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ORIGINAL RESEARCH

Depression and Insomnia Mediate the Link Between Problematic Internet Use and Neuropathic Low Back Pain: Evidence from a Cross-Sectional Survey

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Objective: The spread of digital technologies has drawn attention to the health effects of problematic internet use (PIU). This study investigated whether PIU is associated with chronic low back pain, focusing on its neuropathic component and potential mediating factors.

Methods: A cross-sectional online survey was conducted among part-time university students aged 18–65 years. The questionnaire collected demographic, lifestyle, and health-related data, including depression, insomnia, and pain characteristics. Standardized instruments were used: the Problematic Internet Use Questionnaire (PIUQ), Beck Depression Inventory–Short Form (BDI-SF), Athens Insomnia Scale (AIS), and the painDETECT questionnaire. Statistical analyses included chi-square tests, correlations, hierarchical linear regression, and mediation modeling. Analyses were performed on the full sample (N = 550).

Results: The prevalence of chronic low back pain was 46.2%, with 12.2% reporting neuropathic pain. PIU was present in 8.4% of respondents, depression in 69.6%, and insomnia in 5.8%. Neuropathic pain correlated with PIU ($r = 0.126$, $p < 0.05$), depression ($r = 0.44$, $p < 0.001$), and insomnia ($r = 0.33$, $p = 0.009$). In regression analysis, depression ($\beta = 0.256$, $p = 0.001$), headache/migraine ($\beta = 0.20$, $p = 0.002$), smoking ($\beta = 0.135$, $p = 0.030$), and cardiovascular disease ($\beta = 0.129$, $p = 0.045$) were independent predictors of higher painDETECT scores. PIU was not a direct predictor but exerted significant indirect effects: mediation analyses confirmed indirect paths via depression ($b = 0.08$, $p < 0.001$) and insomnia ($b = 0.05$, $p = 0.009$).

Conclusion: Chronic low back pain is influenced by psychological, sleep, and lifestyle factors. Although PIU was not an independent predictor of neuropathic pain, it indirectly increased risk through depression and insomnia. Screening and management of these comorbidities should be incorporated into prevention and treatment strategies.

Keywords: problematic internet use, neuropathic pain, low back pain, depression, insomnia, mediation

Introduction

The rapid expansion of digital technologies in the 21st century has profoundly transformed daily life, social relationships, work, and education. While the reasonable and goal-directed use of technology can clearly enhance quality of life, its excessive and uncontrolled use—particularly when it occurs in maladaptive ways—may lead to numerous negative consequences.[1]

Problematic internet use (PIU) is one such maladaptive behavior, characterized by excessive engagement in online activities and a loss of control over internet use. Importantly, PIU is not defined solely by the amount of time spent online but also by the quality of usage and its impact on daily functioning. From a clinical perspective, internet use becomes problematic when it interferes with psychological well-being, social relationships, academic or occupational performance, or other aspects of everyday functioning, and when it results in subjective distress.[2,3] The manifestations of PIU are highly diverse and span a wide spectrum of behavioral patterns.[3,4] Despite their differences, these behaviors often

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share common addictive, impulsive, and/or compulsive features that sustain dysfunctional patterns of use and undermine adaptive coping strategies.[5]

The prevalence of PIU varies widely across studies, as definitions, diagnostic criteria, assessment tools, and cultural factors all influence the reported rates.[2] Recent meta-analyses suggest that PIU may affect approximately 7% of the general population.[6] In Hungary, our previous research indicated that PIU affects around 11% of primary school students,[7] up to 19% of secondary school students, and between 4–9% of certain adult groups.[8,9]

In parallel with these findings, an increasing body of evidence has highlighted potential associations between PIU and chronic pain syndromes[10] Individuals living with chronic pain often experience social isolation and may turn to the internet as a coping strategy, which in turn increases the risk of PIU. Conversely, excessive internet use, sedentary behavior, poor posture, and physical inactivity can contribute to the onset and persistence of pain, particularly musculoskeletal complaints such as neck, shoulder, and back pain.[10–12]

Moreover, PIU may worsen chronic pain syndromes through additional mechanisms, such as impaired sleep quality.[13–15] Sleep deprivation, in turn, increases pain sensitivity and disrupts the body's natural regenerative processes.[16–18] Neurobiological research suggests that sleep loss alters the functioning of brain regions involved in pain processing, including the nucleus accumbens and ventral tegmental area,[19] and can trigger inflammatory responses mediated by glial cells.[20,21]

Chronic stress, frequently observed in both PIU and chronic pain, also amplifies pain sensitivity through activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system, which sustain systemic inflammatory processes.[22–24] Neuroplastic changes in stress-related brain regions, such as the hippocampus, amygdala, and prefrontal cortex, further contribute to emotional dysregulation and heightened pain perception.[25,26] Importantly, not only the sources of stress but also its subjective appraisal and the coping strategies employed determine its impact. Psychological factors such as anxiety, depression, and low self-efficacy further reinforce this cycle, shaping both stress responses and pain experience.[27,28] Research among adolescents and young adults consistently shows associations between intensive digital technology use and higher levels of depression, anxiety, and psychological distress.[29–38] Cognitive distortions, such as pain catastrophizing, exacerbate pain perception and disability.[39]

Taken together, stress, sleep and mood disturbances, PIU, and pain are interconnected through mutually reinforcing mechanisms that operate at neurobiological, psychological, and behavioral levels. This multifaceted interplay contributes to the persistence and exacerbation of symptoms, underscoring the need for integrated prevention and treatment approaches.

Low back pain is one of the most common forms of chronic pain syndrome, imposing a substantial social and economic burden on individuals and healthcare systems alike.[40,41] Although up to 70–84% of the population may experience low back pain at some point in their lives,[42] in the vast majority of cases no structural abnormality can be identified, and symptoms typically improve spontaneously within a few weeks.[43] Degenerative changes frequently detected by imaging, such as disc degeneration, are not strongly associated with pain severity or the likelihood of chronification.[44,45] However, in approximately 4–25% of cases, pain becomes chronic, often due to psychosocial factors, iatrogenic influences, or maladaptive coping strategies.[46,47]

Recent research has highlighted that low back pain may not only arise from structural (somatic) causes but can also have a neuropathic origin, or may involve mixed mechanisms.[48,49] Neuropathic pain is often accompanied by nociplastic pain, in which central nervous system pain processing becomes dysfunctional—a process that may be further exacerbated by psychological stressors and maladaptive behavioral patterns. In about one-third of patients with chronic low back pain, neuropathic pain predominates, characterized by alterations in pain-processing pathways.[49–51]

An increasing body of evidence suggests that problematic internet use may indirectly contribute to these negative processes: through prolonged sedentary behavior, sleep disturbances, and psychological distress, it may sustain or exacerbate chronic low back pain.[52–54] Jiang and colleagues[55] demonstrated that increased sedentary time—particularly among individuals with PIU—represents an independent risk factor for the development of chronic low back pain (CLBP). Furthermore, studies conducted during the COVID-19 pandemic identified associations between PIU, reduced physical activity, and lumbosacral pain.[56,57]

Although an increasing number of studies have examined the relationship between problematic internet use (PIU) and chronic pain, targeted investigations specifically addressing the association between chronic neuropathic low back pain (NLBP) and PIU are still lacking. Given that both conditions are associated with significant reductions in quality of life

and work capacity, further research is essential. The aim of our study was to explore the relationship between PIU and chronic neuropathic low back pain, with particular attention to the role of psychological factors—primarily depression and sleep disturbances—in the development and persistence of neuropathic pain. In doing so, we sought to contribute to a better understanding of the potential shared neuropsychological mechanisms linking PIU and NLBP.

Materials and Methods

Study Design and Participants

We conducted a cross-sectional online survey between February and May 2023 among part-time students enrolled at Gál Ferenc University. Participation was voluntary and anonymous. The study protocol was reviewed and approved by the Scientific and Research Ethics Committee of the Medical Research Council (ETT-TUKEB; approval number: BMEÜ/1732–3/2022/EKU). This national-level ethical approval covered all collaborating institutions involved in the study, including the University of Pécs (Medical School, Centre for Occupational Medicine) and Gál Ferenc University. All participants provided informed consent prior to participation. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Data Collection

Demographic data included gender, age, marital status, number of children, educational attainment, and secondary employment status. Health-related risk factors recorded were smoking habits, alcohol consumption, diabetes, hypertension, cardiovascular diseases, chronic low back pain, insomnia, and depression. Internet use variables covered daily duration of use and primary purpose of internet use.

Assessment of Chronic Low Back Pain

Chronic low back pain was assessed using the painDETECT questionnaire, a validated self-report screening tool for identifying neuropathic pain components, particularly in non-malignant musculoskeletal conditions. The questionnaire contains nine items: seven assessing pain quality (eg, burning sensations, tingling, pain triggered by pressure or cold) and two assessing temporal patterns and radiation of pain. Based on the total score, three categories can be distinguished: ≤12 indicates neuropathic pain is unlikely, 13–18 is uncertain and requires further evaluation, and ≥19 strongly suggests neuropathic pain. The sensitivity of painDETECT is approximately 85%, specificity 80%, and internal consistency is acceptable (Cronbach's $\alpha = 0.830$).[50]

Assessment of Problematic Internet Use

Since no universally accepted diagnostic criteria exist for PIU, it is recommended to use continuous scales for assessing excessive internet use.[58,59] We employed the Problematic Internet Use Questionnaire (PIUQ), which was developed through clinimetric and psychometric analyses based on Young's Internet Addiction Test and closely aligns with proposed diagnostic criteria. Its reliability has been confirmed by multiple independent research groups and in our prior studies.[9,58,60] The PIUQ consists of 18 items grouped into three dimensions: obsession, neglect, and control disorder. Each item is rated on a five-point Likert scale (1 = never, 5 = always). A total score above 41 indicates problematic internet use. Internal reliability was excellent in our sample (Cronbach's $\alpha = 0.889$).

Assessment of Depression and Sleep Disturbances

Depression was measured using the Beck Depression Inventory – Short Form (BDI-SF), a nine-item instrument assessing symptoms such as indecisiveness, social withdrawal, fatigue, sleep disturbance, work difficulties, excessive concern with somatic symptoms, pessimism, anhedonia, dissatisfaction, and self-blame. Each item was rated on a four-point Likert scale (1–4). Scores above 9 indicate depression, with severity categorized as mild (10–18), moderate (19–25), and severe (≥26). The Hungarian version has been validated,[61] with Cronbach's $\alpha = 0.830$.

Sleep disturbances were evaluated using the Athens Insomnia Scale (AIS), which consists of eight items: five assess nighttime symptoms, and three assess daytime consequences such as sleepiness or fatigue. Each item was rated on a four-

point Likert scale (0–3). A total score above 6 indicates insomnia.[62,63] The validated Hungarian version was used,[63] with high internal consistency (Cronbach's α = 0.834).

Statistical Analysis

Descriptive statistics (mean ± SD or percentages) were calculated for all variables. Associations between categorical variables were examined using chi-square tests, while Pearson's correlation coefficients were computed for continuous variables.

Predictors of neuropathic pain (painDETECT scores) were analyzed using hierarchical linear regression in three models:

1. **Model 1:** sociodemographic variables.
2. **Model 2:** health-related risk factors and comorbidities.
3. **Model 3:** psychosocial and lifestyle variables (depression, PIU, insomnia).

Finally, mediation analysis was applied to test indirect effects of PIU on neuropathic pain through depression and insomnia. Statistical significance was set at $p < 0.05$.

All analyses were performed on the full survey sample (N = 550). Problematic Internet Use (PIU) was entered as a continuous variable in the correlation, regression, and mediation analyses rather than used for subgroup comparisons. No separate subgroup analyses were conducted based solely on participants with self-reported problematic internet use, as the aim was to assess continuous associations across the entire population.

Results

Sample Characteristics

A total of 550 participants completed the online survey. The vast majority were female (87.3%). Detailed descriptive data on demographics, risk factors, comorbidities, and internet use patterns are provided in Supplementary Tables 1 and 2, and have been reported previously (8).

Prevalence of PIU, Depression, Insomnia, and Chronic Low Back Pain

Problematic internet use was identified in 8.4% of the sample (n = 46). Mild depressive symptoms were reported by 64.4% of participants, while moderate and severe depression were less frequent (5.1% and 0.2%, respectively). Insomnia was reported by 5.8% (n = 32). The overall prevalence of chronic low back pain (CLBP) was 46.2% (n = 254). Within this subgroup, 72.8% had nociceptive pain, 15.0% mixed pain, and 12.2% neuropathic pain (Table 1).

Table 1 Prevalence of Problematic Internet Use, Depression, Insomnia, and Chronic Low Back Pain

Variable	Category	n (%)
Problematic internet use	Yes	46 (8.4)
	No	504 (91.6)
Depression	Normal	167 (30.4)
	Mild	354 (64.4)
	Moderate	28 (5.1)
	Severe	1 (0.2)
Insomnia	Yes	32 (5.8)
	No	518 (94.2)
Chronic low back pain (CLBP)	Present	254 (46.2)
	Absent	296 (53.8)
Pain subtype within CLBP	Nociceptive	185 (72.8)
	Mixed	38 (15.0)
	Neuropathic	31 (12.2)

Univariate Associations

No significant age differences were observed in the prevalence of neuropathic low back pain across the age groups (χ^2 = 3.786, p = 0.876). Chi-square tests also revealed no significant associations between neuropathic pain and other sociodemographic variables, including gender (p = 0.335), marital status (p = 0.322), number of children (p = 0.621), or employment status (p = 0.885) (Table 2). Among health-related risk factors and comorbidities, significant associations with neuropathic pain were observed for drug use (p = 0.027), diabetes (p = 0.048), and headache/migraine (p = 0.033). Other factors, including smoking (p = 0.131), hypertension (p = 0.186), cardiovascular disease (p = 0.287), and history of depression (p = 0.433), were not significantly related to neuropathic pain (Table 3).

Table 2 Relationship Between Demographic Variables and Neuropathic Low Back Pain

(Data %, N = 31)	Neuropathic Pain (%)	p value
Gender χ^2 = 2.188		
Male	16.1 (5)	0.335
Female	83.9 (26)	
Age χ^2 = 3.786		
18-25 years	22.6 (7)	0.876
26-35 years	32.3 (10)	
36-45 years	25.8 (8)	
46-55 years	19.4 (6)	
Marital status χ^2 = 4.678		
Single	16.1 (5)	0.322
Relationship	29 (9)	
Married	54.8 (17)	
Number of children χ^2 = 4.416		
Have no child	45.2 (14)	0.621
1 child	12.9 (4)	
2 children	29 (9)	
More than 3 children	12.9 (4)	
Employment while studying χ^2 = 0.245		
No	16.1 (5)	0.885
Yes	83.9 (26)	

Table 3 Univariate Associations Between Risk Factors, Comorbidities, and Neuropathic Low Back Pain (*p < 0.05)

(Data %, N = 31)	Neuropathic Pain (%)	p value
On regular medication χ^2 = 3.327	41.9 (13)	0.189
Smoker χ^2 = 4.065	32.3 (10)	0.131
Alcohol consumption χ^2 = 1.480	3.2 (1)	0.477
Drug consumption χ^2 = 7.222	3.2 (1)	0.027*
Diabetes χ^2 = 6.074	9.7 (3)	0.048*
Hypertension χ^2 = 3.361	12.9 (4)	0.186
Cardiovascular problems χ^2 = 2.493	12.9 (4)	0.287
Headache/migraine χ^2 = 6.834	41.9 (13)	0.033*
History of depression χ^2 = 1.674	16.1 (5)	0.433

Bivariate correlation analysis showed that higher PainDETECT scores were significantly correlated with PIU ($r = 0.126$, $p < 0.05$), depression scores ($r = 0.44$, $p < 0.001$), and insomnia scores ($r = 0.33$, $p = 0.009$). In addition, age was not a significant predictor in either the hierarchical regression or the mediation models ($p > 0.05$). Therefore, age was retained in the adjusted analyses as a covariate but did not independently influence the outcomes. These associations suggested that both psychological and behavioral factors contributed to the neuropathic pain component (not shown).

Hierarchical Regression Analysis of Predictors of Low Back Pain

To examine predictors of low back pain, a three-step hierarchical linear regression analysis was performed. The first model included sociodemographic variables (gender, age, marital status, number of children, employment), none of which were significantly associated with painDETECT scores. In the second model, health-related risk factors were added, and headache/migraine emerged as a significant predictor ($p = 0.002$). In the third model, lifestyle and psychosocial variables were included. Smoking ($p = 0.030$), cardiovascular disease ($p = 0.045$), and particularly depressive symptoms ($p = 0.001$) were significantly associated with higher painDETECT, reflecting a greater likelihood of neuropathic pain. Depression was the strongest predictor, showing a robust positive association with painDETECT scores. The explanatory power of the models increased from 5% in Model 2 to 13.6% in Model 3 (Table 4).

Mediation Analysis

Mediation analysis revealed that PIU was significantly associated with both higher depression scores ($b = 0.189$, $p < 0.001$) and sleep disturbances ($b = 0.162$, $p < 0.001$). In turn, depression ($b = 0.441$, $p < 0.001$) and sleep disturbances ($b = 0.328$, $p = 0.009$) were independently and significantly associated with higher painDETECT. These findings indicate that PIU exerts an indirect effect on neuropathic pain through depressive symptoms and sleep disturbances (Table 5).

Table 4 Predictors of Neuropathic Low Back Pain Identified by Hierarchical Regression (*p < 0.05)

Dependent: Sum of Low Back Pain Scores (paindetect)	Model I			Model II			Model III		
	B	SE B	β	B	SE B	β	B	SE B	β
Gender	−0.716	1.278	−0.037	−1.126	1.378	−0.058	−0.776	1.318	−0.040
Age	−0.054	0.049	−0.093	−0.046	0.051	−0.079	−0.081	0.071	−0.138
Marital status	−1.155	0.985	−0.077	−0.881	0.978	−0.059	−0.966	0.947	−0.065
Number of children	0.222	0.451	0.041	0.113	0.458	0.021	0.372	0.442	0.069
Employment while studying	−0.393	1.211	−0.022	−0.151	1.195	−0.008	0.069	1.140	0.004
On regular medication				−0.160	0.950	−0.012	−0.325	0.920	−0.025
Smoker				1.777	0.996	0.114	2.100	0.962	0.135*
Alcohol consumption				−1.905	2.457	−0.054	−1.999	2.374	−0.057
Drug consumption				11.347	6.643	0.116	7.473	6.409	0.076
Diabetes				2.099	1.611	0.086	2.248	1.551	0.092
Hypertension				0.886	1.444	0.045	0.582	1.387	0.029
Cardiovascular problems				2.863	1.632	0.117	3.170	1.574	0.129*
Headache/migraine				2.672	0.873	0.200*	2.420	0.856	0.181*
History of depression				0.163	1.081	0.010	−2.078	1.131	−0.125
Time spent online per day							0.055	0.258	0.015
Age of starting with a digital device							0.096	0.082	0.121
Internet addiction							1.672	1.406	0.080
Sum of depression scores							0.433	0.131	0.256*
Sum of sleep disturbance scores							0.159	0.145	0.087
Adjusted R^2	−0.009			0.050			0.136		
F	0.574			1.927*			3.048*		

Table 5 Mediation Analysis of the Indirect Effects of PIU on Neuropathic Pain

Mediator	a-path (X→M)	b-path (M→Y)	Indirect Effect	95% CI	p value
Depression	0.189	0.441	0.08	0.04–0.14	<0.001
Insomnia	0.162	0.328	0.05	0.01–0.10	0.009

Discussion

The aim of this study was to explore the association between problematic internet use (PIU) and chronic neuropathic low back pain (NLBP), while also considering sociodemographic, health-related and psychological factors (depression, sleep disturbance).

We found no association between sociodemographic factors and NLBP, in contrast to previous research that has reported significant gender- and work-related differences in the development of this pain syndrome.[40,41,64] This discrepancy could be due to the relatively low number of patients within this subgroup.

Among health-related risk factors and comorbidities, significant associations with neuropathic pain were observed for drug use, diabetes, and headache/migraine in univariate analysis. The relationship between diabetes and various forms of neuropathic pain has been recognized, and this condition is also strongly linked to mood disorders[65] The association between illicit drug use and chronic low back pain has previously been documented in different studies, including a population-based study from the United States.[66] The underlying mechanisms may involve substance use as a maladaptive coping strategy for persistent pain, as well as shared neurobiological pathways that contribute to both chronic pain and substance use disorders.[66] The emergence of headache/migraine as a predictor suggests that these chronic pain conditions may share common neurobiological mechanisms. Key processes may involve the trigeminovascular system, serotonergic pathways, and cortical sensitization. Since these systems directly influence pain perception and processing, they may explain the overlaps and comorbidities observed across different chronic pain syndromes.[67–70]

Bivariate correlation analysis showed that higher painDETECT scores were significantly correlated with PIU, depression, and insomnia. These associations may indicate that both behavioral and psychological factors contribute to the development of NLBP. The observed correlation between depression and neuropathic pain was particularly strong, which is consistent with previous findings highlighting depression as one of the most important comorbidities of chronic pain.[35] Similarly, the relationship between insomnia and increased pain sensitivity has been documented in both experimental and clinical studies, underscoring the bidirectional interaction between disturbed sleep and pain chronification.[16–22] Although the correlation between PIU and neuropathic pain was weaker, it suggests that maladaptive digital behaviors may play an indirect role, primarily through their impact on psychological well-being and sleep quality.

The hierarchical regression analysis provided a slightly different picture compared to the univariate and bivariate findings. It revealed that headache/migraine, smoking, cardiovascular disease, and depression were significantly associated with neuropathic low back pain. Among concomitant diseases, only migraine remained significant, and its association with a cluster of chronic pain syndromes has been well established as mentioned above.[67–70] Interestingly, smoking and cardiovascular disease were not significantly associated with neuropathic pain in univariate model, they only became significant in the fully adjusted regression model. This apparent discrepancy may be explained by confounding and suppression effects, once psychosocial and health-related factors were simultaneously controlled for, the independent contribution of smoking and cardiovascular disease emerged. Smoking also emerged as a significant predictor of neuropathic pain. Several mechanisms may account for this association: smoking increases inflammatory activity and weakens endogenous analgesic systems. Although nicotine may exert mild short-term analgesic effects, long-term use paradoxically enhances pain perception by promoting the release of pro-inflammatory mediators. Additionally, its impact on the peripheral nervous system may sensitize nociceptive pathways, thereby contributing to the persistence of chronic pain.[71] Similarly, cardiovascular disease was significantly associated with higher painDETECT scores. Chronic circulatory impairment and the resulting tissue hypoxia may increase both peripheral and central sensitization, key processes in the development and maintenance of chronic pain. Furthermore, microcirculatory disturbances and systemic inflammation characteristic of cardiovascular conditions may exacerbate maladaptive pain processing and prolong pain persistence.[65,72]

Our findings highlight depression as the strongest predictor of neuropathic pain, which is consistent with previous research emphasizing the close relationship between mood disorders and chronic pain. Depression not only distorts the subjective perception of pain but also undermines coping abilities, thereby increasing the risk of developing and maintaining chronic pain syndromes.[35]

In contrast to univariate correlations, PIU did not demonstrate a statistically significant direct association with neuropathic low back pain in the fully adjusted regression model. One possible explanation is that neuropathic pain primarily develops through distinct neurobiological mechanisms - such as central sensitization and neuroinflammation - that may be less directly influenced by lifestyle factors like internet use.[28,35] However, the mediation analysis added an important layer of interpretation: PIU was significantly associated with both higher depression and sleep disturbance scores, and these factors in turn independently predicted higher painDETECT scores. This indicates that PIU may not directly contribute to neuropathic pain, but instead exerts its influence through psychological distress and impaired sleep. Our results are also in line with previous meta-analytic evidence showing that problematic internet use is significantly associated with multiple psychiatric comorbidities, including depression and anxiety.[73] Clinically, this highlights the importance of screening and managing depression and sleep problems in individuals with problematic internet use, as targeting these comorbidities could mitigate the risk of neuropathic pain. Another explanation for the lack of a direct effect may be related to age differences: PIU is more characteristic of younger, digitally active populations, whereas neuropathic pain and chronic low back pain are more prevalent among older adults. Although our sample included both younger and older participants, this divergence may have reduced the detectability of a direct association.

A key strength of this study is the use of internationally validated and widely applied instruments (PIUQ, BDI-SF, AIS, and painDETECT), which ensured reliable assessment of problematic internet use, depression, insomnia, and neuropathic pain. The large sample size and the application of multiple statistical methods, including hierarchical regression and mediation analyses, allowed us to disentangle direct and indirect pathways linking PIU and neuropathic low back pain. This comprehensive analytical approach provided novel insights into the mediating roles of depression and sleep disturbances.

Nevertheless, several limitations should be acknowledged. First, the cross-sectional design precludes causal inference; longitudinal studies are required to clarify temporal and causal pathways. Second, all measures were based on self-report questionnaires, which are subject to recall bias and reporting errors, and no clinical diagnoses were confirmed by medical professionals. Third, the number of participants with neuropathic pain was relatively small (n = 31), which may have limited statistical power and increased the risk of type II error. Fourth, the sample was predominantly female (87%), consisting of part-time university students, which limits the generalizability of the findings to the broader population. Fifth, while our regression models identified significant predictors, the overall explained variance remained modest (13.6%), suggesting that additional unmeasured factors—such as genetic, occupational, or environmental influences—also play an important role in the development of neuropathic pain.

Conclusion

In summary, the development and persistence of chronic low back pain is a complex, multidimensional phenomenon. Our findings demonstrate that higher PainDETECT scores—and thus greater likelihood of neuropathic pain—are primarily explained by psychological factors (depression, sleep disturbances) and certain lifestyle and health-related risks (smoking, cardiovascular disease, headache/migraine). Importantly, PIU exerts an indirect effect through its association with depression and sleep disturbances, underscoring the mediating role of these factors. Clinically, our results emphasize the need for regular screening of psychological and sleep-related problems, particularly in patients with problematic internet use. Incorporating targeted psychological and lifestyle interventions into treatment strategies may help reduce the risk of neuropathic pain and contribute to a more comprehensive, personalized approach to managing chronic low back pain.

Disclosure

The authors report no conflicts of interest in this work.

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