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Correlation Between Lipid Profiles and Chronic Low Back Pain: A Systematic Review

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Abstract

Background: Chronic low back pain (CLBP) is a leading cause of disability worldwide. Emerging evidence suggests that metabolic factors, particularly lipid profile abnormalities, may contribute to the pathogenesis of CLBP through vascular, inflammatory, and degenerative mechanisms.

Objective: To systematically review the association between lipid profiles and chronic low back pain.

Methods: A systematic review was conducted in six electronic databases (PubMed, Scopus, Web of Science, Embase, Cochrane Library, and Google Scholar) following PRISMA 2020 guidelines. Observational and genetic epidemiological studies published between 2020 and 2025 examining lipid parameters (total cholesterol, LDL-C, HDL-C, triglycerides, and lipid subclasses) in adults with CLBP were included. Study quality was appraised using a STROBE-based checklist.

Results: Ten studies met the inclusion criteria. Large population-based studies demonstrated that low HDL-C levels and elevated LDL-C/HDL-C ratios were associated with an increased risk of CLBP (odds ratios ranging from approximately 1.15 to 1.34). Mendelian randomization analyses consistently indicated a protective causal effect of genetically higher HDL-C on low back pain risk (OR ≈ 0.92). Lipidomic studies further revealed increased monoacylglycerol and diacylglycerol levels, alongside reduced triacylglycerol levels, suggesting altered lipid metabolism and low-grade inflammation in CLBP.

Conclusion: Dyslipidemia, particularly low HDL-C and atherogenic lipid patterns, is consistently associated with chronic low back pain. Lipid profiles may serve as complementary risk markers in CLBP assessment and support a multidisciplinary management approach integrating metabolic, biomechanical, and lifestyle interventions.

Keywords
Lipid Profile; Low Back Pain; Dyslipidemia; Systematic Review

Introduction

Chronic low back pain (CLBP) is one of the most prevalent musculoskeletal health problems among adults worldwide, affecting both developed and developing countries. CLBP is commonly defined as pain localized in the lumbar region persisting for more than 12 weeks, with varying intensity and frequently accompanied by functional limitations.[1] This condition not only compromises physical functioning but also adversely affects psychological well-being, work productivity, and overall quality of life. Epidemiological evidence consistently identifies CLBP as one of the leading causes of years lived with disability and work absenteeism, resulting in a substantial socioeconomic burden on individuals, families, and healthcare systems worldwide.[2]

Previous research on CLBP has primarily emphasized mechanical factors, such as posture, occupational physical load, and musculoskeletal injury, alongside psychosocial determinants including stress, depression, and job satisfaction. These perspectives have informed a wide range of prevention and management strategies, including physiotherapy-based exercise programs, ergonomic interventions, and psychological therapies.[3,4] However, despite documented benefits in selected patient populations, recurrence rates and persistent symptoms remain relatively high.[5] This persistence suggests that CLBP is a multifactorial condition and that conventional biomechanical and psychosocial approaches alone may be insufficient to fully explain its pathogenesis and clinical course.[6]

In recent years, scientific attention has shifted from viewing CLBP solely as a localized mechanical disorder to conceptualizing it as a condition influenced by systemic biological processes, including low-grade inflammation and metabolic dysregulation. One emerging area of interest is the role of lipid metabolism and dyslipidemia in chronic musculoskeletal pain conditions, including CLBP.[7] A standard lipid profile typically includes total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides, which are well-established markers of cardiovascular and metabolic health. Increasing evidence suggests that abnormalities in these lipid parameters may also modulate inflammatory pathways, tissue degeneration, and nociceptive processing, thereby contributing to the development and persistence of chronic pain states.[8,9]

Several biological mechanisms have been proposed to explain the association between dyslipidemia and spinal degeneration. Abnormal lipid profiles may promote atherosclerotic changes in the small blood vessels supplying the vertebral bodies and intervertebral discs, leading to impaired perfusion and reduced nutrient exchange within disc tissues.[7] These vascular disturbances may accelerate disc degeneration, a key structural contributor to CLBP. Moreover, elevated LDL-C and triglyceride levels, combined with reduced HDL-C, are closely linked to chronic systemic inflammation characterized by increased pro-inflammatory cytokine activity.[10] Such inflammatory processes may exacerbate both peripheral and central sensitization to pain. Dyslipidemia is also frequently associated with obesity and metabolic syndrome, conditions that further increase CLBP risk through excessive mechanical loading and inflammatory pathways.[8]

A growing body of observational and population-based studies has examined the relationship between lipid profiles and CLBP, yielding mixed results. Several investigations report that individuals with elevated triglyceride and LDL-C levels have a higher likelihood of experiencing CLBP compared with those exhibiting normal lipid profiles.[11,12] Conversely, low HDL-C levels have been

associated with increased pain complaints and reduced functional capacity in some populations.[13] However, other studies have reported weak or nonsignificant associations after adjusting for potential confounders such as age, body mass index, and lifestyle factors. These inconsistencies may reflect heterogeneity in pain definitions, lipid measurement techniques, covariate adjustment strategies, and study designs, underscoring the need for a comprehensive synthesis of contemporary evidence.[14]

Despite increasing interest in metabolic contributors to CLBP, no recent review has comprehensively synthesized epidemiological, genetic, and lipidomic evidence on the association between lipid profiles and chronic low back pain within a single analytical framework. Existing reviews predominantly focus on mechanical degeneration or inflammatory pathways without integrating findings from Mendelian randomization studies or lipidomic analyses that may clarify causal relationships and subclass-specific lipid effects. Furthermore, the lack of systematic integration across diverse study designs limits the translation of emerging metabolic evidence into clinical physiotherapy practice and multidisciplinary management strategies.

Therefore, this systematic review aims to synthesize contemporary evidence from observational epidemiological and genetic studies conducted in adult populations (Population) that examine lipid profile parameters, including conventional lipids and lipid subclasses (Exposure), and their associations with chronic low back pain, intervertebral disc degeneration, and related spinal outcomes (Outcome). By integrating findings across epidemiological, Mendelian randomization, and lipidomic research, this review seeks to clarify the strength, direction, and potential mechanisms underlying the relationship between lipid metabolism and CLBP, as well as to highlight implications for prevention and multidisciplinary clinical management.

Methods

Study Design

This study employed a systematic review design guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement.[15] The review aimed to systematically identify, appraise, and synthesize available evidence on the association between lipid profiles and chronic low back pain (CLBP). Although the findings were synthesized narratively, the overall methodological approach followed a systematic review framework, including predefined eligibility criteria, structured literature searches, and transparent study selection procedures.

Protocol and Registration

The review protocol was developed a priori to guide the research questions, eligibility criteria, and analytical approach. However, the protocol was not registered in an international prospective register such as PROSPERO. This review was conducted and reported in accordance with the PRISMA 2020 guidelines to ensure methodological transparency and reproducibility.[15]

Information Sources and Search Strategy

A comprehensive literature search was conducted in six electronic databases: PubMed, Scopus, Web of Science, Embase, the Cochrane Library, and Google Scholar. The search included peer-reviewed articles published between 2020 and 2025 in English or Indonesian. Keywords and Medical Subject Headings (MeSH) terms were combined using Boolean operators (AND, OR), including “chronic low back pain,” “low back pain,” “lipid profile,” “dyslipidemia,” “cholesterol,” “triglycerides,” “HDL,” and “LDL.” An example of the search strategy used in PubMed was as follows: (“chronic low back pain” OR “low back pain”) AND (“lipid profile” OR dyslipidemia OR cholesterol OR triglycerides OR HDL OR LDL). Comparable search strategies were adapted for the remaining databases.

Eligibility Criteria

Studies were included if they met the following criteria: (1) involved adult human participants aged ≥18 years; (2) examined individuals with chronic low back pain, defined as pain persisting for at least 12 weeks; (3) assessed lipid profile parameters, including total cholesterol, LDL-C, HDL-C, triglycerides, or other lipid subclasses; and (4) analyzed associations between lipid profiles and CLBP or related spinal degenerative outcomes. Eligible study designs included observational studies (cross-sectional, cohort, and case–control) and genetic epidemiological studies such as Mendelian randomization analyses.

Exclusion criteria comprised editorials, letters to the editor, conference abstracts without full data, case reports, narrative reviews, studies focusing exclusively on acute or subacute low back pain without differentiation of CLBP, and studies that did not explicitly evaluate both lipid profiles and CLBP outcomes.

Study Selection Process

All records identified through database searching were imported into a reference management system, and duplicate entries were removed. Titles and abstracts were then screened for relevance, followed by full-text assessment based on the predefined inclusion and exclusion criteria. The study selection process followed the PRISMA flow framework, encompassing identification, screening, eligibility assessment, and inclusion of studies in the final review.[15] Any uncertainties regarding study eligibility were resolved through discussion among the authors to reach consensus.

Quality Appraisal / Risk of Bias Assessment

The methodological quality of the included observational studies was appraised using a checklist adapted from the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. The appraisal focused on key domains, including clarity of study objectives, appropriateness of study design, participant selection, measurement of exposure and outcomes, control of confounding variables, and transparency of statistical analysis. Overall, most studies demonstrated moderate methodological quality, with common limitations related to cross-sectional design and residual confounding.

Data Extraction and Synthesis

Data were extracted systematically from each included study, encompassing author and publication year, study design, population characteristics, lipid parameters assessed, outcome measures related to CLBP, and principal findings. Given the heterogeneity of study designs, populations, and outcome measures, a meta-analysis was not feasible. Therefore, a systematic narrative synthesis was undertaken, grouping findings into thematic domains, including epidemiological associations, genetic causal evidence, lipidomic alterations, and clinical implications.

Results

Study Selection (PRISMA Flow)

The study selection process followed the PRISMA 2020 framework and is illustrated in Figure 1. A total of 540 records were identified through database searching. After the exclusion of 320 records that were not within the scope of the review, 220 articles remained for screening. Following title and abstract screening, 144 articles were excluded because they did not examine metabolic or lipid-related factors in chronic low back pain. Consequently, 76 full-text articles were assessed for eligibility. Of these, 66 articles were excluded due to irrelevance to lipid profile outcomes or failure to meet the inclusion criteria. Ultimately, 10 studies were included in the final qualitative synthesis.

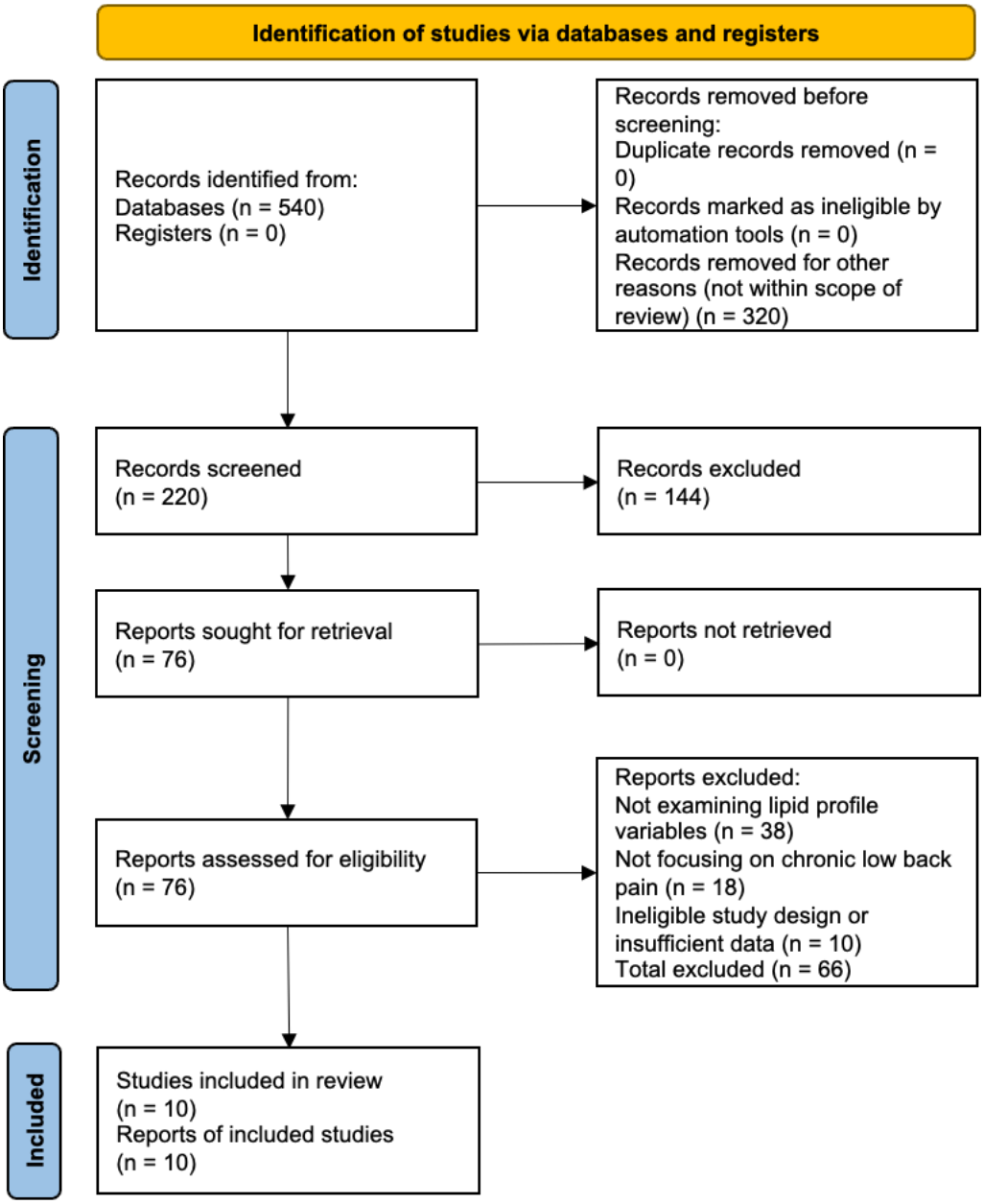

Figure 1. PRISMA 2020 flow diagram of study selection for the systematic review examining the association between lipid profiles and chronic low back pain.

Characteristics of Included Studies

The characteristics and main findings of the included studies are summarized in Table 1. The ten studies, published between 2020 and 2025, encompassed diverse methodological approaches, including large-scale population-based cross-sectional studies, retrospective clinical analyses, Mendelian randomization studies, metabolomic and lipidomic investigations, and descriptive clinical studies. Study populations ranged from small hospital-based cohorts to large national samples exceeding 250,000 participants. Across studies, lipid parameters assessed included conventional serum lipids (total cholesterol, LDL-C, HDL-C, and triglycerides), lipid ratios (LDL-C/HDL-C), and advanced lipid subclasses identified through lipidomic profiling.

Table 1. Summary of Included Studies Examining the Association Between Lipid Profiles and Chronic Low Back Pain

No.	Author & Year	Study Design	Lipid Parameters	Main Findings	Clinical / Scientific Implications
1	Liu et al., 2024 [16]	Drug-target Mendelian randomization (NPC1L1, PCSK9, HMGCR) + NHANES cross-sectional analysis	TC, LDL-C, non-HDL-C	Non-statin users had a higher risk of LBP (OR 1.29). Genetic reduction of TC/LDL-C via NPC1L1 was associated with reduced IVDD risk, while PCSK9 pathways were linked to lower sciatica risk.	Suggests that lipid-lowering mechanisms, particularly NPC1L1 and PCSK9, may reduce spinal degeneration risk; evidence for HMGCR/statins remains inconclusive.
2	Huang et al., 2022 [10]	Retrospective clinical study (n = 302)	TC, TG, LDL-C, HDL-C, LDL-C/HDL-C ratio	In patients without comorbidities, age, HDL-C, and TG were major contributors to disc and facet degeneration severity; in patients with comorbidities, age and BMI predominated.	Dyslipidemia contributes to degeneration severity but is not a sole determinant; metabolic factors should be addressed alongside mechanical risk factors.
3	Kim & Lee, 2024 [11]	Cross-sectional (KNHANES 2010–2012; n = 5,961 women)	Clinical dyslipidemia status	Dyslipidemia was significantly associated with CLBP (OR 1.32; 95% CI 1.14–1.53) after adjustment; hypertension and diabetes were not significant.	Highlights dyslipidemia as an independent metabolic risk factor for CLBP in women.
4	Luo et al., 2024 [1]	Two-sample Mendelian randomization	Genetically predicted HDL-C, LDL-C, TG	Higher genetically predicted HDL-C was causally associated with reduced LBP risk (OR ≈ 0.92); no causal effect for LDL-C or TG.	Supports a protective causal role of HDL-C in low back pain.
5	Bruthans et al., 2023 [17]	Observational clinical study (n = 89)	TC, LDL-C, HDL-C, TG	No overall correlation between lipid levels and pain intensity; correlations with TC and LDL-C emerged only in patients with a positive doctor–patient relationship.	Suggests interaction between biological markers and psychosocial context in pain perception.
6	Pang et al., 2025 [18]	Mendelian randomization with lipidomic features (GeneRISK + FinnGen)	179 lipidomic features across 13 lipid classes	Several phosphatidylcholines, triglycerides, and sterol esters increased IVDD/LBP risk, while certain phosphatidylethanolamines and sphingomyelins were protective.	Demonstrates subclass-specific lipid effects and supports lipidome-based biomarkers.
7	Tang et al., 2024 [19]	Metabolomic & lipidomic case–control study (30 CLBP vs 26 controls)	MAG, DAG, TAG, PS, multiple lipid classes	CLBP patients showed increased MAG, DAG, PS and decreased TAG levels.	Indicates altered lipid metabolism and low-grade inflammation in chronic nonspecific LBP.
8	Zulfikar et al., 2024 [20]	Descriptive clinical case study	Serum LDL-C	Lumbar flexibility improved after Mobilization With Movement + McKenzie exercises despite elevated LDL-C.	Physiotherapy interventions remain effective despite metabolic risk factors.
9	Yoshimoto et al., 2020 [12]	Large cross-sectional study (n = 258,367)	LDL-C, HDL-C, LDL-C/HDL-C ratio	Low HDL-C and high LDL-C/HDL-C ratio were associated with LBP (OR range 1.15–1.34); LDL-C alone was not consistently significant.	Strong epidemiological support for the atherogenic lipid–LBP hypothesis.
10	Ogon et al., 2020 [21]	Cross-sectional imaging study (MRI + ^{1}H-MRS)	IMCL, EMCL	EMCL correlated with age, BMI, muscle degeneration, and spinopelvic alignment, but not pain intensity.	Suggests muscle fat infiltration reflects degeneration rather than pain severity.

Summary of Main Findings

As shown in Table 1, a relatively consistent pattern emerged indicating that atherogenic lipid profiles are associated with chronic low back pain and related spinal degenerative conditions. Large population-based studies reported that low HDL-C levels and elevated LDL-C/HDL-C ratios were significantly associated with an increased risk of low back pain, with adjusted odds ratios generally ranging from approximately 1.15 to 1.34.[12] In contrast, elevated LDL-C alone did not consistently remain significant after full adjustment for confounding factors.

Genetic epidemiological evidence further strengthened these findings**.** Mendelian randomization analyses demonstrated a protective causal effect of genetically higher HDL-C levels on low back pain risk (OR ≈ 0.92)**,** while genetically predicted LDL-C and triglyceride levels showed no consistent causal association with low back pain. Drug-target Mendelian randomization studies also indicated that genetic modulation of cholesterol-lowering pathways involving NPC1L1 and PCSK9 was associated with a reduced risk of intervertebral disc degeneration and sciatica.[22]

Lipidomic and metabolomic studies provided additional mechanistic insights. Patients with chronic nonspecific low back pain exhibited increased levels of monoacylglycerols (MAG), diacylglycerols (DAG), and phosphatidylserines (PS), alongside reduced levels of triacylglycerols (TAG).[19] Subclass-specific lipid analyses further identified certain phosphatidylcholines and sterol esters as risk-enhancing lipid species, whereas selected phosphatidylethanolamines and sphingomyelins appeared to exert protective effects.[18]

Structural Degeneration and Muscle-Related Findings

Several studies indicated that the relationship between lipid profiles and chronic low back pain is more strongly reflected in structural and degenerative changes rather than direct pain intensity. Dyslipidemia, particularly variations in HDL-C and triglyceride levels, was associated with the severity of intervertebral disc and facet joint degeneration, although age and body mass index remained the dominant contributors.[10] Other investigations focusing on muscle composition demonstrated that extramyocellular lipid (EMCL) content in the psoas major muscle was more closely associated with aging, adiposity, reduced muscle cross-sectional area, and altered spinopelvic parameters than with pain intensity itself**.** [21] These findings suggest that lipid-related metabolic disturbances may contribute to CLBP indirectly by promoting degenerative and biomechanical vulnerability rather than acting as immediate determinants of pain severity.

Clinical Intervention Context

Clinical evidence further indicates that metabolic risk factors do not preclude functional improvement through physiotherapy interventions. A descriptive clinical study reported that a combination of Mobilization with Movement and McKenzie exercises resulted in clinically meaningful improvements in lumbar flexibility in an older female patient with CLBP and elevated LDL-C levels.[20] This finding underscores that while dyslipidemia may increase biological vulnerability to degeneration, mechanical and exercise-based interventions remain effective components of CLBP management, reinforcing the multifactorial nature of the condition.

Discussion

Principal Findings

Based on the systematic synthesis of ten studies included in this review (Figure 1 and Table 1), the findings indicate that lipid profiles are meaningfully associated with chronic low back pain (CLBP), although the nature of this relationship is complex and multifactorial. Large-scale epidemiological studies consistently demonstrated that an atherogenic lipid pattern—particularly low HDL-C levels and elevated LDL-C/HDL-C ratios—was associated with an increased risk of low back pain, even after adjustment for major confounding factors such as age, body mass index, and lifestyle behaviors.[12] These associations were not uniformly observed for LDL-C alone, suggesting that lipid balance and ratios may be more informative than isolated lipid parameters.

Genetic epidemiological evidence further strengthened these observations. Mendelian randomization studies summarized in Table 1 demonstrated a protective causal effect of genetically higher HDL-C on low back pain risk (OR ≈ 0.92), supporting a potential biological role of HDL-C beyond its cardiovascular benefits. Drug-target Mendelian randomization analyses additionally indicated that genetic modulation of cholesterol-lowering pathways involving NPC1L1 and PCSK9 was associated with a reduced risk of intervertebral disc degeneration and sciatica.[16] Collectively, these findings suggest that dyslipidemia may contribute not only to systemic vascular disease but also to degenerative processes affecting the lumbar spine.

Biological Mechanisms Linking Dyslipidemia and CLBP

The observed associations between lipid profiles and CLBP are biologically plausible and can be interpreted within a vascular–degenerative framework**.** Dyslipidemia may promote atherosclerotic changes in the microvasculature supplying the vertebral bodies and intervertebral discs, resulting in impaired perfusion and reduced nutrient delivery to disc tissues.[7] Such vascular insufficiency may accelerate disc degeneration, a well-recognized structural contributor to CLBP.

Beyond vascular mechanisms, lipid abnormalities are closely linked to chronic low-grade inflammation. Elevated LDL-C and triglyceride levels, combined with reduced HDL-C, are associated with increased pro-inflammatory cytokine activity, oxidative stress, and endothelial dysfunction.[23] These inflammatory processes may exacerbate both peripheral and central sensitization, thereby lowering pain thresholds and perpetuating chronic pain states.[24] Lipidomic findings summarized in Table 1 further support this interpretation, as patients with chronic nonspecific low back pain exhibited increased monoacylglycerol, diacylglycerol, and phosphatidylserine levels, alongside reduced triacylglycerol levels, indicating altered lipid metabolism and inflammatory signaling.[18,19]

Structural Degeneration and Muscle-Related Considerations

Several studies included in this review suggest that lipid-related metabolic disturbances may be more strongly associated with structural degeneration than with pain intensity per se. Retrospective clinical analyses demonstrated that dyslipidemia—particularly variations in HDL-C and triglyceride levels—contributed to the severity of intervertebral disc and facet joint degeneration, although age and obesity remained dominant determinants.[10] Similarly, imaging studies examining muscle composition revealed that extramyocellular lipid accumulation in the psoas major muscle correlated with aging, higher body mass index, reduced muscle cross-sectional area, and altered spinopelvic alignment, rather than with pain severity itself.[21]

These findings imply that dyslipidemia may increase biomechanical vulnerability by compromising disc integrity and muscle quality, thereby predisposing individuals to CLBP when combined with mechanical stressors such as poor posture, physical inactivity, or occupational loading.[24,25] Thus, lipid abnormalities may act as upstream modifiers of spinal health rather than direct determinants of pain perception.

Interaction Between Metabolic, Mechanical, and Psychosocial Factors

The results summarized in Table 1 highlight that CLBP cannot be explained solely by metabolic or biological markers. One included clinical study demonstrated that correlations between serum lipid levels and pain intensity emerged only in patients reporting a positive doctor–patient relationship, underscoring the influence of psychosocial context on pain reporting and perception.[17] This observation aligns with contemporary biopsychosocial models of chronic pain, which emphasize that biological vulnerability interacts dynamically with psychological and social factors.

Importantly, metabolic risk factors did not preclude functional improvement through physiotherapy interventions. A descriptive clinical study reported clinically meaningful improvements in lumbar flexibility following a combination of Mobilization with Movement and McKenzie exercises in an older patient with CLBP and elevated LDL-C levels.[20,26] These findings reinforce the multifactorial nature of CLBP and suggest that effective management should integrate metabolic risk modification with biomechanical and psychosocial interventions.

Clinical Implications

From a clinical perspective, the findings of this systematic review support the inclusion of lipid profile assessment as part of a comprehensive evaluation in patients with CLBP, particularly those with metabolic risk factors such as obesity or sedentary lifestyles. The consistent association between low HDL-C, unfavorable lipid ratios, and CLBP observed across epidemiological and genetic studies (Table 1) suggests that lipid profiles are not neutral with respect to spinal health.

However, lipid abnormalities should not be interpreted as deterministic predictors of pain. Instead, they may serve as complementary risk markers that inform preventive strategies and multidisciplinary management. Lifestyle interventions aimed at improving lipid profiles—such as regular aerobic physical activity, weight management, and dietary modification—may offer additional benefits when combined with physiotherapy-based exercise programs and postural correction.[27] These recommendations should be interpreted cautiously, as the current evidence base is largely observational.

Study Limitations and Strength of Evidence

This systematic review has several limitations that should be considered when interpreting the findings. First, the predominance of cross-sectional study designs among the included studies limits causal inference. Although Mendelian randomization studies provide stronger causal evidence, their findings are dependent on genetic assumptions that may not fully capture complex environmental interactions. Second, heterogeneity in CLBP definitions, lipid measurement methods, and covariate adjustment strategies across studies may have influenced effect estimates and contributed to variability in reported associations. Additionally, publication bias cannot be excluded, as studies reporting null associations may be underrepresented in the literature. These limitations suggest that while the overall direction of evidence supports an association between dyslipidemia and CLBP, the strength of recommendations for clinical practice should remain moderate.

Conclusion

This systematic review demonstrates that lipid profiles are meaningfully associated with chronic low back pain (CLBP), particularly through an atherogenic pattern characterized by low high-density lipoprotein cholesterol (HDL-C), unfavorable LDL-C/HDL-C ratios, and alterations in specific lipid subclasses. Evidence synthesized from epidemiological studies, Mendelian randomization analyses, and metabolomic and lipidomic investigations consistently supports the role of dyslipidemia in increasing the risk of CLBP, intervertebral disc degeneration, and related spinal conditions. Nevertheless, age, obesity, and mechanical factors remain primary determinants of disease onset and progression.

Importantly, the relationship between lipid metabolism and CLBP appears to be indirect and multifactorial, mediated by vascular insufficiency, low-grade inflammation, structural degeneration, alterations in muscle composition, and psychosocial factors. A more favorable lipid profile—particularly higher HDL-C levels and protective lipid subclasses—may confer resilience against degenerative spinal changes, although lipid parameters alone do not fully explain pain intensity or clinical outcomes.

From a clinical perspective, these findings suggest that lipid profile assessment may serve as a complementary component of comprehensive CLBP evaluation, especially in individuals with metabolic risk factors. Integrating lipid management strategies, such as lifestyle modification and cardiovascular risk reduction, with physiotherapy-based biomechanical interventions and psychosocial support may enhance prevention and management outcomes. However, given the predominance of observational evidence, these conclusions should be interpreted with caution, and further longitudinal and interventional studies are required to establish causality and inform evidence-based clinical guidelines.

Author Contribution

Sulista Putri: Conceptualization, methodology, literature search, data extraction, data synthesis, and manuscript drafting.
Dwi Rosella Komalasari: Supervision, guidance on study design, critical revision of the manuscript, and methodological consultation.
Farid Rahman: Supervision, validation of findings, and manuscript editing.
All authors reviewed and approved the final version of the manuscript.

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Conflict of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this article.

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Ethics Statement

Ethical approval was not required for this study, as it was based exclusively on the analysis of previously published literature and did not involve direct interaction with human participants or access to identifiable individual data.

Data Availability Statement

All data analyzed in this study are derived from published articles included in the systematic review. Further details are available from the corresponding author upon reasonable request.

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