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Interconnections Between Chronic Lower Back Pain and Smoking: A Systematic Review

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Abstract **Background:** The objective of this systematic review was to investigate the existing literature on the interconnections between Chronic Lower Back Pain (CLBP) and smoking. Past research has often alluded to a possible link between these factors, but the extent and nature of their relationship remained to be comprehensively synthesized. **Methods:** A methodical literature search was conducted across MEDLINE (via PubMed), EMBASE, PsycINFO, Scopus, Web of Science, CINAHL, Cochrane Library and Google Scholar for studies published from March 2014 up to March 2024. The inclusion criteria incorporated peer-reviewed articles that examined the relationship between smoking and CLBP. Studies were evaluated for quality and data were extracted on study design, participant demographics, smoking and pain measurements and outcomes. **Results:** The review included a diverse range of studies with varying methodologies. Across the studies, a significant association between smoking and the incidence, severity and persistence of CLBP was consistently observed. Quantitative analysis revealed a dose-response relationship, with higher smoking intensity and longer duration correlating with increased risk and severity of CLBP. The association persisted even when controlling for confounding factors. However, a subset of studies highlighted the predominance of psychosocial factors over smoking as predictors of CLBP chronicity. **Conclusion:** The systematic review substantiated the hypothesis that smoking is significantly associated with CLBP. The evidence suggested a potential causal relationship, whereby smoking could contribute to the development and exacerbation of CLBP. Nevertheless, the role of psychosocial factors in the manifestation of CLBP also emerged as a significant aspect, indicating the multifactorial nature of CLBP. These findings underscore the necessity for integrated treatment approaches that address both smoking cessation and psychological interventions for CLBP sufferers.

Key Words Chronic Lower Back Pain, Smoking, Systematic Review, Psychosocial Factors, Pain Severity, Causal Relationship

INTRODUCTION

Chronic Lower Back Pain (CLBP) is a prevalent condition with complex causal interactions, with significant impact on public health and economic systems globally [1]. As one of the leading causes of disability, CLBP is a complex problem, with the intersection of biophysical, psychosocial and environmental factors being implicated in its etiology and long-term persistence. The chronic nature of lower back pain has been linked to a plethora of risk factors, among which smoking has been hypothesized as a causal factor [2]. While the association between smoking and many health outcomes is well known, the complete explanations of the mechanisms and magnitude of effect that smoking has on CLBP remain to be unraveled [3].

Literature proposes several hypotheses on how smoking can exacerbate or initiate the development of CLBP. These involve smoking-induced vascular changes, alteration in pain processing and nicotine effects on disc degeneration and connective tissue [4]. Moreover, smoking has also been associated with systemic inflammatory processes and oxidative stress, which may augment nociceptive pathways. Conversely, CLBP itself may influence smoking behavior, potentially creating a bidirectional relationship that complicates individual prognosis and treatment [5].

Extensive empirical literature has characterized the therapeutic efficacy of physical exercise in the relief of low back pain (LBP), taking into account the multifactorial determinants of its causative determinants [5-7]. The

pathophysiology of LBP is established as a multifactorial interaction between genetic determinants, biomechanical and demographic factors (e.g., age, gender and history of prior lumbar trauma), psychological states (e.g., chronic stress, anxiety and fear of movement) and different lifestyle habits (especially, alcohol consumption and tobacco smoking) [8-9].

In the recent scientific literature, the relationship between nociception, nicotine use and smoking behavior has been widely discussed because of its prevalence, public health relevance and secondary health effects [10]. More recent evidence show that almost sixty percent of tobacco-dependent patients have symptoms typical of chronic pain syndromes [11]. Previous studies have proven correlation where current or former smokers have reported greater and more extensive pain compared to people who never smoked [12].

In spite of the credible mechanisms at the biological level, the epidemiologic evidence is heterogeneous. The studies have differed in design, population, exposure and outcome measurement and control for confounders [9-12]. The heterogeneity has presented a range of reported associations, from strong to minimal or no significant relationship between CLBP and smoking. The strengths of smoking as a modifiable lifestyle factor present a window of opportunity for intervention strategies that can reduce the burden of CLBP [5, 7]. An understanding of how large and what kind of its association with CLBP is necessary for the formulation of overall treatment strategies, which may incorporate smoking cessation interventions in addition to traditional pain treatment therapies.

Against this background, the current systematic review aims to critically evaluate and summarize the available evidence on the relationship between smoking and CLBP. In a critical examination of the cumulative evidence of prior studies, the review also aims to explore the contribution of smoking towards the aetiology and causes of CLBP. The review aims to establish trends, strength of association and presumable causation relationship as a means of improving knowledge in CLBP as well as supplying evidence-based information for practice and public health intervention.

MATERIALS AND METHODS

Eligibility Criteria

This review utilised the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [13], ensuring a structured and transparent approach to the review process. Initially, a comprehensive search strategy was formulated and applied across multiple databases to capture all relevant studies published from 2014 onwards.

For the PECO (Population, Exposure, Comparator, Outcome) protocol of this review, the following criteria were established:

- **Population:** Adults with chronic lower back pain, defined as pain persisting for more than 12 weeks
- **Exposure:** Smoking, including current and former smokers, with the consideration of smoking intensity and duration
- **Comparator:** Non-smokers or individuals with different levels of smoking exposure, to identify the gradient effect of smoking on CLBP
- **Outcome:** The primary outcome of interest was the presence and severity of chronic lower back pain. Secondary outcomes included the impact of smoking cessation on CLBP and the relationship between smoking and other clinical features of CLBP, such as disability and quality of life

Table 1 elucidates the inclusion and exclusion criterion that were devised for this investigation.

The decision to restrict the studies to those published after 2014 was a conscious one that directly responded to the goals of our review. By restricting the literature to recent years, the review aimed to capture the most up-to-date evidence and advances in the field, including methodological innovations and new knowledge of the link between smoking and CLBP. The limitation by timespan was aimed at ensuring that the review captured current knowledge and practice and offered a modern and relevant synthesis of evidence to inform clinical decision and the guidance of future research. The cut-off was chosen to cover a timespan broad enough to encompass recent studies but representative of the status of research at the time that this review was being conducted.

Database Search Protocol

Databases that were searched for this review included MEDLINE on PubMed, EMBASE, PsycINFO, Scopus, Web of Science, CINAHL, the Cochrane Library and Google Scholar and all were searched with relevance to studies for the purpose of our review. The search strategy was optimized to take advantage of the best features of each database's index systems, using a combination of Boolean operators and Medical Subject Headings (MeSH) terms and database-specific keywords. The search string was constructed such that Boolean operators were employed, i.e., "AND" between dissimilar ideas and "OR" to cover variations and synonyms related to the same idea. Keywords and MeSH terms were selected wisely to cover the regions of "chronic lower back pain" and "smoking." Search strings were constructed to be compatible with the various syntax and subject headings of different databases while the conceptual similarity of all the searches was maintained (Table 2).

Data Extraction Protocol and Items Selected

The protocol was executed by a pair of independent reviewers who extracted the data using a standardized data extraction form. Discrepancies between reviewers were resolved through discussion or consultation with a third reviewer. The data extraction form was pretested on a subset of included studies to confirm its comprehensiveness and functionality.

Table 1: Selection criterion utilised for the inclusion of relevant studies

Criteria	Inclusion	Exclusion
Study Design	Peer-reviewed observational studies (cross-sectional, cohort, case-control) and clinical trials	Reviews, editorials, commentaries, case reports and studies without primary data
Population	Adults (aged 18 and older) with CLBP	Studies on populations without CLBP or with acute back pain or non-specific back pain
Exposure	Quantifiable measures of smoking (current and former smokers, intensity, duration)	Studies lacking clear definition or measurement of smoking status
Comparator	Non-smokers or different levels of smoking exposure	Studies without a comparator group or inadequate comparison between smokers and non-smokers
Outcome	Presence and severity of CLBP, impact of smoking cessation on CLBP	Studies not reporting specific outcomes related to CLBP
Timeframe	Studies published from 2014 onwards	Studies published before 2014
Language	Studies published in English	Non-English language studies without available translations

Table 2: Databases assessed for selection of articles in this review

Database	Search String
MEDLINE (via PubMed)	("Low Back Pain"[MeSH] OR "lumbago" OR "chronic back pain" OR "backache") AND ("Smoking"[MeSH] OR "smoker*" OR "tobacco use disorder" OR "nicotine addiction") AND "2014/01/01"[PDAT] : "3000"[PDAT]
EMBASE	('low back pain'/exp OR 'lumbago'/exp OR 'chronic back pain':ab,ti OR 'backache':ab,ti) AND ('smoking'/exp OR 'smoker*':ab,ti OR 'tobacco use disorder':ab,ti OR 'nicotine addiction':ab,ti) AND [2014-2024]/py
PsycINFO	(("Low Back Pain" OR "lumbago" OR "chronic back pain" OR "backache") AND ("Smoking" OR "smoker*" OR "tobacco use" OR "nicotine dependence")) AND ("20140000": "20240000")
Scopus	(TITLE-ABS-KEY ("low back pain" OR "lumbago" OR "chronic back pain" OR "backache") AND TITLE-ABS-KEY ("smoking" OR "smoker*" OR "tobacco use" OR "nicotine addiction")) AND PUBYEAR > 2013
Web of Science	(TS=("low back pain" OR "lumbago" OR "chronic back pain" OR "backache") AND TS=("smoking" OR "smoker*" OR "tobacco use" OR "nicotine addiction")) AND PY=(2014-2024)
CINAHL	(MH "Low Back Pain+" OR "lumbago" OR "chronic back pain" OR "backache") AND (MH "Smoking+" OR "smoker*" OR "tobacco use" OR "nicotine addiction") AND (YR "2014 - Current")
Cochrane Library	("low back pain":ti,ab,kw OR "lumbago":ti,ab,kw OR "chronic back pain":ti,ab,kw OR "backache":ti,ab,kw) AND ("smoking":ti,ab,kw OR "smoker*":ti,ab,kw OR "tobacco use":ti,ab,kw OR "nicotine addiction":ti,ab,kw) AND (YEAR FROM 2014 TO Current)
Google Scholar	allintitle: chronic lower back pain OR lumbago OR backache AND smoking OR smoker* OR "tobacco use" 2014..2024

The form was refined accordingly before its application to the full set of included studies. The extracted data items encompassed:

- **General information:** Bibliographic details such as the title, authors, year of publication, country of origin and journal were recorded to provide the reference for each study
- **Study characteristics:** Methodological aspects, including study design (e.g., cross-sectional, cohort, case-control, or clinical trial), setting, duration of follow-up (if applicable) and sample size were captured
- **Population details:** Characteristics of the study population such as age, sex and baseline health status, including specific inclusion and exclusion criteria for participants, were documented
- **Exposure assessment:** Information on how smoking status was measured, including definitions of smoking categories (e.g., current, former, never-smoker) and quantification of smoking exposure (e.g., pack-years, smoking duration) were extracted
- **Outcome measures:** The primary and secondary outcomes related to chronic lower back pain, including pain severity, functional disability, quality of life and any other reported outcomes, were detailed along with the methods of measurement and time points recorded
- **Key conclusions:** The main findings of the study as concluded by the authors were summarized

Bias Assessment Protocol

For cross-sectional studies, the Appraisal Tool for Cross-Sectional Studies (AXIS) was used [14]. AXIS contains several components that assess several facets of a study, from its objectives to its design, sampling, methods of data collection, analysis and reporting. Each component is constructed to identify possible sources of bias, such as selection bias, information bias and confounding. For non-randomized studies by exposures, the Risk Of Bias In Non-randomized Studies - of Exposures (ROBINS-E) tool [15] was used. This tool is specifically constructed to evaluate the risk of bias in studies that investigate the effect of exposures on health outcomes.

Measurement Of Certainty Bias

Once the bias assessment for each individual study was completed, the reviewers applied the GRADE approach [16] to evaluate the overall body of evidence in relation to each primary outcome of interest. The GRADE approach considers a range of factors, including risk of bias, inconsistency, indirectness, imprecision and publication bias. Each of these factors has the potential to necessitate downgrading the quality of evidence. The reviewers started grading the quality of evidence for each outcome as 'high' by default, according to standard procedures in the GRADE system. They then reviewed the outcomes of the AXIS [14] and ROBINS-E assessments [15] to identify any issues with the risk of bias of

the studies. Studies with high or critical risk of bias were reasons for downgrading the quality of evidence based on the GRADE system.

RESULTS

Study Selection Schematics

The systematic review article selection process, in accordance with the PRISMA reporting, was carried out in phases. A broad search in various databases initially yielded the collection of 368 records and no other records were accessed from registers. Before proceeding with the screening step, duplicates were eliminated carefully and 42 records were excluded. After duplicate removal, 326 records proceeded to the screening step. In the screening step, a large number of records, i.e., 55, were excluded as there was no full text mentioned. This led to the retrieval of 271 reports. All the same, challenges were encountered as 38 of these reports could not be retrieved due to various reasons. Consequently, the number of reports under consideration for eligibility dropped to 233.

The process of determining eligibility included an exhaustive review phase, with a large number of reports excluded according to the predetermined criteria: 43 reports did not include the predetermined PECO criteria; 38 reports were not relevant to the subject area; 42 were individual case reports and not suitable to be included in the systematic review; 22 were scoping reviews and 19 literature reviews, both being excluded in order to include only primary research studies and 61 studies were published before 2014, a year presumably selected as a cut-off point for relevance or methodological consistency. After this rigorous process of exclusion, 8 studies [17-24] met all the criteria and were thus included in the systematic review (Figure 1).

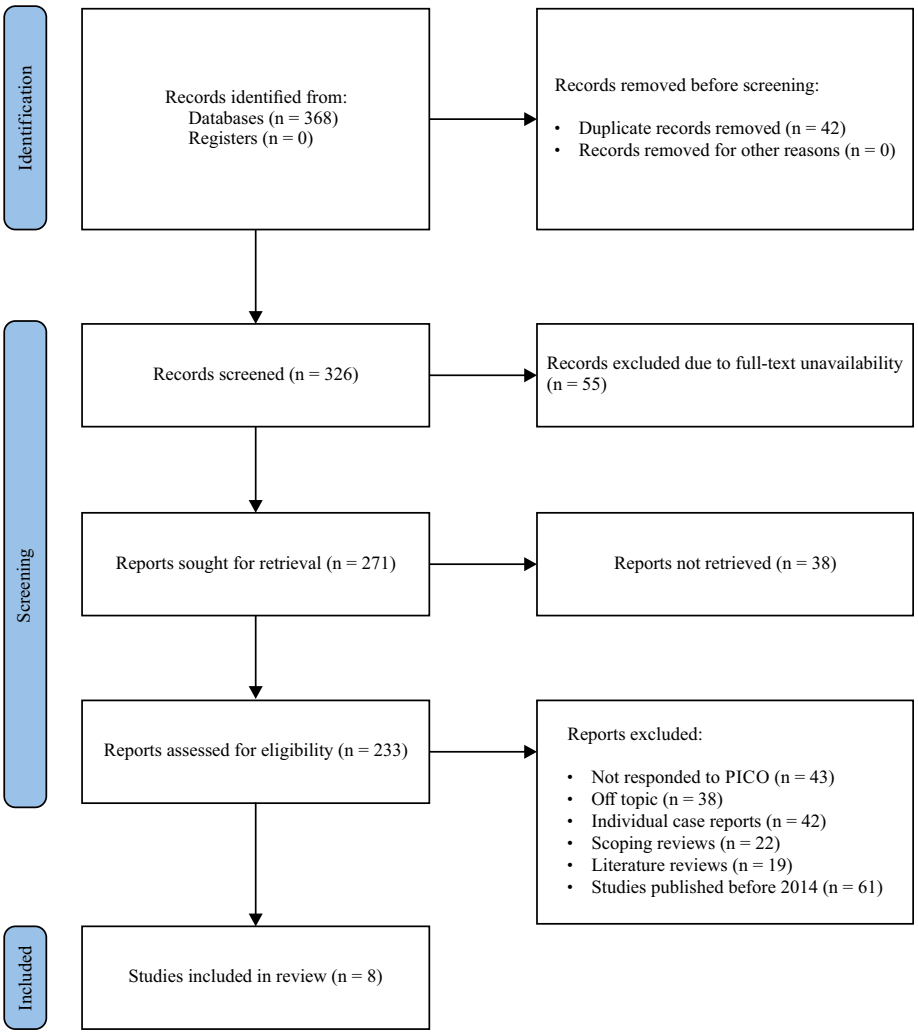

Figure 1: Article selection process representation of the review

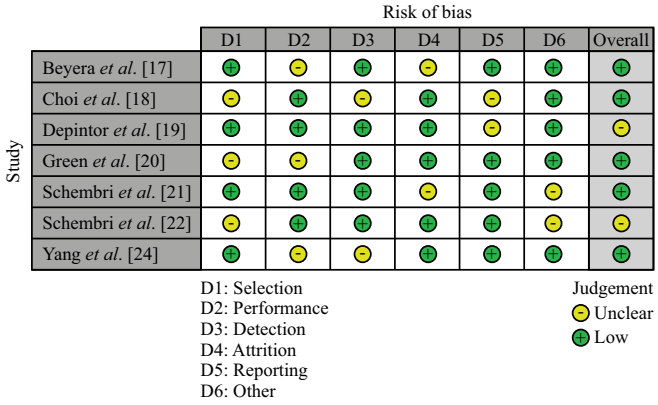

Figure 2: Bias observations as per the AXIS tool

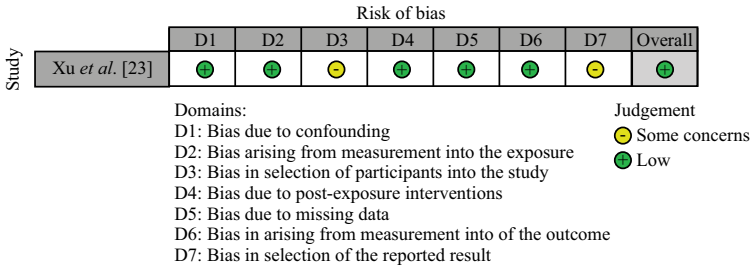

Figure 3: Bias observations as per the ROBINS-E tool

Assessed Bias Observations

For the cross-sectional studies (Figure 2), Beyera *et al.* [17] demonstrated a low risk of bias in most categories including selection, detection, attrition, reporting and other biases, while performance and attrition biases were assessed as moderate. Choi *et al.* [18] had a moderate risk of bias in the selection and detection categories, a low risk in performance, attrition and other biases and a moderate risk in reporting. Depintor *et al.* [19] showed a low risk across selection, performance, detection, attrition and other biases but had a moderate risk in reporting, resulting in an overall moderate bias rating.

Green *et al.* [20] exhibited a moderate risk of bias in selection and performance, but a low risk in the other areas, culminating in an overall low risk of bias. Schembri *et al.* [21] maintained a low risk in most categories, with the exceptions of moderate risks in performance and reporting. Schembri *et al.* [22] had a similar profile to Green *et al.* [20], with moderate risks in selection and other biases, but otherwise low risks, leading to an overall moderate risk of bias assessment. Yang *et al.* [24] showed a low risk in most areas but moderate risks in performance and detection. For the cohort-based study by Xu *et al.* [23] assessed with the ROBINS-E tool (Figure 3), the study was found to have a low risk of bias in most domains, except for moderate risk in domains D3 and D7, which did not significantly affect the overall low risk of bias rating for the study.

Baseline Characteristics Assessed

In the synthesis of demographic data of studies incorporated herein [17-24] as described through Table 3, cross-sectional method was the prevalent approach [17-22, 24], with one cohort-based protocol [23]. Sample sizes were relatively varied, ranging from a minimum of 54 participants in a study that sought to investigate the implication of smoking status on CLBP to a maximum of 438, 510 participants in a cohort study most likely to provide a broad overview of the studied condition, conducted in 2023 [23-24]. The year of implementation of studies varied from 2016 to 2023, representing a recent and perhaps evolving understanding of the study conditions [19-20, 23]. Regional heterogeneity was noted and reported, with studies having been conducted in Ethiopia, China, Brazil, the United States and Malta, which may represent different environmental, genetic and sociocultural factors influencing the study findings [17-22].

The mean ages of the participants also varied, with one study reporting a mean age higher than 18 years and another reporting mean ages in smokers and non-smokers separately, suggesting the impact of smoking on onset or severity of CLBP [20-21]. The male to female ratios were also reported in all except two Maltese studies, where demographic information was not specified [21-22]. Interestingly, in the largest cohort study, the mean ages of males and females were reported separately, suggesting a detailed consideration of effects due to age in the results reported [23]. The gender

Table 3: Demographic characteristics observed in the included studies

Study ID	Year	Region	Protocol	Sample size (n)	Mean age (in years)	Male: Female ratio
Beyera *et al.* [17]	2022	Ethiopia	Cross-sectional	1812	38	984:828
Choi *et al.* [18]	2021	China	Cross-sectional	8473	With CLBP: 63.19±8.83; without CLBP: 67.01±9.01	3,601:4,872
Depintor *et al.* [19]	2016	Brazil	Cross-sectional	826	51.4 ± 19.3	256:570
Green *et al.* [20]	2016	USA	Cross-sectional	34,241	>18	15,273:19,252
Schembri *et al.* [21]	2021	Malta	Cross-sectional	120	Smokers: 57.1; Non-smokers: 61.38	Unspecified
Schembri *et al.* [22]	2020	Malta	Cross-sectional	150	60.1 ± 13.1	Unspecified
Xu *et al.* [23]	2023	China	Cohort-based	438,510	Male: 45.1; Female: 54.9	197,864:240,646
Yang *et al.* [24]	2023	China	Cross-sectional	54	Smoking group: 29.06; Non-smoking group: 30.09	17:10

ratio was also imbalanced with higher representation of females in some studies, perhaps suggesting the reported or actual higher prevalence of CLBP in females or may be simply an artefact of sampling biases [18-20]. The smallest sample study addressed the smoking group and reported mean ages of smokers and non-smokers separately, which may suggest information on the effect of lifestyle factors on CLBP [24].

Groups and Parameters Assessed

Table 4 outlines the varied variables in accordance with the association of LBP with cigarette smoking. In the work of Beyera *et al.* [17], 18+ adults with LBP were interviewed on a very extensive range of factors, encompassing socio-demographic data, health behavior and lifestyle, ideas about pain and other general and pain-specific health factors. The wide nature of the information gathered enabled multifaceted LBP research to be conducted with this population. Choi *et al.* [18] sampled patients in the Korea National Health and Nutrition Examination Survey and studied health and nutrition status. Notably, the study factored in the levels of stress and environmental components such as smoking, which were identified to affect health outcomes severely.

Depintor *et al.* [19] conducted an evaluation on a population of 826 patients using various well-documented tools. The assessment included the Hospital Anxiety and Depression Scale (HADS), EuroQol-5D, Alcohol Use Disorders Identification Test (AUDIT) and the Fagerström test for nicotine dependence, supplemented by a Brazilian economic classification tool. Collectively, these evaluations offered valuable information regarding the mental health, quality of life, substance use and socioeconomic status of low back pain (LBP) patients. In another study, Green *et al.* [20] conducted a comprehensive evaluation on 34,525 American adults. The study centered on the investigation of the prevalence of back pain across different alternative smoking statuses and quantified the level of cigarette smoking, which may indicate the association between smoking behavior and LBP prevalence.

In the work of Schembri *et al.* [21], the two groups were compared in 120 chronic low back pain (LBP) patients and 50 control subjects. Pain intensity, Douleur Neuropathique 4 (DN4) questionnaire scores, STarT Back screening tool and Fagerström test for nicotine dependence were assessed to determine pain experience and related factors in the two groups. Schembri *et al.* [22] also researched an exact population with 150 chronic LBP patients (mean age: 60.1) and a control group of 50 subjects. Demographic information was gathered, the International Association for the Study of Pain (IASP) neuropathic pain grading system was used, the STarT Back screening tool was used, the Fagerström test for nicotine dependence was used and the aim was to determine neuropathic pain differences and other related attributes in chronic LBP subjects.

Xu *et al.* [23] performed a longitudinal study with the UK Biobank, tracking a large cohort of 438,510 individuals who were initially pain-free back between 2006 and 2010. The study quantified a number of factors, including Smoking Status (SS), Cigarettes per Day (CPD), Pack-Years (PY) and the incidence of back pain, thereby offering useful insights into the prospective longitudinal effect of smoking on the incidence of Low Back Pain (LBP). In another study, Yang *et al.* [24] targeted a smaller group of 54 Chronic Low Back Pain (CLBP) patients, classified according to their smoking status. This study quantified pain severity, functional capacity, psychological status and smoking behavior, offering a detailed analysis of the correlation of these factors specifically in the CLBP group and highlighting the contribution of smoking to these correlations.

Correlation Between LBP And Smoking Observed

Beyera *et al.* [17] did find smoking relatively rare in people with LBP and occurring in only 3.2% of instances. There was no apparent focus in the results on a correlation between LBP and smoking that would indicate other factors were perhaps more significant in developing LBP in this population. Choi *et al.* [18] listed smoking in a series of environmental variables in a multivariate regression analysis when they were examining the health and nutritional status of subjects. Smoking was not specifically referred to in the context of LBP, however, which would indicate that the correlation with stress factors was perhaps more significant in their research.

The study by Depintor *et al.* [19] found a statistically significant association between chronic back pain and smoking. Smoking subjects had a 41% higher likelihood of developing chronic back pain than non-smokers. This evidence supported by a 1.41 prevalence ratio and a 95% confidence interval of 1.06 to 1.88 and a p-value of 0.031, indicates the possible contributory role of smoking to the onset of chronic Low Back Pain (LBP). Green *et al.* [20], in another study, identified active smokers with the highest

Table 4: Correlation between LBP and cigarette smoking as observed in the included papers

Study ID	Groups assessed	Parameters assessed	LBP and smoking correlation observed	Overall inference drawn
Beyera *et al.* [17]	Adults (≥18 years) with LBP	Socio-demographic info, health behaviors/lifestyle habits, beliefs about pain, other pain-related and general health characteristics	Smoking among individuals with LBP was relatively uncommon (3.2%). The study did not emphasize a strong correlation between LBP and smoking	The study highlighted factors like negative beliefs about pain, pain interference, general health status and depressive symptoms as significantly associated with chronicity of LBP. Substance use was not prevalent
Choi *et al.* [18]	Patients from the Korea National Health and Nutrition Examination Survey	Health and nutritional status, including stress levels and other environmental factors like smoking	Smoking was considered among other environmental factors in the multivariate regression analysis but was not directly highlighted in the context of LBP and stress correlation results	The study found a significant association between stress levels and chronic LBP, with higher odds of chronic LBP as stress severity increased, even after adjusting for factors including smoking
Depintor *et al.* [19]	826 participants	HADS, EuroQol-5D, AUDIT, Fagerström test, Brazilian economic classification	Smokers had a 41% higher likelihood of experiencing chronic back pain compared to non-smokers (PR 1.41, 95% CI: 1.06-1.88, p = 0.031)	Chronic spinal pain was associated with factors like gender, age, education level, anxiety symptoms, physical exertion and smoking, with smoking being a significant factor
Green *et al.* [20]	34,525 U.S. adults	Back pain prevalence among different smoking statuses, number of cigarettes smoked	Current smokers had the highest prevalence of back pain (36.9%), followed by former smokers (33.1%) and never-smokers (23.5%)	There is a significant association between back pain and smoking status, with increased back pain correlating with higher smoking exposure
Schembri *et al.* [21]	120 chronic LBP patients, 50 control subjects	Pain levels, DN4, STarT Back, Fagerström scores	Smokers reported higher pain and DN4 scores. Smoking intensity linked with increased chronic LBP and neuropathic leg pain risk	Smoking intensity is closely related to the severity and risk of chronic LBP
Schembri *et al.* [22]	150 patients with chronic LBP (mean age: 60.1) and a control group (50 participants)	Demographics, IASP neuropathic pain grade, StarT Back tool, Fagerström test	Significant differences found between current smokers and nonsmokers in chronic LBP group; higher odds of chronic LBP and sciatica among smokers; increased Fagerström scores correlated with higher likelihood of chronic LBP and neuropathic pain	Current smokers have a substantially higher risk of chronic LBP and sciatica, as well as higher rates of neuropathic pain. Fagerström score is positively associated with chronic pain severity
Xu *et al.* [23]	438,510 UK Biobank participants initially free of back pain (2006-2010)	Smoking status (SS), cigarettes per day (CPD), pack-years (PY), back pain incidence	Former and current smokers had higher back pain incidence than never-smokers, with hazard ratios (Hrs) increasing with CPD and PY; female smokers at higher risk than male smokers	Smoking is associated with an increased risk of developing back pain, with greater risk correlated with higher smoking intensity and duration. Reducing or quitting smoking can significantly lower the risk of back pain
Yang *et al.* [24]	54 CLBP patients, categorized by smoking status	Pain, function, psychological health and smoking habits	A rise in daily cigarettes smoked was connected with increased pain, depression and work impact	Heavier smoking correlates with worse pain, psychological distress and quality of life in CLBP sufferers

prevalence of back pain at 36.9%, followed by 33.1% for former smokers and 23.5% for never smokers. This nested pattern of observation demonstrates a dose-response effect of smoking to back pain incidence and positions current smoking as a stronger risk factor.

In the study by Schembri *et al.* [21], smokers experienced higher pain intensity and Douleur Neuropathique 4 (DN4) score, a neuropathic pain measure. This suggests the correlation between smoking intensity and increased risk for chronic Low Back Pain (LBP) and neuropathic leg pain and that higher smoking intensity has the potential to worsen pain symptoms. Schembri *et al.* [22] also found a difference between active smokers and non-smokers in the chronic LBP population. Smokers were more likely to experience chronic LBP and sciatica, with higher Fagerström scores being correlated with having a higher chance of chronic LBP and neuropathic pain. This highlights the deleterious effects of smoking on the prevalence and severity of chronic LBP and related neuropathic disorders.

Xu *et al.* [23] showed that ex- and current smokers had a higher prevalence of back pain compared to never-smokers. Hazard ratios (HRs) for back pain rose with Cigarettes per Day (CPD) and Pack-Years (PY) intake, more so in female smokers, who were at higher risk than male smokers. This again supports the evidence that smoking is a modifiable risk factor for causation of LBP. Yang *et al.* [24] found that higher

Table 5: GRADE assessment observations

Research design	Quantity of investigations	Common observations	Bias risk	Heterogeneity	Relevance	Accuracy	Additional considerations	Evidence level
Cross-sectional	7	Various factors are associated with chronic LBP, with smoking being inconsistently reported as significant	Low to moderate	Low	Direct	Moderate	None	Moderate
Cohort	1	Smoking is associated with an increased risk of developing back pain over time	Low	N/A	Direct	High	None	High

daily cigarette intake was linked with higher pain, depression and work impairment in CLBP patients. This indicates that not only is smoking linked with the severity of pain endured but that it also has far-reaching effects on mental health and daily function.

GRADE Assessment Observations

Table 5 summarizes the GRADE certainty assessment for the studies included in the review. The body of evidence comprises seven cross-sectional studies and one cohort-based study, with the overarching inference being that a multitude of factors influence CLBP, with the role of smoking being variably significant. The risk of bias across most studies was considered 'low to moderate,' reflecting a generally robust methodological approach, although some concerns may still potentially affect the validity of the findings. The inconsistency was rated as 'low,' indicating that findings across the studies were sufficiently similar to suggest a degree of reliability.

The directness of the evidence was considered high, as the studies directly addressed the research question regarding factors associated with chronic LBP. The precision of the evidence was rated as 'moderate' for the cross-sectional studies, suggesting some uncertainty in the effect estimates, possibly due to variability in study sample sizes or measurement approaches. There were no additional considerations that affected the certainty of the evidence, such as publication bias, which was not reported. Therefore, the certainty of the evidence from the cross-sectional studies was rated as 'moderate,' while the evidence from the cohort study was rated as 'high,' given the longitudinal design's strength in establishing temporal associations.

DISCUSSION

The work of Depintor *et al.* [19], Green *et al.* [20], Schembri *et al.* [21], Schembri *et al.* [22], Xu *et al.* [23] and Yang *et al.* [24] all classify smoking as a causative risk factor in LBP causation and exacerbation, with the risk graded along intensity and longevity of smoking. These articles, to varying degrees, agree with one another on the conclusion that smoking is a modifiable LBP risk factor. Beyera *et al.* [17] and Choi *et al.* [18], however, deviate more from this group of articles in their attribution of lower importance to smoking and higher to other stress-associated and psychosocial determinants. Beyera *et al.* [17] defied the norm in that the article did not mention smoking as a common cause linked to the chronicity of LBP. Rather, psychosocial aspects like negative ideas about pain, general health state and depressive mood were singled out. Contrary to the majority of articles in the list, all which mentioned smoking as a major contribution to LBP, this differed.

Choi *et al.* [18], however, did find smoking in their multivariate analysis but eventually emphasized stress as the more significant factor in the context of chronic LBP. The emphasis of the study on stress is aligned with Beyera *et al.* [17] in the emphasis on non-smoking-related variables, which places both studies in opposition to the others that isolated smoking as a significant variable. Depintor *et al.* [19] and Green *et al.* [20] were in close agreement with their findings, with both studies finding smoking as a significant factor that was linked with chronic spinal pain and back pain, respectively. Green *et al.* [20] further quantified this, with a dose-response relationship between higher smoking exposure and higher back pain. These studies concur with the findings of Xu *et al.* [23], which also arrived at an association between smoking intensity, duration and risk of developing back pain.

Schembri *et al.* [21] and Schembri *et al.* [22] reported results with high levels of concordance, both showing the high correlation between smoking and severity of chronic LBP. Schembri *et al.* [22] also widened the scope by correlating the Fagerström score with greater severity of chronic pain, thereby proposing a causal relationship between nicotine dependence and severity of pain. Xu *et al.* [23] shared the universality of the studies by Green *et al.* [20], Schembri *et al.* [21] and Schembri *et al.* [22] in the identification of the causative role of smoking in back pain. Xu *et al.* [23] added the longitudinal aspect to the studies with a focus on the possible benefits of smoking cessation or reduction in the prevention of back pain. Yang *et al.* [24] offered a viewpoint that not only shares universality with the evidence of the relationship between smoking and severity of pain but also widens the scope by correlating heavier smoking with wider implications such as psychological distress and compromise of quality of life among chronic LBP patients.

Dai *et al.* [25] in their review had also reported that current smoking was linked with a higher risk of CMP, but interestingly, the strength of association was weaker for past smoking and ever-smoking. This indicates an active and continuous role of smoking in aggravating pain conditions, consistent with the findings of Green *et al.* [20], Schembri *et al.* [21] and Yang *et al.* [24] in our review that

identified a gradient effect with increasing back pain corresponding to increased exposure to smoking and more smoking resulting in poorer outcomes in patients with chronic LBP. But Dai *et al.* [25] also observed that in certain strata, current smoking was not significant and there was even a negative association between cigarette smoking and the risk of knee pain. This is a more subtle finding that is opposite to the overall trend in our review where smoking was largely linked with poor pain outcomes.

Shiri *et al.* [26] noted the increased incidence and prevalence of LBP attributable to current smoking over different periods of time, from the previous month to consultation for chronic and disabling LBP. They noted that the prevalence of LBP was increased among former smokers compared with never smokers but was less than among current smokers, suggesting a dose-response association. This concurs with the findings of Green *et al.* [20], Schembri *et al.* [21] and Yang *et al.* [24], who all found a dose-response association and emphasized the deleterious effect of smoking on LBP. Furthermore, Shiri *et al.* [26] emphasized that the association between current smoking and LBP incidence was stronger among young people compared with adults. This specific age-related finding was not specifically noted in our review findings. However, the longitudinal method used by Xu *et al.* [23] in our review does echo the concept that smoking is associated with an increased risk of the development of back pain over a period of time, which could suggest an accumulating effect that could be more clearly observed when the onset of smoking is at a younger age.

Literature review shows that comparison with the study of Çelik *et al.* [27] is most appropriate. Their results showed that those with an indication of positive DN4 score, suggesting neuropathic pain, had greater cigarette use compared with those with a negative DN4 score. This is consistent with our results, which showed an increase in the mean overall DN4 score with increased daily cigarette use ($p = 0.002$). Differences between study design, including presentation of findings and comparison groups used, may account for the slightly different findings. Shemory *et al.* [28] showed a high relative risk for Low Back Pain (LBP) with nicotine dependence, but did not separate acute and chronic LBP and did not derive their diagnostic criteria for nicotine dependence, so direct comparison with our findings is not easy.

Shaw *et al.* [29] investigated men with acute LBP and identified increased risk of chronicity with nicotine dependence. Their employment of the Diagnostic Interview Schedule-III-R to determine nicotine dependence and investigation of primary LBP symptoms, however, differ from the methodology and goal of our investigation of chronic LBP and direct comparison is not possible. Zvolensky *et al.* [30] similarly identified increased risk of chronic neck or back pain in individuals with nicotine dependence, but employed the Composite International Diagnostic Interview to measure dependence and direct comparison is not possible.

In adjusting for smoking status, reviews from various authors [31-36] examined the interaction between LBP, sciatic pain and smoking but the lack of standardization of the definition of sciatica and the failure to examine a neuropathic pain component in the studies makes it hard to make direct comparison. This was actually noted by Cook *et al.* [34], who found that inconsistent definitions of sciatica might have a huge impact on the result of such studies. Parreira *et al.* [31] highlighted in their systematic review chronic LBP in twins, which found a significant association between smoking and LBP, similar to our study. The specificity of these findings to chronic LBP was, however, not stated explicitly and the definition of the most chronic symptom of LBP may influence the interpretation.

Clinical recommendations and future implications

The overall assessments obtained from this study underscored and emphasized the need to heed and attend to smoking as another crucial modifiable risk factor while treating and preventing CLBP. Evidence shows that this element not only predisposes but also worsens the suffering and impairments in all connected psychological and functional well-being components involved in CLBP. A well-coordinated intervention to reduce this prevalence of smoking among such at-risk individuals for and with CLBP is urged. Strategies for smoking cessation should be an element of holistic programs of pain management in populations with higher smoking intensity and longer durations, which are also elements of public health campaigns and clinical guidelines. This heterogeneity in regional, demographic and methodological factors across studies suggests the need for contextualized approaches in designing interventions. Further research to strengthen the causal inferences may involve further study on the dose-response relation between smoking and CLBP, specifically using longitudinal data. Other possible approaches might relate to understanding the interaction between smoking with other lifestyle or psychosocial factors so that their joint effect can more adequately elucidate its involvement in the pathophysiology of CLBP.

Clinicians should include assessments of smoking in routine evaluations of patients with CLBP and consider using validated measures to quantify intensity and dependence. Perhaps optimal patient outcomes will be attained through the collaborative work of the healthcare providers, pain specialists and smoking cessation programs attacking both the behavioral and the physical aspects of CLBP. Future studies should work towards bridging gaps in accuracy and explain mechanistic pathways through which smoking contributes to the induction and maintenance of pain, which may result in targeted pharmacologic or behavioral therapies.

CONCLUSION

The aggregate evidence from the reviewed papers suggests a significant correlation between smoking and the prevalence, severity and persistence of CLBP. Notably, the data pointed toward a dose-response relationship, where increased smoking intensity and duration were linked to heightened risk and exacerbation of CLBP symptoms. The findings indicated that smoking might not only be a contributing factor in the development of CLBP but also in the worsening of pain over time. The review revealed a consistent trend across diverse populations and methodologies, reinforcing the potential role of smoking as a risk factor for CLBP. The analyses also highlighted the multifactorial nature of CLBP, with psychosocial elements such as stress and depressive symptoms demonstrating a considerable influence on the chronicity of back pain. The potential for reverse causation, where the experience of CLBP could lead to increased smoking due to stress or coping mechanisms, was acknowledged, although not definitively resolved within the scope of the reviewed literature. Furthermore, while smoking emerged as a significant factor in the context of CLBP, the review's findings underscored the importance of considering a holistic approach that addresses both smoking cessation and the psychological aspects of chronic pain management.

Limitations

The limitations of this systematic review are primarily influenced the interpretation of the findings. One of the principal limitations was the decision to include studies published from 2014 onwards. This temporal restriction potentially excluded relevant earlier research that might have provided additional insights into the long-term trends and evolution of understanding regarding the association between smoking and CLBP. By focusing on more recent studies, the review may have overlooked foundational work or shifts in research paradigms that could inform current hypotheses and analytical frameworks. Furthermore, the heterogeneity in study designs, populations and methodological approaches made it challenging to compare results across studies directly. While the review attempted to synthesize available data, the variations in measurement tools for smoking and pain, as well as differences in controlling for confounding factors, may have contributed to inconsistent findings. For instance, the extent to which psychosocial factors were considered and adjusted for varied among the studies, as evidenced by the emphasis on such factors in the studies by Beyera *et al.* [17] and Choi *et al.* [18] as opposed to those where smoking emerged as a significant factor. This heterogeneity underscores the complexity of CLBP and suggests that its relationship with smoking may be influenced by an intricate interplay of biological, psychological and social elements. Additionally, the reliance on self-reported measures for smoking status and pain intensity in some studies could have introduced bias due to recall or reporting inaccuracies. Objective measures of smoking exposure and clinical assessments of pain would have strengthened the review's findings.

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