work in animal and human studies suggests that the dietary linoleic acid:ALA ratio and dose response to ALA may also be important with regard to the relation between ALA intake and long-chain n–3 PUFAs status (4–7).

Although further explanations for mechanisms to explain the findings in our observational study are of interest, this should not distract from the fact that substantial differences between intakes and status of long-chain n–3 PUFAs were found within our study in the different diet-habit groups, and the proportion of circulating long-chain n–3 PUFAs was higher in non-fish-eaters than would be expected given intake: intake of total n–3 PUFAs in meat-eaters was 72% of that in fish-eaters and in vegetarians was 70%, whereas the status of meat-eaters was only 92% and of vegetarians was 87% of that of fish-eaters. (For intakes of EPA and DHA, meat-eaters consumed only 12% of that of fish-eaters and vegetarians consumed only 5%). Our study is also the largest to date to show such detailed population differences in habitual intake and status, particularly for ALA intake.

Unresolved questions are, first, whether the lower long-chain n–3 PUFA status in non-fish-eating vegetarians observed in this and other studies is of clinical importance and, second, what conditions induce optimum conversion of ALA to long-chain n–3 PUFA in non-fish-eating populations. To answer these questions, further carefully controlled human intervention studies are needed to measure the relation between intake of ALA and long-chain n–3 PUFA status in those with vegan and vegetarian eating patterns that also take into account the background dietary composition, dose response, age, and body weight and that are of sufficient size and duration to answer these important questions.

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REFERENCES

Heritability of serum vitamin D concentrations: twin studies

Dear Sir:

It was with interest that we read the article by Karohl et al (1), which uses a classical twin study design. Serum 25-hydroxyvitamin D [25(OH)D] concentrations were examined in American adult twins to estimate the genetic contribution in determining circulating 25(OH)D concentrations. The difference in correlation of a given phenotype within monozygotic compared with dizygotic twin pairs, or the intraclass correlation (ICC), provides insight into the relative contributions of genetic factors to the phenotype (2). Structural equation modeling and other methods are used to estimate heritability or the proportion of phenotypic variation that is attributable to genetics (2). Elucidating the genetic components involved in regulating 25(OH)D status is a valuable pursuit given the known associations of suboptimal vitamin D status and a wide range of diseases.

Karohl et al (1) reported ICCs for wintertime serum 25(OH)D of $r = 0.69$ for monozygotic twin pairs and $r = 0.29$ for dizygotic twin pairs in a total of 100 twin pairs. Results from a 2008 twin study in the Journal (3) showed ICCs for winter serum 25(OH)D of $r = 0.71$ for monozygotic twin pairs and $r = 0.32$ for dizygotic twin pairs in 99 twin pairs. In the study by Karohl et al, the proportion of trait variation attributable to genetic factors (heritability) was 0.70, which was very similar to the estimate of 0.77 in the study by Orton et al. Only wintertime 25(OH)D concentrations were examined by Orton et al because of the known seasonal fluctuation and highly dominant environmental influence during summer. Given the similarity in subject material, approach, and findings, we find it surprising that the authors did not acknowledge the Orton et al article. Given that it was published in the Journal <2 y ago, we feel it would have been appropriate to mention that the findings confirm those published by Orton et al.

Furthermore, in the Discussion, Karohl et al (1) consider vitamin D metabolic pathway gene candidates potentially responsible for the observed heritability. However, a key step was overlooked. The 1$\alpha$ hydroxylase enzyme (CYP27B1) is responsible for the rate-limiting conversion of 25(OH)D to the active vitamin D metabolite and is involved in regulatory feedback loops. The study by Orton et al (3)
showed that CYP27B1 gene variants were associated with 25(OH)D concentrations ($P < 0.001$). Further studies are consistent with the association of CYP27B1 single nucleotide polymorphisms (which have high linkage disequilibrium) and 25(OH)D (4). We feel that including this previously published finding in their discussion of potential vitamin D genes would have been useful for readers, especially those looking to extend this line of research.

We appreciate that not every previous study can be acknowledged, especially in light of word and referencing limits. We also recognize there are differences between the 2 articles. However, it is beyond our experience that an article with a comparable design and highly similar results and conclusions goes unacknowledged in the same journal.

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REFERENCES


Comment on the article “A saturated fatty acid–rich diet induces an obesity-linked proinflammatory gene expression profile in adipose tissue of subjects at risk of metabolic syndrome”

Dear Sir:

The effect of nutrient composition on human adipose gene expression, independently of body mass index, is largely unknown. It was therefore with great interest that I read the article by van Dijk et al (1) in a recent issue of the Journal. In their article, van Dijk et al report that intervention with a diet rich in saturated fatty acids (SFAs) results in changed expression of 1523 genes in human white adipose tissue, whereas intervention with a diet...