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Favorable effect of modest alcohol consumption to fatty liver disease

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Abstract

We previously reported that modest alcohol consumption was significantly inversely associated with fatty liver disease. Feng *et al* pointed out a discrepancy of statistical significance between our current larger scale cohort and a previous cohort. However, the prevalence of non-alcoholic fatty liver disease was higher in non or minimal drinkers than those in light drinkers in both cohorts. They also argue that some potential co-factors such as soft drink consumption and genetic variations should be discussed.

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Key words: Alcohol; Fatty liver disease; Obesity; Diabetes; Metabolic syndrome

Core tip: We reported the inversed association of modest alcohol consumption with fatty liver disease. However, other potential co-factors were argued to be important. Herein, we reply and discuss these important factors in this letter.

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TO THE EDITOR

Feng *et al* argued against the validity of the category of alcohol consumption we used, however this categorization was made in accordance with standardized methods[1,2]. They also pointed out the discrepancy of statistical significance between our current larger scale cohort and a previous cohort[3-5]. However, the prevalence of non-alcoholic fatty liver disease (NAFLD) was higher in non or minimal drinker than those in light drinker in both cohorts. In the current cohort, the prevalence of NAFLD was higher in non or minimal drinkers than those in light drinkers both in men (36.5%, 2248/6154 *vs* 26.4%, 457/1734) and in women (10.4%, 719/6893 *vs* 5.4%, 22/406)[4]. A similar trend was reported in the previous cohort as follows; the prevalence of NAFLD was higher in non or minimal drinkers than those in light drinkers both in men (28.6%, 170/595 *vs* 23.5%, 464/1977) and in women (10.7%, 105/981 *vs* 8.6%, 73/848)[5]. While statistical significance was not detected in the cohort used in our previous study, however we speculated that the smaller size of the cohort may have reduced the power of the statistical test and thus failed to detect the difference. Moreover, we previously reported that there is a favorable effect of modest alcohol consumption for the development of NAFLD in a prospective cohort study[6]. The adjusted odds ratio of light drinkers for the development of NAFLD was 0.82 (0.59-1.15, $P = 0.26$) in men, and 0.86 (0.51-1.45, $P = 0.56$) in women, respectively. The odds ratio was not statistically significant, but the

possibility of a favorable effect was speculated[6].

Feng *et al*[3] also argue that soft drink consumption or genetic variations may be potential co-factors for the favorable effect of modest alcohol consumption to fatty liver disease. Soft drink consumption is an important risk factor for NAFLD[7]. Unfortunately, in this case we had no data of soft drink consumption. However, a previous study has reported no association between alcohol consumption and soft drink consumption[8]. Thus, the association of NAFLD with soft drink consumption may be independent of those with alcohol consumption.

Genetic variations have been reported as important risk factors for NAFLD and non-alcoholic steatohepatitis[9-13]. The mechanisms of these genetic variations for development of NAFLD are still largely unknown. Thus, future investigations are needed; however, we appreciated Dr. Feng's extension of our discussion regarding the role of genetic variations in the pathophysiology of NAFLD.

In conclusion, we reported the inverted association of modest alcoholic consumption with fatty liver disease in a large scale cross-sectional study. Future large scale prospective studies and basic investigations are needed to clarify the effect of modest alcohol consumption in the development of fatty liver disease.

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