Reviewer’s report

Title: The potential impact of food taxes and subsidies on cardiovascular disease and diabetes burden and disparities in the United States

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Reviewer: Justin White

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This study estimates how joint price changes to key dietary targets affect cardiometabolic disease (CMD) and CMD mortality in the US. The study uses two levels of price subsidies and taxes: 10% and 30%. It further assesses how prices have heterogeneous effects on low SES vs. high SES groups. The authors find that the joint price change would prevent > 3% of CMD deaths under their 10% price change scenario, with a large SES gradient that affects low SES individuals to a far greater extent. The authors conclude that price strategies could be used to prevent a substantial number of CMD deaths.

I believe that the paper has a number of important issues that need to be addressed.

1. Why do the authors assess the effects of jointly changing the prices on all 7 dietary targets, rather than the effect of separately changing the prices of each dietary target? It is not remotely politically feasible. A comparison of the separate price effects for each dietary target seem far more instructive.

Relatedly, a joint 10% increase in some food categories and 10% decrease in others is effectively a 20% relative price change for a given food (and same for the 30% price change). The framing of the joint price change seems misleading.

Further, product-specific taxes are not necessarily the most desirable strategy. Harding and Lovenheim (Forthcoming) find that nutrient-specific taxes will have a significantly larger impact than product-specific taxes, because they are broader-based taxes that affect consumption of a variety of unhealthy foods.

2. There are several issues with the price elasticities used in the paper.

a. The price elasticity estimates are drawn from Afshin et al. (2017), which includes non-US studies. The authors should rely on US-based studies (as opposed to South Africa or elsewhere).

b. A key model assumption is the price sensitivity of low SES vs. high SES groups. The authors' decisions regarding this parameter seems indefensible to me. In the "low gradient" scenario, they draw this parameter from price elasticity differences between high-income countries vs. low-income countries. Cross-country comparisons have little bearing on cross-subgroup price elasticities in the US. In the "high gradient" scenario, the authors draw this parameter from cross-
group differences in Mexico, which has huge societal differences from the US. The authors need to go back to the literature, and find US-specific evidence on differences by SES. For example, Lin et al. (2011) compare the price elasticity of demand for SSBs and other drink categories among low- vs high-income groups in the US. So does Zhen et al. (2011) and other articles. Clements and Si (2015) look at price elasticities of food demand, as have others.

c. The low-gradient and high-gradient scenarios are based on income differences. It is not clear to me how the authors converted the income gradient for price elasticity into their education gradient. This needs to be detailed.

d. The authors assume that the price elasticity by education category is the same for fruits, vegetables, nuts/seeds, and whole grains. Can you the authors review the literature to find more granular estimates?

3. The authors do not account for substitution effects induced by the subsidies/taxes, e.g., switching from SSBs to 100% fruit juice. The authors acknowledge it as a limitation, but it really calls into question the validity of the estimates.

4. The authors acknowledge that their estimates of etiologic effects are based mainly on observational data, and argue that these are supported by a single large RCT that tested the Mediterranean diet and "mechanistic evidence." This obscures the active debate in the research community—e.g., low-carb vs. low-fat vs. low-calorie. Diet comparison trials have found conflicting results, and the mechanistic evidence tends to indicate that sugar has a special metabolic role. I think the authors need to give a more even-handed assessment of potential confounding.

Moreover, the etiologic effect estimates come from an article that is not yet in the public domain. I cannot assess how accurately the effects are estimated. It would help to have more detailed information about how these estimates were derived.

5. What declining proportional effects by age were applied to the etiologic effect data? The authors should be explicit. The cited Singh study uses global data and again relies on observational data as an input. Isn't there higher quality, US-centric data to rely on?

References


**Are the methods appropriate and well described?**
If not, please specify what is required in your comments to the authors.

No

**Does the work include the necessary controls?**
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Yes

**Are the conclusions drawn adequately supported by the data shown?**
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