Sugar-sweetened beverage link to cardiovascular risk factors is unsupported

Dear Sir:

In a recent article, Aeberli et al (1) concluded that low to moderate consumption of sugar-sweetened beverages (SSBs) adversely affects markers of cardiovascular risk. Their conclusion is inappropriate and unsupported for several reasons.

First, the differences between sweetener variables reported in their Table 1 are either insignificant (LDL subclasses IIa, IIIb, Iva, IVb; high-sensitivity C-reactive protein; adiponectin; ghrelin; fasting and postprandial glucose; C-peptide; HOMA2 IR; free fatty acids; aspartate aminotransferase; and alanine aminotransferase) or significant, but so slight as to be of questionable clinical importance (LDL subclasses I, IIb, and IIIa and leptin). The small measured differences may be more a reflection of baseline variability and analytic prowess than true medical consequence.

Second, the authors are justifiably critical of past studies that used unrealistically high sweetener doses that provided 25–60% of energy as fructose to show metabolic anomalies between sugars; to a lesser extent, they are guilty of the same error. To better reflect real-world intakes, Aeberli et al (1) ought to have focused more in their discussion on total rather than added fructose. It can be calculated from data in their Table 2 that fructose variables actually contain substantially higher amounts of total fructose (as percentage of total energy) than reported: 14.0% compared with 6.5% and 18.8% compared with 13.1% (actual compared with reported for moderate fructose and high fructose, respectively). Although lower than amounts tested in previous studies, the actual total fructose amounts used by Aeberli et al are considerably higher than the mean and 90th and 95th percentile fructose intakes (9%, 13.6%, and 15.3% of energy as fructose, respectively) recently estimated by Marriott et al (2) for adult males in their Table 2 that fructose variables actually contain substantially higher amounts of total fructose than true medical consequence.

Third, the experimental design intentionally does not compare conventional, real-world beverage formulations, but this severely limits its applicability to normal dietary practices. SSBs produced in the United States are primarily sweetened with high-fructose corn syrup (HFCS), which was not tested. Sucrose is used in SSBs, but mostly outside the United States. Fructose and glucose, which made up 5 of the 6 sweetener variables, are rarely used alone in SSBs.

And fourth, the authors’ justification for the study is weak. There has been debate for decades about the causal role of SSBs in obesity and related diseases. The cited hypothesis relating developing obesity in the United States with the introduction of HFCS was provocative when first proposed in 2004 (3), largely because HFCS is the primary SSB sweetener in the United States. However, SSB sales, HFCS use, and added fructose have all been in continuous decline since their peak in 1999 (4, 5). Thus, any causal relation that may have initially existed between SSBs, HFCS, and fructose and obesity and related diseases was lost 12 y ago, as the latter continued to increase.

In light of these deficiencies, characterizing the small and clinically questionable effects of mostly non-SSB sweeteners as “potentially harmful effects of low to moderate consumption of SSB on markers of cardiovascular risk” is inappropriate and unsupported by the present study.

The author is a consultant to the food and beverage industry in the area of nutritive sweeteners. His clients include research institutes, food industry councils, trade organizations, and individual companies.

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Reply to JS White

Dear Sir:

We appreciate White’s interest in our recent publication on the effect of sugar-sweetened beverages (SSBs) on human health (1), but we do not agree with his view about our conclusion being unsupported by the results presented.
White pointed out that some of the differences in outcome variables after the consumption of SSBs with different sweeteners were only small in this study, which is true. It is, however, not correct that this was due to baseline differences, because these were controlled for in the statistical model. Furthermore, rather subtle changes were expected with the study design used, and the important fact is that we observed significant differences at all after an intervention that lasted no longer than 3 wk, considering the lifelong consumption of SSBs of most people.

In our discussion we pointed out that the source of fructose seems to be very important. The low-fructose intervention (which aimed at avoiding the consumption of fructose from all sources) did not result in any benefit; in contrast, some variables (such as fasting glucose or high-sensitivity C-reactive protein) were negatively influenced. Consequently, we hypothesized that naturally occurring fructose may have beneficial effects, and we therefore did not focus only on total fructose consumption in the discussion. In fact, in light of these considerations, our approach of using 40 g (medium fructose) and 80 g (high fructose) of added fructose per day in the form of SSBs is very comparable with the mean and 90th and 95th percentiles of added fructose intakes estimated by Marriott et al (2) of 54, 93, and 108 g/d, respectively, for this age group.

It is true that we did not use conventional beverage formulations in our study. However, our aim was to dissect effects of the different sugars, and therefore the sugar amounts in the SSBs, but not the formulations of the drinks, which were aimed at mimicking a real-life situation, as was pointed out in our discussion. Furthermore, we used sucrose in one of the interventions, and this is, to our knowledge, still the most frequently used sweetener in SSBs in Switzerland and Europe. Due to the diversity of real-world SSB formulations (not only with regard to sugars used but also other ingredients), their use in such a study would not allow clear conclusions about the effects of specific substances.

Last but not least, we do not agree with White’s statement that a causal relation that may have existed between obesity and SSB consumption in the past was lost y ago. We all know that it is not one single factor, but a combination of several, that is responsible for the obesity epidemic. Thus, even if it may not be clearly visible from SSB sales, this association may still exist. Furthermore, as stated in our Introduction, the justification for our prospective interventional study was not simply based on the concomitant increase in fructose consumption and obesity and diabetes prevalence, but rather a consequence of the evidence provided by large epidemiologic studies and meta-analyses (3), which was also pointed out in a recent editorial of the Journal (4). The results of those studies clearly point out the importance of SSBs in the development of chronic disease, and this further indicates the need for studies that help toward a better understanding of the effects of the different ingredients used.

In summary, in consideration of the fact that our results provide evidence that short-term ingestion of moderate amounts of SSBs already affects important clinical outcome variables, we think it is reasonable to point out their potential harm to cardiovascular health.

None of the authors had any conflict of interest with regard to this letter.

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Multisector intervention to accelerate reductions in child stunting: an independent critique of scientific method

Dear Sir:

In the December 2011 issue of the Journal, Remans et al (1) sought to measure the effects of a multisector intervention on child malnutrition at village sites in 9 sub-Saharan African countries. That analysis contains important flaws that limit its internal and external scientific validity.

First, the study incorrectly attempts to distinguish trends in malnutrition indicators at the intervention sites from nationwide trends. The flaw arises from comparing child stunting trends at the intervention sites during one time period to nationwide trends during a different and irrelevant time period. It compares data from the intervention sites during 2005–2008 or 2006–2009 (depending on the site) to nationwide trends determined much earlier—average nationwide trends during 1986–2008 (their article’s Figure 3). For all but 2 of these countries, the nationwide data used end in 2006 or earlier.

This error likely tends to overstate the impact of the intervention. This is because although child stunting did not generally decline across sub-Saharan Africa in the late 20th century, child stunting did generally and greatly decline across sub-Saharan Africa during the period of the intervention in question—which was a time of generalized prosperity across the region relative to preceding years. Stunting in children younger than age 2 fell nationwide in all but one of the countries considered by Remans et al (1) for which stunting data are available during the intervention. That is, stunting under age 2 fell during approximately the period 2004–2010 in Tanzania (2), Malawi (3), Rwanda (4), Nigeria (5), and Ghana (6); at the time of this writing, no data spanning the period of the intervention are available for Senegal, Mali, Uganda, or Ethiopia.

Second, a comparison of onsite trends to nationwide trends—even in the correct time period—is likely to further overstate the impacts of the intervention. This is because many of the intervention sites are located in parts of each country in which child stunting is declining even faster than the national trend. Clemens and Demombynes (7) analyzed all 3 of the above-mentioned countries for which data spanning the period of the intervention were available in mid-2010. In all 3 (Ghana, Kenya, and Nigeria), child stunting during the intervention period 1) declined across the rural areas of the large region of each country in which the intervention site was located and 2) declined in that region faster than it declined nationwide. This information was available to Remans et al (1) at the time of writing.

Third, the results of Remans et al (1) are likely to overstate the impacts of the same intervention if it were extended to other village