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Insomnia as a risk factor for temporomandibular disorders: a nationwide population-based cohort study

Heon-Young Kim[1†], Jung-Hyun Park[2†], Gwang Hyun Leem[3], Hyo-Jung Lee[4] and Jin-Woo Kim[2*]

Abstract

Objective Temporomandibular disorders (TMD) significantly impact quality of life, and insomnia has emerged as a potential contributing factor. This study aimed to evaluate the association between clinically diagnosed insomnia and the risk of developing TMD using nationwide data from the Korean National Health Insurance Service (NHIS).

Methods We collected baseline data from 2011 to 2014 using the NHIS database and followed participants until 2019. A total of 358,475 adults without previous insomnia or TMD were analyzed, and propensity score matching (1:3) was performed to create balanced insomnia ($n = 20,813$) and non-insomnia ($n = 62,439$) cohorts. Insomnia was defined using diagnostic codes combined with prescription records. Cox proportional hazards models and Kaplan-Meier estimates were employed to evaluate the TMD incidence over a follow-up period ending in 2019.

Results Individuals with insomnia exhibited a significantly higher incidence of TMD (6.46 vs. 4.48 per 1,000 person-years). Even after adjusting for demographic, lifestyle, and clinical variables, insomnia remained a significant independent predictor of TMD, with a 36% increased risk (HR = 1.36; 95% CI: 1.25–1.49). Kaplan-Meier analysis confirmed a consistently higher cumulative incidence of TMD among insomnia patients ($p < 0.001$).

Conclusion Clinically diagnosed insomnia was found to be associated with an increased risk of developing TMD. However, as this study was based on the NHIS database, causality cannot be definitively established. Nonetheless, these findings suggest that incorporating sleep disturbances assessments into the evaluation and management of TMD may be beneficial. Further prospective studies are warranted to clarify this association and assess the potential benefits of integrated care strategies.

Keywords Insomnia, Sleep quality, Temporomandibular joint disorders, Risk factors

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Introduction

Temporomandibular disorders (TMD) are a group of conditions involving pain and dysfunction in the temporomandibular joint (TMJ) and associated muscles. They are recognized as a leading cause of non-odontogenic orofacial pain and significantly impair quality of life [1]. TMD affects approximately 8–15% of the general population [2], with higher prevalence among adults and the elderly [3]. While the etiology of TMD is multifactorial, emerging evidence underscores the substantial contribution of psychosocial and systemic factors to its onset and progression [4–6].

Among psychosocial influences, insomnia has gained increasing attention as a potential contributor to TMD [7–9]. Defined by persistent difficulty initiating or maintaining sleep with associated daytime impairment [10, 11], insomnia affects approximately 10–20% of adults worldwide [12–14]. In South Korea, the prevalence of sleep disorders has nearly doubled over the past decade [15]. Mechanistically, insomnia may increase TMD vulnerability through pathways involving central sensitization, neuroinflammation, and psychological stress dysregulation [8, 9, 16, 17]. These biological and emotional disturbances can heighten pain sensitivity and exacerbate musculoskeletal conditions, including TMD.

Although several studies have reported associations between sleep disturbances and TMD [6, 16, 18, 19], evidence clarifying whether insomnia increases the risk of TMD remains limited. This may be due in part to the predominance of observational studies with cross-sectional designs or small clinical samples, which restrict causal inference and generalizability [20, 21]. In particular, variations in how insomnia is operationalized—often relying on diagnostic codes alone—may contribute to inconsistent findings [2]. In addition, insufficient adjustment for dental conditions or psychological factors may further complicate the interpretation of observed associations.

To address these gaps, this study aimed to evaluate the longitudinal association between clinically diagnosed insomnia and the risk of developing TMD using nationwide data from the Korean National Health Insurance Service (NHIS). Insomnia was defined based on both diagnostic coding and prescription of hypnotic agents to ensure clinical validity. By excluding individuals with prior insomnia, TMD, or related dental conditions, and by applying propensity score matching (PSM), we aimed to isolate the effect of insomnia on subsequent TMD risk. We hypothesized that individuals with clinically diagnosed insomnia would show an increased risk of developing TMD, regardless of demographic, lifestyle, and medical factors.

Materials and methods

Data source

This study utilized data from the Korean NHIS, which operates a mandatory universal healthcare system covering over 97% of the Korean population. The NHIS database contains comprehensive longitudinal information on demographic characteristics, insurance eligibility, healthcare utilization, diagnoses coded according to the International Classification of Diseases, 10th Revision (ICD-10), procedures, prescription records, and mortality data from 2002 [22]. In addition, the NHIS provides a standardized health screening program conducted every 1 to 2 years to all insured adults aged ≥ 40 years and to employed individuals aged ≥ 20 years. The program collects data on anthropometric measures, blood pressure, laboratory tests, and health-related behaviors, including smoking, alcohol consumption, and physical activity.

For this study we used the National Sample Cohort, a population-based cohort established by the Korean NHIS [23]. The cohort consists of a 2% representative sample (approximately one million individuals) of the Korean population in 2002, selected through stratified random sampling by age, sex, region, and insurance type. All enrollees have been followed annually through claims, eligibility, and death records until 2019. This study was reviewed by the Institutional Review Board of Seoul National University Bundang Hospital (IRB number X-2018-705-901), in accordance with the principles of the Declaration of Helsinki. The requirement for informed consent was officially waived by the IRB, as the study used fully anonymized retrospective data from the NHIS in accordance with national regulations.

Study population and cohort selection

We initially identified 511,396 adults aged ≥ 20 years who participated in AT least one national health screening examination between January 1, 2011, and December 31, 2014. The index date was defined as the most recent health screening for each individual. To identify newly diagnosed cases of insomnia and subsequent TMD, we applied the following exclusion criteria: (1) individuals diagnosed with malignant neoplasms (ICD-10 codes C00–C97) from 2002 to 2014; (2) individuals with prior diagnoses of bruxism (F51.8, F45.8), tooth wear (K03.00–K03.19), or tooth fracture (S02.52–S02.59) before the index date; and (3) individuals diagnosed with either insomnia (F51.0 or G47.0) or TMD (K07.6) prior to or during the index year, which was considered the washout period. After exclusions, a total of 358,475 participants remained for the final analysis.

The insomnia group was defined as individuals with at least one medical claim containing the ICD-10 code G47.0 (disorders of initiating and maintaining sleep) or F51.0 (nonorganic insomnia), recorded as either a

principal or additional diagnosis. To enhance diagnostic specificity, individuals were additionally required to have a prescription for hypnotic medications lasting more than seven consecutive days. These criteria were based on prior Korean studies investigating the prevalence and incidence of insomnia [24].

According to the Korean Clinical Practice Guideline for insomnia, 19 pharmacologic treatments are introduced, of which 12 are approved by the Korean Ministry of Food and Drug Safety [25]. To reduce the risk of misclassification, we excluded medications commonly used for other indications such as anxiety, delirium, pain, or depression. In particular, some agents (e.g., antihistamines) listed in the clinical guideline were excluded because they are widely prescribed for non-insomnia indications, such as allergic conditions. Additionally, other agents (e.g., melatonin, eszopiclone) were not yet approved or commercially available in Korea during the cohort inclusion period. As a result, only six hypnotic agents—doxepin, flunitrazepam, flurazepam, triazolam, zolpidem, and trazodone—were included in the analysis [24]. The control group comprised individuals with no recorded diagnosis of insomnia and no prescription history of hypnotics throughout the study period. As of December 31, 2019, 20,876 participants met the criteria for the insomnia group, and 337,599 were assigned to the control group.

To construct a matched cohort, 1:3 PSM was performed. Individuals in the insomnia group were matched to three controls using a greedy nearest-neighbor algorithm with a caliper of 0.2 Matching was based on key baseline covariates, including age, sex, household income, body mass index (BMI), smoking status, and alcohol consumption. After matching, the final cohort comprised 20,813 individuals with insomnia and 62,439 matched controls who were followed for the development of TMD (Fig. 1).

Variable definition and primary outcome

Baseline characteristics, including age, sex, household income, BMI, smoking habits, and alcohol consumption, were assessed as of the index date. Comorbidities were identified using ICD-10 codes recorded within one year prior to the index date. The comorbid conditions included: hypertension (I10–I15), hyperthyroidism (E05.8, E05.9), hypothyroidism (E03.9), diabetes mellitus (E10, E11.8, E11.9, E13, E14.9), dyslipidemia (E78), myocardial infarction (I21, I22, I25.2), stroke (I60–I63), anemia (D46, D50–D53, D55–D64, D74), asthma (J45.0–J45.9), pneumonia (J12–J18), and chronic obstructive pulmonary disease (COPD; J43.1, J43.2, J43.8, J43.9, J44).

The primary outcome was the incidence of TMD, defined as the first occurrence of an outpatient visit with a diagnosis code of K07.6 after the index date, accompanied by a procedure code corresponding to an analytical assessment of TMD (electronic data interchange code: E9040). Participants were followed from the index date until the occurrence of TMD, death, or December 31, 2019, whichever came first. This definition demonstrated

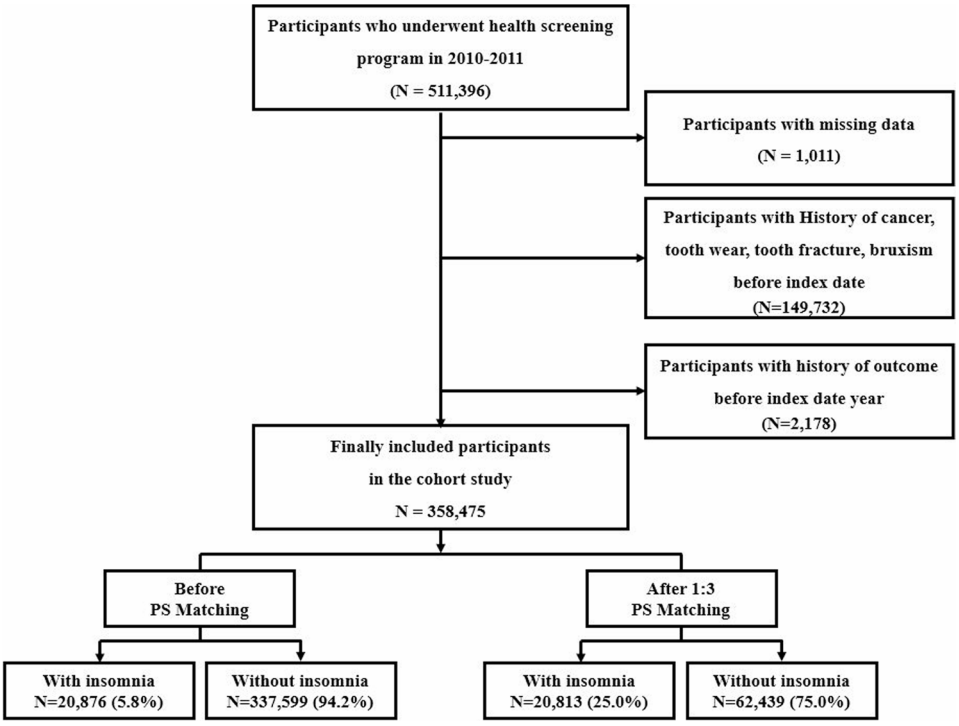


Fig. 1 Flowchart of the study population selection

high diagnostic performance, with a sensitivity of 95.8%, specificity of 92.3%, positive predictive value of 92.0%, negative predictive value of 96.0%, and overall accuracy of 94.0% (see Supplementary Methods for details).

Statistical analyses

We examined baseline characteristics by applying the Student's t-test for continuous variables and the chi-square test for categorical variables to compare the insomnia and control groups. The incidence rate of TMD was estimated as the number of newly diagnosed cases divided by the total person-years (PY) of follow-up. Differences in incidence rates between groups were assessed using the normal approximation test for binomial proportions.

To examine the association between insomnia and the risk of developing TMD, we applied Cox proportional hazards regression models to calculate hazard ratios (HRs) with 95% confidence intervals (CIs). Both unadjusted and multivariable-adjusted models were constructed. Covariates in the adjusted model included age, sex, household income, BMI, smoking status, alcohol consumption, and comorbidities such as hypertension, hyperthyroidism, hypothyroidism, diabetes mellitus, myocardial infarction, stroke, anemia, asthma, pneumonia, and COPD.

To address potential confounding, 1:3 PSM was implemented using a nearest-neighbor algorithm without replacement and with a caliper width 0.2. Covariate balance before and after matching was assessed using standardized mean differences (SMDs), with an SMD < 0.1 considered indicative of adequate balance. The Cox regression analysis was applied to the matched sample to ensure consistency and robustness of the findings.

Kaplan–Meier curves were generated to visualize the cumulative incidence of TMD according to insomnia status. Group differences in survival were tested using the log-rank test. Statistical significance was defined as a two-sided p-values less than 0.05. All analyses were performed using SAS software version 9.4 (SAS Institute Inc., Cary, NC, USA).

We confirmed the proportional hazards assumption for the Cox regression model using Schoenfeld residuals for all covariates, including insomnia. The global test showed no significant violations, and no time-varying effects were observed. The estimated HRs were stable throughout the follow-up period.

Results

Baseline characteristics

Table 1 summarizes the baseline characteristics of participants according to insomnia status. Individuals in the insomnia group were significantly older than those in the control group (mean age 59.07 vs. 47.56 years, $p < 0.001$), and the proportion of women was markedly higher in insomnia group (66.9%) than in the control group ($p < 0.001$). Regarding socioeconomic status, the proportion of individuals receiving medical aid was significantly higher in the insomnia group (3.7%) compared to the control group (1.2%) ($p < 0.0001$).

The distribution of BMI was similar between groups, although minor differences across BMI categories were statistically significant ($p = 0.0273$). Regarding lifestyle factors, the insomnia group had a higher proportion of non-smokers (71.8% vs. 60.0%) and fewer current smokers (17.0% vs. 25.6%) compared to the control group ($p < 0.0001$). Alcohol consumption was more frequent in the insomnia group, particularly in the one to two times per week category (82.3% vs. 71.2%, $p < 0.0001$). Comorbidities such as hypertension, diabetes mellitus, dyslipidemia, anemia, asthma, and COPD were all significantly more prevalent among individuals with insomnia (all $p < 0.0001$). Overall, insomnia was more common among older adults, women, and socioeconomically disadvantaged individuals with a higher burden of chronic illness.

Association between insomnia and TMD risk (before matching)

The relationship between insomnia and TMD incidence in the unmatched sample of 358,475 participants is summarized in Table 2. During the follow-up period, a total of 10,186 TMD cases occurred in the control group and 767 in the insomnia group. The overall incidence rate of TMD was 5.18 per 1,000 PY, with a higher rate observed in the insomnia group compared to controls (6.46 vs. 5.10 per 1,000 PY). The results of the multivariable Cox regression analysis identifying risk factors for TMD are presented in Supplementary Table 1. Insomnia remained a significant independent predictor of TMD (HR: 1.35; 95% CI: 1.25–1.46; $p < 0.001$), even after adjusting for demographic, behavioral, and clinical covariates.

Post-matching characteristics and TMD risk

After PSM, 83,252 participants were included in the matched cohort, comprising 20,813 with insomnia and 62,439 controls. Sociodemographic and lifestyle characteristics were well balanced between groups, with all SMDs below 0.1 (Table 3). The overall incidence rate of TMD in the matched cohort was 4.96 per 1,000 PY. Individuals with insomnia had a higher incidence rate than controls (6.46 vs. 4.48 per 1,000 PY). In the matched sample, the adjusted HR for TMD associated with insomnia was 1.36 (95% CI: 1.25–1.49, $p < 0.001$), confirming a significant and independent association (Table 4). Additional Cox regression results for covariates in the matched cohort are presented in Supplementary Table 2. Findings were consistent with the unmatched analysis.

Table 1 Baseline characteristics of participants according to insomnia diagnosis

Variables	Total (*n* = 358,475)		Insomnia (-) (*n* = 337,599)		Insomnia (+) (*n* = 20,876)		*p*-value
	N or mean	Prop or SD	*N* or mean	Prop or SD	*N* or mean	Prop or SD	
Age	48.23	± 14.52	47.56	± 14.31	59.07	± 13.72	< 0.0001
20–29	37,179	10.4	36,676	10.9	503	2.4	
30–39	62,976	17.6	61,836	18.3	1,140	5.5	
40–49	96,935	27.0	93,602	27.7	3,333	16.0	
50–59	80,078	22.3	74,588	22.1	5,490	26.3	
60–69	46,143	14.5	41,363	14.3	4,780	13.7	
≥ 70	35,164	9.8	29,534	8.8	5,630	27.0	
Sex							< 0.0001
Male	183,279	51.1	176,378	52.2	6,901	33.1	
Female	175,196	48.9	161,221	47.8	13,975	66.9	
Household income							< 0.0001
Insured	353,833	98.7	333,733	98.9	20,100	96.3	
Medical aid	4,642	1.3	3,866	1.2	776	3.7	
BMI	23.85	± 3.44	23.85	± 3.45	23.85	± 3.4	0.0273
< 18.5 (underweight)	14,157	4.0	13,341	4.0	816	3.9	
18.5–22.9 (normal)	131,807	36.8	124,285	36.8	7,522	36.0	
23.0-24.9 (overweight)	85,198	23.8	80,072	23.7	5,126	24.6	
≥ 25.0 (Obese)	127,313	35.5	119,901	35.5	7,412	35.5	
Smoking							< 0.0001
Non-smoker	217,704	60.7	202,708	60.0	14,996	71.8	
Ex-smoking	50,971	14.2	48,631	14.4	2,340	11.2	
Current smoker	89,800	25.1	86,260	25.6	3,540	17.0	
Alcohol							< 0.0001
Non-drinker	77,659	21.7	75,130	22.3	2,529	12.1	
1–2 session/week	257,524	71.8	240,353	71.2	17,171	82.3	
Heavy drinker	23,292	6.5	22,116	6.6	1,176	5.6	
Comorbidity							
Hypertension	79,065	22.1	69,272	20.5	9,793	46.9	< 0.0001
Hyperthyroid	9,103	2.5	7,916	2.3	1,187	5.7	< 0.0001
Hypothyroid	15,364	4.3	13,339	4.0	2,025	9.7	< 0.0001
Diabetes mellitus	33,330	9.3	28,847	8.5	4,483	21.5	< 0.0001
Dyslipidemia	61,057	17.0	52,879	15.7	8,178	39.2	< 0.0001
Myocardial infarction	6,765	1.9	5,684	1.7	1,081	5.2	< 0.0001
Stroke	8,387	2.3	6,707	2.0	1,680	8.1	< 0.0001
Anemia	42,795	11.9	37,521	11.1	5,274	25.3	< 0.0001
Asthma	75,044	20.9	67,333	19.9	7,711	36.9	< 0.0001
Pneumonia	27,237	7.6	23,839	7.1	3,398	16.3	< 0.0001
COPD	11,031	3.1	9,115	2.7	1,916	9.2	< 0.0001

Prop Proportion, *SD* Standard deviation, *COPD* Chronic obstructive pulmonary disease

Table 2 Relationship between insomnia and incidence risk of temporomandibular disorders

	Number of participants	Event rate (%, 95%CI)	Person-years	Incidence rate (per 1,000 PY)	Adjusted HR	*p*-value
All participants	358,475	3.06 (3, 3.11)	2115631.2	5.18		
Insomnia diagnosis						
No	337,599	3.02 (2.96, 3.07)	1996943.2	5.10	(ref)	
Yes	20,876	3.67 (3.42, 3.93)	118688.0	6.46	**1.35 (1.25, 1.46)**	**< 0.0001**

The multivariable model was adjusted for sex, age, household income, BMI, smoking habit, drinking habit, hypertension, hyperthyroid, hypothyroid, diabetes mellitus, myocardial infarction, stroke, anemia, asthma, pneumonia, chronic obstructive pulmonary disease, and insomnia

HR Hazard ratio, *CI* Confidence interval, *PSM* Propensity score matching

Table 3 Relationship between insomnia and incidence risk of temporomandibular disorders after PSM

	Number of participants	Event rate(%, 95%CI)	Person-years	Incidence rate(per 1,000 PY)	Adjusted HR	*p*-value
All participants	83252	2.91 (2.8, 3.03)	488785.6	4.96		
Insomnia diagnosis						
No	62439	2.66 (2.53, 2.78)	370268.0	4.48	(ref)	
Yes	20813	3.68 (3.42, 3.94)	118517.7	6.46	**1.36 (1.25, 1.49)**	**<.0001**

The multivariable model was adjusted for sex, age, household income, BMI, smoking habit, drinking habit, hypertension, hyperthyroid, hypothyroid, diabetes mellitus, myocardial infarction, stroke, anemia, asthma, pneumonia, chronic obstructive pulmonary disease, and insomnia

HR Hazard ratio, *CI* Confidence interval, *PSM* Propensity score matching

Table 4 Baseline characteristics of participants according to insomnia diagnosis after PSM

Variables	Total (*n* = 83,252)		Insomnia (-) (*n* = 62,439)		Insomnia (+) (*n* = 20,813)		SMD
	N or mean	Prop or SD	*N* or mean	Prop or SD	*N* or mean	Prop or SD	
Age	59.16	± 13.51	59.19	± 13.44	59.07	± 13.72	0.009
20–29	1,720	2.1	1,280	2.1	440	2.1	
30–39	4,395	5.3	3,255	5.2	1,140	5.5	
40–49	12,889	15.5	9,556	15.3	3,333	16.0	
50–59	22,111	26.6	16,621	26.6	5,490	26.4	
60–69	19,975	13.5	15,195	13.4	4,780	13.7	
≥ 70	22,162	26.6	16,532	26.5	5,630	27.1	
Sex							0.013
Male	27,175	32.6	20,284	32.5	6,891	33.1	
Female	56,077	67.4	42,155	67.5	13,922	66.9	
Household income							−0.061
Insured	80,736	97.0	60,699	97.2	20,037	96.3	
Medical aid	2,516	3.0	1,740	2.8	776	3.7	
BMI	23.87	± 3.28	23.87	± 3.24	23.86	± 3.39	−0.001
< 18.5 (underweight)	2,802	3.4	2,003	3.2	799	3.8	
18.5–22.9 (normal)	30,057	36.1	22,571	36.2	7,486	36.0	
23.0-24.9 (overweight)	20,776	25.0	15,656	25.1	5,120	24.6	
≥ 25.0 (Obese)	29,617	35.6	22,209	35.6	7,408	35.6	
Smoking							0.035
Non-smoker	60,812	73.1	45,878	73.5	14,934	71.8	
Ex-smoking	8,988	10.8	6,649	10.7	2,339	11.2	
Current smoker	13,452	16.2	9,912	15.9	3,540	17.0	
Alcohol							0.001
Non-drinker	9,978	12.0	7,449	11.9	2,529	12.2	
1–2 session/week	68,733	82.6	51,624	82.7	17,109	82.2	
Heavy drinker	4,541	5.5	3,366	5.4	1,175	5.7	

1:3 ratio propensity score matching (Caliper: 0.2) using age, sex, household income, BMI, smoking, and alcohol consumption. Comorbidities were not included in the matching algorithm

Prop Proportion, *SD* Standard deviation, *BMI* Body mass index, *PSM* Propensity score matching, *SMD* Standardized mean differences

Figure 2 presents the Kaplan–Meier survival curves for TMD according to insomnia status, both before and after PSM. In both analyses, the insomnia group consistently exhibited a higher cumulative incidence of TMD compared to the control group. The group differences were statistically significant in both the unmatched and matched cohorts, as determined by log-rank tests (both $p < 0.001$), reinforcing the observed association between insomnia and TMD across analytic approaches.

Discussion

This study utilized a large, population-based cohort from the Korean NHIS to longitudinally evaluate the relationship between insomnia and the development of TMD. Individuals with insomnia exhibited a consistently higher incidence of TMD than those without, and this difference persisted across various analytic approaches, including multivariable adjustment and PSM. Kaplan–Meier survival analysis also demonstrated a sustained elevation in cumulative incidence among individuals with insomnia, supporting a temporal association between the two conditions.

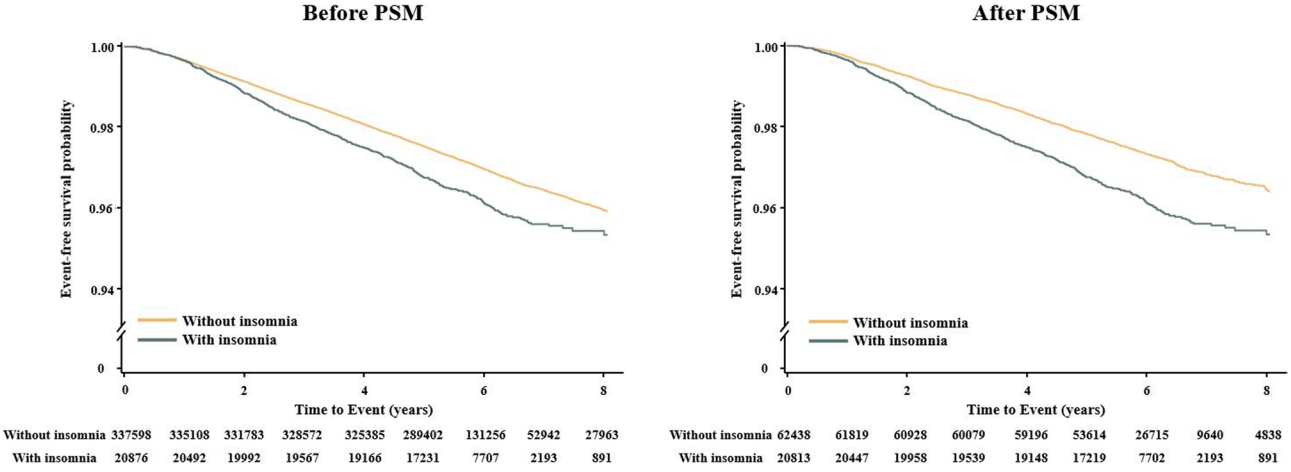


Fig. 2 Cumulative incidence of temporomandibular disorder according to insomnia stats. Kaplan–Meier survival curves comparing the cumulative incidence of temporomandibular disorder between the insomnia and non-insomnia groups. The insomnia group showed a significantly higher incidence over the follow-up period (log-rank test, $p < 0.0001$)

Compared to previous studies that relied solely on diagnostic codes [2], our study employed stricter criteria to identify clinically meaningful insomnia by combining ICD-10 codes with at least one week of continuous hypnotic prescription. We also excluded participants with bruxism, tooth wear, and tooth fractures to reduce diagnostic overlap and reverse causality [26, 27]. The association between insomnia and TMD remained robust across multiple analytic approaches, including multivariable adjustment and PSM.

Notably, a prior nationwide cohort study that reported an increased TMD risk among individuals with sleep disorders [2] relied on broader diagnostic definitions and included a much smaller insomnia population ($n = 6{,}999$), without incorporating treatment information or dental exclusions. In contrast, our study applied stricter diagnostic criteria for both insomnia and TMD, excluded confounding dental conditions, and analyzed over 20,000 individuals with clinically defined insomnia. Despite this narrower definition, our findings were consistent with previous reports [2, 7, 9, 21, 28] and are further strengthened by the large sample size, which enhances statistical power and generalizability.

Mounting evidence suggests that insomnia may contribute to the development and maintenance of TMD through complex interactions between neurophysiological, inflammatory, and psychological mechanisms. One key mechanism is central sensitization, a state of increased excitability in central nociceptive pathways. Sleep disruption may contribute to the development of central sensitization by impairing descending pain inhibitory mechanisms and increasing neural excitability, which can lower pain thresholds and enhance vulnerability to chronic pain such as TMD [29–32]. Another important mechanism is systemic inflammation. Sleep restriction and poor sleep quality are linked to elevated pro-inflammatory markers such as C-reactive protein, IL-1β and IL-6 [33, 34]. These cytokines enhance pain signaling both peripherally and centrally, and have been associated with increased pain severity in TMD patients [35, 36]. Finally, psychological factors may mediate the relationship between insomnia and TMD. Insomnia is a well-known risk factor for depression and anxiety [37, 38], both of which are common in chronic TMD and are associated with symptom amplification [39]. Disrupted sleep can increase stress reactivity, muscle tension, and emotional dysregulation, all of which may further contribute to TMD progression [34, 40–45]. These findings support the role of insomnia as both a potential initiator and perpetuator of TMD-related pain, acting through neural, inflammatory, and psychological dysregulation. Although these mechanisms were not directly measured in our dataset, they are considered plausible hypotheses based on prior literature. Nevertheless, neurophysiological, inflammatory, and psychological alterations associated with insomnia may plausibly heighten pain sensitivity and influence the pathophysiology of musculoskeletal pain conditions, including TMD.

The prevalence of insomnia is steadily increasing worldwide, contributing to a growing public health burden. In the United States, the proportion of adults experiencing sleep disorders rose from approximately 20% in 2011 to 41.7% in 2016 [15], while in South Korea, the prevalence of clinically diagnosed insomnia nearly doubled from 9.4% in 2011 to 17.3% in 2020 [15]. In this context, although the adjusted HRs and absolute incidence of TMD observed in our study were modest, the rising prevalence of insomnia underscores its clinical significance. Previous studies have reported similar trends. For instance, a nationwide cohort study found

a 44% increased risk of TMD among individuals with sleep disorders, with incidence rates of 6.08 versus 3.27 per 10,000 PY [2]. Another study similarly showed that insomnia more than doubled the risk of developing TMD (OR = 2.05) [28].

Taken together, these findings highlight the importance of recognizing the clinical and public health relevance of insomnia. As a modifiable condition with established treatment options—including cognitive behavioral therapy, pharmacologic interventions, and multidisciplinary care—screening for sleep problems may help improve the diagnosis and management of TMD. Nonetheless, sleep disturbances remain underrecognized and undertreated in dental and orofacial pain care. Integrating sleep-focused evaluation into the standard assessment of TMD may facilitate early identification of high-risk individuals and support more comprehensive treatment strategies. This approach may be particularly valuable for patients with complex pain patterns, comorbid psychiatric conditions, or poor response to conventional TMD therapies.

While these results highlight important mechanistic insights and clinical opportunities, they must be interpreted in light of several limitations. First, as this study was based on health insurance claims data, we could not assess insomnia or TMD using objective or standardized tools such as polysomnography, actigraphy, sleep diaries, or validated questionnaires. Likewise, specific clinical information for subclassifying TMD (e.g., disc displacement, masticatory muscle pain, degenerative joint disease) or characterizing the severity and duration of insomnia was unavailable due to limitations of the claims database. In addition, although we defined TMD using both diagnostic (K07.6) and relevant procedural codes to improve diagnostic specificity, patients who did not seek care may not have been captured, potentially leading to an underestimation of true TMD prevalence. Further validation studies and sensitivity analyses are needed to evaluate the diagnostic performance of this definition. Second, our definition of insomnia required at least one week of hypnotic prescriptions in addition to diagnostic codes. While this approach enhances clinical validity, it may have excluded certain subgroups of patients—such as those with mild or subthreshold insomnia symptoms who did not seek medical attention, or those contraindicated for hypnotic medications due to comorbidities or adverse effects. As a result, the prevalence of insomnia may have been conservatively estimated and the included cohort may have been biased toward more severe or pharmacologically managed cases. Third, although we applied strict inclusion and exclusion criteria and adjusted for a wide range of demographic, behavioral, and medical covariates, residual confounding cannot be entirely ruled out. Despite these adjustments, unmeasured factors—such as genetic, psychological, and environmental determinants or chronic pain conditions like fibromyalgia and complex regional pain syndrome—may have influenced the observed association [46]. In addition, due to the limited diagnostic granularity of claims data, we could not account for prior trauma (e.g., whiplash or jaw injuries). Trauma potentially affecting the TMJ is diverse, and applying blanket exclusions based on diagnostic codes may lead to misclassification or over-exclusion, given the lack of reliable information on trauma timing and severity. Sleep apnea, which may influence both insomnia and TMD, was not covered under Korea's health insurance benefits until 2018 and therefore could not be reliably identified or excluded. As a result, individuals with undiagnosed or uncoded sleep apnea may have been included in the cohort. Fourth, the impact of insomnia treatment could not be evaluated. The Korean NHIS database does not contain specific codes for cognitive behavioral therapy for insomnia, which is a first-line treatment recommendation. Lastly, this study was conducted in a Korean population, where stigma surrounding mental disorders may lead to underreporting or avoidance of care, resulting in underdiagnosis in claims data [47]. This gap between actual prevalence and recorded diagnoses has been documented in Korea [48]. Additionally, in the National Sample Cohort, some sensitive diagnostic codes related to mental disorders are masked at the subcategory level, limiting full identification or exclusion of related conditions. We recommend that future research employing more granular clinical records further investigate the potential causal relationship between insomnia and TMD development.

Conclusion

This large-scale population-based cohort study found that clinically diagnosed insomnia was significantly associated with the subsequent development of TMD. Although the use of NHIS claims data limits causal inference, these findings underscore the importance of paying clinical attention to sleep-related complaints in dental and oral and maxillofacial care. Incorporating sleep assessments may help identify patients at risk of TMD. Further prospective studies with detailed clinical evaluations of sleep and pain are warranted to clarify the nature of this relationship and inform preventive strategies.

Abbreviations

TMD	Temporomandibular disorders
TMJ	Temporomandibular joint
NHIS	National Health Insurance Service
ICD	International Classification of Diseases
IRB	Institutional Review Board
PSM	Propensity score matching
BMI	Body mass index
COPD	Chronic obstructive pulmonary disease
PY	Person-years
HR	Hazard ratio

CI Confidence interval
SMD Standardized mean difference

Supplementary Information

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Supplementary Material 1.

Supplementary Material 2.

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Not applicable.

Authors' contributions
H-YK and J-HP were responsible for the initial study conception, design, interpretation, and drafting the manuscript. GHL conducted data acquisition and analysis, and provided significant revisions to the manuscript. H-JL contributed to the overall review of the manuscript and provided general input during manuscript development. J-WK played a key role in the conception and design of the study, contributed to data collection and interpretation, and provided critical feedback during the revision process. All authors reviewed and approved the final manuscript and confirmed their accountability for the work in its entirety.

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Data availability
The data used in this study are available from the National Health Insurance Service (NHIS) database; however, restrictions apply to the public availability of the data used under the license for the current study. Access to the NHIS database for research purposes is granted through official approval, and de-identified data are available upon request from a government agency via the National Health Insurance Sharing Service homepage (http://nhiss.nhis.or.kr).

Declarations

Ethics approval and consent to participate
This study was reviewed and approved by the Institutional Review Board of Seoul National University Bundang Hospital (approval number: X-2108-705-901). All the procedures adhered to the guidelines and principles outlined in the Declaration of Helsinki. The requirement for informed consent was waived because this study used anonymous retrospective data.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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