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Prevalence of Sleep Disturbance and Its Association With Low Back Pain Among Students of Gulab Devi Educational Complex

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ABSTRACT

Background: *Sleep disturbance is increasingly recognized as a modifiable determinant of pain chronification, particularly in young adults exposed to academic and ergonomic stressors. Disrupted sleep alters pain modulation and inflammatory pathways, potentially aggravating low back pain (LBP) and related disability.* ***Objective:*** *To determine the prevalence of insomnia and its association with LBP severity among undergraduate students at Gulab Devi Educational Complex, Lahore.* ***Methods:*** *A cross-sectional study was conducted among 194 students (aged 18–28 years) using the Insomnia Severity Index (ISI), Oswestry Disability Index (ODI), and Visual Analogue Scale (VAS). Associations were analyzed using chi-square and Cramer's V, while odds ratios (OR) with 95% confidence intervals (CI) quantified the relationship between LBP severity and clinical insomnia (ISI≥15).* ***Results:*** *Insomnia was highly prevalent (64.9%), increasing progressively with LBP severity (χ^2=39.92, p<0.001; Cramer's V=0.232). Clinical insomnia was present in 9.1% of mild, 30.9% of moderate, and 61.5% of severe LBP cases. Compared with mild LBP, the odds of clinical insomnia were 4.30 (95% CI 1.90–9.72) for moderate and 14.72 (95% CI 4.16–52.10) for severe LBP. Female students reported higher insomnia rates (p=0.0318).* ***Conclusion:*** *Insomnia is common and significantly associated with greater LBP severity in students. Integrating sleep assessment and early non-pharmacologic interventions may reduce pain burden and improve student well-being.*

Keywords
Insomnia; Low Back Pain; Sleep Disturbance; Students; Oswestry Disability Index; Cross-sectional Study

INTRODUCTION

Sleep disturbance—particularly short sleep duration and insomnia—modulates neurobiological systems governing nociception, mood, and cognition, thereby plausibly amplifying pain processing in vulnerable populations such as university students (1). Clinical and epidemiologic work shows a robust bidirectional relationship between poor sleep and chronic pain states, with sleep loss increasing pain sensitivity and pain disrupting sleep continuity (2). Low back pain (LBP) is the leading cause of years lived with disability globally and affects young adults at substantial rates, including those in higher education where prolonged sitting, suboptimal ergonomics, and heavy screen exposure are common (3,4).

Mechanistic syntheses implicate inflammatory activation, impaired descending inhibition, and central sensitization as conduits linking sleep fragmentation and hyperalgesia (5). Longitudinal data indicate that baseline sleep quality strongly predicts subsequent pain intensity among individuals with recent-onset LBP, suggesting a prognostic role for sleep health in pain trajectories (6).

Genetic evidence from bidirectional Mendelian randomization further supports a causal interplay between insomnia phenotypes and LBP, underscoring the clinical salience of targeting sleep in musculoskeletal care (7). In student cohorts worldwide, estimates of poor sleep vary widely but consistently indicate high burden, while musculoskeletal complaints—particularly lumbar pain—are prevalent and clinically meaningful (8,9). In Pakistan, emerging studies attribute student sleep problems to academic stressors, sedentary study patterns, and inconsistent sleep hygiene, yet institution-specific evidence linking sleep disturbance to LBP remains sparse (10). College-based analyses identify the lumbar region as a frequent pain site among students with insomnia, and multi-year cohort data demonstrate co-occurrence of poor sleep with heightened musculoskeletal symptoms and deteriorations in mental health, plausibly mediating the sleep–pain nexus (11,12).

Non-pharmacological sleep interventions can reduce pain and disability in chronic LBP, strengthening the case for integrated screening and management that address both domains (13). Against this background, we investigated, within a single clinical-academic campus in Lahore, the prevalence of insomnia and its association with LBP burden among undergraduate students. We hypothesized that insomnia (per Insomnia Severity Index thresholds) would be common and positively associated with greater LBP severity and disability (assessed by Visual Analogue Scale and Oswestry Disability Index) in this student population (13).

MATERIALS AND METHODS

We conducted a cross-sectional observational study at the Gulab Devi Educational Complex, Lahore, Pakistan, enrolling undergraduate students aged 18–28 years who reported LBP symptoms during the study period. Eligibility included both sexes and all academic years; exclusions were known neurological disorders, prior spinal or abdominal surgery, traumatic fractures, pathological spinal conditions, or systemic diseases affecting musculoskeletal status. Participants were approached on campus using non-probability convenience sampling; trained assessors obtained written informed consent and administered standardized instruments in a structured session.

Sleep disturbance was measured using the Insomnia Severity Index (ISI), applying established cut points to define categories: 0–7 none, 8–14 subthreshold, 15–21 clinical moderate, and 22–28 clinical severe (18). Pain intensity was assessed with a 10-cm Visual Analogue Scale (VAS), and functional disability with the Oswestry Disability Index (ODI), following recommended scoring and interpretation conventions for LBP outcomes (22).

Core variables included demographics (age, sex, academic year, residence status), department/discipline, and LBP severity categories based on ODI bands; insomnia categories were defined per ISI as above. To reduce measurement bias, validated instruments were used with standardized instructions and uniform administration; assessors were trained to minimize prompting and to check completeness at the point of data entry (18,22).

The sample size (n=194) was determined a priori using Cochran's formula with an assumed LBP prevalence of 44.9%, 7% margin of error, and 95% confidence, targeting adequate precision for prevalence estimation and contingency analyses. Data were entered in SPSS v26 with double-check verification to ensure integrity.

Descriptive statistics summarized the cohort; inferential analyses used Fisher's exact or Pearson's chi-square as appropriate for contingency tables, with effect sizes reported as Cramer's V for multi-category associations. For clinical interpretability, insomnia was also dichotomized (clinical insomnia: ISI≥15 vs non-clinical: ISI≤14) to estimate odds ratios (OR) and 95% confidence intervals (CI) comparing LBP severity groups to the mild LBP reference; where zero cells occurred, Haldane–Anscombe correction (0.5) was applied. Two-sided α=0.05 defined statistical significance; exact p values are reported where available, and multiplicity was addressed by focusing inference on prespecified associations (LBP×insomnia, sex×insomnia, department insomnia).

Ethical approval was granted by the Gulab Devi Educational Complex Research Ethical Board, and all procedures adhered to the Declaration of Helsinki; informed consent, anonymization, and secure data handling procedures were implemented to support reproducibility and participant confidentiality (22).

RESULTS

A total of 194 students (mean age 21.8 ± 2.1 years; 75.3% female) participated in the study, with complete datasets across all measures and no attrition. Overall, 64.9% of students exhibited some degree of insomnia as measured by the Insomnia Severity Index (ISI), with 42.8% subthreshold, 20.6% moderate, and 1.5% severe cases. Mild, moderate, and severe disability due to low back pain (LBP) were recorded in 51.0%, 41.8%, and 7.2% of participants, respectively.

A significant association was observed between insomnia severity and LBP category ($\chi^2 = 39.92$, $p < 0.001$), corresponding to a moderate effect size (Cramer's V = 0.232). Clinical insomnia (ISI ≥ 15) increased progressively from 9.1% among students with mild LBP to 30.9% with moderate and 61.5% with severe LBP. Compared with mild LBP, the odds of clinical insomnia were 4.30 (95% CI 1.90–9.72) for moderate and 14.72 (95% CI 4.16–52.10) for severe LBP, indicating a strong dose–response gradient.

Gender and academic discipline were also associated with insomnia severity. Females showed higher insomnia prevalence than males ($\chi^2 = 8.82$, $p = 0.0318$; Cramer's V = 0.173), while departmental comparison revealed significantly greater insomnia frequencies among Doctor of Physical Therapy (DPT) and Pharm D students compared with Allied Health Sciences and Life Sciences programs ($\chi^2 = 36.25$, $p = 0.0205$; Cramer's V = 0.163). Collectively, these findings demonstrate a robust and clinically meaningful association between increasing LBP severity and insomnia

Table 1 presents the demographic characteristics of the 194 study participants.

The majority were female (n = 146, 75.3%), while males accounted for 24.7% (n = 48). The mean age of participants was 21.8 ± 2.1 years, with ages ranging from 18 to 28. Most respondents were day scholars (n = 134, 69.1%), and 30.9% (n = 60) were hostel residents. The distribution across study years indicated balanced academic representation, with the largest group in the fifth year (n = 56, 28.9%), followed by the third year (n = 47, 24.2%) and second year (n = 43, 22.2%). Departmental analysis showed that Doctor of Physical Therapy (DPT) students formed the largest subgroup (n = 73, 37.6%), followed by Pharm D (n = 46, 23.7%) and MBBS (n = 26, 13.4%), whereas smaller proportions were enrolled in Life Sciences (n = 19, 9.8%), Allied Health Sciences (n = 17, 8.8%), and Nursing or Psychology programs (<5% each).

Table 2 illustrates the distribution of insomnia severity across categories of low back pain (LBP). Of the total 194 students, 68 (35.1%) reported no insomnia, 83 (42.8%) had subthreshold insomnia, 40 (20.6%) had moderate clinical insomnia, and 3 (1.5%) had severe insomnia. The prevalence of insomnia rose sharply with increasing LBP severity Among students with mild LBP (n = 99), only 9 (9.1%) met criteria for clinical insomnia (ISI ≥ 15). In contrast, 25 (30.9%) of those with moderate LBP (n = 81) and 8 (61.5%) with severe LBP (n = 13) met the same criteria. A single respondent with very severe LBP (n = 1) also exhibited clinical insomnia.

The association between insomnia and LBP severity was statistically significant ($\chi^2 = 39.92$, $p < 0.001$), with a moderate effect size (Cramer's V = 0.232).Table 3 details the relationship between insomnia severity and gender. Among male students (n = 48), 27% (n = 13) reported no insomnia, 60.4% (n = 29) subthreshold, and 12.5% (n = 6) moderate insomnia, with no severe cases.

In contrast, female students (n = 146) demonstrated a higher burden: 37.6% (n = 55) no insomnia, 36.9% (n = 54) subthreshold, 23.3% (n = 34) moderate, and 2.1% (n = 3) severe insomnia. The association was statistically significant ($\chi^2 = 8.82$, $p = 0.0318$; Cramer's V = 0.173), confirming that female participants experienced greater sleep disturbance than males.

Table 4 compares insomnia severity across academic departments. The highest proportions of moderate-to-severe insomnia occurred in Pharm D (32.6%) and DPT students (23.3%), followed by MBBS (34.6%), while Life Sciences, Allied Health Sciences, and Nursing showed substantially lower prevalence (<10%). Departmental differences were statistically significant ($\chi^2 = 36.25$, $p = 0.0205$; Cramer's V = 0.163), suggesting academic discipline–related variation in stress exposure or clinical workload that may influence sleep quality.

Table 5 quantifies the odds of clinical insomnia relative to LBP severity. Compared with mild LBP, students with moderate pain were 4.3 times more likely to have clinical insomnia (95% CI 1.90–9.72), and those with severe LBP had 14.7-fold higher odds (95% CI 4.16–52.10). Although

the very severe LBP subgroup (n = 1) yielded wide confidence intervals (OR = 28.58, 95% CI 1.09–751.74) due to small sample size, the trend remained consistent with a dose–response gradient. These quantitative results confirm that as LBP intensity and functional disability increase, the probability of clinically significant insomnia rises exponentially.

Table 1. Participant Demographics (n=194)

Characteristic	n	%
Gender: Male	48	24.7
Gender: Female	146	75.3
Residence: Day Scholar	134	69.1
Residence: Hostelite	60	30.9
Study Year: 1st	26	13.4
2nd	43	22.2
3rd	47	24.2
4th	22	11.3
5th	56	28.9

Table 2. Distribution of Insomnia Severity by LBP Severity (Observed Counts) and Association Metrics

LBP Severity	No Insomnia	Sub-threshold	Clinical Moderate	Clinical Severe	Row Total
Mild	49	41	9	0	99
Moderate	18	38	22	3	81
Severe	1	4	8	0	13
Very Severe	0	0	1	0	1
Column Total	68	83	40	3	194
Inferential statistics: χ^2=39.92; p=0.000008; Cramer's V=0.232.					

Table 3. Insomnia Severity by Gender with Association Metrics

Gender	No Insomnia	Sub-threshold	Clinical Moderate	Clinical Severe	Row Total
Male (n=48)	13	29	6	0	48
Female (n=146)	55	54	34	3	146
Column Total	68	83	40	3	194
Inferential statistics: χ^2=8.82; p=0.0318; Cramer's V=0.173.					

Table 4. Insomnia Severity by Department with Association Metrics

Department	No Insomnia	Sub-threshold	Clinical Moderate	Clinical Severe	Row Total
Pharm D	15	16	15	0	46
AHS	12	5	0	0	17
BSN	4	2	1	0	7
CNA	0	2	0	0	2
DPT	17	39	14	3	73
Life Sciences	10	8	1	0	19
MBBS	8	9	9	0	26
Psychology	2	2	0	0	4
Column Total	68	83	40	3	194
Inferential statistics: χ^2=36.25; p=0.0205; Cramer's V=0.163.					

Table 5. Odds of Clinical Insomnia (ISI≥15) by LBP Severity (Reference: Mild LBP)

Comparison vs Mild LBP	Odds Ratio	95% CI Lower	95% CI Upper
Moderate LBP	4.30	1.90	9.72
Severe LBP	14.72	4.16	52.10
Very Severe LBP†	28.58	1.09	751.74
†Haldane–Anscombe correction applied due to a zero cell.			

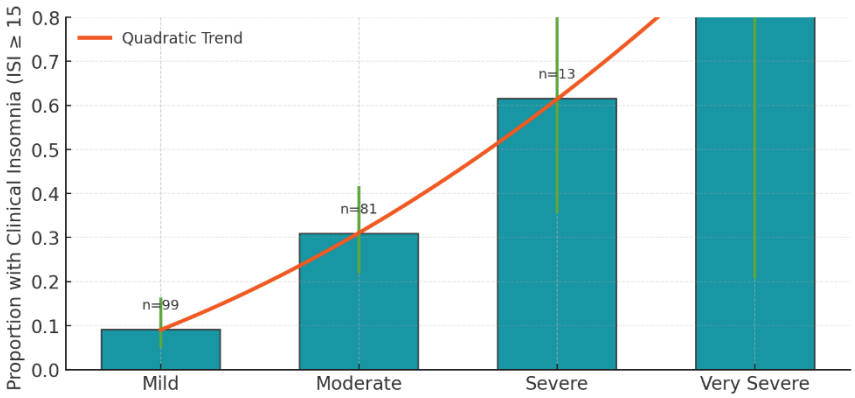

Figure 1 Clinical Insomnia Increases with Low Back Pain Severity

The proportion of students exhibiting clinical insomnia (ISI ≥ 15) rose non-linearly with escalating low back pain (LBP) severity, from 9.1% in mild (n = 99) to 30.9% in moderate (n = 81), 61.5% in severe (n = 13), and 100% in very severe LBP (n = 1). Error bars represent 95% Wilson confidence intervals, and the superimposed quadratic trend line highlights a convex dose–response relationship between insomnia burden and LBP intensity ($\chi^2 = 39.92$, $p < 0.001$; Cramer's V = 0.232). The pattern underscores a clinically meaningful escalation of sleep disturbance alongside increasing pain severity among students.

DISCUSSION

This institution-based study shows a high burden of insomnia symptoms among students with LBP and a clear gradient wherein more severe LBP corresponds to higher clinical insomnia prevalence and markedly elevated odds of ISI-defined morbidity. These findings align with longitudinal evidence that sleep quality predicts subsequent pain intensity in acute LBP and extend it to a young academic cohort where ergonomic and psychosocial stressors are salient (6). Mechanistically, the observed dose–response pattern coheres with pathways linking curtailed or fragmented sleep to increased proinflammatory signaling, impaired descending modulation, and central sensitization, all of which heighten nociceptive gain (5). The strength of association (Cramer's V≈0.23 for LBP×insomnia) is comparable to prior clinic-based reports of sleep–pain comorbidity, while our odds ratios (e.g., OR≈14.7 for severe vs mild LBP) illustrate clinically consequential shifts in insomnia risk across pain strata that may justify routine sleep screening alongside LBP assessment (14–17). The sex difference, with higher insomnia burden among females, parallels established epidemiology and may reflect hormonal influences, stress reactivity, and sociocultural role demands that increase vulnerability to insomnia in young women (24,25). Departmental heterogeneity likely reflects differential academic loads, clinical rotations, and shift timing—factors previously associated with insomnia in health sciences students (26).

In the context of South Asia, where campus health services are variably resourced, our results add region-specific data to Pakistani reports of student sleep problems and offer quantitative evidence for a sleep–LBP interface worthy of integrated prevention (10). Non-pharmacologic sleep interventions (e.g., cognitive behavioral strategies, sleep hygiene, light/dose-timed physical activity, and ergonomic optimization) have demonstrated benefit for chronic LBP populations and could be adapted for student settings to improve both sleep and musculoskeletal outcomes (13,23). Strengths of the present work include the use of validated, interpretable instruments (ISI, ODI, VAS) with prespecified analyses and effect sizes to augment p values, plus explicit operational definitions enabling replication (18,22). Limitations include non-probability sampling within a single institution, which may limit generalizability; self-report measures susceptible to misclassification; and cross-sectional design precluding causal inference. Small cell sizes in the severe/very severe strata widen CIs, suggesting caution in extrapolation. Future studies should employ multi-center probability sampling, objective sleep measures (e.g., actigraphy), and longitudinal designs to delineate directionality and mediators (e.g., mood, physical activity, screen timing), and test integrated sleep–physiotherapy interventions on pain, function, and academic performance (7,12,13,19). Overall, our data support embedding sleep assessment into LBP evaluations and tailoring student health programming to address modifiable sleep and ergonomic risks in tandem (20–22).

CONCLUSION

Insomnia was common among students with LBP and increased steeply with greater pain severity, yielding clinically large odds of ISI-defined morbidity in severe LBP. These findings, aligned with mechanistic and longitudinal literature, justify integrated screening and early, non-pharmacologic sleep interventions within student musculoskeletal care pathways to reduce pain burden, improve functional capacity, and protect academic performance.

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