Intensive dietary counseling does not affect oncologic outcome

Dear Sir:

I read with interest the article by Ravasco et al (1) in the December issue of the Journal, and I was impressed by their Figure 1, which showed the survival of the 3 arms of their study and especially that of group 1 patients (intensive counseling) who had a 5-y survival of ~100%.

I am not aware of any oncologic study involving patients with an advanced colorectal cancer—and hence undergoing a preoperative or postoperative oncologic treatment or a palliative one, as reported in the original article (2)—who had such a survival rate, which perhaps might be observed only in early-stage (Dukes’ A) cancer patients.

I think that differences in survival are better explained by an inhomogeneous distribution of patients with different severity of disease within the 3 arms, and arm 1 included the most favorable cases.

This unfortunately represents an important bias, which invalidates the conclusions of the authors.

The author had no conflicts of interest to disclose.

Amelia Guglielmina Longhi
Via Redi 28
20100 Milan
Italy
E-mail: ameliaconghi@hotmail.com

REFERENCES

doi: 10.3945/ajcn.112.056523.

Reply to AG Longhi

Dear Sir:

Early individualized nutritional counseling during radiotherapy was effective in reducing toxicity and in improving nutritional intake and status, as well as quality of life. After treatment completion and nutritional intervention, the efficacy persisted at the 3-mo follow-up. Similar results were attained by Isenring et al (1), in which individualized counseling compared with standard practice improved the patients’ nutritional status, intake, functional capacity, and quality of life. According to the European Society for Clinical Nutrition and Metabolism guidelines (2) to increase nutritional intake, prevent therapy-associated weight loss, and treatment interruptions.

In our study published in the December 2012 issue of the Journal (3), we show that individualized nutritional counseling has to be implemented adjuvant to antineoplastic treatments, and it should be planned along with the scheduled treatments. The nutritional content of the patient’s diet based on regular foods with appropriate manipulation was important in improving gastrointestinal function and other symptomatic manifestations during treatment and in the medium term. Also, treatment toxicity and symptom incidence and/or severity in patients who received dietary counseling and education were lower, and their improvement in the medium term was faster. After careful consideration and intensive discussion with several international clinical experts in multiple congresses and meetings, we can say that the results we present do show that nutrition improves survival and outcome. Dietary modifications alter bowel functions, such as motility, enzyme secretion, and nutrient absorption; likewise, nutrition modulates gastrointestinal flora whose ecology is central to the pathogenesis of radiation injury severity and therapeutic efficacy. Likewise, antineoplastic treatment can be effective only in well-nourished patients, as shown by the Baracos group (4). The depleted muscle and nitrogen reserves as a consequence of depleted nutrition status and the absence of nutrition intervention may be the mechanism underlying the efficacy of nutrition in improving patient outcomes, thus the suggestion that nutritional intervention and a maintained nutritional status are associated with favorable outcomes in cancer.

Our data include results from a 3-arm clinical trial, and the patient samples were stratified by cancer stage; otherwise, any results would be biased. Clinicians in many centers and clinical statisticians were consulted to help in data interpretation, to confirm that patient samples were homogeneous. We included patients in Dukes’ A, B, and C cancer stages in the 3 arms of the study. This was mandatory to enable any conclusions.

There are now other groups that report on the key role of adequate nutrition in cancer outcomes; studies are under way in Finland and Denmark that point in the same direction and that have reached similar results (H Orell-Kotikangas, unpublished observations, 2013). The references listed (5–11) support that patients with poorer dietary intake, worse nutritional status, and poorer quality of life had a significantly shorter survival and increased incidence of symptoms. Patients should receive nutritional counseling with or without supplements according to their dietary intake at the end of any antineoplastic treatment or surgery. Our studies as well as randomized trials from Isenring et al (1) confirm these associations.

A comprehensive review of the current literature undertaken by Lis et al (5) in 2012 supports the implementation of nutritional screening, assessment, and individualized counseling to correct nutritional derangements in cancer patients, and shows the efficacy of nutrition intervention in any stage of disease (Dukes’ A, B, and C).
Indeed, there is evidence in a range of conditions to support the hypothesis that enabling the provision of the appropriate nutritional therapy leads to improved body weight and fat-free mass and that this generally reflects an improvement in protein and energy status.

The authors had no conflicts of interest to disclose.

Paula Ravasco
Isabel Monteiro-Grillo
Maria Camilo

Laboratory of Nutrition
Unit of Nutrition and Metabolism
Institute of Molecular Medicine
Faculty of Medicine of the University of Lisbon
Avenida Prof Egas Moniz
1649-028 Lisboa
Portugal
E-mail: p.ravasco@fm.ul.pt

REFERENCES
2. Foo SY, Heller ER, Wykrzykowska J, Sullivan CJ, Manning-Tobin JJ, Moore KJ, Gerszten RE, Rosenzweig A. Vascular effects of low-carbohydrate, high-protein (LCHP) diets may be effective in improving markers of cardiovascular risk and thus may be important in diabetes management. In making this conclusion, authors ignored important evidence from animal and human studies.

Foo et al (2) reported that mice fed an LCHP diet developed significantly more atheroma than mice fed a Western diet (5.4% compared with 2.2%, respectively, after 6 wk ($P = 0.004$); and 15.3% compared with 8.8%, respectively, after 12 wk ($P = 0.013$)). In comparison, mice fed a typical chow diet, in which carbohydrates accounted for 65% of calories, had minimal atheroma (0.5% and 1.3% at 6 and 12 wk, respectively). This difference was statistically significant from both the LCHP and the Western diets ($P = 0.01$). Furthermore, the same researchers found that the recovery of perfusion after inducing ischemia in mice fed the LCHP diet was ~39% less compared with mice fed the Western diet at 28 d after surgery ($P = 0.013$). The following are important conclusions made by the authors of that study: “Exacerbated atherosclerosis occurred on the LCHP diet independent of significant alterations in traditional atherogenic serum lipids, serum inflammatory markers and histological indicators of inflammatory infiltration”; “in addition to increasing atherosclerosis in ApoE/mice, the LCHP diet impaired neovascularization”; and “Taken together, these data demonstrate that in animal models LCHP diets have adverse vascular effects not reflected in serum markers and that nonlipid macronutrients can modulate vascular progenitor cells and pathophysiology” (2).

Fleming and Boyd (3) reported similar adverse effects on coronary artery flow with an LCHP diet in humans. Among the 16 individuals who adhered to a diet in which 70% of calories were supplied by carbohydrates, findings showed an improvement in coronary artery disease (CAD) as measured by each of the biomarkers assessed and the recovery of myocardium. On the other hand, both the progression and the severity of CAD, on the magnitude of 39.7%, was observed among the 10 individuals who adhered to an LCHP diet (significantly different from baseline, $P = 0.001$). The LCHP group showed a worsening of independent risk factors, in addition to progression of CAD. These authors concluded that “these results would suggest that high-protein diets may precipitate progression of CAD through increases in lipid deposition and inflammatory and coagulation pathways” (3). Most recently, Merino et al (4) reported similar findings.

Considering that individuals with type 2 diabetes represent a high-risk group for diabetes-related cardiovascular complications, in light of the findings of the above-reviewed studies LCHP diets are hardly appropriate for patients with type 2 diabetes. On the contrary, these results indicate that clinicians should be very cautious in interpreting any improvements in traditional cardiovascular risk markers as beneficial in prevention of these widespread diabetes comorbidities.

The author did not declare any conflicts of interest.

Roman Pawlak

East Carolina University
Rivers West 337
Greenville, NC 27858
E-mail: pawlakr@ecu.edu

REFERENCES
2. Foo SY, Heller ER, Wykrzykowska J, Sullivan CJ, Manning-Tobin JJ, Moore KJ, Gerszten RE, Rosenzweig A. Vascular effects of...


Reply to R Pawlak

Dear Sir:

Pawlak argues that our recent systematic review and meta-analysis (1) did not take into consideration evidence from animal and human studies, which suggest that low-carbohydrate, high-protein (LCHP) diets might have detrimental vascular effects. Pawlak summarizes the findings from one human (2) and one animal (3) study to support this assertion. As Pawlak correctly surmises, neither of these studies looked at dietary interventions in humans with type 2 diabetes and were therefore not within our search criteria.

Foo et al (3) studied mice fed 1 of 3 diets. A “standard chow” (SC) diet high in carbohydrate (60% carbohydrate, 23% fat, and 17% protein), a Western diet (WD; 43% carbohydrate, 42% fat, and 15% protein), or an LCHP diet (12% carbohydrate, 43% fat, and 45% protein). Atherosclerosis progression was assessed in 4 SC, 11 WD, and 11 LCHP mice at 6 wk and in 3 SC, 7 WD, and 7 LCHP mice at 12 wk. At 12 wk, mice fed the SC diet had virtually no atheroma (0.5% of the aortic luminal area), whereas those fed the WD had less atheroma than those fed the LCHP diet (8.8% compared with 15.3% luminal area, respectively). There were no differences between any of the diets in conventional cardiovascular risk factors.

Although these results in a mouse model might not be easily generalizable, Foo et al’s study makes the important point that measuring soft endpoints such as cardiovascular risk markers does not tell the whole story, highlighting the importance of studies of appropriate size and duration in humans to guide recommendations.

The studies included in our analysis are consistent with other animal studies. Raymond et al (4) randomly assigned 48 mice to either a diet high in fat and protein or one high in fat but low in protein. The mice fed the high-protein diet had lower cholesterol and higher HDL cholesterol at 4 wk compared with those fed a low-protein diet.

The 26 individuals in Fleming and Boyd’s study (2) were instructed to follow a specific dietary plan while being actively treated for any abnormalities in lipid profile detected throughout the study. It was found retrospectively that 10 of these patients did not adhere to the prescribed diet but stated that they intermittently consumed a high-protein diet. The nature of this diet is not described and did not form part of the intervention under investigation. Adherence to this undefined “high-protein” diet was intermittent at best, making it impossible to draw any conclusion as to the role of a high-protein diet in the progression of coronary artery disease.

Two randomized trials in obese humans showed that those who consumed an LCHP diet had improved lipid profiles compared with those who consumed a high-carbohydrate, low-fat diet after 1 y (5, 6). These results are consistent with our conclusions, which are suitably judicious and remain unaltered by Foo et al’s (3) interesting work in animals.

The studies included in our systematic review and meta-analysis (1) showed that low-carbohydrate, low–glycemic index, Mediterranean, and high-protein diets were effective in improving various markers of cardiovascular risk in people with diabetes. We argue that these diets may be valid options to consider in people with type 2 diabetes.

None of the authors had a conflict of interest.

Olabukola Ajala
Patrick English
Jonathan Pinkney

Department of Diabetes and Endocrinology
Peninsula College of Medicine and Dentistry
Plymouth PL6 8DH
United Kingdom
E-mail: olabukola.ajala@nhs.net

REFERENCES


Diabetes and diet beverage study has serious limitations

Dear Sir:

In the article “Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidémiologique auprès des femmes de la Mutuelle Générale de l’Education Nationale–European Prospective Investigation into Cancer and Nutrition cohort” (1), the authors examined the associations between sugar-sweetened beverages, artificially sweetened beverages, and type 2 diabetes. We argue against the conclusion in this study that consumption of artificially sweetened beverages increases the risk of developing type 2 diabetes due to the following study limitations.

Poor assessment of beverage consumption. The assessment of sugar-sweetened beverages and artificially sweetened beverages was inadequate. Although the study ran from 1993 to 2007, participants were asked only once in 1993 about their diet beverage consumption patterns. What and how much the
subjects drank likely changed over the course of the study period, but the authors did not account for this. Furthermore, participants were asked to recall their diet beverage consumption patterns for the past year. One year is a very long recall period, which introduces significant recall bias into the study.

**Results are inconsistent with other research on aspartame.** The authors suggested that aspartame might increase glucose or insulin concentrations, but numerous human studies have shown that aspartame does not increase post-prandial glucose or insulin concentrations (2–9). The researchers did not discuss their findings in relation to these studies, which included robust clinical trials. It was also suggested that low-calorie sweeteners may lead to increased preference for sweets and/or enhance appetite; however, neither of these have been shown to be true in scientific studies. Rather, a recent review article found that low-calorie sweeteners do not enhance appetite (10).

**Biologically implausible findings.** It does not seem physiologically possible for low-calorie sweeteners to cause diabetes, and the authors offered no explanations as to why such an association might exist. Middle-aged people are more likely to develop type 2 diabetes regardless of what they drink. Type 2 diabetes is strongly associated with being overweight. When the researchers controlled for BMI, a measure of body weight, the association between diet beverages and diabetes decreased. This finding points to the possibility that the results reported may be related to factors unrelated to diet beverage use, such as total body fat. Individuals seeking to lose weight or to manage their blood sugars often switch to diet beverages. Low-calorie sweetener use might therefore simply be a marker for individuals already on weight-gain or diabetes trajectories, which continued despite their switching to diet beverages. This is the most plausible explanation of these findings, not that the diet beverages caused the overweight or diabetes. Although the authors attempted to control for reverse causality, because it was not a randomized controlled trial reverse causation is still a possible explanation for the findings.

**Other factors may have influenced the findings.** Although researchers tried to control for many variables, it is possible that the findings were due to other factors for which the study was not controlled. This includes, for example, the actual diets of the participants during the years since 1993. The authors also failed to control for risk factors associated with type 2 diabetes, such as metabolic syndrome, hypertriglyceridemia, waist circumference, and coronary artery disease. External factors such as these may likely have influenced the results, which the authors acknowledged.

**Other study limitations.** The study included only women. There was also a large variation in how many diet beverages the women consumed, which makes the data less accurate. The authors admitted they had “limited statistical power in some subcategories,” so the data may be unreliable. Furthermore, ~1 in 4 women in the cohort failed to participate, which has the potential to introduce self-selection bias. Finally, the questionnaire used to determine diabetes diagnosis in the study was administered only every 2–3 y, which may have introduced recall bias into the study.

The authors is the President of the Calorie Control Council, an international association of manufacturers of low- and reduced-calorie foods and beverages, including companies that make alternative sweeteners (eg, intense sweeteners, polyols), fibers, and fat replacers used in those products.

_Haley Curtis Stevens_

Calorie Control Council  
1100 Johnson Ferry Road  
Suite 300  
Atlanta GA 30342  
E-mail: hstevens@caloriecontrol.org

**REFERENCES**


**Artificially and sugar-sweetened beverages and incident type 2 diabetes**

_Dear Sir:_

The French E3N cohort study included 66,118 non diabetic women followed up between 1993 and 2007 (1). Of these, 1369 developed type 2 diabetes (T2D). After multivariate analysis, significant trends in risk of T2D were observed for consumption both of sugar-sweetened beverages (SSBs) and artificially sweetened beverages (ASBs). In the multivariate analyses, a significant association was observed only for the highest consumption categories, with an RR of 1.30 (95% CI: 1.02, 1.66) for 359 mL SSBs/wk and 1.68 (95% CI: 1.19, 2.39) for >603 mL ASBs/wk.

These consumption categories, however, were based on only 73 cases of T2D for SSBs and on 34 cases for ASBs. A key problem of these analyses, in fact, is the definition of quartiles among consumers of sweetened beverages, which, despite consumption
measures defined according to single units of mL/wk, was extremely uneven, ie, the numbers of cases for ASBs were 252, 17, 20, and 34 in subsequent “quartiles.”

An additional concern with reference to ASBs—more than SSBs—is the substantial change in the RR estimates across age-adjusted and subsequent multivariate models. For SSBs, the RR in the highest consumption category was 1.49 in the model adjusted for age only, 1.32 in the simplest multivariate model, and 1.30 in the fully adjusted model (including allowance for BMI).

For ASBs, corresponding values were 3.50 in the age-adjusted model, 2.19 in the simplest multivariate model, and 1.68 in the fully adjusted model. This substantial change in RR estimates across subsequent models indicates that residual confounding (by BMI and/or other factors) may well be present in the multivariate risk estimates for ASBs. This may partly or largely explain the residual apparent association between ASBs and T2D for the 34 women at the highest exposure level.

Evidence from other studies is largely inconsistent. In the Nurses’ Health Study in women (2) and the Health Professionals Follow-Up Study in men (3), significant associations were observed for SSBs but not for ASBs. Two smaller US studies on atherosclerosis (4, 5) found apparent associations for ASBs, with RRs between 1.3 and 1.4 for the highest consumption category, and a positive trend in risk in one study only (4). In the Framingham Heart Study (6) positive associations, of borderline significance, were observed for the highest consumption levels of both SSBs and ASBs.

Even for the highest consumption levels of ASBs, however, the RRs of T2D were systematically below 2, and generally below 1.5, thus leaving open the issue of bias or residual confounding in observational studies, and particularly confounding by BMI. Reverse causation is an additional relevant issue (7, 8), because overweight subjects with possible prediabetic conditions may selectively choose to consume ASBs, thus leaving open the possibility of a false-positive finding between ASBs and T2D risk (9).

The author has received in the past unconditional grant support from the International Sweeteners Association (Brussels, Belgium) for addressing the issue of sweeteners and cancer risk.

Carlo La Vecchia

Department of Epidemiology
Istituto di Ricerche Farmacologiche Mario Negri
Via Giuseppe La Masa, 19
20156 Milan
Italy
E-mail: carlo.lavecchia@marionegri.it

REFERENCES


Reply to HC Stevens and C La Vecchia

Dear Sir:

Our study “Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes in the Etude Epidemiologique auprès des femmes de la Mutuelle Generale de l’Educatiion Nationale–European Prospective Investigation into Cancer and Nutrition cohort” showed that both sugar-sweetened and artificially sweetened beverages (SSBs and ASBs) were associated with an increased risk of type 2 diabetes (T2D) (1). The main strengths of our study were the size of the population (n = 66,118 women free of diabetes at baseline), the long follow-up (from 1993 up to 2007), the use of a validated dietary questionnaire, and the large number of covariates to control for.

Information on SSBs, ASBs, and fruit juice consumption

In 1993, women were asked to report their usual diet using a validated 208-item diet history questionnaire, structured according to the French meal pattern. Information on diet was recorded at baseline, and not updated, so that some women may have changed their consumption of SSBs and ASBs over time. Such a misclassification, however, should only bias our relative risk estimates toward unity, and “true” associations should actually be of greater magnitude.

Categories of ASB and SSB consumption have been defined among consumers of sweetened beverages (ie, women who consume ASBs only, SSBs only, or both). Groups of consumption were then compared with nonconsumers of SSBs or ASBs taken as the reference category for the calculation of HRs.

Associations between ASBs, SSBs, and T2D

Although we observed a decrease in the magnitude of HRs observed when adjusting for multiple covariates, which may be due to a decrease in statistical power, trends in risk remained significant. Most of the variation in HRs was observed when we further adjusted for BMI, which was consistent with our hypothesis that BMI is an intermediate factor in the relation between SSBs, ASBs, and T2D risk. Part of the association between soft drinks and T2D was captured by obesity. But, as stated in the article, we still observed a direct effect of SSBs and ASBs on T2D risk, independent of BMI.
It is very unlikely that the observed associations—for both SSBs and ASBs—were entirely due to residual confounding because models were adjusted for most of the well-known risk factors of T2D.

**Biological implications**

As mentioned in our article’s Introduction, the literature on the effect of ASBs on health is inconsistent. We deliberately considered results from cohort studies only. Previous large cohort studies that were interested in SSB, ASB, and T2D risk have concluded either a significant increased risk (2), which became nonsignificant after controlling for BMI, or no increased risk at all (3). We also clearly stated that no clear biological mechanism was proposed to explain the associations. However, all previous studies had strong limitations, which prevents interpreting their results on ASBs. The most serious limitation was the possibility of reverse causation, possibly due to the fact that ASB consumption has already been shown to be higher in individuals with T2D or prediabetic conditions, such as obesity.

The same hypothesis of reverse causation was evoked by La Vecchia and Stevens with regard to our study. We disagree with them for 2 reasons. First, in a sensitivity analysis, we excluded the 5 first years of follow-up, and results were very similar for both SSBs and ASBs. To our knowledge, our study is the first prospective study that takes into account the reverse causation issue. Second, the E3N cohort is mostly composed of teachers, a relatively health-conscious population with a low proportion of obese participants (3.2% in 1993). The associations we observed were actually driven by women with a BMI (in kg/m²) <25, who therefore developed diabetes without being obese. Consequently, we do not believe that reverse causation could explain the direct association between ASBs and T2D.

**Conclusions**

Even if no clear mechanisms have been proposed to explain the association between ASBs and T2D risk, our study provides additional evidence that high consumption of these beverages is associated with a strong increased risk of T2D. This conclusion will hopefully prompt scientists to conduct biological experiments on components of soft drinks and to replicate our findings in other populations.

The authors declared that they have no competing interests.

*Guy Fagherazzi*

*Françoise Clavel-Chapelon*

Center for Research in Epidemiology and Population Health
INSERM (Institut National de la Santé et de la Recherche Médicale) U1018
Team 9, Nutrition, Hormones and Women’s Health
Institut Gustave Roussy
114 rue Edouard Vaillant
94805 Villejuif Cedex
France
E-mail: clavel@igr.fr

**REFERENCES**


Consumption of artificially and sugar-sweetened beverages and incident type 2 diabetes: methodologic concern about a recent epidemiological study

Dear Sir:

In a recent issue of the Journal, Fagherazzi et al (1) revived the controversy about the safety of intense sweeteners. It has been repeatedly proven that increased sugar intake leads to weight gain (2) and that consumption of sugar-sweetened beverages (SSBs) is significantly associated with an increased risk of type 2 diabetes (T2D; reviews in reference 3). From the data on the E3N French cohort of 66,118 menopausal women, monitored between 1994 and 2007, Fagherazzi et al extend this risk to the consumption of artificially sweetened beverages (ASBs). However, several methodologic weaknesses cast doubt on the validity of their conclusions.

First, it is unlikely that a single diet-history questionnaire from 1993 on eating and drinking habits during the previous year was sufficiently precise to quantify the amount of different beverages consumed during the following 14 y of medical consultations. The reference the authors provided to justify their survey methodology (4) shows a limited reproducibility of the data provided by such a questionnaire, as indicated by low correlation of coefficients between data of 2 diet-history questionnaires recorded at 1-y intervals. This reference also indicates a very poor relative validity because of the systematic differences between data provided by the questionnaires and the data of 12 repeated 24-h recalls. In addition, in the E3N questionnaire, the beverage recording was only partial because consumption at the traditional 2 main meals in France, lunch and dinner, was not taken into consideration.

Another weakness identified was that the questionnaire only allowed respondents to answer either “only SSB consumption,” or “only ASB consumption,” without the possibility of indicating the respective quantity of both beverages that a large proportion of subjects probably consumed. This is confusing and may have introduced a significant distortion in the respective quantification of the SSB and ASB intakes. Furthermore, one diet-history questionnaire carried out at the beginning of the 14 y cannot be said to be sufficient to identify changes in the respondents’ dietary habits. Changes over a significant period of time may have been important to note, considering the commonly broadcast advice of reducing energy intake, particularly sugar intake, by public health stakeholders in France during the follow-up period. In addition, the age and the professional activity of the participants may have favored significant changes. All were female national education employees, aged 52.6 ± 6.6 y at baseline, who stopped their professional activity during the follow-up period. Different French national surveys indicate strong changes in life and eating habits at
this age (5), and body weight frequently increases during this period in women (6).

In this way, it is detrimental that Fagherazzi et al.’s (1) article did not provide any follow-up of the participants’ body weight, especially because they reported twice as much obesity among ASB consumers (6.8%) as in SSB consumers (3.1%). Taking into consideration the results, it must be noted that the relation between sweetener consumption and diabetes was largely reduced after adjustment for confounding variables (model 1), especially after adjustment for energy intake (model 2) and even further reduced after adjustment for BMI (model 3), with a disappearance of the risk for the middle levels of ASB consumption. It is surprising that the risk of T2D associated with ASB intake followed a U-shaped curve, which was significant only in the first quartile (<1 soda every 2 wk) and women in the fourth quartile (weekly consumption of >2 portions) but not for consumers between these levels. Unfortunately, Fagherazzi et al did not give any explanation for this surprising observation, which made hazardous the quadratic spline regression model of risk of T2D presented in their Figure 1. Another tentative conclusion is the argued absence of association of T2D with the consumption of fruit juices. As indicated in the article’s Table 1, after adjustment for the confounding variables including body weight, the OR of diabetes appearance increased significantly for consumers who drank 3–4 portions of fruit juice (448–967 mL) on a weekly basis, in accordance with other works (7).

Finally, an association between the consumption of intense sweeteners and T2D does not indicate a causal relation. As underlined by Elfhag et al (8), a reverse relation seems more probable because overweight or obese persons with prediabetic conditions tend to favor ASBs to SSBs to reduce their sugar consumption. However, by discussing the causal mechanism of T2D induced by ASBs, the authors suggest that intense sweeteners may promote insulin secretion. This claim is supported by a reference from Anton et al (9). Unfortunately, this work showed the contrary, ie, that sparing caloric intake by substituting sugar with intense sweeteners significantly reduced postprandial glucose and insulin concentrations, with no compensation in eating more during the following meals. Numerous other studies confirmed that intense sweeteners do not increase hunger, consumption, or insulin secretion (reviewed in reference 10).

In conclusion, it is possible that methodologic weakness may explain the surprising observation reported by Fagherazzi et al (1) as well as the significant delay between the end of the survey (June 2007) and the publication of the findings (January 2013).

The author, as an expert in physiology and nutrition, is a member of a scientific-reflection group appointed by the French section of the International Sweetener Association.

Marc Fantino
CREA Bio
Faculté de Médecine
7 Boulevard Jeanne D’Arc
21079 Dijon Cedex
France
E-mail: m fantino@u-bourgogne.fr

REFERENCES

Erratum


An error appears in the second paragraph of the Results section, where alleles are reported on the alternative strand and consequently do not match tabulated results. This can be remedied by reporting alleles on the opposite strand in this section of text only as in the corrected version below. An error also appears in Table 2, where genotypes for the SNP rs6596473 were not shown correctly. This error was not in the original manuscript but was introduced during copyediting when the table was reformatted according to Journal style. A corrected version of Table 2 appears on the next page.

The second paragraph of the Results section should read as follows:

“Within the BWHHS minor (m) allele frequencies at SNPs rs10063949(A/Gm), rs6596473(C/Gm), rs6596471(T/Cm), and rs33972313(G/Am) were 0.32, 0.28, 0.25, and 0.03, respectively. rs10063949, rs6596473, and rs33972313 all adhered to Hardy-Weinberg equilibrium (P > 0.3), although rs6596471 showed a nominal departure consistent with a slight overrepresentation of heterozygotes (P = 0.01). Measurements of the degree of LD between these variants are shown in Table 1. The variants rs6596473 and rs33972313 correlated with each other (independent of allele frequency, r² > 0.8), but all other pairwise comparisons showed low LD.’’


(Erratum continued on next page)
**TABLE 2**
Circulating L-ascorbic acid by allelic variation at SCL23A1 in the British Women’s Heart and Health Study

<table>
<thead>
<tr>
<th>L-Ascorbic acid (μmol/L)</th>
<th>GG</th>
<th>GA</th>
<th>AA</th>
<th>AG</th>
<th>CC</th>
<th>CG</th>
<th>TT</th>
<th>TC</th>
<th>Per allele effect</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>47.01 (44.07, 49.96)</td>
<td>—</td>
<td>42.54 (41.15, 43.93)</td>
<td>43.85 (42.40, 45.30)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Genotype at rs10063949</td>
<td>47.01 (44.07, 49.96)</td>
<td>—</td>
<td>42.54 (41.15, 43.93)</td>
<td>43.85 (42.40, 45.30)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>47.01 (44.07, 49.96)</td>
<td>—</td>
<td>42.54 (41.15, 43.93)</td>
<td>43.85 (42.40, 45.30)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Genotype at rs6596473</td>
<td>48.56 (45.31, 51.81)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>42.04 (40.72, 43.36)</td>
<td>44.35 (42.86, 45.83)</td>
<td>—</td>
<td>—</td>
<td>2.86 (1.39, 4.33)</td>
<td>0.0001</td>
</tr>
<tr>
<td>(n = 3365)</td>
<td>48.56 (45.31, 51.81)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>42.04 (40.72, 43.36)</td>
<td>44.35 (42.86, 45.83)</td>
<td>—</td>
<td>—</td>
<td>2.86 (1.39, 4.33)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Genotype at rs6596471</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>45.56 (41.55, 49.58)</td>
<td>—</td>
<td>43.01 (41.73, 44.29)</td>
<td>43.68 (42.17, 45.19)</td>
<td>0.95 (-0.63, 2.53)</td>
<td>0.2</td>
</tr>
<tr>
<td>(n = 3384)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>45.56 (41.55, 49.58)</td>
<td>—</td>
<td>43.01 (41.73, 44.29)</td>
<td>43.68 (42.17, 45.19)</td>
<td>0.95 (-0.63, 2.53)</td>
<td>0.2</td>
</tr>
</tbody>
</table>

1 All values are means (95% CIs) adjusted for age. Means and per allele effects were derived from linear regression.
Erratum


The authors have become aware of some minor errors in this published article. The minor corrections that have been made for this erratum do not affect the title, abstract, interpretation, or conclusions of the article.

In summary, the authors first discovered an error when they realized that they had mislabeled the fatty acids trans 16:1n–7 and trans 16:1n–9 in their work. They have gone back to the 2 laboratories where the biochemical measurement was done to seek clarification and explanation and have also had detailed discussion with their information manager to check their databases. The authors then confirmed that the analysis and results (as in the published article) are correct for the 2 separate trans fatty acids but that the naming of the fatty acids was mixed up. In fact, trans 16:1n–7 ("palmitelaidic acid") was measured in the erythrocyte fraction, whereas trans 16:1n–9 ("trans-7-hexadecenoic acid") was measured in the plasma fraction; however, the authors labeled both as palmitelaidic acid in the article. As their primary aim was to report on the comparison between the plasma and erythrocyte fractions, they have now removed the results for these 2 trans fatty acids and the total trans fatty acids, because they are no longer comparable or relevant, with only one of these trans fatty acids measured in each fraction. Because of this mislabeling error for the trans fatty acids, the authors went back and checked all of their data and results for accuracy and identified and corrected minor typographical errors and inconsistencies in the way in which subtotals of fatty acids were defined across results for Tables 2–4.

The authors are confident that the overall meaning and message of this published article remain unchanged; the focus was to report on a comparison of a full suite of fatty acids measured in 2 different blood fractions (plasma and erythrocyte) in relation to incident diabetes.

The specific minor corrections in the text and tables are as follows:

1) All references to 22 fatty acids in the manuscript should now change to 21 fatty acids (eg, on page 1215, column 2, line 9 within the section “Fatty acid measurement in plasma and erythrocyte-membrane phospholipid fractions”; on page 1221, column 1, antepenultimate line within the “Discussion” section; on page 1221, column 2, line 2 of the second paragraph within the “Discussion” section).

2) Page 1216, column 1, lines 5–6 within the section “Fatty acid measurement in plasma and erythrocyte-membrane phospholipid fractions”—the sentence should read as follows: “... TFA: only a single fatty acid was available in both plasma and erythrocyte-membrane fractions—trans 18:1n–9 (elaidic acid).”

3) Page 1216, column 2, lines 16–17 within the section “Statistical analyses”—the sentence should read as follows: “... sensitivity analyses were performed: first, prevalent cancer and cardiovascular disease (n = 39) ...” This does not change our results for Table 4 because the n = 39 were correctly included as a covariate in logistic regression analyses.

4) Page 1217, column 1, lines 2–4 within the section “Fatty acid distribution by incident diabetes status”—the sentence should read as follows: “Fatty acid distribution derived by FFQ was not significantly different by incident diabetes status except for palmitic acid.”

5) Page 1217, column 1, lines 10–12 of the second paragraph within the section “Fatty acid distribution by incident diabetes status”—the sentence should read as follows: “Linoleic acid and arachidonic acid were the greatest contributors of n–6 PUFAs in plasma and erythrocyte.”

6) In the footnote 2 to Table 2, and in a new footnote 2 to be added to Tables 3 and 4, the following sentence needs to be inserted: “Only elaidic acid (trans 18:1n–9) is available in both plasma and erythrocyte fractions; thus, no total trans fatty acid values are shown.”


(Erratum continued on next page)
Baseline fatty acid composition (%) and fatty acid product-to-precursor ratio derived by food-frequency questionnaire and as measured in plasma or erythrocyte-membrane phospholipid by incident diabetes status: the EPIC (European Prospective Investigation into Cancer and Nutrition)-Norfolk study

<table>
<thead>
<tr>
<th>Method of fatty acid measurement</th>
<th>Food-frequency questionnaire</th>
<th>Plasma phospholipid</th>
<th>Erythrocyte-membrane phospholipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty acids</td>
<td>Noncases (n = 184)</td>
<td>Cases (n = 195)</td>
<td>Noncases (n = 184)</td>
</tr>
<tr>
<td>Saturated fatty acids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myristic acid (14:0)</td>
<td>4.08 (3.19, 4.87)</td>
<td>4.12 (3.24, 5.12)</td>
<td>0.35 (0.27, 0.43)</td>
</tr>
<tr>
<td>Palmitic acid (16:0)</td>
<td>37.71 (33.80, 41.42)</td>
<td>38.36 (35.62, 42.59)</td>
<td>0.18 (0.14, 0.21)</td>
</tr>
<tr>
<td>Stearic acid (18:0)</td>
<td>---</td>
<td></td>
<td>26.84 (25.00, 28.48)</td>
</tr>
<tr>
<td>Monounsaturated fatty acids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palmitoleic acid (16:1n–7)</td>
<td>1.26 (1.11, 1.40)</td>
<td>1.26 (1.11, 1.44)</td>
<td>---</td>
</tr>
<tr>
<td>Oleic acid (18:1n–9)</td>
<td>29.51 (27.25, 31.78)</td>
<td>29.83 (27.41, 32.25)</td>
<td>---</td>
</tr>
<tr>
<td>Nervonic acid (24:1n–9)</td>
<td>---</td>
<td></td>
<td>---</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Linoleic acid (18:2n–6)</td>
<td>14.36 (10.76, 18.44)</td>
<td>13.58 (10.31, 17.91)</td>
<td>---</td>
</tr>
<tr>
<td>γ-Linolenic acid (18:3n–6)</td>
<td>0.03 (0.02, 0.04)</td>
<td>0.03 (0.02, 0.04)</td>
<td>---</td>
</tr>
<tr>
<td>Arachidonic acid (20:4n–6)</td>
<td>0.08 (0.07, 0.11)</td>
<td>0.09 (0.07, 0.11)</td>
<td>0.50 (0.45, 0.56)</td>
</tr>
<tr>
<td>Adrenic acid (22:4n–6)</td>
<td>---</td>
<td>---</td>
<td>0.18 (0.14, 0.21)</td>
</tr>
<tr>
<td>Elaidic acid (18:1n–9)</td>
<td>4.28 (3.51, 5.22)</td>
<td>4.17 (3.35, 5.12)</td>
<td>---</td>
</tr>
<tr>
<td>Fatty acid product to precursor ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Δ^6-Desaturase (20:4n–6/20:3n–6)</td>
<td>---</td>
<td>---</td>
<td>2.28 (1.90, 2.61)</td>
</tr>
<tr>
<td>Δ^6-Desaturase (20:3n–6/18:2n–6)</td>
<td>---</td>
<td>---</td>
<td>0.18 (0.14, 0.21)</td>
</tr>
<tr>
<td>Δ^9-SCD1 (16:1n–7/16:0)</td>
<td>---</td>
<td>---</td>
<td>0.03 (0.02, 0.03)</td>
</tr>
<tr>
<td>Δ^9-SCD2 (18:1n–9/18:0)</td>
<td>0.78 (0.68, 0.86)</td>
<td>0.82 (0.70, 0.95)*</td>
<td>---</td>
</tr>
</tbody>
</table>

1 Values are medians; interquartile ranges in parentheses. SCD, stearoyl-CoA desaturase; ---, data not available for food-frequency questionnaire. P values were estimated by using a Wilcoxon’s rank-sum test. *P < 0.05, **P < 0.01, ***P < 0.0001.

2 Individual monounsaturated fatty acids and trans fatty acids derived by food-frequency questionnaire were not available. Only elaidic acid (trans 18:1n–9) is available in both plasma and erythrocyte fractions; thus, no total trans fatty acid values are shown.

(Erratum continued on next page)
TABLE 3
Spearman’s correlation coefficients between fatty acids derived by food-frequency questionnaire (FFQ) and those measured in plasma or erythrocyte-membrane phospholipid at baseline among noncases (n = 184): the EPIC (European Prospective Investigation into Cancer and Nutrition)–Norfolk study

<table>
<thead>
<tr>
<th>Fatty acids</th>
<th>FFQ vs plasma phospholipid</th>
<th>FFQ vs erythrocyte-membrane phospholipid</th>
<th>Plasma phospholipid vs erythrocyte-membrane phospholipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty acids</td>
<td>r</td>
<td>P value</td>
<td>r</td>
</tr>
<tr>
<td>Saturated fatty acids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myristic acid (14:0)</td>
<td>0.06</td>
<td>0.45</td>
<td>0.27</td>
</tr>
<tr>
<td>Pentadecanoic acid (15:0)</td>
<td>0.39</td>
<td>$\leq 0.0001$</td>
<td>0.42</td>
</tr>
<tr>
<td>Palmitic acid (16:0)</td>
<td>0.09</td>
<td>0.21</td>
<td>0.07</td>
</tr>
<tr>
<td>Heptadecanoic acid (17:0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stearic acid (18:0)</td>
<td>$-0.14$</td>
<td>0.07</td>
<td>$-0.04$</td>
</tr>
<tr>
<td>Oleic acid (18:1n–7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palmitoleic acid (16:1n–7)</td>
<td>0.31</td>
<td>$\leq 0.0001$</td>
<td>0.34</td>
</tr>
<tr>
<td>Monounsaturated fatty acids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n–3</td>
<td>0.27</td>
<td>0.0002</td>
<td>0.31</td>
</tr>
<tr>
<td>n–6</td>
<td>0.27</td>
<td>$\leq 0.0001$</td>
<td>0.31</td>
</tr>
<tr>
<td>Eicosapentaenoic acid (20:5n–3)</td>
<td>0.33</td>
<td>$\leq 0.0001$</td>
<td>0.29</td>
</tr>
<tr>
<td>Docosapentaenoic acid (22:5n–3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Docosahexaenoic acid (22:6n–3)</td>
<td>0.29</td>
<td>$\leq 0.0001$</td>
<td>0.40</td>
</tr>
<tr>
<td>Linoleic acid (18:2n–6)</td>
<td>0.32</td>
<td>$\leq 0.0001$</td>
<td>0.28</td>
</tr>
<tr>
<td>$\gamma$-Linolenic acid (18:3n–6)</td>
<td>$-0.08$</td>
<td>0.30</td>
<td>$-0.07$</td>
</tr>
<tr>
<td>Arachidonic acid (20:4n–6)</td>
<td>0.04</td>
<td>0.60</td>
<td>0.02</td>
</tr>
<tr>
<td>Dihomo-$\gamma$-linolenic acid (20:3n–6)</td>
<td>0.02</td>
<td>0.76</td>
<td>0.02</td>
</tr>
<tr>
<td>Elaidic acid (18:1n–9)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^1$ -- data not available for FFQ.
$^2$ Only elaidic acid (trans 18:1n–9) is available in both plasma and erythrocyte fractions; thus, no total trans fatty acid values are shown.

(Erratum continued on next page)
### TABLE 4

Odds ratios (95% CIs) of developing type 2 diabetes across tertiles (T) of fatty acid composition and fatty acid product-to-precursor ratio derived by food-frequency questionnaire or measured in plasma or erythrocyte-membrane phospholipid (*n* = 383): the EPIC (European Prospective Investigation into Cancer and Nutrition)–Norfolk study

<table>
<thead>
<tr>
<th>Method of fatty acid measurement</th>
<th>Food-frequency questionnaire</th>
<th>Plasma phospholipid</th>
<th>Erythrocyte-membrane phospholipid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>T2 vs T1</td>
<td>T3 vs T1</td>
<td>T2 vs T1</td>
</tr>
<tr>
<td>Saturated fatty acids</td>
<td>1.79 (1.01, 3.19)</td>
<td>1.68 (0.94, 3.02)</td>
<td>1.39 (0.79, 2.46)</td>
</tr>
<tr>
<td>Myristic acid (14:0)</td>
<td>1.12 (0.64, 1.97)</td>
<td>1.29 (0.72, 2.29)</td>
<td>1.74 (0.97, 3.10)</td>
</tr>
<tr>
<td>Pentadecanoic acid (15:0)</td>
<td>1.57 (0.89, 2.79)</td>
<td>1.11 (0.63, 1.98)</td>
<td>1.07 (0.60, 1.90)</td>
</tr>
<tr>
<td>Palmitic acid (16:0)</td>
<td>1.67 (0.95, 2.96)</td>
<td>2.28 (1.28, 4.08)*</td>
<td>1.08 (0.61, 1.91)</td>
</tr>
<tr>
<td>Heptadecanoic acid (17:0)</td>
<td>—</td>
<td>0.60 (0.34, 1.06)</td>
<td>0.68 (0.38, 1.20)</td>
</tr>
<tr>
<td>Stearic acid (18:0)</td>
<td>1.70 (0.96, 3.03)</td>
<td>1.16 (0.65, 2.07)</td>
<td>1.05 (0.59, 1.81)</td>
</tr>
<tr>
<td>Monounsaturated fatty acids</td>
<td>0.88 (0.50, 1.55)</td>
<td>0.46 (0.27, 0.87)*</td>
<td>1.52 (0.88, 2.69)</td>
</tr>
<tr>
<td>Palmitoleic acid (16:1n–7)</td>
<td>0.87 (0.49, 1.52)</td>
<td>1.02 (0.57, 1.83)</td>
<td>1.37 (0.76, 2.45)</td>
</tr>
<tr>
<td>V vaccenic acid (18:1n–7)</td>
<td>—</td>
<td>0.73 (0.41, 1.28)</td>
<td>0.99 (0.53, 1.74)</td>
</tr>
<tr>
<td>Oleic acid (18:1n–9)</td>
<td>—</td>
<td>0.73 (0.41, 1.28)</td>
<td>0.99 (0.53, 1.74)</td>
</tr>
<tr>
<td>Eicosenoic acid (20:1n–9)</td>
<td>—</td>
<td>0.73 (0.41, 1.28)</td>
<td>0.99 (0.53, 1.74)</td>
</tr>
<tr>
<td>Nervonic acid (24:1n–9)</td>
<td>—</td>
<td>0.73 (0.41, 1.28)</td>
<td>0.99 (0.53, 1.74)</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids</td>
<td>0.95 (0.53, 1.68)</td>
<td>0.73 (0.41, 1.30)</td>
<td>0.50 (0.28, 0.90)</td>
</tr>
<tr>
<td>n-3</td>
<td>0.82 (0.47, 1.44)</td>
<td>0.71 (0.40, 1.27)</td>
<td>0.70 (0.40, 1.23)</td>
</tr>
<tr>
<td>α-Linolenic acid (18:3n–3)</td>
<td>0.74 (0.42, 1.31)</td>
<td>0.72 (0.40, 1.28)</td>
<td>0.88 (0.50, 1.54)</td>
</tr>
<tr>
<td>Eicosapentaenoic acid (20:5n–3)</td>
<td>0.94 (0.54, 1.66)</td>
<td>0.66 (0.37, 1.18)</td>
<td>1.35 (0.76, 2.40)</td>
</tr>
<tr>
<td>Docosapentaenoic acid (22:5n–3)</td>
<td>—</td>
<td>0.74 (0.42, 1.32)</td>
<td>0.88 (0.50, 1.56)</td>
</tr>
<tr>
<td>Docosahexaenoic acid (22:6n–3)</td>
<td>0.83 (0.47, 1.47)</td>
<td>0.63 (0.35, 1.13)</td>
<td>0.79 (0.45, 1.39)</td>
</tr>
<tr>
<td>Arachidonic acid (20:4n–6)</td>
<td>0.71 (0.40, 1.27)</td>
<td>0.73 (0.41, 1.30)</td>
<td>1.11 (0.62, 1.99)</td>
</tr>
<tr>
<td>Linoleic acid (18:2n–6)</td>
<td>0.73 (0.41, 1.31)</td>
<td>0.72 (0.40, 1.29)</td>
<td>0.75 (0.42, 1.34)</td>
</tr>
<tr>
<td>γ-Linolenic acid (18:3n–6)</td>
<td>0.89 (0.50, 1.57)</td>
<td>0.60 (0.34, 1.07)</td>
<td>1.66 (0.94, 2.94)</td>
</tr>
<tr>
<td>Eicosadienoic acid (20:2n–6)</td>
<td>—</td>
<td>0.85 (0.47, 1.52)</td>
<td>0.41 (0.23, 0.74)*</td>
</tr>
<tr>
<td>Dihomo-γ-linolenic acid (20:3n–6)</td>
<td>0.45 (0.26, 0.81)</td>
<td>0.73 (0.40, 1.31)</td>
<td>1.03 (0.58, 1.82)</td>
</tr>
<tr>
<td>Arachidonic acid (20:4n–6)</td>
<td>0.55 (0.31, 0.97)</td>
<td>0.87 (0.49, 1.56)</td>
<td>0.98 (0.55, 1.74)</td>
</tr>
<tr>
<td>Adrenic acid (22:4n–6)</td>
<td>—</td>
<td>1.13 (0.63, 2.00)</td>
<td>1.16 (0.64, 2.08)</td>
</tr>
<tr>
<td>trans Fatty acids</td>
<td>0.78 (0.44, 1.38)</td>
<td>0.96 (0.55, 1.69)</td>
<td>0.86 (0.49, 1.53)</td>
</tr>
<tr>
<td>Elaidic acid (18:1n–9)</td>
<td>—</td>
<td>0.86 (0.49, 1.53)</td>
<td>0.73 (0.41, 1.29)</td>
</tr>
<tr>
<td>Fatty acid product to precursor ratio</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Δ9-Desaturase (20:4n–6/20:3n–6)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Δ9-Desaturase (20:3n–6/18:2n–6)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Δ9-SCD1 (16:1n–7/16:0)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Δ9-SCD2 (18:1n–9/18:0)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*1 Values are odds ratios (95% CIs) for diabetes comparing the highest (T3) with the lowest (T1) tertiles and the middle (T2) with the lowest (T1) tertiles of the distribution for each fatty acid derived by food-frequency questionnaire and measured in plasma or erythrocyte-membrane phospholipid. T1 represents the reference category. SCD, stearoyl-CoA desaturase; —, data not available for food-frequency questionnaire. Multivariable logistic regression analyses were performed and adjusted for age, sex, family history of diabetes, BMI, smoking status, physical activity, and alcohol intake. *P for trend < 0.05.

*2 Only elaidic acid (trans 18:1n–9) is available in both plasma and erythrocyte fractions; thus, no total trans fatty acid values are shown.

**Erratum (Continued)**