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Letter to the Editor: Moderate alcohol use in fatty liver disease; don't throw the cabernet out with the bathwater.

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List of abbreviations:

NAFLD: Non-alcoholic fatty liver disease

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To the Editor:

We congratulate Dr Aberg and colleagues for a significant contribution to the literature on the important topic of moderate alcohol use in fatty liver disease (NAFLD) (1). Approximately 30% of the population of the Western world has obesity and/or other complications of insulin resistance and the metabolic syndrome. Due to the magnitude of this problem worldwide, palatable lifestyle therapies that are affordable, available and acceptable are necessary to curtail preventable early deaths. Moderate alcohol has the potential to fill this space, although knowledge remains limited.

The most common cause of death in patients with NAFLD is cardiovascular disease. Aberg's paper clearly demonstrated that low-to-moderate alcohol consumption reduced cardiovascular events by greater than 20% and all-cause mortality by a similar magnitude. As hepatologists, we are rightly concerned about variables that influence liver outcomes in NAFLD. However, we should be similarly engaged in the discussion of lifestyle interventions that offer extra-hepatic benefits, as Aberg's data suggest moderate alcohol does. Moderate alcohol drinking in this population does carry some risks; including increasing the risk of hepatocellular and breast cancer, but at the same time reduces the risk of renal cell carcinoma and lymphoma. It may also reduce the risk of developing diabetes, stroke, dementia and cardiovascular disease (2). The net effect is positive as demonstrated by Aberg with reduced all-cause mortality. Aberg's data therefore does not argue against the use of moderate alcohol in this population but for it.

The association between low level alcohol consumption, particularly wine, and the development of liver disease itself, remains unanswered from Aberg's dataset. In fact, for low dose alcohol consumption (0-9g per day) there was no significant link between alcohol intake and liver events, and the association for 10-19g/day of alcohol and liver events was only seen in non-wine drinkers. In a previous large cohort study, wine consumption up to 10g per day reduced the risk of hepatic steatosis by 50% (3). A case-control study examining histology and gene array also found that low-level alcohol may suppress gene expression involved in the immune response that drives steatohepatitis (4). Given the small numbers of patients in the

current study reporting low-level wine consumption, it was likely underpowered to assess benefit. Furthermore, information on significant confounders including baseline hepatic fibrosis diabetic control and coffee intake was not available.

Further well-defined studies are required to assess the impact of low-moderate alcohol consumption on liver disease and cardiovascular outcomes before this can be recommended as a lifestyle intervention. Similarly, alcohol's narrow therapeutic window can be difficult to achieve.

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