Review of: "Alcohol consumption and the risk of gastric intestinal metaplasia in a U.S. Veterans population"

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In a cross-sectional study, Holmes et al. investigated the association between average lifetime total alcohol consumption and as captured by drink type with prevalent gastric intestinal metaplasia. The authors also looked at interactions with smoking and H. pylori infection, however this does not add much to the message of the paper. The authors concluded that no quantity of alcohol when assessed alone as exposure variable, irrespective of beverage type, confers increased risk of gastrointestinal metaplasia. This rather shocking conclusion dismisses any relationship between alcohol and gastric intestinal metaplasia, which is in fact a precursor of gastric cancer. At the same time, the authors do not deny the well-established link between alcohol (as a class 1 carcinogen) and gastric cancer. This simply does not make sense.

The reasons why the authors were able to reach the above conclusions is due to the analytical approach followed, be it deliberate or not. In fact, the pro-alcohol literature, irrespective of the outcome measure, all use these approaches and published in high-ranking journals. These approaches forces the J-shaped associations with outcome measures, causing and underestimation of the risks associated with even low to moderate alcohol consumption. This must must simply stop.

These approaches are:

1. The use of non-drinkers or never drinkers as a reference group. Usually, a large proportion of this group consists of participants who abstain from drinking due to poor health reasons and resultant associations will be underestimated or even show protection, as we often see with cardiovascular disease, especially coronary artery disease. The authors do provide continuous analysis in Figure 1, however, provides no odds ratios or 95% confidence intervals for the linear association in the figure, or in the text.
2. Combining drink types. Alcohol captured from beer/cider and spirits are not comparable to that of wine, with wine (both red and white) showing no association and even protection (perhaps due to polyphenols), whereas beer/cider and spirits show linear positive relationships with health outcomes. Combining alcohol captured from all drink types as exposure variable results in dilution or underestimation of the risk associated with alcohol intake, specifically low to moderate alcohol

consumption which is causing all the confusion.

The authors therefore applied the above to be able to dismiss any associations with different drink types and total alcohol consumption in categorical analysis using non-drinkers as reference group (table 2), and in continuous analysis (figure 1) combining all drink types and therefore showing an underestimated linear association between alcohol intake and gastric intestinal metaplasia.

Unless the authors can confirm their findings after excluding all non-drinkers and analyse their data separately for alcohol captured from non-wine and wine beverages in continuous analysis, the findings from this paper should be ignored and the paper even retracted.