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Commentary

Alcohol and steatosis: The Japanese paradox — Authors' reply☆

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We thank Lonardo et al. (2016) for their comments on our meta-analysis on the relationship between alcohol consumption and hepatic steatosis (Roerecke et al., 2016). We would like to clarify our position on overall risk associated with alcohol consumption and specifically on the risk of hepatic steatosis. First, as we have stated in our paper, we are in agreement with Lonardo et al. that a distinction between alcoholic and non-alcoholic hepatic steatosis has little meaning because alcohol is only one of the risk factors for liver disease, including non-alcoholic fatty liver disease. Different dimensions of alcohol consumption should be distinguished in this regard as non-regular binge drinking among moderate drinkers seems to increase the risk for hepatic steatosis. However, other risk factors also play a role, and the interactions among all risk factors determine the overall risk for liver injury. The large heterogeneity we observed in countries other than Japan underlines that risk factors other than alcohol play a substantial role in risk for hepatic steatosis and precludes us from clearly identifying the role alcohol plays in the development of hepatic steatosis. Thus, categorizing the etiology of liver injury based on one risk factor does not make much sense.

Second, when talking about risk associated with alcohol consumption, it makes little sense to use words such as safe. Rather, risk needs to be examined for each disease outcome separately because pathways and risk differ from disease to disease, and the same amount of alcohol may be associated with a low risk for one disease outcome but with high risk for another. For example, the risk for breast cancer is elevated no matter how little alcohol is consumed (Shield et al., 2016), and no level of alcohol consumption is safe in this case. Consequently, guidelines for alcohol consumption are considered 'low-risk' drinking guidelines (Stockwell & Room, 2012), i.e. no level of alcohol consumption is safe.

Third, the term paradox gives evidence that we do not yet fully understand the etiology of liver disease and the role alcohol plays in it. Any comprehensive theory of alcohol's effect on hepatic steatosis or other forms of liver disease should be able to explain the difference in associations our meta-analysis has shown.

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