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Bidirectional association between eating disorder and temporomandibular joint disorder: A retrospective longitudinal nationwide population-based cohort study



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KEYWORDS

Eating disorder; Temporomandibular disorder; Temporomandibular joint disorder; Anorexia nervosa; Bulimia nervosa **Abstract** Background/purpose: An increasing body of evidence indicates correlations between the symptoms of temporomandibular disorder and those of eating disorder (ED). However, further investigation is required to elucidate the temporal and causal relationships between the aforementioned disorders. Materials and methods: This retrospective cohort study was conducted using data from the

Taiwan National Health Insurance Research Database. Temporomandibular joint disorder (TMJD) was analyzed both as the cause and consequence of ED. We collected the data (from January 1, 1998, to December 31, 2011) of patients with antecedent TMJD (N = 15,059) or ED (N = 1219) and their respective controls (1:10), matched by age, sex, income level, residential location, and comorbidities. This study included patients who had received a new diagnosis of an ED or a TMJD between January 1, 1998, and December 31, 2013. Cox regression models were used to assess the risk of ED or TMJD development in patients with antecedent TMJD or ED.

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Results: TMJD patients had an approximately 3.70-fold (95 % confidence interval [CI]: 1.93 –7.10) risk of ED development. Similarly, patients with ED had an approximately 4.78-fold (95 % CI: 2.52–9.09) risk of TMJD development. Subgroup analyses based on ED subtypes indicated antecedent TMJD and bulimia nervosa as the predictors of increased bulimia nervosa and TMJD risks (hazard ratios: 6.41 [95 % CI: 2.91 to 14.11] and 5.84 [95 % CI: 2.75 to 12.41]), respectively.

Conclusion: Previous TMJD and ED are associated with increased risks of subsequent ED and TMJD; these findings suggest the presence of a bidirectional temporal association between TMJD and ED.

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Introduction

Temporomandibular disorders (TMDs) are a set of diseases and disorders characterized by alterations in the structure, function, or physiology of the masticatory system; these disorders may be associated with other systemic and comorbid medical conditions.¹ TMDs are divided into 2 categories: pain-related disorder and temporomandibular joint disorder (TMJD).^{2,3} TMDs have multifactorial complex pathophysiology and have been associated with various risk factors.⁴ Psychological factors are widely known to play important roles in the onset and maintenance of TMJD symptoms.^{4,5} TMJD symptoms have well-known and detrimental psychological consequences. An increasing body of evidence suggests that the associations between TMD and various psychosocial factors are bidirectional^{6,7} and that psychosocial factors contribute to the onset and duration of TMD symptoms.^{6,7}

Eating disorders (EDs), such as anorexia nervosa (AN) and bulimia nervosa (BN), are mental illnesses that are associated with significant morbidity and mortality.⁸ Although AN and BN have overlapping characteristics, which primarily include an excessive preoccupation with body shape and weight, they are distinct disorders.⁹ AN is characterized by persistent caloric restriction, resulting in underweight. AN involves an intense fear of weight gain and a distorted perception of body weight or shape.⁹ BN is characterized by recurrent episodes of excessive food consumption, known as binge eating, followed by compensatory behaviors, such as self-induced vomiting, aimed at preventing weight gain. ED not otherwise specified (ED-NOS) is a residual ED category that is reserved for clinically severe EDs that do not satisfy the diagnostic criteria for AN or BN,¹⁰ and that is later labeled "Other Specified Feeding and Eating Disorders".¹¹ Individuals of any age, sex, ethnicity, socioeconomic background, and weight can develop an ED.¹² Despite the significant psychosocial and physical morbidity and mortality rate associated with ED, patients with ED often conceal or deny their illness and avoid seeking professional help.¹³

Patients with ED often have parafunctional oral habits, such as tongue thrusting during the day and at night and intensive gum chewing.^{14,15} They are more likely to

experience dry or cracked lips, mouth burning, and parotid gland enlargement than are individuals without any ED.¹⁶ These patients also exhibit increased prevalence of dental erosion, attrition and caries, and orofacial symptoms (eg, facial pain and jaw tiredness);¹⁴⁻¹⁷ higher rates of increased size of salivary glands and gingival recessions;¹⁸ a higher sensitivity to muscle palpation;¹⁴ and a reduced mouth opening capacity than do individuals without any ED¹⁵ In a study by Goldberg et al., 60.9 % of all patients with ED were experiencing or had recently experienced some form of facial pain.¹⁹ This finding was supported by Souza et al.²⁰ In addition to masticatory myofascial pain, there was a higher prevalence of pain complaints in other parts of the body in ED patients than in the controls.²⁰ However, whether patients with TMD are susceptible to ED remains unknown. In a study by Gatchel et al., the lifetime prevalence of ED in patients with acute or chronic TMD was 5.9%or 6.0%, which was not higher than the base rate (0.1%) in the general population.^{21,22}

Patients with ED often exhibit the symptoms and signs of TMD. However, whether ED is a cause or consequence of TMD remains unclear. The only studies on this topic have had a cross-sectional design.^{14,15,17,19,20} The temporality of the relationship between ED and TMD must be clarified through a cohort study. Thus, the present retrospective, nationwide population-based cohort study was conducted to investigate the temporal association between ED and TMD. We hypothesized that the relationship between ED and TMJD is bidirectional and that TMJD is both a cause and a consequence of ED.

Materials and methods

The details of the methods are provided in the supplementary materials. In the present study, we analyzed demographic, socioeconomic, clinical, and medical data from the Longitudinal Health Insurance Database of the Taiwan National Health Insurance Research Database (NHIRD) that encompasses >99% of the population's healthcare data, including diagnostic codes (ICD-9-CM). The study obtained Institutional Review Board approval, with informed consent waived due to the use of encrypted, de-identified data.

Study 1: antecedent temporomandibular joint disorder and subsequent eating disorder

The TMJD cohort included individuals diagnosed with TMJD (ICD-9-CM codes: 524.60–524.69) between 1998 and 2011 by dentists. New ED cases (AN, BN, ED-NOS) were diagnosed during follow-up (from enrollment to December 31, 2013) by psychiatrists. Individuals with prior ED diagnoses before enrollment were excluded. Controls were matched for age, sex, income, location, and comorbidities (1:10).

Study 2: precedent eating disorder and subsequent temporomandibular joint disorder

The ED cohort encompassed individuals diagnosed with any ED (AN [ICD-9-CM code: 307.1], BN [ICD-9-CM code: 307.51], and ED-NOS [ICD-9-CM code: $307.5 \times$ except 307.51]) between 1998 and 2011 by psychiatrists. New TMJD cases were diagnosed during follow-up (from enrollment to December 31, 2013) by dentists. Individuals with prior TMJD diagnoses before the enrollment were excluded. Controls were matched for age, sex, income, location, and comorbidities (1:10).

Covariates

Both studies considered potential confounding factors, including medical and psychiatric comorbidities, smoking, Charlson Comorbidity Index (CCI) scores, and all-cause clinical visits. Urbanization level was categorized into 5 levels (from Level 1 [most urbanized] to Level 5 [least urbanized]).

Sample size and power estimation

The optimal sample sizes were 105,202 for Study 1 and 9409 for Study 2.

Statistical analysis

Statistical analyses used F-tests for continuous variables and Pearson chi-square tests for nominal variables. Cox regression models (adjusting for demographics, comorbidities, CCI, and clinical visits) calculated hazard ratios. Significance was set at P < 0.05.

Results

Study 1: antecedent temporomandibular joint disorder and subsequent eating disorder

Study 1 involved 15,059 patients with TMJD and 150,590 matched controls (Table 1). No significant difference was noted in age, sex, comorbidities (including dentofacial anomalies and depressive disorder), income level, or urbanization level between the TMJD and control groups. However, CCI score and all-cause clinical visits were significantly higher in the TMJD group than in the control group.

The incidence of ED in the TMJD group was 1.0 % (15 out of 15,059), which was significantly higher than that in the control group (0.2 % [33 out of 150,590]; P < 0.001). The time to ED development in the TMJD group was significantly shorter than that in the control group (Fig. 1a; log-rank P < 0 0.001). The incidence rate of BN was higher in the TMJD group than in the control group (TMJD group: 0.7 %, n = 11; control group: 0.1 %, n = 16; P < 0.001; Table 1). However, no significant difference was observed in the incidence rate of AN or ED-NOS between the TMJD and control groups. In the TMJD group, the hazard ratio for ED development was 3.70 (CI: 1.93 to 7.10) and for BN development was 6.41 (CI: 2.91 to 14.11; Table 2).

Study 2: precedent eating disorder and subsequent temporomandibular joint disorder

Study 2 involved 1219 patients with ED and 12,190 matched controls (Table 3). No significant difference was noted in age, sex, comorbidities (including dentofacial anomalies and depressive disorder), income level, urbanization level, or all-cause clinical visits between the ED and control groups. However, CCI score was significantly higher in the ED group than in the control group. The ED group had a significantly higher incidence of a subsequent TMJD than did the control group (ED group vs control group: 11.5 % vs 2.5 $\%_{00}$, respectively; P < 0.001). The time-to-TMJD occurrence in the ED group was significantly shorter than that in the control group (Fig. 1b; log-rank P < 0.001). The hazard ratio for TMJD development was 4.78 (CI: 2.52 to 9.09) for individuals with any ED, 5.84 (CI: 2.75 to 12.41) for those with BN, and 6.13 (CI: 2.34 to 16.06) for those with ED-NOS (Table 4).

Discussion

Patients with TMJD exhibited a 3.7-fold increase in the risk of ED, and patients with ED exhibited at a 4.78-fold increase in the risk of TMJD. These findings remained significant even after covariate adjustment. Our results indicate that ED increases the risk of TMJD and vice versa, suggesting a bidirectional association between ED and TMJD.

Notably, >50 % of all patients with ED experience some form of facial pain.^{15,19,20} Patients with ED are more likely to experience facial pain, tiredness in the jaw, increased sensitivity to muscle palpation, and reduced mouth opening capacity than are matched controls.^{14–17} TMD-associated pain, signs, and symptoms were significantly higher among patients with ED than among matched controls.¹⁵ Souza et al. demonstrated that ED was associated with a diagnosis of TMD.²⁰ Our result of a temporal bidirectional association between ED and TMJD is consistent with the findings of the aforementioned cross-sectional studies and indicates that ED and TMJD both precede and result from each other.

The overall incidence rate of AN in Taiwan is 1.1 per 100,000, which is lower than the rate in Western countries^{23,24} Due to the low incidence rate of AN in Taiwan and the strict matching process in the present study, no suitable patients with AN were identified from the Longitudinal Health Insurance Database. The relationship between ED and TMJD, with one increasing the likelihood of the other,

	Individuals with TMJD	Controls	P value	
	(n = 15,059)	(n = 150,590)		
Age at enrollment (year)s, mean \pm SD	37.51 (16.94)	37.51 (16.94)	0.977	
Male, n (%)	4894 (32.5)	48,940 (32.5)	>0.999	
Comorbidities, n (%)				
Dentofacial anomalies	437 (2.9)	4370 (2.9)	0.997	
Diabetes mellitus	1187 (7.9)	11,870 (7.9)	0.999	
Dyslipidemia	2277 (15.1)	22,770 (15.1)	0.999	
Obesity	256 (1.7)	2560 (1.7)	0.997	
Depressive disorder	1328 (8.8)	13,280 (8.8)	0.999	
Alcohol use disorder	245 (1.6)	2450 (1.6)	0.996	
Substance use disorder	236 (1.6)	2360 (1.6)	0.996	
Smoking	222 (1.5)	2220 (1.5)	0.996	
CCI score, mean \pm SD	1.78 (1.99)	1.31 (1.77)	<0.001	
Urbanization level, n (%)			>0.999	
1 (most urbanized)	2387 (15.9)	23,870 (15.9)		
2	3615 (24.0)	36,150 (24.0)		
3	1177 (7.8)	11,770 (7.8)		
4	1022 (6.8)	10,220 (6.8)		
5 (most rural)	6858 (45.5)	68,580 (45.5)		
Income-related insured amount (per month)			>0.999	
≤ NT\$15 840	6733 (44.7)	67,330 (44.7)		
NT\$15 841 to NT\$25 000	3971 (26.4)	39,710 (26.4)		
\geq NT\$25 001	4355 (28.9)	43,550 (28.9)		
ED incidence, n (%)	15 (1.00)	33 (0.22)	<0.001	
Anorexia nervosa	0 (0.0)	0 (0.0)	_	
Bulimia nervosa	11 (0.73)	16 (0.11)	<0.001	
ED not otherwise specified	4 (0.27)	17 (0.11)	0.112	
Age at diagnosis of ED (years), mean \pm SD	39.20 (13.64)	33.99 (12.27)	0.195	
Duration between enrollment and diagnosis (years), mean \pm SD	4.77 (3.97)	6.58 (3.61)	0.127	
All-cause clinical visits (times per year), mean \pm SD	11.97 (12.26)	8.54 (9.63)	<0.001	

Table 1 Demographic characteristics of and ED incidence in patients with this and matched con	Table 1	Demographic charac	cteristics of and ED) incidence in j	patients with I	MJD and	matched cont	irols
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ED: eating disorder; TMJD: temporomandibular joint disorder; SD: standard deviation; NT\$: new Taiwan dollar; CCI: Charlson Comorbidity Index.



Fig. 1 Survival curves for the development of ED in patients with precedent TMJD and matched controls (1a) and for subsequent TMJD in patients with antecedent ED and matched controls (1b). TMJD, temporomandibular joint disorder; ED, eating disorder.



 Table 2
 Risk of a subsequent ED in patients with TMJD and matched controls.^a

	ED		Anorexia nervosa		Bulimia nervosa		ED-NOS	
	N (‰)	HR (95 % CI)	N (‰)	HR (95 % CI)	N (‰)	HR (95 % CI)	N (‰)	HR (95 % CI)
Patients with TMJD	15 (1.0)	3.70 (1.93-7.10)	0 (0.0)	n/a	11 (0.7)	6.41 (2.91-14.11)	4 (0.3)	0.56 (0.09-3.64)
Matched controls	33 (0.2)	1 (reference)	0 (0.0)	1 (reference)	16 (0.1)	1 (reference)	17 (0.1)	1 (reference)

CI: confidence interval; ED: eating disorder; ED-NOS: eating disorder not otherwise specified; HR: hazard ratio; N: number of subject; n/ a: not applicable; TMJD: temporomandibular joint disorder.

^a Cox regression model adjusted for demographic characteristics, physical and mental comorbidities, Charlson Comorbidity Index scores, and all-cause clinical visits. Boldfaced values indicate statistical significance.

was primarily influenced by BN (Tables 2 and 4). BN is characterized by recurrent episodes of binge eating accompanied by inappropriate compensatory behaviors aimed at preventing weight gain.⁹ These compensatory behaviors include self-induced purging; extreme dietary restriction; and misuse of laxatives, diuretics, or other medications.²⁵ Repeated self-induced vomiting is accompanied by sudden and extreme mouth opening, which stretches the temporomandibular joints and masticatory muscles and tendons. Repeated application of minor forces on the stomatognathic apparatus is a form of microtrauma.^{14,26} Frequent self-induced purging can lead to an increased sensitivity to muscle palpation and the development of myofascial symptoms.¹⁴ Additionally, it may cause dislocations or subluxations of the condyle.²⁶ Therefore, frequent self-induced vomiting in individuals with BN may serve as a predisposing, initiating, or perpetuating factor for a TMD.^{14,26,27} Other possible reasons for explaining the bidirectional relationship between BN and TMD may be the co-occurrence of other comorbidities, particularly psychiatric comorbidities. BN and TMD are often accompanied by other mental illnesses.^{6,28} However, in this study, we found that the increased risks of BN and TMJD 'persisted even after adjustments were made for comorbidities related to internal medicine, external medicine, and psychiatry. The analysis also considered the effect of comorbidity loading. This indicates that the bidirectional relationship between BN and TMJD is independent of these comorbidities. Further research is warranted to investigate the causal relationship between BN and TMJD.

The strength of this study lies in its cohort design, which allowed us to investigate the reciprocal relationships between ED and TMJD. Furthermore, we rigorously adjusted for potential confounding factors, encompassing physical and mental comorbidities and pertinent psychosocial variables (eg, income and urbanization levels) in all our analyses. Our study has some limitations. First, because of its retrospective design, our study was susceptible to information and interpretation biases and unable to provide enough evidence to demonstrate a causal relationship between TMJD and ED. To validate the bidirectional associations between ED and TMJD, prospective studies should be conducted using internationally accepted diagnostic systems for both ED and TMJD. A prospective approach that also considers all cause factors of TMJD and ED may minimize information bias and enhance the credibility of findings. Second, TMJD and ED were diagnosed on the basis of ICD-9-CM codes retrieved from the NHIRD, in which disease diagnoses are clinically oriented and driven by treatment needs.²⁹ However, the clinical examinations and diagnostic instruments used for TMJD, as mentioned in the NHIRD, were not standardized or calibrated consistently among examiners across Taiwan's clinics.³⁰ Obtaining standardized diagnostic information for a nationwide population-based cohort study is challenging.³¹ Consequently, the diagnoses of TMJD and ED in our study may differ from and be less reliable than those established in other studies involving questionnaire surveys, self-report analyses, or standardized diagnostic criteria and procedures (e.g., the Research Diagnostic Criteria for Temporomandibular Disorders,

	Patients with ED	Controls	P value			
	(n = 1219)	(n = 12, 190)				
Age at enrollment (years), mean \pm SD	27.98 (9.88)	28.07 (9.95)	0.779			
Male, n (%)	105 (8.6)	1050 (8.6)	0.995			
Types of EDs, n (%)						
Anorexia nervosa	170 (13.9)					
Bulimia nervosa	682 (55.9)					
ED not otherwise specified	367 (30.2)					
Comorbidities, n (%)						
Dentofacial anomalies	10 (0.8)	100 (0.8)	>0.999			
Diabetes mellitus	34 (2.8)	340 (2.8)	0.991			
Dyslipidemia	85 (7.0)	850 (7.0)	0.995			
Obesity	60 (4.9)	600 (4.9)	0.993			
Depressive disorder	908 (74.5)	9080 (74.5)	>0.999			
Alcohol use disorder	52 (4.3)	520 (4.3)	0.993			
Substance use disorder	77 (6.6)	770 (6.6)	0.994			
Smoking	32 (2.6)	320 (2.6)	0.990			
CCI score, mean \pm SD	1.26 (1.46)	1.12 (1.34)	<0.001			
Urbanization level, n (%)			>0.999			
1 (most urbanized)	209 (17.2)	2090 (17.2)				
2	313 (25.7)	3130 (25.7)				
3	81 (6.6)	810 (6.6)				
4	71 (5.8)	710 (5.8)				
5 (most rural)	545 (44.7)	5450 (44.7)				
Income-related insured amount (per month)			>0.999			
\leq NT\$15 840	677 (55.5)	6770 (55.5)				
NT\$15 841 to NT\$25 000	343 (28.2)	3430 (28.2)				
\geq NT\$25 001	199 (16.3)	1990 (16.3)				
Incidence of TMJDs, n (‰)	14 (11.48)	30 (2.46)	<0.001			
Age at TMJD diagnosis (years) mean \pm SD	33.66 (9.72)	37.56 (13.78)	0.346			
Duration between enrollment and diagnosis (years), mean \pm SD	5.07 (3.91)	6.88 (4.52)	0.206			
All-cause clinical visits (times per year), mean \pm SD	10.17 (10.71)	9.61 (9.44)	0.054			

ED: eating disorder; TMJD: temporomandibular joint disorder; SD: standard deviation; NT\$: new Taiwan dollar; CCI: Charlson Comorbidity Index.

Table 4	Risk of a subsequent TMJD in patients with ED or
matched (ontrols. ^a

	TMJD	
	N (‰)	HR (95 % CI)
Patients with ED Anorexia nervosa Bulimia nervosa Eating disorder—not otherwise specified	15 (1.0) 0 (0.0) 9 (13.2) 5 (13.6)	4.78 (2.52–9.09) n/a 5.84 (2.75–12.41) 6.13 (2.34–16.06)
Control group	30 (2.5)	1 (reference)

CI: confidence interval; ED: eating disorder; HR: hazard ratio; N: number of subject; n/a: not applicable; TMJD: temporomandibular joint disorder.

^a Cox regression model adjusted for demographic characteristics, physical and mental comorbidities, Charlson Comorbidity Index scores, and all-cause clinical visits. Boldfaced values indicate statistical significance. Diagnostic Criteria for Temporomandibular Disorders, and Mini International Neuropsychiatric Interview for major psychiatric disorders).^{29,32} Third, the data analyzed in this study primarily comprised medical records rather than data obtained from field surveys. Hence, the information used may not fully represent the general population, which includes individuals who may be reluctant to seek medical assistance or who have less severe conditions than those seeking health-care services. Fourth, the symptomatic profiles of TMJD, including parafunctional behaviors, pain location and intensity, jaw function limitations, and painrelated disability, were not assessable using the NHIRD data. Consequently, we could not establish any correlation between individual TMJD symptoms and ED. Fifth, the size of each ED subgroup was relatively small, which might have inflated effect size estimations and the generalizability of our results. Finally, we did not investigate the effects of treatments or interventions for preexisting TMJD on ED onset or the effects of ED on TMJD onset. The absence of this data might have resulted in the inclusion of patients

who were resistant to standard treatments for TMJD and ED, potentially influencing our findings.

In conclusion, we provided evidence for the coexistence of ED and TMJD and demonstrated that the temporal relationship between these two disorders is bidirectional. Psychiatric care providers must inquire about the presence of TMD symptoms in patients with ED because TMD symptoms may negatively influence ED treatment. Dental professionals should identify the clinical presentations of ED and consider its potential effect when evaluating and treating patients with TMJD.²⁶ The potential bidirectional association between ED and TMJD indicates the need for integrating an ED screening and treatment approach into the protocol for the evaluation and management of patients with TMJD.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jds.2023.11.010.

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