Influence of sleep disturbances on temporomandibular disorders, somatic, and negative emotional symptoms in young adults: A cross-sectional study

Carolina D Marpaung[1,2*]
Ni Luh Dewi[1]
Yenny Pragustine[1]
Isya Hanin[1]
Hani Yuliasari[1]
Enrita D Rahmadini[3]

[1]Department prostodonsia Faculty of Dentistry, Universitas Trisakti, Indonesia
[2]Department of Orofacial Pain and Dysfunction, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and Vrije Universiteit Amsterdam, Amsterdam, the Netherlands
[3]Departement Pediatric Dentistry, Faculty of Dentistry,, Universitas Trisakti, Indonesia

***Correspondence**
Email | carolina@trisakti.ac.id

Submitted | 11 Februari 2026
Revised | 24 March 2026
Accepted | 28 April 2026
Published | 30 April 2026
DOI: 10.24198/jkg.v38i1.66392

p-ISSN 0854-6002
e-ISSN 2549-6514

Citation | Marpaung CD, Dewi NL, Pragustine Y, Hanin Y, Yuliasari H, Rahmadini ED. Influence of sleep disturbances on temporomandibular disorders, somatic, and negative emotional symptoms in young adults: A cross-sectional study. J. Kedokt. Gigi Univ. 2026;38(1):61-71.
DOI: 10.24198/jkg.v38i1.66392

ABSTRACT

Introduction: While temporomandibular disorders (TMDs), sleep disturbances, and psycho-emotional symptoms are interrelated, it remains unclear whether sleep disturbances are a contributing factor to TMDs or a result of chronic pain and somatic symptoms. The objective of this study was to analyze the influence of sleep disturbances on TMD, somatic, and negative emotional symptoms in young adults. **Methods**: A cross-sectional study was conducted on young adults from a private university in Indonesia. Sleep disturbances were assessed using the Pittsburgh Sleep Quality Index (PSQI), TMD symptoms using the quintessential five TMD symptoms (5Ts), and somatic symptoms using the Patient Health Questionnaire-15 (PHQ-15). Psychological distress and coping strategies were measured using the Depression, Anxiety, Stress Scales-21 (DASS-21) and the Brief Coping Orientation to Problems Experienced Inventory (Brief-COPE). Data were analyzed using the Chi-square test, Kruskal-Wallis, Spearman correlation, and logistic regression (p=0.05). **Results**: Among the 456 participants (mean age 22.4 ± 1.5 years), 58.2% reported TMD symptoms. Participants with high sleep disturbance had significantly higher TMD pain, somatic symptoms, dysfunctional coping, and psychological distress compared to those with low sleep disturbance. Somatic symptoms and dysfunctional coping were moderately correlated with general distress, anxiety, and stress. Multivariate analyses indicated that the odds of sleep disturbance was increased by somatic symptoms (OR=1.18;95% CI=1.12-1.24) and stress (OR=1.10;95% CI=1.04-1.17). **Conclusion**: Sleep disturbances are not only linked to TMD but also contribute to increasing the severity of somatic symptoms and psycho-emotional symptoms.

Keywords

psychological distress, sleep disturbance, somatic symptoms, temporomandibular joint disorder, negative emotional symptoms.

Pengaruh gangguan tidur terhadap gangguan temporomandibula, somatik, dan gejala emosional negatif pada dewasa muda: sebuah studi potong lintang

ABSTRAK

Pendahuluan: *Gangguan temporomandibular (TMD), gangguan tidur, dan gejala psiko-emosional saling berkaitan, namun masih belum jelas apakah gangguan tidur merupakan faktor yang berkontribusi terhadap TMD atau justru merupakan akibat dari nyeri kronis dan gejala somatik. Tujuan dari penelitian ini adalah menganalisis pengaruh gangguan tidur terhadap gangguan temporomandibula, somatik, dan gejala emosional negative pada dewasa muda.* ***Metode:*** *Partisipan dari studi potong lintang ini merupakan dewasa muda yang direkrut dari sebuah universitas swasta di Indonesia. Gangguan tidur dinilai menggunakan Pittsburgh Sleep Quality Index (PSQI), gejala TMD dengan lima gejala utama TMD (5Ts), dan gejala somatik menggunakan Patient Health Questionnaire-15 (PHQ-15). Tekanan psikologis dan strategi koping diukur menggunakan Depression, Anxiety, Stress Scales-21 (DASS-21) dan Brief Coping Orientation to Problems Experienced Inventory (Brief-COPE). Analisis data dilakukan dengan uji Chi-square, Kruskal-Wallis, korelasi Spearman, dan regresi logistik (α=0,05).* ***Hasil:*** *Sebanyak 58,2% dari 456 partisipan melaporkan mengalami gejala TMD (usia rata-rata 22,4 ± 1,5 tahun). Partisipan dengan tingkat gangguan tidur yang tinggi menunjukkan tingkat nyeri TMD, gejala somatik, koping disfungsional, dan tekanan psikologis yang secara signifikan lebih tinggi dibandingkan dengan mereka yang memiliki gangguan tidur rendah. Gejala somatik dan koping disfungsional menunjukkan korelasi sedang dengan tekanan psikologis umum, kecemasan, dan stres. Analisis multivariat menunjukkan bahwa kemungkinan terjadinya gangguan tidur meningkat seiring dengan meningkatnya gejala somatik (OR = 1,18; 95% CI = 1,12–1,24) dan stres (OR = 1,10; 95% CI = 1,04–1,17).* ***Simpulan:*** *Gangguan tidur tidak hanya berkaitan dengan TMD, tetapi juga berkontribusi terhadap peningkatan keparahan gejala somatik dan psiko-emosional.*

Kata kunci

Tekanan psikologis, gangguan tidur, gejala somatik, gangguan sendi temporomandibula, emosi negative.

INTRODUCTION

Sleep disturbances among young adults have emerged as a significant public health concern, with studies showing an increase in insomnia, irregular sleep patterns, and diminished sleep quality within this demographic.[1] Epidemiological studies have indicated that approximately 40% of young adults encounter clinically significant sleep-related difficulties, which are further correlated with academic pressures, occupational demands, and extensive technology use.[2,3] The high correlation between inadequate sleep and mental health issues has also been demonstrated within this demographic, which is already predisposed to elevated negative psychological factors, including anxiety and stress.[4]

Pain, as defined by the International Association for the Study of Pain (IASP), is "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.[5] This definition highlights the multifaceted nature of pain, which extends beyond a mere physiological reaction to encompass a complex phenomenon involving biological, psychological, and social (biopsychosocial) determinants. Psychosocial factors significantly influence the neurochemistry of pain, particularly among young adults, by modulating the functionality of essential neural pathways and neurotransmitter systems integral to pain processing. Furthermore, psychosocial determinants play a pivotal role in influencing pain perception by affecting emotional, cognitive, and behavioral responses to pain.[6,7]

Negative emotional factors have been recognized for their potential to increase pain sensitivity and significantly contribute to the onset of chronic pain syndromes. These psychological states augment neural activity within the brain's pain-processing regions.[6] Chronic pain often results in persistent activation of the nervous system, which may precipitate central sensitization—a pivotal mechanism that underlies nociplastic pain.[8] Central sensitization is defined by an increased sensitivity of the central nervous system (CNS) to nociceptive stimuli, resulting in an heightened perception of pain, even in the absence of tissue damage.[9]

Moreover, the continuing nature of pain may lead to somatization, in which psychological distress is expressed through physical manifestations. Chronic pain can induce elevated levels of anxiety and depression, which may augment bodily awareness and exacerbate the perception of somatic symptoms, including fatigue, dizziness, or gastrointestinal discomfort. This interplay may establish a feedback loop wherein chronic pain and psychological distress mutually reinforce each other, thereby further exacerbating both somatization and nociplastic pain.[10] As a result, many young adults report recurrent somatic symptoms that drive frequent healthcare use and functional impairment even when no clear cause is found.

Temporomandibular disorders (TMDs) encompass a range of conditions that affect the temporomandibular joint (TMJ), the masticatory muscles, and associated anatomical structures. These disorders are characterized by discomfort in the jaw joint and surrounding tissues, restricted jaw movement, and joint sounds, such as clicking or popping, that occur during jaw activities.[11] The etiology of TMDs is complex and multifactorial, encompassing biomechanical, psychological, and genetic components.[11] Recent investigations have increasingly highlighted the bidirectional relationship between TMDs and psychological variables, including stress, anxiety, and depression, which may intensify pain perception and contribute to the persistence of symptoms.[12]

Moreover, sleep disturbances are commonly reported among TMD patients, indicating a multifaceted interaction among pain, sleep, and psychological health.[13] The question remains, particularly among Indonesian young adults, whether sleep disturbances are direct contributors to the onset of TMDs or if they emerge as a consequence of chronic pain and somatic symptoms. The hypotheses were: (a) Young adults with sleep disturbance would have significantly greater TMD, somatic, and psycho-emotional symptoms severity. (b) Sleep disturbances are correlated with TMD, somatic, and psycho-emotional symptoms;

and (c) The presence of TMD, somatic, and psycho-emotional symptoms increases the odds of sleep disturbance. Therefore, the objective of this study was to analyze the influence of sleep disturbances on TMD, somatic, and negative emotional symptoms in young adults.

METHODS

This observational study recruited young adults using a convenience sampling approach at a private university in Indonesia during September 2024 to March 2025. Eligible participants were university students aged 18-24 years who demonstrated proficiency in the English language, as verified by their academic transcript showing a minimum English grade of B and provided informed consent. We excluded those with a documented history of orofacial trauma or orthognathic surgical procedures, as well as individuals currently receiving treatment for significant physical and/or mental health disorders.

Recruitment announcements were disseminated via the university intranet and complemented by in-person invitations at the University clinic. The announcement described the study aims, procedures, estimated time commitment, data confidentiality, and emphasized that participants were voluntary and participants could withdraw at any time without consequences. Interested students enrolled through the provided link and completed online consent before accessing the questionnaire. The online document included demographic details, Pittsburgh Sleep Quality Index (PSQI), the quintessential five TMD symptoms (5Ts), Patient Health Questionnaire-15 (PHQ-15), Depression, Anxiety, Stress Scales-21 (DASS-21), and the short version of Coping Orientation to Problems Experienced Inventory (Brief-COPE).

A minimum of 363 subjects was deemed essential for the research. This figure was calculated using an online sample size calculator, considering a 95% confidence interval, a 5% margin of error, a student demographic comprising 20,800 individuals, and an estimated prevalence of 60% for TMD symptoms among young adults, as established in prior studies. TMD symptoms were evaluated a 30-day period using the 5Ts screening tool, which was derived from the Symptom Questionnaire (SQ) of the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD).[11,14] The items in the 5T screening tool include two pain-related symptoms (TMJ/masticatory muscle pain and headache) and three non-pain TMD dysfunctions (TMJ sounds, closed locking, and open locking). Participants were categorized as "5Ts-negative" if they responded negatively to all five items and as "5Ts-positive" if they responded positively to any of the five items. The "5Ts-positive" group was further subdivided into individuals with TMD pain and TMD dysfunction.

Somatic symptoms were assessed over a 30-day period using the PHQ-15 instrument. The PHQ-15 consists of fifteen items associated with significant somatic symptom manifestations, which represent the expression of psychological distress through somatic symptoms.[15] These items encompass bodily pain, fatigue, gastrointestinal issues, cardiopulmonary concerns, and various other complaints. They are graded on a 3-point scale, where the options are "not bothered at all" (0 points), "bothered a little" (1 point), and "bothered a lot" (2 points). Total PHQ-15 scores are calculated, with higher scores indicating greater somatic symptom burden or somatization.

Psychological distress was assessed with the Depression, Anxiety, and Stress Scale-21 (DASS-21), which comprises seven items pertaining to each of the subscales for depression, anxiety, and stress.[16] The DASS-21 has good measurement properties, and responses are rated on a 4-point scale, ranging from "did not apply to me at all" (0 points) to "applied to me very much or most of the time" (3 points). The depression, anxiety and stress subscales have been shown to demonstrate good reliability, with coefficients above 0.80. Rasch analysis supported the unidimensionality of each subscale, with explained raw

variance ranging from 53.9% to 60.2%.[16] Subscale scores are calculated, with higher scores indicating higher depression, anxiety, and stress levels.

The coping styles and techniques were analyzed using the Brief Coping Inventory (BCI), which includes twenty-eight items that reflect the extent to which participants utilize fourteen different coping strategies when encountering daily life stressors.[17] The items are rated on a four-point Likert scale, ranging from 'I haven't been doing this at all' (corresponding to 1 point) to 'I've been doing this a lot' (equating to 4 points).

The fourteen strategies for coping are divided into three major coping styles, namely: problem-focused coping (which includes active coping, instrumental support, and planning), emotion-focused coping (which entails acceptance, emotional support, humor, positive reframing, and religion), and dysfunctional coping (which includes behavioral disengagement, denial, self-distraction, self-blame, substance use, and venting).

While the scores for the fourteen coping strategies were derived by summing the scores for their respective two designated questions, the adjusted scores for the various coping styles were computed by aggregating the item scores and dividing the total by the number of questions involved. Both sets of scores pertain to habitual coping orientations, with elevated scores signifying a more extensive utilization of specific coping strategies or styles. The quality and disturbances of sleep were assessed over a 30-day period using the Pittsburgh Sleep Quality Index (PSQI), which comprises a total of 19 items.[18] The PSQI is psychometrically sound, and items are generally graded on a 4-point scale, ranging from "not during the past month/very good" (0 points) to "three or more times a week/very bad" (3 points). Total and component Pittsburgh Sleep Quality Index (PSQI) scores are calculated according to established protocols, whereby elevated scores denote poorer overall sleep quality and an increase in sleep-related dysfunction, respectively.

A cumulative PSQI score of 5 points or greater indicates the presence of suboptimal sleep.[19] In this study, participants were categorized as having good sleep quality (low disturbance) when the global PSQI score was <5, and poor sleep quality (high disturbance) when the global PSQI score was ≥ 5. This dichotomization followed the standard PSQI cutoff for clinically relevant sleep disturbance.[18-19]

Statistical analyses were conducted using SPSS software Version 27.0 (IBM Corporation, Armonk, New York, USA), with a significance threshold set at 0.05. Qualitative data were represented as frequencies along with proportions, and assessed using the Chi-square test. Quantitative data were presented as means with standard deviations (SDs), along with medians and interquartile ranges (IQRs), and assessed for normality using the Shapiro-Wilk test. Missing data were evaluated for extent and patterns; cases with missing outcome data were excluded, and the remaining analysis used complete cases (listwise deletion) because missingness was low (<5%). Given their non-normal distribution, the Kruskal-Wallis test and post-hoc Mann-Whitney U tests, as well as Spearman's rank-order correlation, were utilized.

The strength of correlation coefficients (r_s) was designated as weak, moderate, or strong based on predetermined cut-off thresholds of 0.1, 0.4, and 0.7. Univariate and multivariate logistic regression analyses were conducted to ascertain the factors influencing sleep disturbance. In the multivariate model, factors that did not achieve significance were excluded through a stepwise variable selection approach with a threshold of $p<0.10$. Stepwise selection was used to obtain a parsimonious model and reduce overfitting given the number of candidate predictors relative to events. The results were represented as odds ratios (ORs) along with their associated 95% confidence intervals (95% CIs).

RESULTS

Out of the 456 eligible young adults who consented to participate, 92 were excluded due to incomplete data. Consequently, the final sample comprised 364 participants, with a

mean age of 22.4±1.5 years. While 41.8% (152/364) of the participants were "5Ts-negative" (TN), 58.2% (212/364) were "5Ts-positive" (TP). The HS group exhibited significantly elevated levels of TMD pain, as did the TMD mixed groups (Table 1).

Table 1. Demographic characteristics of the study population.

Variables	Total	Low sleep disturbance (LS)	High sleep disturbance (HS)	P-value*
Total n (%)	364 (100)	182 (50.0)	182 (50.0)	-
Age				0.065
Mean (SD)	22.38 (1.46)	22.49 (1.42)	22.27 (1.48)	
Median (IQR)	22.00 (3.00)	23.00 (2.00)	22.00 (2.25)	
Presence of TMDs n (%)	212 (58.2)	95 (52.2)	117 (64.3)	**0.019**
TMD Pain n (%)	63 (17.3)	29 (46.0)	34 (54.0)	**0.024**
TMD Dysfunction n (%)	71 (19.5)	35 (49.3)	36 (50.7)	0.070
TMD Mixed n (%)	78 (21.4)	31 (39.7)	47 (60.3)	**0.019**

*SD = Standard deviation; IQR = Interquartile range. Results of *Mann-Whitney U and ^Chi-square tests. Bold indicates P <0.05.*

Table 2. Mean and median physical and psycho-emotional symptom scores for participants with low and high sleep disturbance.

Physical and psycho-emotional variables	Low sleep disturbance (LS)	High sleep disturbance (HS)	P-value*	Differences
TMD symptoms				
TMD pain				
Mean (SD)	1.31 (0.54)	1.68 (0.73)	0.032	LS<HS
Median (IQR)	1.00 (1.00)	2.00 (1.00)		
TMD dysfunction				
Mean (SD)	1.57 (1.00)	1.47 (1.03)	0.262	-
Median (IQR)	1.00 (1.00)	1.00 (0.00)		
TMD Mixed				
Mean (SD)	3.61 (1.43)	4.04 (2.22)	0.703	-
Median (IQR)	4.00 (2.00)	3.00 (3.00)		
Somatic symptoms				
Total PHQ-15				
Mean (SD)	4.65 (4.10)	9.45 (6.03)	<0.001	LS<HS
Median (IQR)	3.00 (5.00)	8.50 (9.00)		
Coping methods				
Problem focused				
Mean (SD)	17.90 (3.54)	17.51 (2.97)	0.170	-
Median (IQR)	18.00 (4.00)	18.00 (4.00)		
Emotion				
Mean (SD)	28.10 (5.21)	27.92 (4.34)	0.566	-
Median (IQR)	28.00 (7.00)	28.00 (6.00)		
Dysfunction				
Mean (SD)	23.05 (4.14)	24.42 (4.36)	0.005	LS<HS
Median (IQR)	23.00 (6.00)	24.00 (6.00)		
Negative emotional symptoms				
Total-DASS (TD)				
Mean (SD)	10.56 (8.74)	16.45 (9.80)	<0.001	LS<HS
Median (IQR)	9.00 (11.25)	15.00 (14.00)		
Depression (DD)				
Mean (SD)	3.26 (4.91)	5.07 (5.31)	<0.001	LS<HS
Median (IQR)	1.50 (4.00)	3.00 (5.00)		
Anxiety (AD)				
Mean (SD)	3.44 (3.02)	5.42 (3.46)	<0.001	LS<HS
Median (IQR)	3.00 (4.00)	5.00 (5.00)		
Stress (SD)				
Mean (SD)	4.99 (3.98)	7.54 (3.85)	<0.001	LS<HS
Median (IQR)	5.00 (5.00)	7.50 (5.00)		

*SD = Standard deviation; IQR = Interquartile range. Results of *Mann-Whitney U test. Bold indicates P <0.05.*

Table 2 displays the mean and median TMD symptoms, somatic symptoms, coping methods, and psychological distress scores among individuals with low (LS) and high (HS) sleep disturbance. TMD pain, somatic symptoms, dysfunctional coping strategies, and all negative emotional symptoms scores were higher in the HS group compared to the LS.

Table 3 presents the correlations between the physical and psycho-emotional variables. Somatic symptoms were moderately correlated with TMD pain, general distress, anxiety, and stress (r_s=0.439-0.467). Similarly, dysfunctional coping was moderately correlated with general distress, anxiety, and stress (r_s = 0.405-0.465). Table 4 shows the outcomes of univariate and multivariate logistic regression analyses.

Univariate analysis identified significant association between sleep disturbance and TMD pain (OR=1.43; 95% CI=1.15-1.78), TMD mixed (OR=1.19; 95% CI =1.05-1.35), somatic symptoms (OR=1.21; 95% CI=1.15-1.27), general distress (OR=1.07;95% CI =1.05-1.10), depression (OR=1.08; 95% CI=1.03-1.13), anxiety (OR=1.21; 95% CI= 1.13-1.30), stress (OR = 1.18; 95% CI = 1.12-1.25), and dysfunctional coping (OR = 1.08; 95% CI = 1.03-1.14). However, multivariate analysis identified somatic symptoms (OR = 1.18; 95% CI = 1.12-1.24) and stress (OR = 1.10; 95% CI = 1.04-1.17) as the primary risk factors of high sleep disturbance.

Table 3. Correlations between physical and psycho-emotional variables.

Variables	TPain	TDys	TMix	TP	TD	DD	AD	SD	PC	EC	DC
TPain	-										
TDys	0.298**	-									
TMix	**0.776****	**0.788****	-								
TP	**0.440****	0.125*	0.353**	-							
TD	0.154**	0.087	0.152**	**0.448****	-						
DD	0.056	0.012	0.049	0.362**	**0.633****	-					
AD	0.239**	0.120*	0.213**	**0.467****	**0.876****	**0.483****	-				
SD	0.139**	0.070	0.134*	**0.439****	**0.924****	**0.600****	**0.791****	-			
PC	0.047	-0.052	-0.017	0.053	0.043	-0.076	0.097	0.059	-		
EC	0.040	-0.012	0.014	0.097	0.058	-0.054	0.115*	0.063	**0.733****	-	
DC	0.128*	0.026	0.089	0.348**	**0.465****	0.382**	**0.405****	**0.428****	0.341**	0.346**	-

*TPain = TMD Pain; TDys = TMD Dysfunction; TMix = TMD Mixed; TP = Total Physical Symptom PHQ15; TD = Total DASS-21; DD = Depression; AD = Anxiety; SD = Stress; PC= Problem Coping; EC= Emotional Coping; DC= Dysfunctional Coping, Results of Spearman's correlation. *indicates p <0.05, ** indicates p <0.01, and Bold indicates correlation coefficient >0.4.*

Table 4. Physical and psycho-emotional variables associated with sleep disturbance.

	Univariate		Multivariate	
Variables	**Odds ratio (95% CI)**	**P-value***	**Odds ratio (95% CI)**	**P-value^**
TMD Pain	1.43 (1.15 – 1.78)	**0.001**		
TMD Dys	1.14 (0.94 – 1.38)	0.192		
TMD Mixed	1.19 (1.05 – 1.35)	**0.006**		
Total PHQ-15 (TP)	1.21 (1.15 – 1.27)	**<0.001**	1.18 (1.12 – 1.24)	**<0.001**
Total DASS (TD)	1.07 (1.05 – 1.10)	**<0.001**		
Depression	1.08 (1.03 – 1.13)	**0.001**		
Anxiety	1.21 (1.13 – 1.30)	**<0.001**		
Stress	1.18 (1.12 – 1.25)	**<0.001**	1.10 (1.04 – 1.17)	**0.002**
Dysf COPE (TC)	1.08 (1.03 – 1.14)	**0.003**		

*Results of *univariate and ^multivariate logistic regression analyses. Bold indicates P <0.05.*

DISCUSSION

This study identified several significant associations between sleep disturbance, TMD symptoms, somatic symptom burden, coping strategies, and negative emotional symptoms among young adults. More than half of the participants screened positive for at least one TMD symptom. Participants with high sleep disturbance had a significantly higher frequency of TMD symptoms and mixed TMD presentations than those with low sleep disturbance, as shown in Table 1.

Subjects with high sleep disturbance showed higher scores for TMD pain, somatic symptoms, dysfunctional coping strategies, and psycho-emotional factors as shown in Table 2. Interestingly, TMD dysfunction scores did not differ significantly between the low and high sleep disturbance groups, suggesting that sleep disturbance in this sample was more closely related to the pain component of TMD than to non-pain dysfunction symptoms. Numerous studies have shown that sleep plays a critical role in modulating pain perception and managing physical symptoms, with growing evidence highlighting its bidirectional relationship with pain.[13,21,22]

Poor sleep quality can heighten pain sensitivity by altering neural pathways involved in nociception, such as increasing activity in the amygdala and reducing prefrontal cortex regulation. In contrast, chronic pain often disturbs sleep, creating a vicious cycle that exacerbates both conditions.[21] Furthermore, sleep deprivation has been shown to lower pain thresholds and increase the severity of physical symptoms, such as fatigue and musculoskeletal discomfort.[22] The present findings are consistent with previous reports of an association between subjective sleep disturbance and painful TMD; however, the cross-sectional design does not allow determination of whether sleep disturbance preceded TMD pain or resulted from it.

Sleep is a fundamental biological process that influences somatic symptoms, coping strategies, and emotional regulation. Studies have consistently demonstrated that insufficient or disrupted sleep exacerbates somatic complaints, including chronic pain, fatigue, headaches, and gastrointestinal disturbances.[23] The use of PHQ-15 in this study was based on the common use of this tool to detect common somatic symptoms that contribute to conditions like somatic symptom disorder (SSD), depression, and anxiety.[15, 24]

In addition, the participants with high sleep disturbance reported significantly higher TMD pain, somatic symptom burden, dysfunctional coping, depression, anxiety, and stress scores, although TMD dysfunction, problem-focused coping, and emotion-focused coping did not differ significantly between groups (Table 2).

Correlation analyses further demonstrated that somatic symptoms were moderately correlated with TMD pain and negative emotional symptoms, whereas dysfunctional coping was moderately correlated with psychological distress, anxiety, and stress (Table 3). In the final multivariable model, somatic symptom burden and stress remained independently associated with high sleep disturbance (Table 4). Somatic symptoms were also closely related to TMD pain, negative emotional factors, and dysfunctional coping strategies (see Table 3). This relationship might be due to the central sensitization mechanism in chronic pain, where prolonged nociceptive input and psychological distress lead to maladaptive neuroplastic changes, resulting in heightened pain sensitivity, widespread somatic symptoms, and emotional dysregulation.[9]

This was also indicated in previous studies showing higher central sensitization to be closely associated with psychological distress in patients with chronic orofacial pain.[25] Moreover, longitudinal data have shown that pre-existing psychological distress and increased nociceptive sensitivity can drive transitions into chronic orofacial pain states, consistent with maladaptive neuroplastic changes.[26] Nevertheless, central sensitization was not directly assessed in the present study; therefore, this mechanism should be considered a possible explanation rather than a demonstrated finding.

This study also indicated that negative emotional factors, namely depression, anxiety,

and stress, are closely linked to dysfunctional coping strategies (e.g., catastrophizing, avoidance) through maladaptive cognitive and neurobiological mechanisms as shown by the significant correlation in Table 3. In particular, dysfunctional coping showed moderate correlations with general psychological distress, anxiety, and stress, indicating that less adaptive coping responses were more apparent among participants with greater emotional symptom burden. Chronic emotional distress impairs cognitive-emotional regulation by diminishing prefrontal cortex control over limbic system activity, particularly the amygdala, thereby promoting maladaptive responses to stressors.[6]

This dysregulation is further compounded by hypothalamic-pituitary-adrenal (HPA) axis dysfunction, wherein prolonged cortisol elevation perpetuates emotional distress and reinforces ineffective coping patterns.[27] Consequently, a cyclical relationship emerges wherein negative emotions foster dysfunctional coping, which in turn exacerbates emotional and physical distress. However, because dysfunctional coping did not remain in the final multivariable model, it should be interpreted as a related feature of psychological burden rather than an independent factor associated with sleep disturbance in this sample.

Sleep has also been shown to play a crucial role in maintaining emotional stability and enhancing the effectiveness of coping strategies. Sleep disturbances may negatively impact cognitive functions, including attention, memory, and executive functioning, which are vital for adaptive coping abilities.[28] Individuals experiencing poor sleep are more likely to engage in maladaptive coping strategies, such as avoidance or emotional suppression, rather than utilizing proactive and effective approaches.[29]

Additionally, sleep deprivation is strongly associated with negative emotional states, including increased anxiety, depressive symptoms, and emotional dysregulation, which further compromise coping efficacy and exacerbate somatic symptom severity, as demonstrated by a longitudinal study.[29] Consistent with these findings, the high sleep disturbance group in the present study reported significantly higher dysfunctional coping, depression, anxiety, and stress scores than the low sleep disturbance group (Table 2). Among these variables, stress remained significantly associated with high sleep disturbance in the final multivariable model (Table 4).

Temporomandibular disorders (TMD), like other musculoskeletal pain conditions, can significantly disrupt sleep quality by causing persistent discomfort and altering sleep architecture.[30] In this study, participants with high sleep disturbance showed a significantly higher frequency of overall TMD symptoms and mixed TMD presentations than those with low sleep disturbance (Table 1). The significant difference in mixed TMD presentations may indicate that participants experiencing both pain and dysfunction represent a group with greater overall symptom burden. Patients often experience difficulty falling asleep, frequent awakenings, and reduced restorative sleep stages, including deep sleep and REM sleep.

This disturbance may be caused by the activation of the sympathetic nervous system by nociceptive signals, leading to an increase in arousals and preventing the body from achieving a relaxed state conducive to sleep.[31] Moreover, the bidirectional relationship between TMD pain and sleep disturbances creates a vicious cycle. Poor sleep quality can lower pain thresholds and increase pain sensitivity, making the pain feel more intense and further disrupting sleep.[21] As the present study used self-reported sleep disturbance rather than objective sleep assessment, changes in sleep architecture cannot be confirmed from these data.

Dysfunctional coping strategies refer to maladaptive ways of managing stress or emotional distress, such as avoidance, denial, substance use, or excessive rumination, which often exacerbate rather than alleviate the underlying problem.[17] These strategies are particularly relevant to sleep disturbances, as they can increase psychological arousal and emotional distress, making it more difficult to fall asleep or stay asleep. TMD pain and dysfunctional coping strategies are closely interrelated, as individuals experiencing chronic pain often adopt maladaptive coping mechanisms to manage their discomfort.[32]

In the present study, dysfunctional coping scores were significantly higher among

participants with high sleep disturbance and were correlated with TMD pain, somatic symptoms, and negative emotional symptoms (Tables 2 and 3). However, dysfunctional coping was significant only in the univariate analysis and was not retained in the final multivariable model (Table 4). This finding suggests that its relationship with sleep disturbance may be partly explained by the accompanying somatic symptom burden and stress.

The findings in Table 4 indicate that somatic symptoms and psychological distress were the variables that remained independently associated with high sleep disturbance in the final multivariable model. Although TMD pain, mixed TMD symptoms, depression, anxiety, total psychological distress, and dysfunctional coping were associated with high sleep disturbance in the univariate analyses, these variables were not retained in the final model. This suggests that somatic symptom burden and stress may account for an important part of the relationship between sleep disturbance and the other physical and psycho-emotional symptoms observed in this sample.

Somatic manifestations, particularly musculoskeletal pain, activate nociceptive pathways that disrupt sleep continuity by increasing microarousals and altering sleep architecture through thalamocortical dysregulation. Concurrently, stress-related physiological arousal may contribute to prolonged sleep latency and poorer perceived sleep quality.[23] However, these possible mechanisms were not directly examined in this study, and the findings should therefore be interpreted as associations rather than causal pathways.

Notwithstanding these findings, several limitations should be acknowledged when interpreting the results. This study included university students, a population characterized by high negative emotional factors despite relatively low pain severity.[2] This demographic characteristic may potentially influence the observed relationship between psychological distress and somatic symptoms. While the association between pain and negative emotional factors is well established in clinical populations, the current study design restricts the generalizability of this relationship.

In addition, the cross-sectional design prevents conclusions regarding the temporal or causal direction of the associations among sleep disturbance, TMD symptoms, somatic symptoms, and negative emotional symptoms. The use of convenience sampling from a single private university, together with the English-language eligibility criterion, may further limit the generalizability of the findings to other young adult populations in Indonesia. All variables were assessed using self-reported screening instruments; therefore, the findings reflect symptom burden rather than clinically confirmed TMD diagnoses or objectively measured sleep disturbances. Moreover, 92 consenting participants were excluded because of incomplete data, which may have introduced selection bias. Finally, additional variables that may influence sleep quality and TMD symptoms, such as sex, body mass index, caffeine consumption, screen exposure, academic workload, and diagnosed sleep disorders, were not examined in the present analysis.

Several limitations of this study relate to the cross-sectional nature. Reliance of self-reported measures and voluntary recruitment of students preclude causal inferences and limit the generalizability of the results to other young-adult population. Consequently, future studies should evaluate these relationships in more diverse young adult populations and among patients with clinically confirmed TMD pain. Longitudinal research is also needed to determine whether sleep disturbance precedes increases in TMD pain, somatic symptom burden, and stress, or whether these symptoms contribute to subsequent deterioration in sleep quality.

CONCLUSION

Sleep disturbances in young adults are not only related to TMD but also to an increased occurrence of somatic symptoms and psycho-emotional symptoms. This suggests a

multifaceted relationship in which sleep issues may exacerbate both physical and emotional health problems. Moreover, somatic symptoms and stress were shown to increase the odds of sleep disturbance. The association between sleep disturbances, TMD, and psychological symptoms in this study indicates that addressing sleep disturbances could have a positive impact on mitigating both physical and psychological symptoms, ultimately leading to improved overall health and quality of life among young adults. Implications of this study are guidance the identification of sleep disturbances may facilitate a more comprehensive understanding of the patient's symptom burden and support management approaches that consider concurrent somatic and psychological symptoms. Future longitudinal studies are warranted to clarify the direction and clinical relevance of these relationships.

Acknowledgement : None

Author Contribution: Conceptualization, C.M and N.L.D.; methodology, C.M., N.L.D., and Y.P; software, C.M and Y.P.; validation, C.M., N.L.D., Y.P., and I.H.; formal analysis,,C.M., N.L.D. , and Y.P. ; investigation, C.M and N.L.D.; resources, C.M and N.L.D.; data curation, C.M and N.L.D; writing—original draft preparation, C.M.; writing—review and editing, C.M., N.L.D., Y.P., I.H.; visualization, C.M., H.Y., and N.L.D.; supervision, C.M.; project administration, H.Y. and E.R.; funding acquisition, C.M. and Y.P. All authors have read and agreed to the published version of the manuscript.

Funding: This study was funded by Universitas Trisakti with grant number: 0142/PUF/FKG/2021-202.

Ethical Approval: This research was carried out in accordance with the Helsinki declaration, and was approved by the Ethics Committee for health research at Faculty of Dentistry, Universitas Trisakti (No: 031/S3/KEPK/FKG/6/2023, date of approval: June 8th, 2023).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study

Data Availability Statement: Research data are available upon request to the corresponding author.

Conflicts of Interest: The authors declare no conflict of interest.

REFERENCES

1. Bruce ES, Lunt L, McDonagh JE. Sleep in adolescents and young adults. Clin Med (Lond). 2017;17(5):424–428. https://doi.org/10.7861/clinmedicine.17-5-424.
2. Al Salmani AA, Al Shidhani A, Al Qassabi SS, Al Yaaribi SA, Al Musharfi AM. Prevalence of sleep disorders among university students and its impact on academic performance. *Int J Adolesc Youth*. 2020;25(1):974–81. https://doi.org/10.1080/02673843.2020.1815550.
3. Li Y, Bai W, Zhu B, Duan R, Yu X, Xu W, et al. Prevalence and correlates of poor sleep quality among college students: a cross-sectional survey. *Health Qual Life Outcomes*. 2020;18(1):210. https://doi.org/10.1186/s12955-020-01465-2.
4. Clement-Carbonell V, Portilla-Tamarit I, Rubio-Aparicio M, Madrid-Valero JJ. Sleep quality, mental and physical health: a differential relationship. *Int J Environ Res Public Health*. 2021;18(2):460. https://doi.org/10.3390/ijerph18020460.
5. Raja SN, Carr DB, Cohen M, Finnerup NB, Flor H, Gibson S, et al. The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. *Pain*. 2020;161(9):1976–82. https://doi.org/10.1097/j.pain.0000000000001939.
6. Edwards RR, Dworkin RH, Sullivan MD, Turk DC, Wasan AD. The role of psychosocial processes in the development and maintenance of chronic pain. J Pain. 2016;17(9 Suppl):T70–92. https://doi.org/10.1016/j.jpain.2016.01.001.
7. Cheatle MD. Biopsychosocial approach to assessing and managing patients with chronic pain. Med Clin North Am. 2016;100(1):43–53. https://doi.org/10.1016/j.mcna.2015.08.007.
8. Kosek E, Cohen M, Baron R, Gebhart GF, Mico JA, Rice ASC, et al. Do we need a third mechanistic descriptor for chronic pain states? *Pain*. 2016;157(7):1382–6. https://doi.org/10.1097/j.pain.0000000000000507.
9. Curatolo M. Central sensitization and pain: pathophysiologic and clinical insights. Curr Neuropharmacol. 2024;22(1):15–22. https://doi.org/10.2174/1570159X20666221012112725.
10. Löwe B, Toussaint A, Rosmalen JGM, Huang WL, Burton C, Weigel A, et al. Persistent physical symptoms: definition, genesis, and management. Lancet. 2024;403(10444):2649–2662. https://doi.org/10.1016/S0140-6736(24)00623-8.
11. Schiffman E, Ohrbach R. Executive summary of the Diagnostic Criteria for Temporomandibular Disorders for clinical and research applications. J Am Dent Assoc. 2016;147(6):438–445. https://doi.org/10.1016/j.adaj.2016.01.007.
12. Slade GD, Ohrbach R, Greenspan JD, Fillingim RB, Bair E, Sanders AE, et al. Painful temporomandibular disorder: decade of discovery from OPPERA studies. J Dent Res. 2016;95(10):1084–1092. https://doi.org/10.1177/0022034516653743.
13. Sanders AE, Akinkugbe AA, Bair E, Fillingim RB, Greenspan JD, Ohrbach R, et al. Subjective sleep quality deteriorates before development of painful temporomandibular disorder. *J Pain*. 2016;17(6):669–77. https://doi.org/10.1016/j.jpain.2016.02.004.
14. Yap AU, Zhang MJ, Zhang XH, Cao Y, Fu KY. Viability of the quintessential five temporomandibular disorder symptoms as a screener. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2022;133(6):643–9. https://doi.org/10.1016/j.oooo.2021.11.009.
15. Hybelius J, Kosic A, Salomonsson S, Wachtler C, Wallert J, Nordin S, Axelsson E. Measurement properties of the Patient Health Questionnaire-15 and Somatic Symptom Scale-8: a systematic review and meta-analysis. JAMA Netw Open. 2024;7:e2446603. https://doi.org/10.1001/jamanetworkopen.2024.46603.
16. Lee J, Lee EH, Moon SH. Systematic review of the measurement properties of the Depression Anxiety Stress Scales–21 by applying

updated COSMIN methodology. Qual Life Res. 2019;28(9):2325–2339. https://doi.org/10.1007/s11136-019-02177-x.
17. Huda N, Lin YK, Shaw MK, Hsu YY, Chang HJ. Psychometric properties and cross-cultural adaptation of the Indonesian version of the Brief COPE in a sample of advanced cancer patients. PLoS One. 2022;17(11):e0275083. https://doi.org/10.1371/journal.pone.0275083.
18. Setyowati A, Chung MH, Yusuf A, Haksama S. Validity and reliability of the Indonesian version of the Pittsburgh Sleep Quality Index in adolescents. Int J Nurs Pract. 2021;27(5):e12856. https://doi.org/10.1111/ijn.12856.
19. Mollayeva T, Thurairajah P, Burton K, Mollayeva S, Shapiro CM, Colantonio A. Pittsburgh sleep quality index as screening tool: a meta-analysis. *Sleep Med Rev*. 2016;25:52–73. https://doi.org/10.1016/j.smrv.2015.01.009.
20. Akhtar P, Ma L, Waqas A, Naveed S, Li Y, Rahman A, et al. Prevalence of depression among university students in low and middle income countries (LMICs): a systematic review and meta-analysis. J Affect Disord. 2020;274:911–919. https://doi.org/10.1016/j.jad.2020.03.183
21. Lavigne GJ, Sessle BJ. Neurobiology of orofacial pain and sleep interactions. *J Dent Res*. 2016;95(10):1109–16. https://doi.org/10.1177/0022034516648264.
22. Klasser GD, Almoznino G, Fortuna G. Sleep and orofacial pain. *Dent Clin North Am*. 2018;62(4):629–56. https://doi.org/10.1016/j.cden.2018.06.005.
23. Herrero Babiloni A, De Koninck BP, Beetz G, De Beaumont L, Martel MO, Lavigne GJ. Sleep and pain: mechanisms and insights. *J Neural Transm*. 2020;127(4):647–60. https://doi.org/10.1007/s00702-019-02067-z.
24. Yap AU, Kurniawan F, Pragustine Y, Marpaung C. Temporomandibular disorder and somatic symptoms. *Acta Odontol Scand*. 2024;83:340–7. https://doi.org/10.2340/aos.v83.40776.
25. Sewerin P, Waliszewska-Prosol M, Petrasova A, Bort M, Seweryn M, Straburzynski M, et al. Central sensitisation, anxiety and depressive symptoms in chronic masticatory pain. *J Oral Rehabil*. 2025. https://doi.org/10.1111/joor.70007.
26. Ohrbach R, Slade GD, Bair E, Rathnayaka N, Diatchenko L, Greenspan JD, et al. Premorbid and concurrent predictors of TMD onset and persistence. Eur J Pain. 2020;24(1):145–158. https://doi.org/10.1002/ejp.1472.
27. Vachon-Presseau E, Centeno MV, Ren W, Berger SE, Tétreault P, Ghantous M, et al. Emotional brain in chronic pain. *J Dent Res*. 2016;95(6):605–12. https://doi.org/10.1177/0022034516638027.
28. Griggs S, Harper A, Hickman RL Jr. A systematic review of sleep deprivation and neurobehavioral function in young adults. Appl Nurs Res. 2022;63:151552. https://doi.org/10.1016/j.apnr.2021.151552.
29. Otsuka Y, Itani O, Matsumoto Y, Kaneita Y. Coping strategies and insomnia. *Sleep*. 2022;45(2):zsab244. https://doi.org/10.1093/sleep/zsab244.
30. Slade GD, Ohrbach R, Greenspan JD, Fillingim RB, Bair E, Sanders AE, et al. Painful TMD: OPPERA studies. *J Dent Res*. 2016;95(10):1084–92. https://doi.org/10.1177/0022034516653743.
31. Berger M, Vakulin A, Hirotsu C, Marchi NA, Solelhac G, Bayon V, et al. Association between sleep microstructure and incident hypertension in a population-based sample: the HypnoLaus study. J Am Heart Assoc. 2022;11(14):e025828. https://doi.org/10.1161/JAHA.121.025828.
32. Yap AU, Marpaung C. Psychological distress and coping in TMD. *Acta Odontol Scand*. 2023;81(6):456–63. https://doi.org/10.1080/00016357.2023.2172076.
33. Yap AU, Marpaung C. Psychological factors in TMD and somatization. *Int J Prosthodont*. 2024;37(6):605–14. https://doi.org/10.11607/ijp.8590