Original Article

Metabolic dysfunction associated fatty liver disease (MAFLD) and serum vitamin D concentration in South Korea

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Background and Objectives: Non-alcoholic fatty liver disease (NAFLD) has recently been renamed as metabolic dysfunction-associated fatty liver disease (MAFLD) by the Asian Pacific Association for the Study of the Liver (APASL) to reflect metabolic dysfunction. Vitamin D regulates free fatty acid flux from the periphery to the liver. The association MAFLD and vitamin D has been controversial. We investigated the association of MAFLD, nutrient intake, and vitamin D status in South Korean adults. **Methods and Study Design:** We analyzed patient responses from the Korea National Health and Nutrition Examination survey (KNHANES) 2010-2011. The disease group was selected as per the latest guidelines. Steatosis was evaluated by the fatty liver index (FLI). Frequency analysis was performed on general characteristics. We compared differences in nutritional status using complex sample adjusted chi-square tests and generalized linear models. After adjusting for age, complex sample logistic regression analysis was used to examine the relationship between MAFLD and vitamin D. **Results:** Aspartate aminotransferase (AST), alanine aminotransferase (ALT), LDL, triglyceride, creatinine, glucose, nutrient intake, and serum 25(OH)D concentrations were significantly elevated while HDL was reduced in the disease group than in the control group. The OR for 25(OH)D was 1.015 (95% CI: 1.004-1.026, $p<0.0001$). However, MAFLD presented no significant association with vitamin D concentration (OR 1.010, 95% CI: 0.985-1.037, $p=0.431$) after adjusting for age. **Conclusions:** We found no significant relationship between MAFLD and serum vitamin D concentration in South Korean adults.

Key Words: Vitamin D, MAFLD, NAFLD, metabolic dysfunction, fatty liver

INTRODUCTION

The most common chronic liver disease is characterized by fat accumulation in the liver without significant alcohol intake or drug use; it was previously referred to as non-alcoholic fatty liver disease (NAFLD).[1] The definition of NAFLD was vague and controversial because it relied on diagnosis by exclusion of other overt causes. There is now a need for a new definition that includes the findings of NAFLD; however, the new definition should allow for the presence of metabolic disease, regardless of whether the presence of liver disease is caused by alcohol or other factors. Thus, in 2020, the Asian Pacific Association for the Study of the Liver (APASL) announced the more clinically practical definition, metabolic dysfunction associated fatty liver disease (MAFLD).

Among the general population, the prevalence of NAFLD is approximately 20-30%. NAFLD is a metabolic disorder resulting from obesity, which occurs due to consumption of a nutritionally insufficient and energy dense diet along with a lack of physical activity.[2-4] Although NAFLD has a generally good prognosis, some patients may progress to having serious liver diseases, such as non-alcoholic steatohepatitis (NASH), non-alcoholic fatty liver-associated cirrhosis, and hepatocellular carcinoma; these are clinically important diseases affecting multiple organs, such as the heart and kidneys.[5,6] With the global increase in obesity and metabolic syndrome, the prevalence of NAFLD is increasing worldwide, and it is gradually becoming a serious social and economic problem.[7]

In the past, NAFLD could be diagnosed with an ultrasound, computerized tomography (CT), magnetic resonance imaging, or a liver biopsy, and it was generally suspected in patients who did not have a history of alcohol use or a metabolic disease like diabetes.[1] In contrast, the diagnosis of MAFLD is more systematic. Hepatic steatosis is diagnosed by histological or imaging tests or biomarkers in addition to the presence of at least one of the following three criteria: (1) overweight or obesity, (2) type 2 diabetes mellitus, or (3) clinical evidence of metabolic disturbance, such as abdominal obesity or abnormal lipid or glycemic profile.[8] As a tool to evaluate steatosis, the fatty liver index (FLI) was introduced as a serological panel that can diagnose fatty liver without liver biopsy

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and imaging tests. The FLI is calculated using BMI, waist circumference, and serum triglyceride and γ-glutamyltransferase (GGT) concentrations.[9]

Recently, interest in the roles of various nutrients in fatty liver disease has increased, particularly for vitamin D. Vitamin D is an essential lipophilic molecule required for the regulation of bone metabolism through regulation of calcium and phosphorus metabolism. Vitamin D is synthesized in the skin through reactions that require absorption of UV light and is later activated by hydroxylation in the kidneys and liver.[10] Vitamin D has pleiotropic effects and is known to reduce free fatty acid-induced insulin resistance in peripheral tissues and hepatocytes.[11,12] Therefore, we applied the new concept of MAFLD as a metabolic disorder beyond the existing concept of NAFLD and evaluated the association between MAFLD and nutrient intake, particularly vitamin D, in a large sample of South Korean adults diagnosed with fatty liver according to their FLI.

METHODS

Study population

Research was conducted with the approval of the Wonkwang University Hospital Institutional Review Board (approval number 2021-07-009). Consent from all participants was not required as we reviewed only publicly available data for this research. We guarantee that we will not use the information for any purpose other than the current research study. Among the participants of the 2010-2011 Korea National Health and Nutrition Examination Survey (KNHANES), 11,998 adults aged ≥19 years were included. A total of 10,506 patients were eventually analyzed after excluding: patients whose values for the calculation of the FLI for fatty liver diagnosis were not investigated or were missing, those for whom a 24-h dietary pattern recall method was not undertaken, and those with chronic viral hepatitis and other liver diseases (Figure 1).

Diagnosis of MAFLD

Participants were included in the MAFLD group if (1) the BMI and FLI were ≥23 and ≥60, respectively; (2) the BMI was <23, although there were two or more metabolic risk abnormalities, and the FLI was ≥60; or (3) the patient had diabetes mellitus, and the FLI was ≥60.[8]

Calculation of the FLI

The FLI was calculated according to the following formula.[9]

FLI = 1 / (1 + exp (–x)) × 100, where x = 0.953 × loge (triglyceride) + 0.139 × BMI + 0.718 × loge (γ-glutamyltransferase) + 0.053 × (waist circumference) – 15.745

FLI ≥60 was considered fatty liver.

Clinical and laboratory assessments

General characteristics, such as sex, age, smoking, alcohol consumption, blood test results (aspartate aminotransferase [AST], alanine aminotransferase [ALT], creatinine, fasting glucose, total cholesterol, triglyceride, LDL cholesterol [LDL-C], HDL cholesterol [HDL-C], and serum vitamin D concentrations), and nutrient intake were evaluated. Education level was inferred from socioeconomic characteristics. Smoking was classified as "yes" by combining the responses "every day," "sometimes," and "no" for "smoking in the past but not now." Alcohol consumption was not investigated.

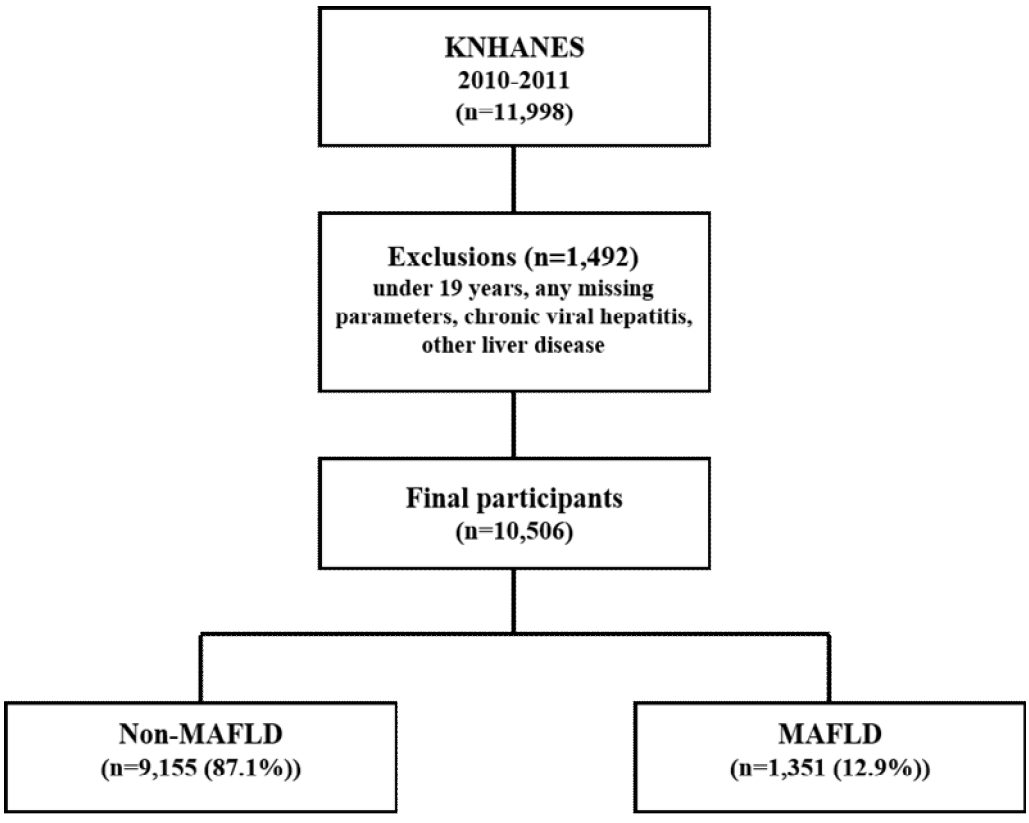

Figure 1. Flowchart of participant inclusion and exclusion.

Nutrient intake was given by the intake of total energy (kcal), water (mL), protein (g), fat (g), carbohydrate (g), fiber (g), ash (g), calcium (mg), phosphorus (mg), iron (mg), sodium (mg), potassium (mg), vitamin A (μg RAE), carotene (μg), retinol (μg), thiamin (mg), riboflavin (mg), niacin (mg), and vitamin C (mg), along with blood vitamin D concentrations (ng/mL). Any associations of nutrient intake constituents with MAFLD were analyzed. The concentration of vitamin D in the blood was measured in the form of 25(OH)D concentrations using a 1470 Wizard gamma counter (PerkinElmer, Turku, Finland) and 25-Hydroxy Vitamin D 125I RIA kit (DiaSorin, Stillwater, OK, USA).

Statistical analyses

The data were analyzed using the SPSS for Windows Version 26.0 software program (SPSS Inc., Armonk, NY, USA), and statistical significance was defined as p value <0.05. Since the KNHANES provides complex survey data, a complex and weighted sample analysis was performed. Weights were applied according to the guidelines for using raw data from the Korea Center for Disease Control and Prevention. The general characteristics of the study subjects were analyzed using the complex sample plan for frequency analysis. The complex sample Rao–Scott adjusted chi-square test and complex sample generalized linear model t-test were used to compare differences between general characteristics and nutrient intake according to MAFLD. A complex sample logistic regression test was used to see if the variables related to MAFLD were affected and the OR was calculated; change was shown after adjusting for age.

RESULTS

Participants

Our study included 10,506 participants, comprising 4,299 men (49.9%) and 6,207 women (50.1%), of whom 1,351 (12.9%) were in the MAFLD group according to the new MAFLD diagnostic criteria.

Clinical and laboratory assessments

Demographic and substance use indicators, tests of liver function and glycemic control, and nutritional status of study participants are presented according to MAFLD status in Table 1. Participants in the MAFLD group were older and had higher rates of drinking and smoking than those in the control group. Levels of exercise performed did not differ between the groups. In the MAFLD group, the concentrations of AST, ALT, total cholesterol, LDL-C, triglyceride, fasting glucose, and creatinine were significantly higher. In particular, the concentration of triglyceride in the MAFLD group was more than double that in the control group. On the other hand, the HDL-C concentration was significantly lower in the MAFLD group. For most nutrients, intake was significantly higher in the MAFLD group than in the non-MAFLD group, with the exception of carotene, retinol, and vitamin C, which did not differ significantly. Serum vitamin D concentrations tended to be <20 ng/mL, indicating vitamin D deficiency in both groups (17.47±0.18 ng/mL for non-MAFLD vs 18.07±0.26 ng/mL for MAFLD, p=0.010).

Statistical analyses

To investigate the association between MAFLD and metabolic functions and nutrient status, the OR for each factor reported in Table 1 was calculated (Table 2). The OR was significantly elevated for the following parameters: ALT, LDL-C, triglyceride, fasting glucose, and creatinine. Among these, creatinine showed the greatest elevation in risk at 8.5. In the MAFLD group, the OR of each nutrient examined was significantly higher, with the exception of vitamin C (OR 1.0001, 95% CI: 0.999-1.001, p=0.704).

We adjusted the metabolic profiles and nutrients, including vitamin D, for age (Table 3). High fasting glucose, triglyceride, LDL-C, ALT, and creatinine concentrations were correlated with an increased risk of MAFLD. Specifically, elevated creatinine was associated with a 4.3-fold increase in the likelihood of MAFLD occurrence. Sodium intake (OR 1.0001, 95% CI: 1.00002-1.0001, p=0.007) was also significantly associated with MAFLD. In contrast, our study found no significant association of vitamin D with MAFLD (OR 1.010, 95% CI: 0.985-1.037, p=0.431).

DISCUSSION

Our study is the first to analyze South Korean adults according to the new concept of MAFLD, which reflects metabolic disease status, as opposed to the previously vague concept of NAFLD. We evaluated nutrient intake and especially the association between MAFLD and vitamin D; however, no significant relationship between the two was identified. There are currently few studies on MAFLD, although many studies have been conducted on the relationship between NAFLD and vitamin D. We, therefore, aim to reconcile our results with those of many other studies showing conflicting results regarding this relationship.

Experimental evidence has demonstrated that vitamin D plays a role in immune modulation; cell differentiation and proliferation; and regulating inflammation, fibrogenesis, and metabolism.[13] On the other hand, vitamin D deficiency is associated with obesity, sedentary lifestyle, and insulin resistance.[14]

Many studies have found associations between NAFLD and vitamin D concentrations. Barchetta et al[15] studied adults with normal liver enzyme concentrations, assessed by FLI and ultrasound, and reported that NAFLD was strongly associated with low blood concentrations of vitamin D, independent of diabetes, metabolic syndrome, and insulin resistance. Seo et al[16] found that the vitamin D concentration was lower among subjects with diabetes and insulin resistance, independent of the presence of visceral obesity, which was evaluated using CT scans. The OR for NAFLD increased sequentially with decreasing vitamin D concentration quartile in the diabetes group, even after adjusting for visceral fat. According to the study by Chung et al[17] in 2016, vitamin D concentrations were inversely correlated with NAFLD, even at normal concentrations. A 2017 study by Park et al[18] evaluated the relationship between NAFLD and vitamin D in 7,514 South Korean adult men and women and found an association that depended on sex, as only men showed a positive association. Wang et al[19] demonstrated in 2016 that Chinese men with vitamin D deficiency had

Table 1. Clinical characteristics of the non-MAFLD and MAFLD groups

Variables	Total subjects (n=10,506)	MAFLD		*p* value
		No=9,155	Yes=1,351	
Sex				
Male	4299 (49.9)	3396 (45.7)	903 (75.6)	<0.0001
Female	6207 (50.1)	5759 (54.3)	448 (24.4)	
Educational level				
≤Elementary	2649 (18)	2284 (17.9)	365 (18.7)	0.004
Middle	1163 (10.3)	967 (9.7)	196 (13.5)	
High	3425 (38.2)	3019 (38.6)	406 (35.5)	
≥College	3116 (33.5)	2746 (33.7)	370 (32.4)	
Alcohol drinking				
No	4925 (41.1)	4488 (43.4)	437 (26.7)	<0.0001
Yes	5378 (58.9)	4484 (56.6)	894 (73.3)	
Smoking				
No	8395 (74.3)	7500 (76.9)	895 (58.6)	<0.0001
Yes	1971 (25.7)	1529 (23.1)	442 (41.4)	
physical activity (moderate)				
No	9336 (90.1)	8142 (90.1)	1194 (90.1)	0.997
Yes	1021 (9.9)	878 (9.9)	143 (9.9)	
Walking				
No	6399 (60.6)	5548 (60.1)	851 (63.6)	0.056
Yes	3952 (39.4)	3468 (39.9)	484 (36.4)	
Age (years)	45.3±0.32	44.5±0.32	47.0±0.50	<0.0001
AST (IU/L)	22.3±0.15	20.9±0.13	31.0±0.58	<0.0001
ALT (IU/L)	22.0±0.23	19.2±0.19	39.0±0.95	<0.0001
Total cholesterol (mg/dL)	188±0.51	185±0.51	206±1.25	<0.0001
LDL cholesterol (mg/dL)	112±0.70	0.19±0.01	0.41±0.02	<0.0001
HDL cholesterol (mg/dL)	49.5±0.18	50.6±0.19	43.2±0.33	<0.0001
Triglyceride (mg/dL)	132±1.46	110±0.88	265±6.30	<0.0001
Fasting glucose (mg/dL)	96.3±0.29	94.3±0.28	109±0.95	<0.0001
Creatinine (mg/dL)	0.84±0.003	0.82±0.003	0.91±0.01	<0.0001
Total energy (kcal)	2090±14.3	2060±14.3	2362±38.1	<0.0001
Water (g)	1065±12.5	1045±13.1	1239±33.4	<0.0001
Protein (g)	75.7±0.68	75.0±0.73	85.0±1.6	<0.0001
Fat (g)	43.9±0.55	43.7±0.55	49.0±1.5	<0.0001
Carbohydrate (g)	328±1.98	326±2	345±5.05	<0.0001
Fiber (g)	7.55±0.09	7.49±0.09	8.35±0.19	<0.0001
Ash (g)	20.9±0.18	20.6±0.18	23.8±0.45	<0.0001
Calcium (mg)	526±4.87	520±4.95	586±14.3	<0.0001
Phosphorus (mg)	1227±8.32	1216±8.61	1359±21.1	<0.0001
Iron (mg)	15.5±0.19	15.3±0.19	17.6±0.52	<0.0001
Sodium (mg)	5175±48.5	5086±50.7	6062±121	<0.0001
Potassium (mg)	3136±25.1	3114±25.8	3453±59.5	<0.0001
Vitamin A (μgRE)	854±16.2	848±17.2	989±39.6	<0.0001
Carotene (μg)	4360±91.8	4344±97.3	5003±224	0.004
Retinol (μg)	121±5.41	113±3.93	174±31.7	0.055
Thiamin (mg)	1.4±0.01	1.4±0.01	1.6±0.03	<0.0001
Riboflavin (mg)	1.3±0.01	1.3±0.01	1.4±0.03	<0.0001
Niacin (mg)	17.9±0.16	17.7±0.17	20.2±0.4	<0.0001
Vitamin C (mg)	110±1.44	111±1.57	112±2.96	0.707
Serum 25(OH)D (ng/mL)	17.6±0.18	17.5±0.18	18.1±0.26	0.010

MAFLD: metabolic associated fatty liver; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDL: low-density lipoprotein; HDL: high-density lipoprotein; 25(OH)D: 25-hydroxyvitamin D.
Values are presented as means ± standard errors or unweighted numbers/weighted numbers (weighted %).
Percentages were weighted using the Korea National Health and Nutrition Examination Survey 2010-2011 sampling weights.
p-value was taken by the complex sample Rao-Scott adjusted chi-square test or complex sample generalized linear t-test.

significantly more hepatic fat than those with a normal serum vitamin D concentration; they also found that this inverse relationship was independent of age, BMI, or components of metabolic syndrome. Conversely, other study has reported that vitamin D is inversely correlated with NAFLD in both men and women.[20] Another analysis found that vitamin D concentration was only affected in patients with NAFLD who were also obese.[21] A study from Ukraine found that individuals with type 2 diabetes, diagnosed with NAFLD by ultrasound, were associated with vitamin D3 deficiency in the high FLI and hepatic steatosis index group.[22]

In contrast, many studies have found no association between NAFLD and vitamin D concentrations, as we have presently found. Barchetta et al[23] reported in 2012 that NASH, confirmed by liver biopsy, was not correlated with the blood concentrations of vitamin D. Similarly, Li et al[24] in China using ultrasound for diagnosis and Jeong et al[25] in South Korea using abdominal ultrasound and chemiluminescence immunoassay for diagnosis, also

Table 2. Crude odds ratio for general and biochemical characteristics, nutrient intake, and serum vitamin D concentration by MAFLD

Variables	Odds ratio	95% CI	*p* value
Sex (male)	3.694	3.184-4.285	<0.0001
Educational level	1.087	0.908-1.303	0.363
≤Elementary			
Middle	1.437	1.157-1.784	0.001
High	0.955	0.791-1.152	0.628
Alcohol drinking	2.106	1.800-2.465	<0.0001
Smoking	2.356	2.015-2.754	<0.0001
AST (IU/L)	1.067	1.055-1.079	<0.0001
ALT (IU/L)	1.062	1.053-1.071	<0.0001
Total cholesterol (mg/dL)	1.015	1.013-1.017	<0.0001
LDL cholesterol (mg/dL)	3.104	2.679-3.596	<0.0001
HDL cholesterol (mg/dL)	0.938	0.930-0.945	<0.0001
Triglyceride (mg/dL)	1.015	1.014-1.016	<0.0001
Fasting glucose (mg/dL)	1.023	1.019-1.027	<0.0001
Creatinine (mg/dL)	8.533	5.426-13.418	<0.0001
Total energy (kcal)	1.0003	1.0002-1.0004	<0.0001
Water (g)	1.0003	1.0002-1.0004	<0.0001
Protein (g)	1.004	1.003-1.006	<0.0001
Carbohydrate (g)	1.001	1.001-1.002	<0.0001
Fiber (g)	1.02	1.006-1.035	0.005
Ash (g)	1.02	1.015-1.025	<0.0001
Calcium (mg)	1.0005	1.0003-1.0007	<0.0001
Phosphorus (mg)	1.0004	1.0002-1.0005	<0.0001
Iron (mg)	1.012	1.005-1.02	0.002
Sodium (mg)	1.00007	1.00005-1.00009	<0.0001
Potassium (mg)	1.0001	1.00008-1.0002	<0.0001
Vitamin A (μgRE)	1.0001	1.00004-1.0002	0.001
Carotene (μg)	1.00001	1.000005-1.00002	0.002
Retinol (μg)	1.0003	1.00003-1.0006	0.027
Thiamin (mg)	1.228	1.149-1.312	<0.0001
Riboflavin (mg)	1.238	1.128-1.358	<0.0001
Niacin (mg)	1.019	1.013-1.025	<0.0001
Vitamin C (mg)	1.0001	0.999-1.001	0.704
Serum 25(OH)D (ng/mL)	1.015	1.004-1.026	<0.0001

MAFLD: metabolic associated fatty liver; CI: confidence interval; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDL: low-density lipoprotein; HDL: high-density lipoprotein; 25(OH)D: 25-hydroxyvitamin D.
p-value was taken by the complex samples logistic regression test.

Table 3. Age-adjusted odds ratio for general and biochemical characteristics, nutrient intake, and serum vitamin D concentration by MAFLD

Variables	Odds ratio	95% CI	*p* value
Age (years)	1.009	0.996-1.021	0.164
Serum 25(OH)D (ng/mL)	1.01	0.985-1.037	0.431
Fasting glucose (mg/dL)	1.011	1.003-1.019	0.007
Total cholesterol (mg/dL)	0.989	0.977-1.001	0.07
Triglyceride (mg/dL)	1.014	1.012-1.017	<0.0001
LDL cholesterol (mg/dL)	1.017	1.004-1.031	0.013
ALT (IU/L)	1.044	1.032-1.055	<0.0001
Creatinine (mg/dL)	4.329	1.506-12.446	0.007
Carbohydrate (g)	0.999	0.997-0.99995	0.041
Sodium (mg)	1.0001	1.00002-1.0001	0.007
Retinol (μg)	1.0003	0.999-1.001	0.056

MAFLD: metabolic associated fatty liver; CI: confidence interval; 25(OH)D: 25-hydroxyvitamin D; ALT: alanine aminotransferase; LDL: low-density lipoprotein.
p-value was taken by the complex samples logistic regression test.

found no relationship between NAFLD and vitamin D. There were no differences in the incidence of NAFLD according to vitamin D quartiles or sex.

In a 2013 study by Catena et al,[26] NAFLD was not associated with vitamin D deficiency in hypertensive patients without additional cardio-metabolic risk. In 2016, Patel et al[14] analyzed the relationship between vitamin D concentration, hepatic gene expression for vitamin D-related genes, and histological severity of NAFLD confirmed by liver biopsy. Vitamin D concentration is affected by light skin color and seasonal effects, nutritional supplementation, BMI, age, and gender, all of which were

considered. There were no differences in vitamin D blood concentrations between the NAFLD and control groups, and there was no association with histological severity of NAFLD that had been confirmed by biopsy rather than imaging. Furthermore, the current author's previous study on NAFLD, diagnosed using CT and FLI, also showed no correlation between NAFLD and vitamin D in 785 health check-up subjects, consistent with the present findings.[27]

Previous studies have inconsistently shown associations between vitamin D and NAFLD depending on the presence of underlying diseases, such as diabetes and hypertension, race, sex, method of vitamin D measurement, and diagnostic modalities of fatty liver. Our study differs in that we have applied the new MAFLD concept. Nevertheless, we have not found an association between vitamin D and MAFLD, suggesting that new diagnostic criteria by themselves have not clarified the relationship between vitamin D and MAFLD. Thus, we indicate that using the new diagnostic criteria for MAFLD is not adequate to guarantee that an association between vitamin D and MAFLD will be evident.

Excessive sodium intake by the respondents of the KNHANES is evident from the data and reflects the general South Korean diet. An increase in awareness regarding the associated risks of a high sodium diet is essential. Elevated sodium can lead to an increase in blood pressure, a component of metabolic syndrome. Choi et al[28] reported in 2016 that dietary sodium intake was associated with NAFLD, and Huh et al[29] reported in 2015 that high dietary sodium intake was associated with NAFLD and hepatic fibrosis. In our study, high sodium intake also appears to increase the risk of MAFLD. Additionally, creatinine, which is related to renal function, significantly increases the risk of MAFLD when compared with other factors. Thus, high sodium intake as well as serum creatinine may act as predictors of MAFLD, although further research is needed to ascertain the exact role of these factors. NAFLD is also known to increase the risk of chronic kidney disease by causing a decrease in renal function.[30] However, MAFLD more accurately predicts the risk of occurrence of chronic kidney disease and significant liver fibrosis than that of NAFLD.[31,32]

MAFLD is multifactorial. Dietary macronutrients and micronutrients can influence MAFLD pathogenesis. Excessive macronutrient intake causes insulin resistance and proinflammation. On the other hand, micronutrients are assumed to play a supportive role, but still less evident.[33]

Our study has some limitations. First, we attempted to use the most recent data available, although the latest data available was from 2010-2011 as this was the last time GGT had been included in the KNHANES. Second, since this is a cross-sectional study, causal relationships are unclear. Third, FLI was used instead of biopsy, and fourth, various confounding variables were not controlled for.

Although previous clinical studies provided evidence regarding an association of vitamin D in the pathogenesis of NAFLD, we found no such association for MAFLD. Studies have shown various results in different settings, and simple comparisons are difficult. A significant challenge ahead is to determine reasons for the vast variability in reports of the association between MAFLD and vitamin D. This may be due to different diagnostic criteria for vitamin D deficiency, different methods of measuring vitamin D concentration, and different patient populations. Therefore, it is necessary to collect various research results in the future to discern this relationship.

Host, environmental, and genetic factors for vitamin D synthesis and metabolism, definition of vitamin D deficiency, methods of measuring 25(OH)D, and diagnostic methods may interfere with the evaluation of the association between MAFLD and vitamin D; these potential confounders should be controlled accordingly.[13]

In the future, as the newly established concept of MAFLD gains precedence, additional studies using the latest data will be required. We anticipate that the data in our study may be used as a baseline reference for future MAFLD studies.

AUTHOR DISCLOSURES

The authors declare no conflict of interest.

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