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Vague relationship between alcohol consumption and metabolic syndrome in nonobese people

Kei Nakajima, Masafumi Saito

Kei Nakajima, Masafumi Saito, Division of Clinical Nutrition, Department of Medical Dietetics, Faculty of Pharmaceutical Sciences, Josai University, 1-1 Keyakidai, Sakado, Saitama 350-0295, Japan
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Correspondence to: Kei Nakajima, MD, PhD, Division of Clinical Nutrition, Department of Medical Dietetics, Faculty of Pharmaceutical Sciences, Josai University, 1-1 Keyakidai, Sakado, Saitama 350-0295, Japan. keinaka@josai.ac.jp
Telephone: +81-49-2717260 Fax: +81-49-2717260
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Abstract

Fatty liver, including non-alcoholic fatty liver disease, is closely associated with metabolic syndrome (MS). Thus, the presence of fatty liver without MS in some conditions may be clinically important. Many studies have shown that compared with no or occasional alcohol intake, moderate alcohol consumption is associated with lower prevalence rates of hypertension and type 2 diabetes, and lower levels of circulating C-reactive protein, a valuable marker for MS and insulin resistance. Considering these findings, light to moderate alcohol consumption has theoretical benefits on fatty liver and MS. Fatty liver, including non-alcoholic fatty liver disease, may be more clinically important than MS, particularly in non-obese individuals, because fatty liver can develop before MS in several conditions, such as regular alcohol consumers. Furthermore, most of the currently used MS criteria are unable to detect "true MS" because of variations in multiple factors such as age, height, medications, and complications.

Key words: Alcohol consumption; Non-alcoholic fatty liver disease; Metabolic syndrome; Adult treatment panel Ⅲ

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TO THE EDITOR

We read the recent article by Hamaguchi *et al*[1] with much interest. The authors showed in a cross-sectional study that the effects of alcohol consumption differed between metabolic syndrome (MS) and fatty liver, and that light to moderate alcohol consumption had a favorable effect on fatty liver, but not on MS, in Japanese men and women. Habitual alcohol consumption, which generally impairs fatty acid oxidation and stimulates lipogenesis in the liver[2,3], substantially influences morbidity, all-cause mortality, and cardiovascular mortality rates[4,5]. Thus, the findings reported by Hamaguchi *et al*[1] are impressive in terms of public health and scientific interest. As described by the authors, fatty liver, including non-alcoholic fatty liver disease (NAFLD), is closely associated with MS and is a hepatic manifestation of MS or insulin resistance. This is based on the concept that MS is a leading cause for fatty liver in the cause-effect relationship.

In addition, many studies have shown that moderate alcohol consumption is associated with a low incidence of adverse health outcomes associated with hypertension[5] and type 2 diabetes[6], and with lower levels of cir-

culating C-reactive protein (CRP)[7,8], an important marker for MS, obesity, and insulin resistance[9,10]. The association between moderate alcohol consumption and lower CRP was independent of body mass index (BMI)[9,10] and alcohol-related effects on lipids[10].

Considering these findings, light to moderate alcohol consumption is hypothesized to have beneficial effects on fatty liver and MS. Plausible explanations for the lack of a beneficial effect of light to moderate alcohol consumption on the risk of MS include the characteristics of the subjects studied, the criteria used to define MS, or food patterns specific for light to moderate alcohol consumers. Of particular note, NAFLD also occurs, albeit relatively infrequently, in normal weight people who commonly have metabolic abnormalities and insulin resistance[11,12]. Most of the subjects in the study by Hamaguchi *et al*[1] were of normal weight (mean BMI 23.2-23.5 kg/m^2 for men and 20.9-21.4 kg/m^2 for women), in whom the prevalence of MS is likely to be reduced when waist circumference is dichotomized according to the criteria for MS with a fixed threshold. High waist circumference is necessary for the diagnosis of MS according to the International Diabetes Federation (IDF) criteria for MS, but not the adult treatment panel (ATP)-Ⅲ criteria for MS. This may help to explain the lower prevalence of people fulfilling the IDF criteria than the ATP-Ⅲ criteria in that study. By contrast, fatty liver can be diagnosed using ultrasound techniques irrespective of manufacturer-specific criteria, which likely resulted in the unexpected finding that more than half of the subjects with fatty liver did not have MS.

Furthermore, although the authors claimed in the discussion that there was no significant association between alcohol consumption and BMI > 25.0 kg/m^2, the prevalence of BMI > 25.0 kg/m^2 was actually lower, particularly in females with light to moderate alcohol consumption. This suggests that the observed association might be mediated by lower BMI, which is also affected by dietary patterns and nutrient intake in such alcohol consumers[13,14]. Sub-analyses of subjects divided into specific groups (e.g., overweight/obese groups) or analyses controlling for BMI, waist circumference, and dietary patterns (as assessed by food frequency questionnaires, for example) are needed to clarify whether the observed association is independent of obesity, MS criteria, or dietary patterns.

Taken together, the results of the study by Hamaguchi *et al*[1] provide meaningful insight into the etiological differences in metabolic abnormalities between liver and visceral adipose tissue, in which lipolysis is reduced by acetate produced in the liver[15]. In addition, the results indicate that fatty liver, including NAFLD, may be clinically more important than MS, particularly in non-obese people, because fatty liver can develop before MS in some conditions and that MS criteria are often unable to detect "true MS". The authors propose that fatty liver without MS is an important disease in the general population. Similarly, Stefan *et al*[16] proposed that fatty liver may be more important than visceral fat for the discrimination of benign obesity that is not accompanied by insulin resistance.

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