

Dietary carbohydrate intake and mortality: reflections and reactions

In their Article in *The Lancet Public Health* (September 2018), Sara Seidelmann and colleagues¹ use data from a long-term prospective cohort study and do a meta-analysis of other related cohorts to assess the association between carbohydrate intake and mortality. Our main problem with this Article¹ was regarding the confounders that are related to an individual's consumption of carbohydrates and to that individual's associated mortality. Some of these confounders were included as control variables, but since it is impossible to account for all possible confounders, some confounders necessarily went unobserved. This problem is illustrated in the figure.

In any observational study, unobserved confounders prevent the identification of a causal relationship between carbohydrate intake and the risk of associated mortality. In this context, any statistical association between these two variables cannot be argued to be causal, no matter the level of statistical significance.

What could be an unobserved confounder? One example could be whether individuals tended to have highly variable bodyweight due to extreme diets (ie, diets that are too low or too high in carbohydrate intake), which was not observed in this study. Such variability in bodyweight is associated with an increased risk of diabetes and increased cardiometabolic risk factors.^{3,4} Without a variable that measured whether a respondent dieted in this way (and there is no indication that Seidelmann and colleagues controlled for this confounder), the estimated association between carbohydrate consumption and related mortality could be biased. Hence, instead of concluding that diets that are either too low or too high in carbohydrates cause a higher risk of associated mortality, in fact,

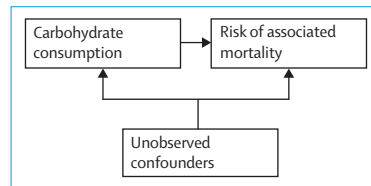


Figure: A directed acyclic graph showing the identification problem

Carbohydrate consumption is presumed to increase the risk of associated mortality and unobserved confounders are presumed to cause both.² That unobserved confounders are associated with both carbohydrate consumption and associated mortality is an example of the identification problem that almost always plagues empirical research done with non-experimental data: that correlation is not causation.

bodyweight variability due to extreme diets would be responsible.

Some of our concerns could be easily assuaged with the inclusion of more information about the analysis, including the regression results for the Atherosclerosis Risk in Communities study itself instead of merely presenting only the adjusted odds ratios of interest and one figure. Additionally, robustness checks on the results could be presented, including falsification tests.⁵

With the increase in computing power, decreasing costs of data storage, and increased use of big data in the health sciences,⁶ we would have expected additional analyses to support the findings.

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ABF declares no competing interests.

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