A change (ie, eliminating all meat) made by a small number of the population could have a greater impact than a large change in diet (ie, reduction in meat) made by the majority of the population. From a public health perspective, this magnitude would still require a major shift in dietary habits for the current dietary intake in the United Kingdom (3). A reduction of even this magnitude lent to currently eaten. The diet contained only 372 g meat/wk (equivalent to 60% of the current average dietary intake in the United Kingdom (3)).

Vegetarian diets were included in the sample diet but in smaller quantities than the current average dietary intake. However, we also recognize that this would require data on the proportions of animal-based to plant-based foods within these dishes, which is widely eaten in the United Kingdom but show how altering the proportions of animal-based to plant-based foods within these dishes can achieve a significant reduction in GHGEs. The sample menu may appear to contain a lot of meat, but the menu needs to be viewed alongside the actually quantity of animal products listed in our Table 3, which shows that these dishes contain only small amounts of meat and more plant-based foods (1). Furthermore, it is important to consider the impact of dietary changes on the whole diet rather than focus on specific food groups so as to avoid possible unintended consequences, as shown by other recent work. Vieux et al (4) showed that the magnitude of reduction in GHGEs by limiting the amount of meat in the diet was dependent on the caloric density of the replacement food; an isocaloric replacement of meat with fruit and vegetables, for example, could increase GHGEs of the total diet.

It is important to remember that the diet described in our article was only one example of how dietary requirements for health and a reduction in GHGEs could be achieved, and they could be achieved with many different combinations of food, including with vegetarian diets. No food items were deliberately excluded from the diet; rather, a combination of food items was optimized to meet dietary requirements and to minimize GHGEs. The article served to show, first, a methodology for optimizing different aspects of the diet, with a scope to include additional factors where data are available, and second, that it is possible to draw synergies between dietary requirements for health and minimizing GHGEs. We would support the need for more research in this area to understand all of the issues and interactions associated with sustainable diets.

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Reply to I Hawkins and J Sabaté

Dear Sir:

Hawkins and Sabaté raise an important issue about the use of the terms sustainable and healthy diets. We agree that given the complex, multidimensional nature of these terms, especially sustainable, it is important in any discussion to be explicit about to what exactly it refers. The concept of a sustainable diet can have many different definitions relating to health, environment, economic and social factors, etc, and within each of these domains the meaning can differ depending on the context in which it is being used. As stated in our article (1), we included only one element of environmental sustainability, greenhouse gas emissions (GHGEs), but we acknowledged that there are a multitude of other elements that need to be considered in future research for a truly sustainable diet. Ideally, a comprehensive definition such as the UN Food and Agriculture Organization’s definition of a sustainable diet (2) would be used. However, we also recognize that this would require data on the many different aspects of sustainability for a wide range of food items, and for many of these variables currently there are few or no data at this level of detail. We would support the need for future research to address this issue. In the context of the diet, we would argue that sustainability can be used only in a relative manner. Our analysis was designed not to define a sustainable diet but to assess whether reductions in GHGEs could be achieved by realistic dietary change at the population level while still achieving dietary requirements for health. In terms of a “healthy” diet, we defined healthy as meeting UK government dietary requirements for health.

Hawkins and Sabaté question the role of meat and animal products in a sustainable diet. In our study, meat was included in the diet (1), but we were not suggesting that this was necessary to achieve nutrient requirements; rather, we discussed some of the complexities of ensuring that the right combination of food was in a vegetarian diet to meet micronutrient requirements. Vegetarian diets can meet dietary requirements for health and generally will have lower GHGEs. Our approach, however, was to take into account current dietary patterns of the UK population so that proposed changes to the diet could be seen as realistic. In the UK National Diet and Nutrition Survey, only 5% of participants reported being vegetarian in 2001 and 2% in 2008–2011 (3), and for this reason meat was included in the sample diet but in smaller quantities than currently eaten. The diet contained only 372 g meat/wk (equivalent to 4 servings/wk), which is 60% of the current average dietary intake in the United Kingdom (3). A reduction of even this magnitude would still require a major shift in dietary habits for the majority of the population. From a public health perspective, a moderate change in diet (ie, reduction in meat) made by the majority of the population could have a greater impact than a large change (ie, eliminating all meat) made by a small number of people. We therefore tried to maintain menus and dishes that are

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REFERENCES


Re: Consumption of artificial sweetener– and sugar-containing soda and the risk of lymphoma and leukemia in men and women

Dear Sir:

With the use of data from the Nurses’ Health Study (NHS) and the Health Professionals Follow-Up Study (HPFS), which included 1324 non-Hodgkin lymphomas (NHLs), 285 multiple myelomas (MMs), and 339 cases of leukemia, Schernhammer et al (1) report, in men only, some excess RR of NHLs related to consumption of both sugar-sweetened (RR = 1.66 for ≥1 serving/d) and diet (RR = 1.31) sodas, including mainly aspartame-containing beverages. The RRs were also elevated in men only for MMs (for sugar-sweetened sodas: RR = 1.76, NS; for diet sodas: RR = 2.02). For leukemia, there was no significant association in sex-stratified analyses, and the RR for ≥1 diet sodas/d was 1.42, which was of borderline significance, in both sexes combined. For NHLs and MMs there was no association in women and, more importantly, there was no association in the pooled analyses of both sexes combined.

The absence of overall excess risk of NHLs and MMs among consumers of aspartame-containing beverages in both sexes combined weighs against the existence of a real association. Mechanistic interpretation of apparent associations in subsets of these data only is therefore open to criticism. Schernhammer et al (1) speculate that aspartame–derived methanol would have a different rate of conversion to formaldehyde in men and women due to different enzymatic activation of alcohol dehydrogenase (ADH) type 1 in men and women. No association, however, was observed in men with heavier alcohol consumption (≥6 g/d; RR of NHLs = 0.96 for ≥2 drinks of diet soda/d). However, ADH is largely substrate induced, and women are less frequently (heavy) alcohol drinkers. Consequently, the observation that any association would be restricted to non- or light-drinking men, but was not observed among heavier drinking men and in women overall, is difficult to interpret simply in terms of unbound ADH activity and consequent formaldehyde serum concentrations. More important, any association between formaldehyde and lymphoid cancer risk remains open to discussion (2, 3), particularly when formaldehyde is administered by oral route (4).

The possible association between aspartame-containing beverages and hematopoietic malignancies has also been considered in the NIH-AARP Diet and Health Study cohort (5), which included 1279 lymphoid neoplasms. The highest cutoff quintile of estimated aspartame intake was 143 mg/d for men in the HPFS and 129 mg/d for women in the NHS. The NIH-AARP cohort was able to analyze considerably higher aspartame intake amounts: the overall multivariate RRs of all lymphoid neoplasms combined were 0.98 for 200–400 mg/d, 1.06 for 400–600 mg/d, and 0.95 for ≥600 mg/d. In the same NIH-AARP study, the RRs for the highest consumption amounts were 0.77 for Hodgkin lymphoma, 1.03 for MM, and between 0.77 and 1.25 for various types of NHL and leukemia, none of which were significant. Thus, despite the higher intakes considered, in the NIH-AARP study no association was observed between aspartame and lymphoid cancer risk.

The NHS and the HPFS are well-conducted, large-scale cohort studies with valid measures of cancer incidence. Still, before drawing a conclusion from these studies that soft drinks and/or diet sodas are associated with the risk of selected lymphoid neoplasms, readers must be convinced that the observed associations are real and consequently likely to be replicated in other studies, rather than random observations. This requires considering the details of study design, possible residual confounding, the mechanistic foundation of the observed association, previous results from similar studies, the strength of the associations, and the consistency of the results across multiple strata of covariates (6).

Taking all of these factors into account, the available epidemiologic evidence on low-calorie sweetened sodas—and specifically aspartame—and lymphoid neoplasms does not support the existence of a material association. Mechanistic speculation based on strata-specific analyses in relation to a potential carcinogenic effect of oral formaldehyde is also open to criticism.

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REFERENCES


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