

and 14 years of followup was highly statistically significant (Refs. 21 and 38). In a Finnish study, the dose response relationship of *trans* fat with risk of CHD death was significant ( $p = 0.004$ ), but was not significant for risk of major coronary event ( $p = 0.158$ ) (Ref. 20). In a study of U.S. men, the dose response relationship was significant after statistical adjustment for major CHD risk factors ( $p = 0.01$ ) but was not significant after additional adjustment for dietary fiber ( $p = 0.2$ ) (Ref. 19). Therefore, the prospective studies were consistent with a dose-response relationship, although the relationship was not statistically significant in all analyses. Moreover, as discussed previously in this section, FDA's quantitative estimate of health benefits was not based on the prospective studies, but was based on the regression equations summarizing the results of the intervention feeding studies (tables 8 and 9 in this document and 64 FR 62746 at 62757–62770). The regression equations summarizing the effect of *trans* fat on LDL-C and HDL-C in the intervention studies did show a dose response relationship, as discussed in the November 1999 proposal and noted in section IV of this document. Additionally, the regression equations used by FDA in this document and in the November 1999 proposal were for purposes of making a quantitative estimate of the health benefits as part of an economic analysis and are consistent with newer regression equations in a study published in 2001 (Ref. 130). Therefore, FDA does not agree with the comment that it is not possible to calculate health benefits because there is no dose-response relationship for the adverse effects of *trans* fat.

FDA disagrees with the comment that the health benefits estimate did not account for other CHD risk factors. In the health benefits estimate, FDA used the factors shown in table 8 to calculate the amount of CHD risk associated with the expected amount of change in LDL-C and HDL-C. These factors were derived from large population studies of serum lipids and CHD risk, in which statistical methods accounted for other positive and negative risk factors for CHD.

Regarding the comment about the level of *trans* fat intake in the intervention studies, Section IV of this document explains that, because of uncertainty in intake estimates, caution must be exercised to avoid over-interpretation of the available dietary intake estimates and their relationship to the *trans* fat levels used in the intervention trials. However, in response to the comment, FDA notes

some specific examples of intervention studies with lower *trans* fat intake. One example is the study of Judd et al., 1998 (Ref. 34), which found a significant increase in LDL-C with a difference in *trans* fat intake of 1.5 percent of calories between the *trans* fat test diet (3.9 percent of calories from *trans* fat) and the comparison diet (2.4 percent of calories from *trans* fat). Another example is the study of Lichtenstein and coworkers (Ref. 82) which studied six test diets and reported a positive coefficient, i.e., a linear trend, for the association of the change in LDL-C levels among diets with the change in *trans* fat intake (including *trans* fat changes of 0.4 percent and 2.8 percent of calories). Such a linear trend does suggest that *trans* fat intakes below 3 percent of calories may influence LDL-C levels, and thus, CHD risk. Therefore, significant increases in LDL were found in specific intervention studies with *trans* fat intake at or below the reported average intake for the U.S. population.

FDA disagrees with the comment that disclosure of 0.5 g *trans* fat or greater in a food product has no public health importance and that health benefits may not result from labeling of *trans* fat present in relatively small amount in individual foods. As described earlier in sections III and V of this document, FDA does not need to demonstrate adverse health effects of 0.5 g *trans* fat in a food product in order to justify requiring disclosure of 0.5 g *trans* fat on food labels. Rather, FDA determined that the consistent provision of *trans* fat information on foods consumed throughout the day is of public health importance and can assist consumers in maintaining healthy dietary practices. Further, FDA has determined that the absence of *trans* fat information on foods requiring mandatory labeling would be misleading. However, for the purposes of economic analysis, the health benefits of decreasing *trans* fat intake by 0.5 g can be estimated quantitatively. In a 2,000 calorie diet, 0.5 g *trans* fat corresponds to approximately 0.2 percent of energy. (This correspondence holds because 1 g of fat = 9 kcal, so  $(0.5 \times 9 \times 100)/2000 = 0.2$  percent of energy). Using the factors in table 8, replacement of 0.2 percent of energy from *trans* fat with *cis*-monounsaturated fat would decrease CHD risk by 0.29 percent based on LDL-C and 0.57 percent based on LDL-C and HDL-C. Because CHD is so common in the U.S. population, a relatively small decrease in risk corresponds to a large number of cases and deaths avoided and large dollar value of such benefits, as shown in the example in section IX.A of

this document. Awareness of *trans* fat contributions from food products containing 0.5 g and above will assist individual consumers in maintaining healthy dietary practices, reducing the average 2.6 percent of energy from *trans* fat consumed throughout the day.

FDA agrees with the comments that average saturated fat intake in the United States is about 5 times greater than average *trans* fat intake. FDA stated in the November 1999 proposal that it did not want to distract consumers from years of dietary guidance messages about saturated fat (64 FR 62746 at 62755). But the potential health benefits from decreasing *trans* fat intake compared with decreasing saturated fat intake do not depend solely upon the average total amount of each in the diet. The potential health benefits also depend upon the feasibility of decreasing intake of saturated fat compared with *trans* fat. Average U.S. saturated fat intake in 1980 was about 13 percent of energy and decreased to 11 or 12 percent of energy by the mid-1990s (Ref. 113). Many additional heart attacks and deaths might be prevented if saturated fat intake could be decreased to the recommended less than 10 percent of energy. The targeted decrease in saturated fat intake of one or two percent of energy can be compared with the average *trans* fat intake of 2 percent of energy from partially hydrogenated fats and oils. Labeling of *trans* fat will create new potential for decreased *trans* fat intake by providing an incentive to food manufacturers to reduce the amount of *trans* fat in their products and by providing consumers with information they need to include *trans* fat content in their food purchasing decisions.

(Comment 40) Among the comments that supported the potential public health benefits of *trans* fat labeling, many noted that benefits would result from provision of *trans* fat information on product labels so that consumers could incorporate this information into their purchasing decisions. Several comments also specifically noted the likelihood that *trans* fat labeling would result in reformulation of products to be lower in *trans* fat, and suggested that the public health benefits would be large because reducing *trans* fat intake as a result of reformulation requires little effort by consumers. However, some comments did not agree that *trans* fat labeling would be read or understood by consumers, or that the labeling would affect purchasing decisions. These comments suggested that the net health benefits of *trans* fat labeling would be much smaller than FDA's estimate. Other comments did not agree that