to establish whether this lower amount of soy protein is, as suggested by the findings by Rosell et al, efficacious. Some evidence suggests that it is. In fact, the FDA did not establish 25 g/d as the required intake for cholesterol reduction because evidence suggested that lower amounts were not efficacious but rather because few trials used amounts lower than this.

Of relevance to this issue are 2 clinical trials conducted after 1999 that examined the possible dose-response relation between soy protein and cholesterol reduction. One trial used 20, 30, 40, and 50 g/d (5) and the other 30 and 50 g/d (6). In neither trial were the higher doses more hypocholesterolemic than the lower ones. These findings certainly allow for the possibility that fairly low intakes of soy protein are efficacious. Epidemiologic observations in addition to those of Rosell et al are consistent with this possibility.

For example, in a cross-sectional study, Ho et al (7) found that among Hong Kong Chinese men (n = 500) (but not women), there...
was a statistically significant negative linear relation between soy protein intake and LDL-cholesterol concentrations even though mean soy protein intake was <7 g/d. Similarly, a Japanese cross-sectional study by Nagata et al (8) found that soy protein intake was inversely related to cholesterol concentrations among men (n = 1242). In that study, the relation also existed for women (n = 3596). Soy protein intake was slightly higher in the study by Nagata et al than in the Chinese one, but overall mean (g/d) soy protein intakes for men and women were only 8.0 and 6.9, respectively. These studies are especially noteworthy because Western soy consumers are more likely to differ from their neighbors in ways that affect cholesterol than are Asians, for whom soy is a common dietary staple.

Of course, epidemiologic findings are not evidence of causal relations. There is reason to doubt that the lower cholesterol concentrations observed by Rosell et al are causally related to soy intake. The LDL-cholesterol concentration of the postmenopausal women in the fourth quartile of soy protein intake was ≈15% lower than that of the women in the first quartile, but nearly one-half (6%) of that difference occurred between the first and second quartiles, even though mean soy protein intakes in those quartiles were only 0.1 and 0.8 g/d, respectively (1). The biologic plausibility of such low soy protein intakes affecting cholesterol concentrations is unclear.

Theoretically, in free-living populations, soy foods displace foods in the diet that are higher in saturated fat, but Rosell et al controlled for intakes of saturated fat, polyunsaturated fat, and cholesterol. Furthermore, such low amounts of soy foods would displace negligible amounts of cholesterol-raising foods. Alternatively, it may be that the types of soy foods consumed by the subjects in the study by Rosell et al (1) and in the Asian epidemiologic studies (7, 8) cited previously are more hypocholesterolemic than are the isolated soy proteins used in most clinical trials (9). This possibility needs to be researched and is currently the focus of some debate within the soy research community.

Nevertheless, ample justification appears to exist for establishing via clinical trials the cholesterol-lowering effects of small amounts of soy protein. Such research has public health implications because at the population level even small decreases in cholesterol can significantly reduce heart disease mortality. However, establishing a dose-response relation between soy protein and cholesterol reduction, or even showing that small amounts of soy protein lower cholesterol, will require fairly large sample sizes. This is because the differences among groups are likely to be modest and also because the results of recently conducted trials examining the cholesterol-lowering effects of soy protein have been inconsistent. Some of this inconsistency likely stems from the small sample sizes used in many of these trials. Thus, the needed research will be expensive.

Still, it would be unfortunate for such research not to be conducted. Research showing that easily consumed amounts of soy protein are hypocholesterolemic will help to provide the basis for crafting a dietary message that consumers will be more likely to embrace than the current FDA recommendation. Heart disease rates may be reduced not only as a result of the cholesterol-lowering effects of soy protein but also because, as noted previously, soy foods can help to displace higher-saturated-fat foods in the diet. Furthermore, soybean isoflavones may have their own independent coronary benefits (10).

MM is a consultant to the soy food industry; his clients include the Archer Daniels Midland Co and the United Soybean Board. JWE serves as a member of the Scientific Advisory Panel for the United Soybean Board and has received food products from The Solae Company for human trials. He does not have any financial interests in any companies that produce soy food ingredients.

Mark Messina
Nutrition Matters, Inc
439 Calhoun Street
Port Townsend, WA 98368
E-mail: markm@olympus.net

John W Erdman Jr
Food Science and Human Nutrition
University of Illinois
449 Bevier Hall
905 South Goodwin Ave
Urbana, IL 61801
E-mail: jwerdman@uiuc.edu

REFERENCES

Vitamin A equivalency estimates: understanding apparent differences

Dear Sir:

Haskell et al (1) recently reported estimates of the vitamin A equivalency factors of β-carotene in oil, spinach, and orange-fleshed sweet potato that differ from those found by our laboratories (2–6). They reported factors of 6:1, 10:1, and 13:1, respectively, whereas we reported estimates of ≈2.7:1, 26:1, and 12:1, respectively (2–6). The estimates for yellow, orange, and red fruit and vegetables (YORFV) are almost the same, but those for β-carotene in oil and dark-green leafy vegetables (DGLV) are...