Original Research Article

Dietary Patterns and Anxiety Symptoms: Population-Level Evidence from the 2024 Korea National Health and Nutrition Examination Survey

Jiyun Lim

Seoul Academy, 422 Yeoksam-ro, Gangnam-gu, Seoul 06200, Republic of Korea

ABSTRACT

This study explores the associations between dietary habits and anxiety disorders at the population level. Although there has been increasing scholarly attention to the biological mechanisms, such as the gut-brain axis, and social mechanisms, such as socio-economic status, through which anxiety disorders emerge, few empirical studies have examined the dietary factors that may improve or impair anxiety disorders at the population level. Thus, this study hypothesizes that several risk and protective factors related to dietary habits are strongly associated with anxiety disorders and tests this relationship using data from the 2024 Korea National Health and Nutrition Examination Survey (KNHANES), published by the Korea Disease Control and Prevention Agency, consisting of the final analytical sample of 4,406 respondents. The regression analyses with multiple models reveal no statistically significant associations between nutrient-level diet factors, such as daily intake of saturated fat, omega-6 fatty acid, sodium, sugar, omega-3 fatty acid, and fiber, and anxiety symptoms. However, the results also reveal consistent significance of some socioeconomic, health, and behavioral factors, including income, health access, health literacy, smoking, and sleep, which all exhibited p-values less than 0.05 across different models. These results are discussed from the perspective of the fundamental cause theory. Key improvements in research design and data collection to test the associations between diet and anxiety at the population level are discussed. Also, the broader implications of how public health policy should intervene in the rising prevalence of anxiety are presented.

Keywords: Anxiety; Risk Factors; Protective Factors; KNHANES; Mental Health Epidemiology; Western Diet

INTRODUCTION

Anxiety is a psychological disorder closely linked to contemporary lifestyle. Anxiety has become highly prevalent in contemporary society, regardless of age, gender, or ethnicity. Defined by fear, or avoidance of threats, and panic attacks, it is reported that the disorder affects 4.4% of the population in the world (1). Anxiety appears in different severities and types, posing further detrimental effects on major depressive disorders and adverse cardiac events (2).

With the widespread prevalence of anxiety in contemporary societies, a growing body of literature has attempted to find biological causes, such as genetic predisposition, as evidenced by the fact that 30-40% anxiety symptoms are heritable from their parents,

Corresponding author: Jiyun Lim, E-mail: jylim8605@gmail.com.

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certain genes, such as PDE4B, which is known to be associated with anxiety (3), and psychological antecedents, such as stress, or childhood adversity (4, 5). Among these determinants, dietary habits have begun to garner significant attention as they are deeply embedded in modern lifestyles and thus are deemed to influence diverse physical and mental outcomes.

While the importance of dietary habits is well known in shaping one's health outcomes, limited studies have been dedicated to answering the question of what specific dietary factors can improve or impair anxiety symptoms, which is one of the most pressing public mental health issues in contemporary societies. Particularly, characterized by high intake of risk dietary factors, such as salt, saturated fat, sugar, and low intake of fiber and unsaturated fat, Western dietary habits have been proposed to have an effect on increasing anxiety levels (6–8). However, there is still a lack of empirical research proving population-level evidence that shows associations between Western dietary habits and anxiety prevalence. Theoretical and empirical studies that evidence the associations between diet and anxiety are abundant. However, most of the existing studies rely on clinical evidence or cohort designs, making the results limited to only a small sample size and leaving the population-level evidence underexplored (9).

To test the associations between diet and anxiety, it is important to start by identifying the biological mechanisms and the dietary factors that play a role in these mechanisms. While diverse biological mechanisms have been explored, one of the prominent mechanisms that explains the link between dietary habits and anxiety disorders is the gut-brain axis. The gut-brain axis refers to "bidirectional communication between the central and enteric nervous systems, linking the brain's emotional and cognitive centers with peripheral intestinal function," which passes through multiple biological channels, including the nervous, immune, endocrine, and metabolic systems (10). In this communication, gut microbiota play an important role because it is essential in producing neurotransmitters such as serotonin and gamma-aminobutyric acid (GABA) and regulating anxiety, mood, cognition, and pain (11). Clapp *et al*. (12) note that any dysregulations in the axis can serve as a reliable sign of major depression and anxiety, and diet can contribue to such dysregulations.

While the exact mechanisms through which diet influences anxiety vary by nutrient, the literature suggests specific nutrients contribute to the disruption of neurotransmitters and metabolites, and thus the gut-brain axis. For example, a high-saturated-fat di*et al*ters gut diversity, increasing Firmicutes or Bacteroidetes while decreasing beneficial microbiota, such as Prevotella (13). In their animal study, de Noronha *et al*. (13) conducted a controlled experiment where rats were. treated with either a standard control diet (CD) or 45% high-fat diet (HFD) to see if the difference in their diet changes the microbiome composition and anxiety behaviors. They found that the CD group showed the abundance of beneficial microbiota, such as Lactobacillus and Prevotella, whereas the HFD group had the abundance of detrimental ones, including Blautia and Dorea. The difference in anxiety behaviors between the two groups was also confirmed. Additionally, a high-sugar diet is known to affect neural systems and thus emotional regulation by changing the gut microbiome. As dietary sugars can take multiple forms, including glucose, fructose, sucrose, polyols, and non-nutritive sweeteners, existing studies have attempted to empirically test whether high intake of each sugar form can influence the gut-brain axis and thus anxiety symptoms. For example, Do *et al*. (14), in their experiment with male mice, a high-glucose diet (HGD) for 12 weeks led to a loss of gut microbiome diversity with decreased Bacteroidetes and increased Proteobacteria. Moreover, HGD-fed animals showed increased gut permeability and intestinal inflammation. The literature reports similar phenomena for fructose and sucrose in animal studies (15, 16). On the contrary, previous studies evidence that a low fiber diet can reduce the production of short-chain fatty acids (SCFAs), which are involved in energy production and the regulation of the gut-brain axis (17). In particular, Han *et al*. (18) document that information moves from the gut to the brain with involvement of microbial metabolites such as SCFAs, implying the negative association between fiber intake and anxiety symptoms.

Overall, these studies suggest strong biological mechanisms through which nutrient-level diet factors can be associated with anxiety symptoms. However, there still exists a large room for exploration of whether such associations can be applied to humans because many studies relied on animal studies or tested only a single nutrient's association. Thus, building on the emerging literature of Western diet and mental health (6–8), the current study identifies key nutrient-level risk and protective diet factors and aims to test their associations with anxiety symptoms at the population level. Thus, this study attempts to answer whether the independent associations of diet factors can be confirmed in a human population and thus test whether such independent

associations are significant enough to be detected once controlling for other confounding factors that are known to be associated with anxiety.

It is important to note that individuals' socioeconomic backgrounds can serve as separate mechanisms through which dietary habits are associated with mental health outcomes. In a wide range of literature, socioeconomic status is proposed to influence mental health issues significantly, whose severity is more pronounced in low-income families. For example, studies propose that people of low socioeconomic status tend to consistently buy or store processed foods, which significantly reduces their grocery budget. However, these processed foods often contain nutrient imbalances, characterized by high risk and low protective dietary factors, and ultimately influence the gut microbiome and mental health (19). This finding provides direct evidence of the socioeconomic mechanism through which diet and mental health are associated. However, socioeconomic status conditions the relationship not only because of the budget constraint but also because of food-related stress. Particularly, food insecurity causes anxiety due to the lack of guaranteed meals later. Food insecurity occurs in households that cannot adequately meet their dietary needs, and the reasons can include insufficient income and other social factors that prevent them from accessing appropriate food, including geographic disadvantages or limited dietary literacy (20). These results clearly indicate that individual socioeconomic status can closely link diet and anxiety through diverse channels, including income and food-related stress.

In addition, the literature on behavioral clustering posits that, apart from socio-economic status, behavioral and psychological factors often manifest together, and thus some of these behavioral factors can serve as strong predictors of dietary habits and anxiety. One of the most well-known clusters includes unhealthy diet, physical inactivity, poor sleep, and smoking. Thus, while an unhealthy diet can independently influence anxiety symptoms, clustered factors that are accompanied by a poor diet can also serve as the second channels through which diet and anxiety are associated (21).

In conclusion, prior research demonstrates consistent associations between dietary habits and anxiety in various mechanisms. However, existing studies still lack population-level evidence to bolster the results of small-sample studies. Moreover, while dietary habits are strongly associated with cultural background, non-Western contexts have been underexplored, providing limited insights into how dietary habits are associated with anxiety in different cultural contexts. Therefore, this study aims to explore the relationship between risk and protective diet factors and anxiety symptoms in a non-Western context, which is South Korea by using the 2024 Korea National Health and Nutrition Examination Survey (KNHANES) data, which contained the final analytical sample of 4,406 respondents.

METHODS AND MATERIALS

To explore the relationship between dietary factors and anxiety symptoms at the population level, the current study used the 2024 Korea National Health and Nutrition Examination Survey (KNHANES), which was designed to assess the health and nutritional status of the Korean population. It is a nationally administered health and nutrition survey conducted by the Korea Disease Control and Prevention Agency, starting from 1998. Since 2008, the Korea Disease Control and Prevention Agency has utilized Mobile Examination Centers (MECs) that travel to each survey location with a standardized environment and equipment and collect a variety of health measures to better understand the multifaceted nature of public health (22). The 2024 dataset was collected throughout 2024, and the final raw dataset was published in 2025, which allowed for the analysis conducted in this study.

While the original dataset contained 6,929 respondents, this study utilized the final analytical sample of 4,406 respondents. Once identifying relevant independent, dependent, and control variables that may influence the diet-anxiety association from the dataset, the author recoded invalid and non-response values and separated them from valid, response values, and selected only cases with complete answers for the main regression model, which yielded the final sample of 4,406. The final sample only included adults who were 19 years old and older at the time of the survey because dietary patterns are not subject to individual choices, and socioeconomic status, such as income and education level, and behavioral factors, such as smoking and alcohol use, cannot meaningfully be considered in the younger population. There can be concerns about selection bias because the number of respondents significantly decreased from the full sample of 6,929 to the final analytical sample of 4,406. Especially, if the two samples are systemically different from one another, the selection bias can be more pronounced. Thus, the author compared descriptive statistics of the full vs. the analytical sample and checked whether there existed a systematic difference between

them. The comparison confirmed no systemic difference across major dependent, independent, and control variables, which reduces the concerns of selection bias. For the robustness check, the sample included 1,551 complete cases who reported using nutrition labels when consuming food because those who do so were more likely to avoid recall bias in the survey process.

The dependent variable used was the total anxiety symptom score (mh_GAD_S). This variable was constructed by summing scores from seven anxiety diagnostic items, each of which can take values from 0 to 3. Thus, a respondent can be considered experiencing no anxiety symptoms at all if the total score is 0 and full anxiety if the score is 21. The main independent variables encompassed nutrient-level dietary factors surveyed in the dataset. To precisely establish the expected directions of associations between diet factors and anxiety symptoms, these variables were classified into risk and protective factors. Risk dietary factors included saturated fat intake (N_SFA), omega-6 fatty acid intake (N_N6), sodium intake (N_NA), and sugar intake (N_SUGAR), which were expected to have positive associations with anxiety symptoms, and Protective dietary factors included omega-3 fatty acid intake (N_N3) and dietary fiber intake (N_TDF), which were predicted to have negative associations with anxiety symptoms. All nutrient variables were measured as daily intake; sodium was measured in milligrams per day, while the other dietary variables were measured in grams per day. In addition, this study constructed a composite Western Dietary Score (WDS) to capture the overall balance between dietary risk and protective factors. The WDS was based on six nutrient-level variables: saturated fat intake (N_SFA), omega-6 fatty acid intake (N_N6), sodium intake (N_NA), sugar intake (N_SUGAR), omega-3 fatty acid intake (N_N3), and dietary fiber intake (N_TDF). Because these variables were measured on different scales, each component was first standardized into a z-score. The four risk factors—saturated fat, omega-6 fatty acids, sodium, and sugar—were entered in the positive direction. The two protective factors—omega-3 fatty acids and dietary fiber—were reverse-coded by multiplying their standardized values by -1. The WDS was then calculated as the mean of the six standardized components: WDS = mean[z(N_SFA), z(N_N6), z(N_NA), z(N_SUGAR), -z(N_N3), -z(N_TDF)]. Therefore, higher WDS values indicate a more risk-oriented dietary profile. All independent variables, with brief justifications for their associations with anxiety, are summarized in Table 1.

Table 1. *Risk and Protective Diet Factors and Their Expected Influence on Anxiety. Risk/protective classification is theoretical. Expected direction refers to the hypothesized association with anxiety symptoms.*

Variable	Description	Classification	Expected direction	Rationale
N_SFA	Saturated fat intake	Risk factor	Positive	High saturated fat intake could cause pro-inflammatory changes, reduce gut microbiota diversity, and serotonin dysregulation, contributing to anxiety-like behaviors.
N_N6	Omega-6 fatty acid intake	Risk factor	Positive	Intake of omega-6 may trigger inflammation and dysbiosis, increasing possibility of anxiety.
N_NA	Sodium intake	Risk factor	Positive	Huge amount of sodium intake causes systemic inflammation and gut barrier dysfunction, which are connected to mental health problems.
N_SUGAR	Sugar intake	Risk factor	Positive	Intake of refined sugar disrupts gut microbiota balance, increases gut permeability and neuroinflammation, which are linked to anxiety symptoms.
N_N3	Omega-3 fatty acid intake	Protective factor	Negative	Omega-3 fatty acids have anti-inflammatory effects, and improve neurotransmitter synthesis, which lower anxiety risk.
N_TDF	Dietary fiber intake	Protective factor	Negative	Fiber makes short-chain fatty acid (SCFA) production, more active, improves gut barrier function, and supports beneficial microbiota, which prevent anxiety.
WDS	Western Dietary Score	Composite index	Positive	Mean of standardized risk components and reverse-coded protective components. Higher values indicate higher dietary risk.

The models also included demographic, socio-economic, health-status, and lifestyle controls that may confound the association between dietary intake and anxiety symptoms. Demographic and socioeconomic controls included sex, age, individual income quintile, household income quintile, education level, economic activity, private health insurance status, unmet medical needs, and health literacy. Sex was coded as male or female, while age was measured in years. Individual and household income were measured in five-level quintile categories, and education was measured as elementary school or lower, middle school, high school, or college or higher. Economic activity indicated whether the respondent was employed or unemployed. Private health insurance and unmet medical needs were coded as binary variables. Health literacy was measured using M_HL_S in the original dataset, a total health-information-understanding score based on 10 items, ranging from 10 to 40, with higher scores indicating greater health literacy. Health-status controls included obesity status, diabetes status, high cholesterol, high triglycerides, and hypertension, based on the KNHANES health classifications, which follow the WHO classification standard. Lifestyle controls included aerobic physical activity, smoking status, drinking status, and weekday sleep duration. Aerobic physical activity indicated whether respondents met the weekly aerobic activity guideline, which is 2.5 hours of aerobic activity per week. Smoking status was categorized as never smoker, former smoker, current occasional smoker, or current daily smoker. In addition, drinking status was categorized as never drinker, no drinking in the past year, less than once per month, about once per month, 2–4 times per month, 2–3 times per week, or 4 or more times per week. Lastly, weekday sleep duration was measured in hours.

The analysis models employed ordinary least squares regression because the dependent variable was a continuous anxiety symptom score, ranging from 0 to 21. By considering the control variables, the models were able to estimate adjusted associations between dietary variables and anxiety symptoms. However, due to the cross-sectional nature of the dataset, the results from inferential analyses with the models should not be interpreted as causal effects. Rather, the models aimed to test general associations between diet and anxiety at the population level. In particular, Model 1 included all six dietary variables to separately estimate the associations between individual dietary risk and protective factors and anxiety symptoms. The model included saturated fat, omega-6 fatty acids, sodium, sugar, omega-3 fatty acids, and dietary fiber simultaneously, along with the full set of control variables, as shown in the following regression equation, Model 1. This specification allowed the analysis to assess whether any individual dietary factor was independently associated with anxiety symptoms after adjustment for the control variables (denoted as X_i). Model 2 regression was specified by replacing the individual dietary variables with the composite Western Dietary Score (WDS). This model tested whether an overall risky dietary profile with high intake of risk factors and low intake of protective factors was associated with anxiety symptoms. The same control variables were included in Model 2 to compare with Model 1 and see whether any controls were consistently associated with anxiety across different models.

$$\textit{(Model 1) } Anxiety_i = \beta_0 + \beta_1 SFA_i + \beta_2 N6_i + \beta_3 Sodium_i + \beta_4 Sugar_i + \beta_5 N3_i + \beta_6 Fiber_i + \gamma X_i + \epsilon_i$$

$$\textit{(Model 2) } Anxiety_i = \beta_0 + \beta_1 WDS_i + \gamma X_i + \epsilon_i$$

For the robustness of the results, the study used the subsample that consisted of 1,551 complete cases who reported using nutrition labels when consuming food. The same specification strategies identified in the Model 1 and 2 were applied to this subsample to see if the results could be replicated in a more robust sample. The inferential results are reported separately in Table 5 as Models 3 and 4.

Before conducting the analyses, it was necessary to evaluate a potential heteroskedasticity issue because ordinary least squares regression assumes constant error variance. Thus, heteroskedasticity was assessed by using the Breusch–Pagan test. The Breusch–Pagan test evaluates whether the variance of the residuals is constant, and a statistically significant result provides evidence of heteroskedasticity (23). The test rejected the null hypothesis across the four models (Model 1: $p < 0.001$, Model 2: $p < 0.001$, Model 3: $p = 0.008$, and Model 4: $p = 0.004$). In turn, heteroskedasticity-consistent robust standard errors were used for statistical inference, as this approach yields more reliable estimates when the constant-variance assumption is violated (24). To evaluate potential multicollinearity concerns, VIF/GVIF diagnostics were also conducted. Because the models included categorical variables with multiple levels, adjusted generalized variance inflation factor values were examined. As a rule of thumb, VIF values below 5 indicate low multicollinearity concern, values between 5

and 10 suggest moderate collinearity, and values above 10 may indicate serious concern (25). The diagnostics revealed that the maximum adjusted GVIF values were 1.760 in Model 1, 1.702 in Model 2, 1.566 in Model 3, and 1.485 in Model 4, suggesting low multicollinearity concerns across the four models. Thus, it was concluded that it was safe to include all theoretically and empirically relevant variables that were selected in this study.

The results are reported with estimates of the coefficients (unstandardized coefficients, *b*), robust standard errors, standardized coefficients (β), and statistical significance (p-value) in the inferential result tables. In addition to unstandardized coefficients, standardized coefficients were calculated to compare the relative strength of predictors measured on different scales. While *b* indicates the size of associations of anxiety symptoms with the unit increase in the real independent and control variables, β indicates the size of associations with a one standard deviation increase in the independent and control variables.

RESULTS

The current study aims to investigate the relationship between risk and protective diet factors and anxiety symptoms, accounting for socio-economic and behavioral factors at the population level, using the Korea National Health and Nutrition Examination Survey, 2024. While it is not possible to examine the direct influence of diet factors on anxiety due to the cross-sectional design of the survey data, this study attempts to test whether significant associations can be found between diet factors, socio-economic status, behavioral factors, and anxiety at the individual level. While the original KNHANES 2024 dataset contains 6,929 individuals, the current study used a sample of 4,406 respondents who completed the survey items used in the main analysis, including the dependent anxiety variable, independent diet factor variables, and socio-economic, chronic disease, and behavioral control variables. In other words, the sample used in the main analysis was obtained after excluding observations with missing values on the variables in the main regression models.

Tables 2 and 3 report the overall characteristics of the analytical sample used for the final inferential analyses. The sample exhibited 2.03 of mean anxiety score (SD = 3.40) out of 21 full score, indicating a relatively low level of anxiety symptoms in the sample. For risky dietary intake, the mean saturated fat intake was 14.96 g/day (SD = 12.02), omega-6 fatty acid intake was 10.55 g/day (SD = 8.10), sodium intake was 3,168.00 mg/day (SD = 1,821.46), and sugar intake was 57.04 g/day (SD = 40.53). For protective dietary factors, mean omega-3 fatty acid intake was 1.94 g/day (SD = 1.89), and mean dietary fiber intake was 25.31 g/day (SD = 13.57). The average age was 53.73 (SD = 16.34), which indicates that the sample well represents the whole South Korean population across age groups. While the high level of health literacy score (M = 29.94, SD = 5.19) suggests a relatively strong capability of the sample to manage their dietary habits

Table 2. *Continuous Variable Descriptive Statistics for Main Analytic Sample. Descriptive statistics are based on the main complete-case analytic sample used in the fully adjusted main models (N = 4,406).*

Variable	N	Mean (SD)	Median	Range
Anxiety symptoms (GAD-7 sum score)	4406	2.03 (3.40)	0.00	0.00-21.00
Western Dietary Score	4406	-0.02 (0.39)	-0.08	-2.22-3.49
Saturated fat intake (g/day)	4406	14.96 (12.02)	12.00	0.23-175.89
Omega-6 fatty acid intake (g/day)	4406	10.55 (8.10)	8.59	0.22-100.48
Sodium intake (mg/day)	4406	3168.00 (1821.46)	2844.89	95.75-16573.57
Sugar intake (g/day)	4406	57.04 (40.53)	48.40	0.04-401.78
Omega-3 fatty acid intake (g/day)	4406	1.94 (1.89)	1.43	0.01-24.41
Dietary fiber intake (g/day)	4406	25.31 (13.57)	22.89	0.00-125.96
Age	4406	53.73 (16.34)	56.00	19.00-80.00
Health literacy score	4406	29.94 (5.19)	30.00	10.00-40.00
Weekday sleep duration (hours)	4406	6.61 (1.26)	7.00	1.00-12.00

Table 3. *Descriptive Statistics of Lifestyle, Health, and Socio-economic Background.*

Sex (sex)	**Male**	**Female**					4406 (100%)
	1833 (41.6%)	2573 (58.4%)					
Individual Income (incm5)	**Lower**	**Lower-Middle**	**Middle**	**Middle-Upper**	**Upper**		
	802 (18.2%)	890 (20.2%)	898 (20.4%)	915 (20.8%)	901 (20.4%)		4406 (100%)
Household Income (ho_incm5)	**Lower**	**Lower-Middle**	**Middle**	**Middle-Upper**	**Upper**		
	607 (13.8%)	796 (18.1%)	915 (20.8%)	1031 (23.4%)	1057 (24.0%)		4406 (100%)
Education level (edu)	**Elementary school graduate or lower**	**Middle school graduate**	**High school graduate**	**College or higher**			
	704 (16.0%)	448 (10.2%)	1444 (32.8%)	1810 (41.1%)			4406 (100%)
Economic Activity (EC1_1)	**Yes (employed)**	**No (Unemployed)**					
	2715 (61.6%)	1691 (38.4%)					4406 (100%)
Private health insurance coverage (npins)	**Yes**	**No**					
	3704 (84.1%)	702 (15.9%)					4406 (100%)
Unmet medical needs (M_2_yr)	**Yes**	**No**					
	361 (8.2%)	4045 (91.8%)					4406 (100%)
Obesity (HE_obe)	**Underweight**	**Normal Weight**	**Pre-obesity**	**Class 1 Overweight**	**Class2 Overweight**	**Class 3 Overweight**	
	196 (4.4%)	1651 (37.5%)	998 (22.7%)	1275 (28.9%)	245 (5.6%)	41 (0.9%)	4406 (100%)
Diabetes (HE_DM_HbA1c)	**Normal**	**Pre-Diabetes**	**Diabetes**				
	2402 (54.5%)	1345 (30.5%)	659 (15.0%)				4406 (100%)
Experience of hypercholesterolemia (HE_HCHOL)	**No**	**Yes**					
	2990 (67.9%)	1416 (32.1%)					4406 (100%)
Experience of hypertriglyceridemia (HE_HTG)	**No**	**Yes**					
	3945 (89.5%)	461 (10.5%)					4406 (100%)
Experience of hypertension (HE_HP)	**Normal**	**Elevated Blood Pressure**	**Prehypertension**	**Hypertension**			
	1993 (45.2%)	348 (7.9%)	607 (13.8%)	1458 (33.1%)			4406 (100%)
Aerobic Physical Activity (pa_aerobic1)	**less than 2.5 hours per week**	**More than 2.5 hours per week**					
	2393 (54.3%)	2013 (45.7%)					4406 (100%)

Continued Table 3. *Descriptive Statistics of Lifestyle, Health, and Socio-economic Background.*

Currently Smoking Regularly	**Never Smoker**	**Former Smoker**	**Current Occasional Smoker**	**Current Daily Smoker**			
	2770 (62.9%)	1011 (22.9%)	99 (2.2%)	526 (11.9%)			4406 (100%)
Frequency of drinking over a year	**Never drinker**	**No drinking in past year**	**Less than once per month**	**About once a month**	**2-4 times per month**	**2-3 times a week**	**More than 4 times a week**
	514 (11.7%)	787 (17.9%)	862 (19.6%)	520 (11.8%)	948 (21.5%)	562 (12.8%)	213 (4.8%)
Nutrition Label Use	**Does not use nutrition labels**	**Uses nutrition labels**	**NA**				
	2018 (45.8%)	1551 (35.2%)	837 (19.0%)				4406 (100%)

and mental issues, 6.61 hours of average weekday sleep duration points to the potential health issues related to sleep and psychological control.

Regarding sex, individual, and household income, the analytical sample is well-balanced, suggesting that the results of this study can be generalized to the entire population. 73.9% of the sample had high school or higher education, which reinforces the sample's high average health literacy level. In addition, 84.1% of the sample reported private health insurance coverage, and only 8.2% reported unmet medical needs, suggesting strong institutional medical care for the sample. However, substantial portions of the sample experienced chronic diseases, such as obesity (35.4%), diabetes (15.0%), and hypertension (33.1%), which allowed for testing the association between diverse chronic diseases and anxiety. Regarding health behaviors, while only 14.1% of respondents were current smokers and 17.6% drank more than 2-3 times a week, the proportion of respondents who met the aerobic physical activity guideline was only 45.7%, suggesting a mixed health-related behavioral profile of the sample. Overall, the descriptive analysis suggests sufficient variability across the variables in the sample, allowing for reliable regression analyses.

Table 4 reports the main regression results, where Model 1 estimated the association between individual dietary factors and anxiety, while Model 2 tested the association between the composite Western Dietary Score index and anxiety. Theoretical explanations predicted that risk dietary factors—saturated fat, omega-6 fatty acids, sodium, and sugar—would be positively associated with anxiety score, whereas protective dietary factors—omega-3 fatty acids and dietary fiber—would exhibit negative associations. Contrary to expectations, the regression results confirmed no significant associations. Although omega-6 fatty acid (b = 0.013, robust SE = 0.013, β = 0.030) and sugar intake (b = 0.001, robust SE = 0.002, β = 0.017) produced the expected positive direction, the associations were not statistically significant (p = 0.328 and 0.402, respectively). Saturated fat intake and sodium intake showed negative (b = -0.005, robust SE = 0.006, β = -0.017) and near-zero associations (b = -0.000, robust SE = 0.000, β = -0.008) with non-significance (p = 0.455 and 0.710, respectively), which also contradicts the expected theoretical associations. The protective dietary factors were also not statistically significant: omega-3 fatty acid intake showed almost no association with anxiety symptoms (b = 0.003, robust SE = 0.031, β = 0.002, p = 0.914), and dietary fiber intake was negative but nonsignificant (b = -0.001, robust SE = 0.006, β = -0.006, p = 0.810). Overall, in Model 1, while omega-6 fatty acid, sugar intake, and dietary fiber intake showed the theoretically expected directions, none of the dietary factors were associated with anxiety in a statistically significant way at the population level. These results suggest that individual dietary indicators had limited independent explanatory power for anxiety symptoms after adjustment for broader demographic, socioeconomic, health, and lifestyle factors. This interpretation is reinforced by the fact that the model explained only 9.8% of the variance in anxiety symptoms in the sample ($R^2 = 0.098$, adjusted $R^2 = 0.088$).

To see the overall impact of Western dietary habits, characterized as high intake of risk nutrient factors and low intake of protective nutrient factors, the Model 2 tested the association between the Western Dietary Score (WDS) and anxiety. Because higher WDS score values

indicate higher intake of risk dietary factors and lower intake of protective dietary factors, WDS was expected to show a positive association with anxiety symptoms. The coefficient for WDS was positive, as theories expected, but not statistically significant (b = 0.071, robust SE = 0.157, β = 0.008, p = 0.652). These results indicate that respondents with more Westernized nutrient profiles did not report significantly higher anxiety symptoms than those with less Westernized diet habits after controlling for demographic, socio-economic, health, and behavioral factors. Reinforcing the non-significant association, Model 2 explained only 9.7% of the variance in anxiety symptoms (R^2 = 0.097, adjusted R^2 = 0.088). In conclusion, neither the individual dietary factors nor the composite dietary score provided strong evidence of an independent diet–anxiety association in the main models with the population-level data.

However, it is important to note some of the significant associations between demographic, socio-economic, health, and behavioral factors and anxiety symptoms in the models. For example, female respondents reported significantly higher anxiety symptoms than male respondents in both models (Model 1: b = 1.003, robust SE = 0.153, β = 0.295, p < 0.001; Model 2: b = 1.004, robust SE = 0.150, β = 0.295, p < 0.001), suggesting that sex differences were more strongly related to anxiety symptoms than any dietary variable. In addition, the regression results of household income, education, and employment showed the importance of socio-economic disparity in anxiety symptoms in the sample. For example, compared with respondents in the lowest household-income group, respondents in the middle household-income group reported lower anxiety symptoms (Model 1: b = -0.610, robust SE = 0.274, β = -0.179, p < 0.05), and respondents in the highest household-income group reported nearly one point lower anxiety scores (Model 1: b = -0.994, robust SE = 0.372, β = -0.292, p < 0.01). These results suggest that household-level socioeconomic resources were more consistently associated with anxiety symptoms than individual nutrient intake.

The analysis results also suggested the importance of access to health care and health literacy for mental health. In the two models, the variables related to healthcare access and health literacy were among the strongest predictors of anxiety symptoms. Respondents without unmet medical needs reported substantially lower anxiety symptoms than those with unmet medical needs (Model 1: b = -1.940, robust SE = 0.242, β = -0.570, p < 0.001; Model 2: b = -1.937, robust SE = 0.242, β = -0.569, p < 0.001). The size of this association, evaluated by β, which indicates an increase in anxiety score with a one-standard-deviation increase in the predictor, was larger than the β coefficients for all dietary variables, indicating that access to healthcare was more strongly associated with anxiety symptoms than dietary intake factors. Private health insurance also showed a significant association. Respondents without private health insurance reported higher anxiety symptoms than those with private insurance (Model 1: b = 0.394, robust SE = 0.168, β = 0.116, p < 0.05; Model 2: b = 0.389, robust SE = 0.169, β = 0.114, p < 0.05), further suggesting that healthcare-related resources may be important for understanding anxiety symptoms. Higher health literacy was also consistently associated with lower anxiety symptoms (Model 1: b = -0.069, robust SE = 0.011, β = -0.105, p < 0.001; Model 2: b = -0.069, robust SE = 0.011, β = -0.105, p < 0.001). This suggests that respondents with a higher ability to understand and use health information tended to experience lower anxiety symptoms.

As reported in Table 4, several behavioral and health factors were also more consistently associated with anxiety symptoms than dietary factors. Compared with never smokers, former smokers, current occasional smokers, and current daily smokers all reported higher anxiety symptoms. In particular, Former smokers had higher anxiety symptoms (Model 1: b = 0.625, robust SE = 0.155, β = 0.184, p < 0.001), as did current occasional smokers (b = 0.811, robust SE = 0.401, β = 0.238, p < 0.05) and current daily smokers (b = 0.850, robust SE = 0.207, β = 0.250, p < 0.001). These findings indicate that smoking status was more strongly associated with anxiety symptoms than any of the dietary variables. In addition, several drinking-status categories were also positively associated with anxiety symptoms relative to never drinking. Respondents who reported no drinking in the past year (b = 0.726, robust SE = 0.194, β = 0.213, p < 0.001), drinking less than once per month (b = 0.463, robust SE = 0.180, β = 0.136, p < 0.01), drinking two to four times per month (b = 0.420, robust SE = 0.184, β = 0.123, p < 0.05), drinking two to three times per week (b = 0.490, robust SE = 0.213, β = 0.144, p < 0.05), and drinking four or more times per week (b = 0.746, robust SE = 0.319, β = 0.219, p < 0.05) reported higher anxiety symptoms than never drinkers. Furthermore, sleep behavior was found to be significantly associated with anxiety symptoms. Weekday sleep duration was negatively associated with anxiety symptoms (Model 1: b = -0.187, robust SE = 0.049, β = -0.069, p < 0.001; Model 2: b = -0.189, robust SE = 0.049, β = -0.070, p

Table 4. *Main Regression Models Predicting Anxiety Symptoms. Note. Values are unstandardized coefficients with robust standard errors in parentheses; β denotes standardized coefficient. Reference categories include male, lower income quintile, elementary-or-less education, employed, private insurance yes, unmet medical need yes, normal blood pressure, never smoker, and never drinker. Other controls included in the model are not shown if nonsignificant and not central. † p < 0.10, * p <0.05, ** p < 0.01, *** p < 0.001.*

Predictor	Model 1 b (Robust SE)	Model 1 β	Model 2 b (Robust SE)	Model 2 β
Saturated fat intake	-0.005 (0.006)	-0.017		
Omega-6 fatty acid intake	0.013 (0.013)	0.030		
Sodium intake	-0.000 (0.000)	-0.008		
Sugar intake	0.001 (0.002)	0.017		
Omega-3 fatty acid intake	0.003 (0.031)	0.002		
Dietary fiber intake	-0.001 (0.006)	-0.006		
Western Dietary Score			0.071 (0.157)	0.008
Female	1.003 (0.153)***	0.295	1.004 (0.150)***	0.295
Age	-0.046 (0.006)***	-0.222	-0.046 (0.005)***	-0.220
Individual income: upper	0.565 (0.300)†	0.166	0.587 (0.300)†	0.172
Household income: middle	-0.610 (0.274)*	-0.179	-0.617 (0.272)*	-0.181
Household income: middle-upper	-0.612 (0.317)†	-0.180	-0.612 (0.315)†	-0.180
Household income: upper	-0.994 (0.372)**	-0.292	-1.004 (0.370)**	-0.295
Education: college or higher	0.378 (0.206)†	0.111	0.406 (0.204)*	0.119
Unemployed	0.227 (0.110)*	0.067	0.227 (0.109)*	0.067
No private health insurance	0.394 (0.168)*	0.116	0.389 (0.169)*	0.114
No unmet medical need	-1.940 (0.242)***	-0.570	-1.937 (0.242)***	-0.569
Health literacy score	-0.069 (0.011)***	-0.105	-0.069 (0.011)***	-0.105
High cholesterol	0.246 (0.121)*	0.072	0.250 (0.121)*	0.073
Elevated blood pressure	-0.334 (0.174)†	-0.098	-0.338 (0.174)†	-0.099
Former smoker	0.625 (0.155)***	0.184	0.625 (0.156)***	0.184
Current occasional smoker	0.811 (0.401)*	0.238	0.816 (0.401)*	0.240
Current daily smoker	0.850 (0.207)***	0.250	0.834 (0.207)***	0.245
No drinking in past year	0.726 (0.194)***	0.213	0.724 (0.194)***	0.213
Drinking: < once/month	0.463 (0.180)**	0.136	0.459 (0.179)*	0.135
Drinking: 2–4 times/month	0.420 (0.184)*	0.123	0.407 (0.184)*	0.119
Drinking: 2–3 times/week	0.490 (0.213)*	0.144	0.465 (0.212)*	0.137
Drinking: ≥4 times/week	0.746 (0.319)*	0.219	0.708 (0.318)*	0.208
Weekday sleep duration	-0.187 (0.049)***	-0.069	-0.189 (0.049)***	-0.070
N	4406		4406	
R^2	0.098		0.097	
Adjusted R^2	0.088		0.088	
RMSE	3.233		3.234	

< 0.001), meaning that each additional hour of weekday sleep was associated with approximately 0.19 points lower anxiety score. Some health factors were also found to be associated with anxiety symptoms. For example, high cholesterol (Experience of hypercholesterolemia, HE_HCHOL) was positively associated with anxiety symptoms (Model 1: b = 0.246, robust SE = 0.121, β = 0.072, p < 0.05), while other health-status indicators were less consistent. Taken together, these results suggest that anxiety symptoms were more strongly associated with behavioral and health-related factors than with the dietary indicators.

To assess the robustness of the results, the current study used a partial sample consisting of respondents who reported using nutrient labels when consuming food. The analysis of this partial sample could yield more robust results because it was likely to avoid recall bias in the survey process. Table 5 reports the regression results with this partial sample, with Model 3 estimating the association between individual diet factors and anxiety symptoms and Model 4 testing the association between the Western Dietary Score (WDS) and anxiety symptoms. Overall, the robustness results were broadly consistent with the main models.

Most individual dietary variables remained nonsignificant, including saturated fat, omega-6 fatty acids, sodium, sugar, and dietary fiber. Only omega-3 fatty acid intake showed a marginal negative association with anxiety symptoms (b = -0.085, robust SE = 0.051, β = -0.047, p < 0.10). The association was in the expected protective direction but did not reach conventional statistical significance (p < 0.05). In addition, the Western Dietary Score remained nonsignificant in this label-user subsample (b = 0.188, robust SE = 0.277, β = 0.021). Taken together, even among respondents who reported using nutrition labels, there was limited evidence that dietary risk and protective factors were independently associated with anxiety symptoms.

However, consistent with the main results, contextual variables, including demographic, socio-economic, health, and behavioral variables, were found to be associated with anxiety symptoms in this sub-sample. Particularly, female sex, younger age, unmet medical needs, lower health literacy, smoking status, and shorter weekday sleep duration remained more consistently associated with anxiety symptoms. For example, female respondents reported higher anxiety symptoms (Model 3: b = 1.196, robust SE = 0.252, β = 0.344, p < 0.001), while respondents without unmet medical needs reported lower anxiety symptoms (Model 3: b = -1.481, robust SE = 0.389, β = -0.425, p < 0.001). Higher health literacy (Model 3: b = -0.095, robust SE = 0.019, β = -0.124, p < 0.001) and longer weekday sleep duration (Model 3: b = -0.274, robust SE = 0.086, β = -0.093, p < 0.01) remained also associated with lower anxiety symptoms.

Overall, the results provided limited evidence that dietary risk and protective factors were independently associated with anxiety symptoms. This finding did not support the theoretical expectation that risk dietary factors would be positively associated with anxiety symptoms and protective dietary factors would be negatively associated with anxiety symptoms. The composite Western Dietary Score was also nonsignificant in both samples. Instead, anxiety symptoms were more consistently associated with contextual variables, including sex, age, household income, healthcare access, health literacy, smoking status, drinking status, and sleep duration. These findings suggest that anxiety symptoms in this population were more strongly explained by demographic, socioeconomic, healthcare-access, and behavioral factors than by the dietary indicators included in the study.

Table 5. *Robustness Models Among Nutrition-Label Users.*

Predictor	Model 3 b (Robust SE)	Model 3 β	Model 4 b (Robust SE)	Model 4 β
Saturated fat intake	0.001 (0.009)	0.005		
Omega-6 fatty acid intake	0.022 (0.017)	0.056		
Sodium intake	-0.000 (0.000)	-0.030		
Sugar intake	-0.000 (0.003)	-0.004		
Omega-3 fatty acid intake	-0.085 (0.051)†	-0.047		
Dietary fiber intake	0.004 (0.010)	0.014		
Western Dietary Score			0.188 (0.277)	0.021

Continued Table 5. *Robustness Models Among Nutrition-Label Users.*

Predictor	Model 3 b (Robust SE)	Model 3 β	Model 4 b (Robust SE)	Model 4 β
Female	1.196 (0.252)***	0.344	1.204 (0.254)***	0.346
Age	-0.050 (0.009)***	-0.214	-0.050 (0.009)***	-0.216
Unemployed	0.364 (0.191)†	0.105	0.368 (0.190)†	0.106
No private health insurance	0.681 (0.362)†	0.196	0.681 (0.361)†	0.196
No unmet medical need	-1.481 (0.389)***	-0.425	-1.477 (0.388)***	-0.424
Health literacy score	-0.095 (0.019)***	-0.124	-0.094 (0.019)***	-0.123
Former smoker	0.836 (0.260)**	0.240	0.820 (0.259)**	0.236
Current occasional smoker	1.548 (0.713)*	0.445	1.499 (0.714)*	0.431
Current daily smoker	0.710 (0.366)†	0.204	0.677 (0.365)†	0.194
No drinking in past year	0.556 (0.341)	0.160	0.572 (0.343)†	0.164
Drinking: 2–3 times/week	0.819 (0.395)*	0.235	0.796 (0.393)*	0.229
Drinking: ≥4 times/week	1.723 (0.977)†	0.495	1.721 (0.977)†	0.495
Weekday sleep duration	-0.274 (0.086)**	-0.092	-0.271 (0.086)**	-0.091
N	1551		1551	
R^2	0.112		0.110	
Adjusted R^2	0.084		0.085	
RMSE	3.279		3.283	

DISCUSSION

This study aimed to examine whether dietary risk and protective factors were associated with anxiety symptoms at the population level with the KNHANES data. The purpose was to find the population evidence for the emerging literature on diet and mental health (6, 9, 26). However, the empirical analyses found that there was limited evidence for independent associations between nutrient-level dietary factors and anxiety symptoms after controlling for demographic, socio-economic, health, and behavioral factors. Nevertheless, the results require cautious interpretations because the null findings do not mean no relations between diet and anxiety and broader mental health issues. Rather, the results should be interpreted in the way that this study's dietary factors, such as sodium and sugar intake, may have weak independent explanatory power in this cross-sectional population data, which has also been discussed in the literature (9, 26). While this study could not confirm diet-anxiety associations in the population data, several demographic, socio-economic, health, and behavioral factors, including sex, age, household income, healthcare access, health literacy, smoking, drinking, and sleep duration, emerged as critical and consistent predictors of anxiety symptoms in this population. This result emphasizes the idea that anxiety symptoms are not only shaped by individual-level cognitive and biological causes but also largely influenced by broader social and behavioral contexts (21, 27, 28).

This study was conducted based on theoretical expectations that risk dietary factors, including saturated fat, omega-6 fatty acids, sodium, and sugar, would show positive associations with anxiety symptoms, while protective dietary factors, including omega-3 fatty acids and fiber, would show negative associations with anxiety symptoms. Theoretically, a poor diet negatively influences gut diversity, which induces dysregulations in the gut-brain axis and thus can lead to major depression and anxiety (7, 8). However, the regression results did not support this expectation as none of the individual diet risk and protective factors were found to be statistically significant in several models. While the results are inconsistent with theoretical expectations, they are also reasonable because prior research often shows the associations of mental health with overall dietary

patterns. As the present study focused on only a small number of individual diet factors and their collective influence, these individual nutrients may not fully capture the impact of broader dietary habits on mental health documented in the literature (26, 29). Specifically, Hu (29) argues that a single nutrient may underestimate or obscure the impact of diet on mental health because nutrients are often consumed in combinations and their impacts are interconnected, which justifies the limited explanatory power of nutrient-level models. While the current study attempted to capture the impact of overall dietary patterns by building the WDS composite index with available diet variables in the data, even the index may not have been enough to reflect the complex interplay between nutrients and represent the overall diet patterns (26, 29). This suggests that WDS was only a simple nutrient-based score rather than a comprehensive diet-pattern measure.

It should be noted that, regardless of insignificant associations between diet factors and anxiety in the analyses, the gut-brain axis remains an important theoretical framework through which the relationship between diet and mental health can be understood. Also, this study did not aim to verify this mechanism because the KNHANES does not provide data on microbiome, inflammatory, or neurotransmitter biomarkers (10, 11). Gut-brain axis studies already provide rich and plausible biological explanations for the link between diet and mental health, especially through neural, immune, endocrine, microbial, and metabolic pathways (11, 12). The current study's results only demonstrate that biological mechanisms are difficult to translate into population-level associations using cross-sectional survey data. While the biological mechanisms in the literature are primarily based on experimental, animal, and clinical evidence, this study only employed observational survey data in a specific year. Thus, this study neither confirms nor refutes gut-brain axis mechanisms, but it reveals the difficulty of translating biological mechanisms into population-level associations. However, future studies may include biomarkers, microbiome data, and inflammatory markers in the data to clearly test the biological mechanisms at the population level, or design a longitudinal survey to find causal evidence of the relationships between diet and anxiety.

However, this study provides strong empirical evidence for the importance of socioeconomic and health access factors in mental health. The current study broadly supports the fundamental cause theory (30), which suggests that one's socioeconomic status shapes multiple health outcomes by determining the level of access to resources, knowledge, and treatment. Empirically, household income was consistently associated with anxiety symptoms across the models, reinforcing the significance of available resources in determining individual mental health. In addition, private health insurance and unmet medical needs served as strong predictors of anxiety symptoms. This result is consistent with the theoretical explanations that not only material barriers but also the psychological burden that comes from the inability to gain access to treatment and protection can contribute to an increase in anxiety symptoms (27, 30). Although not directly tested in this study, Gundersen & Ziliak (27) discussed that food insecurity could increase anxiety symptoms through psychological mechanisms, which bolsters this study's findings on the relationship between socioeconomic and health access factors and mental health, and partly explains the mechanism through which diet can influence anxiety.

The importance of health literacy is also worth noting. While low socio-economic status and limited health access served as risk factors, health literacy was empirically proven to be a protective factor that can reduce anxiety symptoms. The finding is consistent with the idea that understanding health information may help individuals better detect and interpret symptoms and navigate health-related uncertainty (31). However, this protective factor should also be interpreted as a social determinant of health rather than individual traits because knowledge is often unequally distributed across socio-economic classes (30). In addition, the high explanatory power of health literacy partly explains why diet factors were not significantly associated with anxiety in the study's models. An individual with high health literacy tends to maintain healthy dietary habits and health behaviors, and thus, the independent explanatory power of diet factors can be significantly reduced once health literacy is accounted for (21, 31).

Health-related behaviors also deserve attention. In the analyses, former, occasional, and daily smokers reported significantly higher anxiety symptoms than non-smokers. Furthermore, several drinking-status categories were positively associated with anxiety compared with the never-drinking category. While there can be diverse biological causal mechanisms through which smoking and drinking can cause anxiety, the results should rather be interpreted as the importance of broader behavioral patterns. Existing literature suggests that health behaviors, such as smoking and

physical activities, are closely related to socio-economic status in the way that individuals with low economic status tend to smoke more and engage in less physical activity (21). Furthermore, these behavioral patterns are often clustered with poor dietary habits. Therefore, it is plausible that the strong associations between smoking and drinking behavior and anxiety symptoms could further reduce the explanatory power of individual diet factors in the models. Particularly, the cross-sectional nature of the data cannot verify the causal relationship between smoking, drinking, and anxiety. But the results provide broad support for the relevance of lifestyle factors to anxiety, as explored in the literature (31).

While the previously discussed factors can be clustered into a broad individual socioeconomic profile that may significantly influence mental health, sleep duration is an independent factor with additional implications. In the analyses, weekday sleep duration was found to be negatively associated with anxiety symptoms, meaning that longer sleep was associated with lower anxiety scores. Although sleep duration is part of the broader individual behavioral profile, it is distinct from other significant factors, such as income, health literacy, smoking, and drinking, because it can be determined independently of individual socioeconomic conditions. Thus, while other factors provide a broader social and policy implications into why policy intervention is necessary to improve mental health among low socio-economic classes, the significant result of sleep duration points to the importance of individual lifestyle patterns.

It should be noted that the cultural context of the population may have played a significant role in the associations between diet patterns and anxiety symptoms. While existing literature suggests the general risks of Western dietary habits, this dietary pattern may not work in the same way in the Korean cultural context. For example, high sodium intake can come not only from industrially processed food but also from traditional food in the Korean diet. Thus, even individuals with overall healthy dietary patterns can report high-sodium intake, which may have further reduced the explanatory power of diet factors. Particularly, Yun *et al*. (32) studied the relationship between nutrient intakes, dietary habits, and anxiety symptoms among Korean adults, suggesting that even a dietary profile that is widely considered healthy in the Korean context is closely associated with anxiety symptoms. Furthermore, the widespread fermented food culture in Korea can also obscure the relationship between Western dietary habits and anxiety. For example, Selhub *et al.* (33) argue that fermented foods can often promote the generation of probiotics and microbiota diversity, leading to positive mental health outcomes. However, in the Korean context, fermented foods are often high in salt, and thus their net effect on mental health can be questionable. Thus, future research should clearly distinguish the sources of nutrients, for example, traditional food and Western-type ultra-processed food, to appropriately reflect cultural context in the relationship between diet and anxiety (32, 33). For example, survey questionnaires can be adapted to reflect cultural context and enable control for it in empirical tests (34).

Further limitations in measurement are worth mentioning. First, the survey data are highly prone to recall and reporting biases. Because, in the KNHANES data, the dietary intake was measured through survey-based dietary variables by asking respondents to recall their recent dietary behaviors in measurable numerical terms, it is highly plausible that errors were widely involved in the reporting process (35, 36). Second, social desirability bias may have played a role. As poor dietary habits can be subject to social judgment, the respondents may have underreported their undesirable diet intake and overreported desirable diet intake, which negatively influences the quality of the data (37). In fact, the limitation of dietary assessment in epidemiological studies is widely discussed, particularly when the measurement relies on respondents' recall rather than objective observational measurement (36). These sources of bias in measurement are inherent limitations of the data, which may have contributed to producing non-significant associations in the models. Particularly because individual nutrients may have small associations with anxiety, given a broader set of demographic, socioeconomic, health, and behavioral contributors, even a small measurement error can significantly attenuate the statistical power of the diet variables (35, 37). Thus, future research should use more objective measurements by conducting observational field studies or diary studies. This improvement may help find the population-level evidence for the relationship between diet and anxiety.

The limitation of cross-sectional data should also be mentioned. Because the survey was conducted at a specific time point, the data cannot reveal the temporal order between diet intake and anxiety, and therefore their potential causal relationships. While existing theoretical mechanisms, particularly the gut-brain axis, reveal biological causality between the two, the nature of the cross-sectional data prevented the study from testing the causality. Even if the study found strong associations

between diet intake and anxiety symptoms, the results could not support causality because there could be reverse causality in the empirical tests with cross-sectional data (5, 35). For example, while a poor diet can lead to poor mental health, individuals with poor mental health can engage in a poor diet, conversely. Konttinen *et al*. (38) discuss emotional eating as a mechanism through which psychological distress can shape food choices, which explains the reverse causality. Therefore, future research should employ a longitudinal design of data collection to clarify whether diet influences anxiety or the reverse case is true (5, 38).

Although the current study could not confirm the biological link between diet and anxiety at the population level, it provides rich implications that anxiety prevention largely depends on socio-economic conditions, healthcare access, health literacy, and health behaviors rather than individual diet correction (27, 30). From a broad policy perspective, dietary improvement can still be valuable for public health, but public intervention should integrate dietary intake and broader environmental conditions, including health access, health literacy, smoking, alcohol, and sleep (21, 27, 30, 31). In establishing public health policy, the responsibility for mental health issues should not solely be attributed to individual choices, such as dietary patterns. Rather, it should be recognized that such individual choices are often shaped by and interact with social conditions that individuals cannot easily control. This is where public policy should play an important role.

CONCLUSION

The study aimed to examine whether theoretically predicted associations between diet and anxiety can be confirmed at the population level. While individual nutrient-level diet factors were found to be non-significant, contrary to expectations, this study contributes to the literature and public health policy in several ways. First, this study is the first of its kind to provide population-level evidence for the relationship between diet and anxiety in the Korean context, a non-Western context, by using the KNHANES data. By investigating a non-Western context, this study highlights the importance of considering cultural context and using refined measurements in exploring diet and mental health. Second, rather than providing evidence for the overall dietary effect, this study shows that nutrient-level dietary factors had only limited independent associations with anxiety after controlling for a wide range of demographic, socioeconomic, health, and behavioral factors. This finding helps policymakers decide where to put policy priority in establishing public health policy. Third, this study contributes by clarifying that dietary factors are not isolated determinants of mental health, but rather, they can be shaped by and interact with broader socio-economic context.

As information about diet and health spreads more widely and extensively across online spaces, the topic of diet and mental health is gaining popularity among scholars, policymakers, and the general public. It is undeniable that diet plays a significant role in determining one's mental health, and the widespread adoption of Western dietary habits warrants caution against poor dietary practices. However, such dietary factors or individual choices should not be interpreted in isolation, as doing so can remove the complex social mechanisms that may influence such choices. Thus, while exploring the individual influence of diet is highly valuable in microscopic studies, policymakers and the general public should also pay more attention to social conditions that shape individual choices and, in return, health outcomes.

CONFLICT OF INTEREST

The author declares that there are no conflicts of interest related to this work

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