


# Let's Require the "T-Word"

 See also Galea and Vaughan, p. 602; Hernán, p. 616; Begg and March, p. 620; Ahern, p. 621; Chiolero, p. 622; Glymour and Hamad, p. 623; and Hernán, p. 625.

In this issue of *AJPH*, Hernán (p. 616) argues that we should stop avoiding the “C-word”—causality—in articles about observational studies when the research question is a causal question. We agree that authors should clearly specify their purpose in the introduction, including whether the goal is characterization, risk stratification, or assessment of causation, to ensure use of distinct and appropriate statistical model building for descriptive, predictive, or causal questions. However, the interpretation of findings from an observational study assessing relations needs to maintain use of associational language to reduce the likelihood of misinterpretation from the media and the general public. Media coverage, for example, on the benefits of drinking a glass of red wine a day (based on the “French paradox”) resulted in increased red wine sales in the United States in the 1990s.<sup>1</sup> Imagine how much worse this misinterpretation would be if stronger causal language were used in Discussion sections. For red wine and reduced risk of coronary heart disease, a likely explanation for the observed protective associations is confounding by higher socioeconomic position, better health status, and greater ability to

delay gratification, which enable consumption of one glass of red wine per day and reduced risk of coronary heart disease. Mendelian randomization studies have not found a protective effect of moderate alcohol use on coronary heart disease.<sup>2</sup>

Furthermore, we disagree on many levels with the general notion that imagining an observational study as testing a causal effect in a randomized trial is a useful exercise. It fails to distinguish between the theoretical model and its testing, between an intervention and the mechanism by which it operates, and between the different sources of bias. This type of thinking results in claims that models that use statistical techniques such as inverse probability weights mimic a randomized controlled trial, increasing their use in the literature without clear consideration for best practices.<sup>3</sup> All statistical approaches to analyzing observational data for causal questions assume sufficiently measured and adjusted confounders and predictors of missing data, when historically, many adjusted models from observational studies have identified exposures as beneficial, which were later found to be harmful or to have no effect.<sup>4</sup> A focus on bias from confounding and missing data

also may divert attention from pervasive biases that can occur from selection into the study dependent on exposure and outcome. For example, a population representative study inevitably excludes people who have already succumbed to a harmful exposure and who cannot easily be re-created by extrapolating from the survivors, even with the use of inverse probability weighting.

In summary, we agree fully with the importance of being clear about the purpose of a study in the Introduction. However, we do not agree with using language in interpretation of results that suggests that an observational study alone has fulfilled its purpose and correctly identified a causal effect. Moreover, what may be more important than adding the “C-word” to the Introduction is to require authors to add the “T-word”—that is, to explain their underlying theory of causal mechanism, whether it is the underlying biology or the underlying social structures and systems that clarify why the authors hypothesize that

exposure  $x$  causes outcome  $y$ , so that we start off with questions that are most likely to yield effective interventions. Furthermore, requiring an explanation of the causal theory would increase the likelihood of collaboration across disciplines. *AJPH*

Heidi E. Jones, PhD, MPH  
C. Mary Schooling, PhD

## CONTRIBUTORS

Both authors contributed equally to this editorial.

## REFERENCES

1. Burros M. Eating well. *New York Times*. March 4, 1992. Academic OneFile. Available at: [http://link.galegroup.com/apps/doc/A174814924/AONE?u=cuny\\_ccny&sid=AONE&xid=1a25d4d7](http://link.galegroup.com/apps/doc/A174814924/AONE?u=cuny_ccny&sid=AONE&xid=1a25d4d7). Accessed January 30, 2018.
2. Holmes MV, Dale CE, Zuccolo L, et al. Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. *BMJ*. 2014;349:g4164.
3. Austin PC, Stuart EA. Moving towards best practice when using inverse probability of treatment weighting (IPTW) using the propensity score to estimate causal treatment effects in observational studies. *Stat Med*. 2015;34(28):3661–3679.
4. Smith GD, Lawlor DA, Harbord R, Timpson N, Day I, Ebrahim S. Clustered environments and randomized genes: a fundamental distinction between conventional and genetic epidemiology. *PLoS Med*. 2007;4(12):e352.

## ABOUT THE AUTHORS

Heidi E. Jones is with the Department of Epidemiology and Biostatistics, City University of New York School of Public Health, New York, NY. C. Mary Schooling is with the Department of Environmental, Occupational and Geospatial Health Sciences, City University of New York School of Public Health.

Correspondence should be sent to Heidi E. Jones, PhD, MPH, Associate Professor, Department of Epidemiology and Biostatistics, City University of New York School of Public Health, 55 W 125th St, New York, NY 10027 (e-mail: [heidi.jones@sph.cuny.edu](mailto:heidi.jones@sph.cuny.edu)). Reprints can be ordered at <http://www.ajph.org> by clicking the “Reprints” link.

This editorial was accepted February 2, 2018.

doi: 10.2105/AJPH.2018.304365