Reply to S Somerset

Dear Sir:

Our hypothesis-based analyses showed that a high intake of anthocyanins was associated with an 8% reduction in risk of hypertension, and the magnitude of the reduction in risk was greater in participants aged ≤60 y (1). To best capture dietary flavonoid intakes, we used a cumulative intake approach starting with our 1990 questionnaire, which covered a broader range of flavonoid-based foods from the habitual diet. Such observational data highlight associations between different flavonoid subclasses and a range of health endpoints to help develop research priorities and optimize the design of randomized controlled trials.

The US Department of Agriculture database, like Somerset’s food analyses, highlights several rich dietary sources of specific flavonoids, but because our analyses focused on habitual intakes in US participants, it was not surprising that some foods that contain high amounts of some flavonoid constituents were not good dietary sources in our population group because the quantity and frequency of consumption were low. To calculate flavonoid amounts in foods that were grouped together in our questionnaire, we used grocery store and population survey data of US consumption patterns to allocate intake data for each individual food within that grouping. For mixed dishes, such as vegetable pizza, we followed standardized recipes and allocated flavonoid values to the individual ingredients.

We used a food-frequency questionnaire (FFQ) and categorized our study participants into quintiles of intake; previous data have shown that when quintiles of flavonoid-rich foods, such as fruit and vegetables, are included in a linear regression model, regression coefficients are similar for intakes estimated with 2 dietary assessment instruments: a 7-d diary and an FFQ (2). Any additional measurement error associated with our FFQ would bias our findings toward the null and suggest that the risk reduction we report with increased anthocyanin intake is likely to be an underestimation of the true association.

Neither of the authors had a conflict of interest to declare.

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REFERENCES


Lack of evidence for increased ω-linolenic acid metabolism in vegetarians

Dear Sir:

In their article in the November issue of the Journal, Welch et al (1) address a very important issue. Vegans and vegetarians are limited with regard to food sources of the preformed long-chain omega-3 polyunsaturated fatty acids (LC n–3 PUFAs) docosahexaenoic acid (DHA; 22:6n–3) and eicosapentaenoic acid (EPA; 20:5n–3), and intakes can be very low. However, there are several fundamental problems with this study that lead us to question the conclusions of the authors.

The main premise of this study is that various types of vegetarians have greater ω-linolenic acid (ALA) metabolism as shown by a higher bloodstream content of its metabolites such as EPA and DHA. This contention is not well borne out by the data presented in the authors’

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TABLE 1

Amounts of selected flavonoids (in mg/100 g) in the edible portion of particular foods

<table>
<thead>
<tr>
<th>Food</th>
<th>Quercetin</th>
<th>Cyanidin</th>
<th>Petunidin</th>
<th>Delphinidin</th>
<th>Pelargonidin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red plum</td>
<td>1.85</td>
<td>4.73</td>
<td>2.21</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Black plum</td>
<td>12.45</td>
<td>39.68</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Peach</td>
<td>0.68</td>
<td>1.61</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Eggplant</td>
<td>0</td>
<td>0.02</td>
<td>0</td>
<td>13.76</td>
<td>0.02</td>
</tr>
<tr>
<td>Zucchini</td>
<td>0.47</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

1 Data are from reference 3.

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Neither of the authors had a conflict of interest to declare.

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Table 5. For example, in men, plasma values for DHA were lowest in male vegans and vegetarians, and these were virtually the same as in the meat-eating group. In vegan men, even though ALA intake was the highest in any group and may have led to an increased EPA level, the docosapentaenoic acid n–3 was the lowest value found in any of the dietary groups. The plasma of women vegans exhibited the highest value for DHA of any group, and this is the apparent cornerstone of this report. However, this was not confirmed by the findings in vegetarian women who had the lowest value for DHA in any of the female dietary groups. Further doubt about the main finding concerning the vegan DHA value in women was evident from the very large SD value of 211, which was nearly as large as the mean value of 286. This SD dwarfs the difference in mean values between vegans and meat-eating women, which was only 45.

A second issue is that ratios of precursors to products from a fatty acid profile have no relation to fatty acid metabolism, and any such inference is incorrect. This was most clearly shown in a stable isotope–labeled ALA (18:3n–3) study by Pawlosky et al. (2) in which it was shown that the incorporation of the stable isotope label had increased in several cases, even though the levels of the end products had declined or stayed the same. In other words, the metabolism appeared to go in the opposite direction from that predicted by a precursor-product relation. In short, calculations made from fatty acid compositional data alone cannot be used to suggest that there is an increased metabolism of ALA to EPA or DHA.

One possible factor that might be predicted to lead to a higher ALA conversion to its LC-PUFA metabolites would be a lower intake of linoleic acid (LA) in vegetarians. However, the authors report that LA intake was highest in vegetarian and vegan men and women (Table 1). In fact, the highest intake in any group was observed in male vegetarians, which was 25% higher than that observed in meat-eating men. Clearly then, a low LA intake in vegetarians cannot provide a basis for a claim of increased ALA metabolism.

Finally, the n–3 intakes reported for the entire study population are much lower than in other reports from this geographical area. The European Food Safety Authority (3) reports that the intake of total n–3 PUFAs for the general UK population is up to twice that reported by Welch et al. With regard to UK vegetarians, previous estimates indicate an order of magnitude higher intake of DHA than that reported by Welch et al. (4). The nutrient database used in the current study to analyze food records may have contributed to this discrepancy. Several foods are reported by the authors to contain EPA but zero DHA, which rarely, if ever, occurs. In addition, food tables often do not give amounts <0.1 g/100 g, which can cause errors in the calculation of total DHA. Finally, vegetable items from the food-frequency questionnaire are reported to supply EPA; however, because plants lack the enzymes to elongate and desaturate ALA, it is not possible for these vegetables to contain EPA. It is not surprising to find a database that is lacking reliable data with regard to the composition of LC n–3 PUFAs. In the United States, for example, the US Department of Agriculture’s National Food and Nutrient Analysis Program was created out of necessity to achieve “long-sought improvements in the nutrient values in the National Nutrient Databank System” (5).

n–3 Fatty acids are among the nutrients cited by the program as in need of better estimation. In short, although we do not question that the LC n–3 PUFA intake of vegetarians and vegans is low, the extremely low intake reported in the current study may have been influenced by nutrient database inaccuracies. Without accurate intake information, correlations between intake and DHA plasma concentrations become irrelevant. A failure to find a correlation between DHA intake and plasma concentrations in the face of imprecise intake data should not be considered evidence of increased in vivo synthesis of DHA from ALA.

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doi: 10.3945/ajcn.111.012211.

Reply to N Salem Jr and CN Kuratko

Dear Sir:

Unfortunately, there were a number incorrect assumptions and interpretations made by Salem and Kuratko, which need to be rectified.

Our study was focused on both fish-eaters and all non-fish-eaters, including a large group of non-fish-eating meat-eaters, in a UK population, as is clearly stated in our article (1). We agree that the data in Table 5 are as described; however, it is clear that we investigated the relative relation between long-chain n–3 polyunsaturated fatty acid (LC n–3 PUFA) status and α-linolenic acid (ALA) intake. This product-precursor ratio is described in our Tables 6 and 7, after accounting for the known factors that would affect this ratio.

Intakes of LC n–3 PUFAs reported in our study are a little lower than in certain other reports, but few data for individual LC n–3 PUFA [eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)] exist to make comparisons, as noted in our article (1). The higher intakes of LC n–3 PUFAs quoted by Salem and Kuratko included ALA within the total and so are not directly comparable with our data. Our data for total LC n–3 PUFAs did not include 18:4n–3 (stearidonic acid) or docosapentaenoic acid, unlike some food-composition databases (2, 3). With regard to comparative intakes of DHA in vegetarians, we are not clear why Salem and