has both EFAs, but most soybean oil in foods is hydrogenated and thereby deplete of n−3 fatty acids. Furthermore, many manufacturers have shifted production toward low-fat foods and removed soybean oil from their products (eg, salad dressings and sauces). Thus, a major source of EFAs is disappearing from the market.

Commercials and government advertisements create the perceived desirable goal of eating no fat. "Today, the all-important index of healthfulness for many people is fat content. As a result, health-conscious consumers have anointed all kinds of improbable products as nutritionally correct" (eg, pretzels, licorice, and candy) (2). In 1994 we were interviewed by > 100 reporters in the United States and abroad. It was their consensus that media and advertisements encourage people to eat extremely low-fat diets. Fat is presumed to be bad, and therefore less of it is better. Consumers are not told that EFAs are essential. Under Food and Drug Administration and consumer protection statutes in most states, failure to explain the need for and role of EFAs would be considered a serious misrepresentation that would subject the perpetrator to severe fines.

Individuals eating mostly very-low-fat foods based on processed carbohydrates without oil supplements may have difficulties obtaining an adequate intake of EFAs if they follow the recommendations of the USDA pyramid. Because of huge variability in PUFA intake, millions of people do not eat enough PUFAs, even if the "average individual" intake is adequate. For example, a slim woman eating 1500 kcal/d (≈6279 kJ) who follows the USDA *Food Guide Pyramid* recommendations easily obtains 700 kcal (≈2930 kJ) from breads, pasta, and cereals. The remaining 800 kcal (≈3349 kJ) may come from vegetables, fruits, chicken, and low-fat dairy products. From these foods she cannot possibly get 15–20 g n−3 and n−6 EFAs/d.

The USDA notes that the *Food Guide Pyramid* reflects up-to-date knowledge of nutrition. However, the implicit and explicit assumptions and citations in the USDA *Food Guide Pyramid* are scientifically incorrect, because they are based on obsolete recommendations. It is irrelevant that the USDA believes that Americans eat enough EFAs or that EFA deficiency is not prevalent. What matters are proper blood test results. We analyzed the fatty acid profiles of > 500 adults from the Framingham Heart Study. More than 20% had biochemical evidence of n−3 or n−6 deficiency (3) with ≥ 5% having a severe deficiency. We found that EFA abnormalities are the most significant nutritional factor in elevated ratios of total to high-density lipoprotein cholesterol (4, 5).

We propose that nutritional requirements for EFAs should be expressed as absolute grams per kilogram body weight per day rather than as a percentage of daily energy intake, as recommended by most public health organizations. Otherwise, individuals who eat < 1500 kcal/d (≈6279 kJ) would meet the USDA recommended guidelines for EFA intake but could become EFA-deficient.

In our clinical experience, the amounts of PUFAs recommended by the USDA are too low for most adult Americans. By establishing a somewhat arbitrary recommendation for EFA intake, the USDA is assured that consumers meet their guidelines. Our clinical experience and research data indicate that the USDA nutrition recommendations, coupled with misleading food labels, are a major contributory factor to cardiovascular disease. Unless drastic changes are made in nutrition policy to emphasize the need for EFAs, we are afraid that current policies may increase morbidity and mortality.

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Nutrition, thyroid hormones, body temperature, and mortality of elderly patients with acute illnesses

Dear Sir:

Nogues et al (1) studied biological markers of nutritional status (serum albumin, prealbumin, transferrin, and cholesterol) or thyroid status (thyroid stimulating hormone, thyroxine, triiodothyronine, and reverse triiodothyronine) and clinical variables (loss of body weight and rectal temperature) for their association with the death of elderly patients presenting with an acute illness. I would like to take issue with the authors' data analyses and the interpretation they made of their results.

Several statistical errors plague the paper. The authors stated that they calculated the study sample size to achieve a statistical power of 5%. They probably meant a power of 95% (and a probability of type II error < 5%). The authors used unpaired Student's *t* tests even though, for some variables, the assumptions of Gaussian distribution and homoscedasticity (similar variance in both study groups) were not satisfied. For example, the loss of body weight (expressed as a percentage) cannot be expected to show a Gaussian distribution. For some variables, the SD varied by a factor of two between the two compared groups (eg, reverse triiodothyronine and serum prealbumin between survivors and nonsurvivors). The authors did not indicate what criteria were used for the inclusion and retention of variables in the stepwise discriminant analysis. In the reported equation thus obtained, it is not clear what units were used (the units μmol/L and ng/mL for reverse triiodothyronine both appear in different parts of the article). The predictive performance of the discriminant equation was evaluated on the same sample of patients that was used for its development. A reduced predictive performance would be observed in an independent sample (2). The transferability of the equation is further limited by the imperfect agreement between different assays of serum hormones (a different equation would be

required for another assay method for reverse triiodothyronine).

Several of the reported CIs for the means of the studied variables are clearly incorrect. For example, the CI for serum albumin in nonsurvivors (33.2–36.3 g/L) does not include the mean value (31.1 g/L). If this CI is correct then the reported statistically significant difference from the mean of survivors is erroneous since the CIs for the two groups would show a great overlap. For other results, the mean value is either at the lower or the higher limit of the reported CI rather that at the center as expected.

The authors also made mistakes in the conversion of their results to Système International (SI) units. With a molecular weight of ≈54 000 Da, serum prealbumin does not reach mol/L amounts as reported (the frequently reported reference range of 100–400 mg/L corresponds to 1.85–7.4 μmol/L). Similarly, the range of serum albumin concentrations reported in their Figure 1 is 10-fold too high (g/dL units are reported instead of the correct g/L units).

Contrary to the interpretation by the authors, I do not interpret the association between low serum albumin and mortality as primarily reflecting a nutritional deficit. As acknowledged by the authors, serum albumin is also a negative acute-phase reactant. It is also modified by nonnutritional means in some of the pathologies affecting the studied patients (fluid disturbances, congestive heart disease, and cirrhosis). If a nutritional deficit was the primary source of decreased albumin, it would be expected that the other nutritional markers would also be significantly modified. The observed alterations of total thyroid hormones are also difficult to interpret. Modifications of the concentration or binding affinity of the hormone-binding proteins can intervene in nonthyroidal illnesses. The authors did not measure the free hormone concentrations (the active fraction). They did mention the measurement of thyroxine-binding globulin in their Methods' section but these results are not reported.

Contrary to the suggestion of the authors, supplementation with thyroid hormones have not been proven effective in patients with nonthyroidal illnesses (3, 4). Finally, interpretation of body temperatures as a prognosis variable requires that we take into account the normal diurnal variations. Rectal temperatures of 36.6 °C can be observed in normal individuals in the early morning. Careful calibration of the thermometers would be required to achieve good discrimination between normal and abnormal body temperatures when a diagnosis criterion of < 36.5 °C for hypothermia is used.

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Reply to J Massé

Dear Sir:

We thank Massé for having thoroughly read our paper on the relation between thyroid hormones, nutrition, and body temperature in elderly people. He alludes to several numerical and unit errors that are acknowledged in an erratum that follows in this issue (1).

Massé is right in pointing out that it might have been preferable to use a nonparametic test to compare some of the variables [eg, percentage weight loss and serum prealbumin and reverse triiodothyronine (rT_3)] between survivors and nonsurvivors (table 3) since they do not appear to have a Gaussian distribution. When these variables were compared with the Mann-Whitney *U* test, the results were as follows: percentage weightloss, $P = 0.051$; prealbumin, NS; rT_3, $P = 0.007$.

Rather than suggesting a new predictive model based on admission parameters, Figure 1 was meant to graphically illustrate the probability of death in our population according to values (extreme low and high) for rT_3, rectal temperature, and serum albumin. For the stepwise discriminate analysis only variables with an *F* value ≥4 were retained. The clinical meaning of the drawing is independent of the units used to build the equation and would not be altered by results yielded by different rT_3 assays.

The difficulties in interpreting serum albumin as a nutritional marker are made clear in the Discussion section of our article and have been thoroughly discussed elsewhere (2, 3). We feel, though, that albumin may have a nutritional significance in our elderly population for the three reasons put forward in our article. In addition, body weight and prealbumin were significantly lower in hypoalbuminemic patients (data not given in the manuscript). This finding reinforces our suggestion that the serum albumin concentration at admission may have been influenced by the preillness nutritional status.

Thyroxine-binding globulin was also measured in our patients. It tended to parallel changes in T_3 concentrations, being significantly lower in the 31 patients with low T_3 ($P = 0.0001$). For this reason we assumed that total hormone concentrations would quite accurately reflect free hormone concentrations.

Correlation between rectal temperature and APACHE II scores, thyroid hormone concentrations, and death rates make it difficult to accept Massé's criticism that hypothermia may be a normal phenomenon linked to diurnal variation of body temperature. We think it represents a true and clinically relevant finding linked to an altered response to injury and associated with a high death rate. Other articles referenced in our manuscript also support this assertion.