Dear Sir:

In a study recently reported in the Journal, Morcillo et al (1) examined the interaction between intestinal fatty acid–binding protein 2 (FABP2) polymorphism and the intake of dietary fats; they determined that the FABP2 Thr54 polymorphism was associated with insulin resistance in subjects with a high intake of sunflower oil. Weiss et al (2) confirmed that, among sedentary nondiabetic persons following a low-fat diet, FABP2 Thr54 carriers have lower glucose tolerance, lower insulin action, and higher lipid oxidation rates than do Ala54Ala carriers. These results were very interesting to our group, which had studied the effect of FABP2 polymorphism on delta-6 desaturase (D6D) activity in obese children (3).

In our study of 32 obese children with a mean (±SD) age of 12.0 ± 3.0 y, the allele frequencies were 0.66 and 0.34 for the FABP2 Ala54 and Thr54 polymorphisms, respectively. Among the FABP2 genotypes, no significant difference in age, body mass index, fasting glucose, insulin, or serum lipoproteins was observed. The content of arachidonic acid (AA) in fasting plasma was significantly lower in Thr54Thr carriers (0.05 ± 0.02) than in Ala54Ala (0.07 ± 0.02) carriers. These results were very interesting to our group, which had studied the effect of FABP2 polymorphism on delta-6 desaturase (D6D) activity in obese children.

Recently, the linkage between D6D activity and obesity was investigated by Warenjo et al (4). This study found that the risk of being overweight was increased by 60% for each 1-SD increase in D6D activity, which was estimated by the fatty acid profile of serum cholesteryl esters. Our group also studied D6D activity in children and found that the D6D was activated in obesity (5). The hypothetical mechanism proposed in rodent animal models is that the increased oxidation of AA must be met by compensatory activation of D6D (6). However, the activities of D6D and delta-5 desaturase are not modified in insulin-resistant animals, even though insulin is a strong activator of these enzymes (7, 8).

In an earlier study (5), our group found that ≈25% of obese children had low plasma AA content (>1 SD below the mean) and a low ratio of AA to linoleic acid, probably because of an impaired compensatory production of AA. In addition, the obese children with low AA content had higher insulin concentrations, although their percentage body fat, waist circumference, and leptin concentrations were similar to those in obese children with normal AA content. AA itself affects the insulin sensitivity of adipocytes because AA can act as a ligand for peroxisome proliferator–activated receptor-γ (9). Our results are compatible with the suggestion by Das (10) that impaired D6D activity is associated with insulin resistance, and they may partly explain the metabolic heterogeneity of obesity. Furthermore, our recent study of FABP polymorphism suggests that Thr54Thr may be a predisposing factor for the impaired D6D activation in obesity. We speculate that the modulation of the absorbed fatty acid composition by FABP2 genotype may affect D6D activity and AA content and, thereby, insulin sensitivity.

None of the authors had a personal or financial conflict of interest.

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Reply to T Okada et al

Dear Sir:

First, we thank Okada et al for their interest in our article. In a study of 32 obese children (1), they found that the content of arachidonic acid (AA) in fasting plasma and the delta-6 desaturase (D6D) activity estimated by the (18:3/18:2) ratio were significantly lower in Thr54Thr carriers than in Ala54Thr or Ala54Ala carriers. This finding is interesting, because it provides information about the role played by the various fatty acid desaturases in the risks of obesity and the metabolic syndrome (2–4). However, clinical and epidemiologic studies on fatty acid desaturase activity cannot be evaluated without also considering diet and the metabolic status of the study subjects (2). In our study, we (5) examined the fatty acid composition of the plasma phospholipids and used an analysis of variance model to evaluate the association between the fatty acid composition and the Ala54Thr polymorphism of the FABP2 gene. The main variable was FABP2 polymorphism (Ala54Aa, Ala54Thr, or Thr54Thr), and the independent variables were sex, obesity (body mass index [BMI]; in kg/m²) > 30, the presence of a carbohydrate metabolism disorder (olive or sunflower), and the type of dietary cooking oil (oatmeal or sunflower), with age as a covariable. The variance in lipids was explained by age (P = 0.02 and P < 0.0001, respectively), whereas the variance in the n−6 fatty acids was explained by the type of dietary fatty acids (P = 0.001). The Ala54Thr polymorphism contributed significantly to the explanation for the variance in the monounsaturated fatty acids of the plasma phospholipids (P = 0.02) and the delta-9-desaturase activity (measured by the 16:0/16:1 ratio) (P = 0.01) after adjustment for age, sex, the presence of obesity, the type of oil consumed, and the presence of any carbohydrate metabolism disorder (Table 1). The D6D activity, however, measured by the AA/18:2 ratio, was explained by the type of dietary fatty acid (P = 0.02) and age (P = 0.02), but not by Ala54Thr polymorphism. The study was undertaken within the context of an epidemiologic study representative of a culturally homogeneous ethnic population for whom 20% of dietary calories come from the consumption of monounsaturated fatty acids (6), and with the controls and the controls coming from the same population, thereby avoiding the possibility of selection bias. The results highlight the difficulty of speaking about fatty acid desaturase activity independently of information about the type of dietary fatty acid and about the metabolic status.

None of the authors had a personal or financial conflict of interest.

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REFERENCES


An error in the US Department of Agriculture nutrient database results in vitamin A values that are 6 times too high

Dear Sir:

We wish to inform the readership of the Journal of a serious error in the vitamin A values listed in the US Department of Agriculture nutrient database. We have identified a discrepancy in the US Department of Agriculture nutrient database results in vitamin A values that are 6 times too high. We encourage all researchers to be aware of this potential error and to cross-check their findings with more reliable sources. We recommend using alternative sources of information to ensure accurate data for their research.

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