

Family History of Low Back Pain is Associated with occurrence of Low Back Pain, Poor Quality of Life, and Lumbar Morphometric Changes: A Cross-Sectional Study

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Abstract

The relationship between family history of low back pain (LBP), health status and spinal radiological features is unclear yet important. From December 2021 to July 2024, this cross-sectional study analyzed 811 participants (392 without and 419 with a family history of LBP) to explore the relationship between family history of LBP, LBP intensity, disability (Roland–Morris Disability Questionnaire [RMDQ]), life quality (the Short Form-36 [SF-36]) and morphological changes (intervertebral disc degeneration [IDD], facet joint degeneration [FJD], modic changes [MDs], and Spondylolisthesis), using logistic and linear regression models. Results indicated family history of LBP was significantly associated with a higher odds of LBP (OR 1.91, 95% CI 1.42, 2.57, $p < 0.01$), lower quality of life (B -3.52, 95% CI -5.79, -1.25, $p < 0.01$), more severe intervertebral disc (B 0.53, 95% CI 0.13, 0.93, $p = 0.01$) and facet joint degeneration (B 0.41, 95% CI 0.14, 0.69, $p < 0.01$) than those without, especially if multiple family members were affected (LBP risk: OR 3.41; SF36:B -7.03; IDD:B 0.92; FJD:B 0.76; all $p < 0.01$). In conclusion, a family history of LBP, especially involving multiple relatives, correlates with increased LBP prevalence, poorer quality of life, and more severe spinal degeneration.

Introduction

Low back pain (LBP) exhibits a remarkably high prevalence worldwide and ranks among the top contributors to disability¹. Furthermore, 13% transition to chronic LBP, with one third of these individuals experiencing moderate to severe levels of burden due to chronic LBP^{2,3}. Once back pain becomes chronic, it can lead to worsened physical function, mental health issues, and substantial healthcare cost⁴. Considering the increasing prevalence of LBP, it has become a crucial area of research.

The etiology of LBP is inherently complex, with genetic factors suggested to play an important role in its development^{5,6}. Prior studies revealed that individuals with family history of LBP are at a higher risk of developing LBP themselves^{7–9}. Focusing on a female cohort, Livshits et al. observed elevated LBP concordance in twin pairs through a cross-sectional design¹⁰. Similarly, Masiero et al., carried out an observational study, revealing a strong association between positive family history and the prevalence of LBP in adolescents¹¹. However, detailed investigations into the relationship between family history of LBP, LBP characteristics, and health indicators remain insufficient.

Imaging of the lumbar spine is used as tool in the evaluation and management of LBP. Previous studies have suggested that certain lumbar morphology abnormalities serve as the source of LBP, including intervertebral disc degeneration (IDD), facet joint degeneration (FJD), modic changes (MCs), among others^{12–15}. The relationship between family history of LBP and these lumbar morphometric changes has not been well studied. However, previous studies indicate that genetic factors or family history of LBP play a significant role in IDD. This indicates that IDD may act as a pathway through which genetic influences contribute to LBP¹⁶.

Hence, this study examined how a familial LBP background relates to LBP intensity and health indicators, including disability and quality of life. In addition, we further assessed its potential link to structural spinal

degeneration.

We hypothesized that: i) individuals with a family history of LBP, particularly those with multiple affected relatives, would exhibit a significantly higher risk of experiencing LBP and poorer clinical health indicators compared to those without a family history; ii) a family history of LBP would be positively associated with greater morphology abnormalities, such as IDD, FJD, MCs and spondylolisthesis.

Materials and Methods

Study sample

This study was designed as a cross-sectional secondary analysis of the prospectively established Berlin Back Study (research unit FOR5177) and was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Adult participants recruited between December 2021 and July 2024 who reported available information on the LBP status of their immediate family members were included in this study. In the Berlin Back Study, individuals were not eligible if they had conditions that could interfere with spinal assessment, pain phenotyping, or study safety. These conditions included participation in professional or elite sports, acute infection, substance abuse, pregnancy, a BMI greater than 28 kg/m², central or peripheral neurological disorders, local inflammation or infection affecting the assessment region, vertebral fractures, osteoporosis, malignant disease or bone metastases, previous spinal surgery, treatment with medications likely to influence musculoskeletal function (e.g., antiepileptic agents, long-acting antihistamines, systemic glucocorticoids, or immunosuppressants), rheumatic or other active systemic diseases, severe visceral disorders posing a risk during testing, and lower-extremity deformities or conditions that could alter gait or spinal loading. For the present secondary analysis, participants were excluded if MRI data were unavailable, if information on family history of LBP was unclear, if key covariate information was missing (body mass index [BMI]), or if they had other types of back pain or an uncertain pain classification. Based on the criteria outlined above, 811 participants were included in the final study analysis. Further details are presented in **Figure-1**.

Ethics statement

All experimental protocols were approved by the Ethics Committee of Charité—Universitätsmedizin Berlin (approval number: EA1/059/21) and were conducted in strict accordance with the Declaration of Helsinki. Furthermore, written informed consent was obtained from all subjects before their inclusion in the research.

Questionnaire and Clinical Evaluation

All participant related data were anonymized. Data on individual characteristics were collected using a questionnaire that incorporated items related to demographic information, lifestyle habits and back pain conditions.

Clinical orthopedic physicians conducted anthropometric measurements of the spine and assessed back pain characteristics, including the presence or absence of back pain, primary or secondary pain locations, as well as the intensity of the pain. The recruited participants were categorized into two groups: those without back pain (no-BP) and those with identified pain primarily between the lower rib margin and the gluteal folds (LBP). The LBP group comprised participants with either chronic low back pain (cLBP), defined as daily pain persisting for more than 12 weeks, or intermittent low back pain (iLBP), defined as recurrent LBP with periods of complete remission. For the present analysis, participants with cLBP and iLBP were combined into a single LBP group, reflecting a symptomatic phenotype characterized by persistent or recurrent low back pain.

The Numeric Rating Scale (NRS), the Roland–Morris Disability Questionnaire (RMDQ), and the Short Form-36 (SF-36) were used to assess pain intensity, disability, and quality of life, respectively, as described previously^{17–19}.

Characteristics of family history of LBP

Participants were inquired about the history of LBP among their immediate family members (i.e., parents, siblings, and children), including potential causes and treatments received (open-ended questions). The family history characteristics were organized and summarized based on participants reported information. According to origin of family member with LBP, participants were classified into four subgroups: i) no family history; ii) maternal family history; iii) paternal family history; and iv) sibling family history. In addition, participants were categorized into three subgroups based on the number of family members with LBP: i) no family history of LBP; ii) single affected family member; and iii) multiple affected family members (two or more family members with LBP). According to whether family members had undergone surgical treatment for LBP, participants were also classified into three subgroups: i) without a family history of LBP, ii) with a positive family history but no surgery, and iii) with any immediate family member who had undergone LBP surgery⁸.

Medical Imaging Evaluation

All participants underwent lumbar spine scans using a 3.0 Tesla Lumina MRI machine (Siemens AG). The acquisition parameters for sagittal and transverse MRI sequences were described in detail in a previous study²⁰.

Several common lumbar morphometric changes were assessed, including those involving the intervertebral discs, facet joints, and vertebrae. IDD and FJD were assessed using the criteria of the Pfirrmann classification (score range 1–5) and Fujiwara Grading (score range 1–4), respectively^{21,22}. MCs and spondylolisthesis were assessed based on previous reports and classified into two categories: 0 indicating absence and 1 indicating presence^{23,24}. The total scores of IDD and FJD were calculated by summing the scores from L1/L2 to L5/S1 segments. The presence of MCs or spondylolisthesis was defined as the occurrence of MCs or spondylolisthesis at any level from L1/L2 to L5/S1 segments.

Imaging abnormalities were independently assessed by two physicians trained in MRI interpretation. To eliminate potential measurement bias, all MRI data were anonymized prior to analysis.

Statistical Analysis

The demographic characteristics of the study population were described for the overall population, as well as for those with absence and presence of family history of LBP, using standardized mean differences (SMDs), where values < 0.1 denoted a good balance. To assess the association between individual LBP status and family history of LBP, we performed multiple binary logistic regressions to evaluate odds ratios (ORs). Differences in pain intensity, functional disability, and quality of life between no family history controls and various family history subgroups were evaluated via linear regression analyses. In addition, logistic regression and linear regression models were used to evaluate the associations between family history of LBP and lumbar imaging abnormalities, represented as binary variables (MCs and spondylolisthesis) and continuous variables (IDD and FJD), respectively. Potential confounders, including age, sex, BMI, smoking habits, and alcohol consumption frequency, were controlled across all regression frameworks^{25–27}, with individuals lacking a family history of LBP serving as the reference group.

Statistical procedures were executed using IBM SPSS Statistics (version 27.0). To indicate the precision of our estimates, 95% confidence intervals (CIs) were reported for all ORs in logistic regression models and for unstandardized beta coefficients (B) in linear regression models. P-values have to be interpreted cautiously in this exploratory analysis. Interpretation of results is based on effect estimates and 95%CI.

Results

The final analytical sample comprised 458 women (56.5%) and 353 men (43.5%), a total of 811 participants, with an average age of 42.3 (standard deviation, SD: 11.9 years, **Table 1**). Among these participants, 419 (51.7%) were reported to have a confirmed family history of LBP, whereas 392 (48.3%) did not. The population characteristics were further reported based on participants' family history of LBP. Except for sex distribution (SMD = 0.123), all baseline covariates were well balanced between participants with and without a family history of LBP (SMD < 0.1).

Table 1 The demographic characteristics categorized by the presence or absence of participants with family history of LBP

Demographic characteristics	Entire population	Participants with family history of LBP		SMD
		absence	presence	
No. of participants, n (%)	811	392	419	
Age (years), mean (SD)	42.3 (11.9)	41.8 (11.9)	42.7 (11.9)	0.076
BMI (kg/m^2), mean (SD)	23.2 (2.4)	23.2 (2.4)	23.3 (2.4)	0.042
Sex, n (%)				
Men	353 (43.5)	183 (46.7)	170 (40.6)	0.123
Women	458 (56.5)	209 (53.3)	249 (59.4)	
Drinking, n (%)				
≤ once per week	597 (73.6)	294 (75.0)	303 (72.3)	0.061
> once per week	214 (26.4)	98 (25.0)	116 (27.7)	
Smoking, n (%)				
No	731 (90.1)	353 (90.1)	378 (90.2)	0.006
Yes	80 (9.9)	39 (9.9)	41 (9.8)	

Abbreviations LBP: low back pain, SD: standard deviation, BMI: body mass index, SMD: standardized mean difference

Participants reporting a positive family history had higher odds of experiencing LBP than those lacking a family history (OR 1.91, 95% CI: 1.42, 2.57, $p < 0.01$, **Table 2**). When further considering the source of the family history of LBP, we found associations among participants with paternal (OR 2.30, 95% CI 1.59, 3.32, $p < 0.01$), maternal (OR 1.83, 95% CI 1.28, 2.61, $p < 0.01$), and sibling (OR 2.90, 95% CI 1.64, 5.15, $p < 0.01$) history of LBP, respectively. Odds of experiencing LBP between individuals with maternal versus paternal family history were similar (OR 0.77, 95% CI 0.47, 1.26, $p = 0.30$). When family history was further stratified by surgical history, participants with affected relatives who had undergone spine surgery showed higher odds of LBP than those without a family history (OR 1.93, 95% CI 0.79, 4.73, $p = 0.15$). In addition, across categories of the number of family members with history of LBP, individuals with multiple affected family members had higher odds of experiencing LBP (OR 3.41, 95% CI 2.00–5.79, $p < 0.01$).

Table 2 The association between the participants with family history of LBP and the presence of LBP

Family history characteristics	LBP cases / Total (%)	OR for LBP (95% CI)	<i>P</i> value
Reference group			
No family history of LBP	227/392 (57.9%)	1.00 (Ref.)	
Origin of family history of LBP			
With family history of LBP	304/419 (72.6%)	1.91 (1.42, 2.57)	< 0.01
With paternal family history	171/227 (75.3%)	2.30 (1.59, 3.32)	< 0.01
With maternal family history	161/225 (71.6%)	1.83 (1.28, 2.61)	< 0.01
With sibling family history	68/85 (80.0%)	2.90 (1.64, 5.15)	< 0.01
Paternal vs. maternal family history	/	0.77 (0.47, 1.26)	0.30
Family history of LBP and surgery			
Family history of LBP but no surgery	285/393 (72.5%)	1.91 (1.41, 2.58)	< 0.01
Family history of LBP and surgery	19/26 (73.1%)	1.93 (0.79, 4.73)	0.15
Number of family members with LBP			
Single affected family member	212/307 (69.1%)	1.62 (1.18, 2.22)	< 0.01
Multiple affected family members	92/112 (82.1%)	3.41 (2.00, 5.79)	< 0.01

Adjusted for age, body mass index, sex, smoking status, and drinking frequency

‘No family history of LBP’ was set as the reference group

Abbreviations LBP: low back pain, OR: odds ratio, CI: confidence interval

A positive family history was associated with poorer quality of life, reflected by lower SF-36 scores (B -3.52, 95% CI -5.79, -1.25, $p < 0.01$) (**Table 3**). The effects were more pronounced among individuals with affected siblings (B -7.54, 95% CI -11.52, -3.57, $p < 0.01$) and those with multiple affected family members (B -7.03, 95% CI -10.48, -3.57, $p < 0.01$). There were no relevant differences observed in average LBP intensity or disability levels, as assessed by the NRS and RMDQ.

Table 3 Association between family history of LBP and LBP intensity, disability, and quality of life.

Family history characteristics	Mean LBP intensity		RMDQ scores		SF-36 scores	
Origin of family history of LBP	B (95% CI)	<i>P</i>	B (95% CI)	<i>P</i>	B (95% CI)	<i>P</i>
With family history of LBP	-0.09 (-0.40, 0.23)	0.59	-0.48 (-1.18, 0.22)	0.18	-3.52 (-5.79, -1.25)	<0.01
With paternal family history	-0.19 (-0.56, 0.18)	0.31	-0.86 (-1.65, -0.07)	0.03	-3.07 (-5.78, -0.35)	0.03
With maternal family history	0.05 (-0.32, 0.41)	0.81	-0.30 (-1.13, 0.54)	0.49	-3.94 (-6.67, -1.20)	<0.01
With sibling family history	0.03 (-0.48, 0.54)	0.91	-0.13 (-1.28, 1.03)	0.83	-7.54 (-11.52, -3.57)	<0.01
Family history of LBP and surgery						
No family history of LBP	Reference		Reference		Reference	
Family history of LBP but no surgery	-0.13 (-0.45, 0.19)	0.42	-0.61 (-1.32, 0.10)	0.09	-3.30 (-5.60, 0.99)	<0.01
Family history of LBP and surgery	0.58 (-0.27, 1.42)	0.18	1.49 (-0.38, 3.37)	0.12	-7.06 (-13.75, -0.36)	0.04
Number of family members with LBP						
No family history of LBP	Reference		Reference		Reference	
Single affected family member	-0.20 (-0.55, 0.14)	0.25	-0.61 (-1.37, 0.16)	0.12	-2.25 (-4.71, 0.20)	0.07
Multiple affected family members	0.18 (-0.27, 0.62)	0.43	-0.18 (-1.16, 0.81)	0.72	-7.03 (-10.48, -3.57)	<0.01

Adjusted for age, body mass index, sex, smoking status, and drinking frequency

SF-36 scores were analyzed across all participants (n=811), whereas the Mean LBP intensity and RMDQ scores were exclusively analyzed among participants with LBP (n=531)

'No family history of LBP' was used as the reference group

Abbreviations LBP: low back pain, RMDQ: Roland–Morris Disability Questionnaire, SF-36: the Short Form-36

Family history of LBP had a more pronounced spinal degeneration, including IDD (B 0.53, 95% CI 0.13, 0.93, $p = 0.01$) and FJD (B 0.41, 95% CI 0.14, 0.69, $p < 0.01$), with similar patterns observed for paternal, maternal, and sibling family history (**Table 4**). Individuals reporting affected relatives who had undergone spine surgery showed the strongest associations with spinal degeneration, with higher IDD (B 2.00, 95% CI 0.91,

3.09, $p < 0.01$) and FJD scores (B 0.84, 95% CI 0.09, 1.58, $p = 0.03$). Similarly, participants with multiple affected family members had higher IDD (B 0.92, 95% CI 0.32, 1.52, $p < 0.01$) and FJD scores (B 0.76, 95% CI 0.36, 1.17, $p < 0.01$). No relevant associations were found between family history of LBP and MCs (OR 1.15, 95% CI 0.84, 1.58, $p = 0.38$) or spondylolisthesis (OR 0.92, 95% CI 0.42, 2.05, $p = 0.84$) across the analyses.

Table 4 The association between participants with family history of LBP and common lumbar morphometric changes as observed on MRI

Family history characteristics	IDD ^a		FJD ^a		MCs ^b		Spondylolisthesis ^b	
Origin of family history of LBP	B (95% CI)	<i>P</i>	B (95% CI)	<i>P</i>	OR (95% CI)	<i>P</i>	OR (95% CI)	<i>P</i>
With family history of LBP	0.53 (0.13, 0.93)	0.01	0.41 (0.14, 0.69)	<0.01	1.15 (0.84, 1.58)	0.38	0.92 (0.42, 2.05)	0.84
With paternal family history	0.47 (0.00, 0.94)	0.048	0.36 (0.05, 0.68)	0.02	1.19 (0.82, 1.73)	0.36	1.12 (0.43, 2.95)	0.82
With maternal family history	0.71 (0.23, 1.19)	<0.01	0.60 (0.27, 0.92)	<0.01	1.16 (0.80, 1.69)	0.43	1.11 (0.44, 2.83)	0.82
With sibling family history	0.69 (0.04, 1.34)	0.04	0.57 (0.13, 1.00)	0.01	1.19 (0.71, 1.98)	0.52	1.00 (0.27, 3.74)	0.99
Family history of LBP and surgery								
No family history of LBP	Reference		Reference		Reference		Reference	
Family history of LBP but no surgery	0.43 (0.02, 0.84)	0.04	0.39 (0.12, 0.66)	<0.01	1.11 (0.81, 1.53)	0.52	1.00 (0.45, 2.22)	0.99
Family history of LBP and surgery	2.00 (0.91, 3.09)	<0.01	0.84 (0.09, 1.58)	0.03	1.96 (0.85, 4.54)	0.12	/	/
Number of family members with LBP								
No family history of LBP	Reference		Reference		Reference		Reference	
Single affected family member	0.39 (-0.04, 0.82)	0.08	0.29 (0.00, 0.57)	0.048	1.07 (0.76, 1.51)	0.69	0.63 (0.25, 1.64)	0.35
Multiple affected family members	0.92 (0.32, 1.52)	<0.01	0.76 (0.36, 1.17)	<0.01	1.36 (0.86, 2.16)	0.19	1.94 (0.68, 5.56)	0.22

Separate regression models were constructed across all participants (n=811) for each MRI outcome variable.

Model 'a': linear regression; Model 'b': logistic regression. all models adjusted for age, body mass index, sex, smoking status, and drinking frequency

'No family history of LBP' was used as the reference group

Abbreviations IDD: intervertebral disc degeneration, FJD: facet joint degeneration, MCs: modic changes, LBP: low back pain, OR: odds ratio, CI: confidence interval

Discussion

Given that the family history of LBP is relatively widespread, with previous studies reporting a prevalence exceeding 30%^{8,9}, we explored its associations with individual LBP odds, clinical features, and changes in lumbar morphology. The main findings are as follows: i) Family history of LBP exhibited higher odds of experiencing LBP, especially with multiple affected family members; ii) Individuals with LBP family history reported lower quality of life, but no relevant difference in pain intensity and disability level; iii) Family history of LBP was associated with greater spinal degeneration, including IDD and FJD.

Our results show that individuals reporting a family history of LBP are 1.91 times more likely to experience it themselves than those without. This is in line with the results of earlier classic twin studies and epidemiological surveys. For instance, Battié et al., based on the famous Twin Spine Study, noted that genetic factors explain about 30%-46% of the variability in disc degeneration and back pain, rather than just structural degeneration or occupational loading^{16,28}. MacGregor et al. analyzed data from a registry of 1,064 volunteers from the TwinsUK cohort, demonstrating that genetic factors play a significant role in the variability of LBP (52–68%) and neck pain (35–58%) in adults²⁹. Similarly, Livshits et al. also used data from 2,256 women twins in the UK cohort and found an odds ratio of 2.2 for experiencing LBP when the individual had a dizygotic co-twin¹⁰.

However, unlike most previous studies that defined family history only as “yes” or “no”^{8,10,16,28–30}, a unique strength of this study is that we analyzed the familial loading of LBP and the specific links to fathers, mothers, and sibling. We found that when there are at least two people in the family affected, the person's LBP odds go up to 3.41 times. On the one hand, this cumulative association may suggest a polygenic inheritance pattern, where the accumulation of risk alleles (such as COL9A2 or VDR) increases susceptibility. On the other hand, this cumulative effect could be partly explained by shared environmental factors within families. Family members share many behavioral characteristics, including diet, physical activity patterns, occupational tendencies, socioeconomic status, and psychological perception of pain, all of which substantially contribute to LBP risk^{31,32}.

When we look at the specific source of family history, our subgroup analysis explored different lineage associations. The results showed that a family history of the mother (OR 1.83) or the father (OR 2.30) demonstrated similarly higher odds. This lack of difference is consistent with an Autosomal Polygenic Inheritance pattern, rather than sex-linked inheritance³³. It could mean LBP risk is linked to the

accumulation of many small effect traits from both parents, which follow Battié et al.'s finding that familial background is the primary determinant of spinal structural fragility²⁸.

Furthermore, Individuals with a sibling history of LBP showed a notably high association (OR 2.90), which had even higher odds than those with maternal or paternal family history. We consider it could be attributed to shared environmental factors, because siblings usually share more similar childhood environments, dietary habits, exercise patterns, and even psychological coping mechanisms for pain compared to their parents. Hestbaek et al., in a study of adolescents and young adults, also noted that back pain concordance between siblings increases with age, likely due to the combined effect of genes and shared early lifestyles like sedentary behavior and types of physical activity³⁴. Therefore, our data emphasizes that asking about siblings' back pain during clinical visits may have higher predictive value than just asking about parents, which is often overlooked in past screenings. Interestingly, our analysis of the family surgery history subgroup revealed a potential dissociation between the susceptibility to LBP and the severity of structural degeneration. Compared to individuals lacking a familial LBP history, those with a family history of LBP but without surgical intervention in affected relatives had higher odds of experiencing LBP (OR 1.91, $p < 0.01$). Similarly, those with a family history of both LBP and surgery showed a similar effect size for LBP odds (OR 1.93), but this did not reach statistical significance, possibly due to limited statistical power from a smaller sample size in this specific subgroup, rather than the definitive absence of an association.

Besides, we observed an interesting dissociation phenomenon in clinical symptoms. Although the individuals with a family history, especially those with affected siblings or multiple affected members, reported a significantly lower quality of life, their NRS and RMDQ were similar compared to those without family history of LBP. This might be explained through a bio-psycho-social model: i) the transmission of pain may be psychological as well as physiological. Individuals with a family history may have watched relatives suffer from back pain since childhood. This could lead to stronger fear-avoidance beliefs or catastrophizing, causing them to perceive pain as a long-term, inevitable state extending reported duration and lowering their quality of life, especially in psychological domains³⁵; ii) this may reflect an adaptation mechanism. Long-term exposure to a family environment of back pain might lead to pain habituation or increased pain interference thresholds within the family context; iii) they do not report higher acute pain scores, but the chronic burden still damages their overall health perception. Matsui et al. noted similar findings, suggesting genetics influence the occurrence and duration of back pain more than just the severity³⁰. This is consistent with what Amorim et al. found in the HUNT study. They confirmed that parental chronic spinal pain is an independent risk factor for poor prognosis and slow recovery in children³⁶. It also echoes the work of Zadro et al., though they additionally revealed reduced physical activity levels and poorer rehabilitation outcomes in the older cLBP patients with a family history of LBP by a randomized controlled study in Australia⁹.

At the anatomical level, our MRI data showed that a family history of LBP is associated with more severe structural degeneration (IDD and FJD). But it's important to note that these imaging findings should be interpreted with caution as morphometric markers rather than as exact structural causes of LBP. Because

degenerative lumbar spine changes are highly prevalent even in asymptomatic individuals, MRI abnormalities can't be directly considered as a structural cause of pain³⁷. The absolute values of the regression coefficients for IDD and FJD were generally modest, suggesting that family history contributes to structural degeneration burden, but is unlikely to be a dominant determinant of cumulative MRI scores by itself. This aligns with some previous points that both IDD and FJD are multifactorial phenotypes influenced not only by familial predisposition³⁸, but also by age, body composition, mechanical loading, occupational exposure, smoking, and physical activity^{39–41}. Even so, the MRI findings remain important, suggesting that familial effects may not be limited to the level of pain reporting, but at least to some extent reflected in spinal structure. In this sense, poorer quality of life without markedly higher NRS, together with greater structural degeneration burden, may indicate that family history is associated with a more chronic or cumulative vulnerability, rather than simply increased pain intensity at a single time point.

A particularly remarkable finding was the stronger association observed for participants with affected relatives who had undergone spine surgery. Despite the similar odds for clinical LBP onset, participants with affected relatives who had undergone spine surgery exhibited drastically worse structural degeneration scores, with the coefficients for IDD and FJD being more than double those of the non-surgery family history group. This may suggest that surgery in family members captures a subgroup with a heavier familial burden of spinal disease, potentially reflecting a more severe degenerative phenotype rather than merely the occurrence of pain symptoms. It should be emphasized that this interpretation should remain cautious, because family members' surgical histories are shaped not only by biological disease severity but also by healthcare access, treatment preferences, and differences in surgical indications^{42–45}. In addition, the sample size of the surgical family-history subgroup is relatively small, which may limit the precision and make the result unstable. Regardless, this pattern is attractive at both clinical and biological levels, since it suggests that not all positive family histories carry the same meaning. A general family history of LBP may be associated with modest increases in structural burden, whereas a family history marked by spine surgery may identify families at the more severe end of the degenerative spectrum.

Additionally, a graded familial loading pattern was also observed for both IDD and FJD. Participants with only one affected relative showed only weak or borderline associations with IDD and FJD, whereas those with multiple affected relatives demonstrated stronger associations. This suggests that the cumulative burden of family history may be more informative than a simple binary classification of family history as present or absent. A higher number of affected relatives may reflect a more stable familial predisposition, including both shared genetic susceptibility and long term shared environmental exposures, such as physical workload patterns, lifestyle factors, health behaviors, and learned responses to pain^{46,47}. This observation is also compatible with the clinical findings described above, where affected siblings and multiple affected family members were particularly associated with poorer quality of life. Overall, these results suggest that familial burden may be reflected not only in broader clinical impact but also in the burden of MRI structural degeneration.

In contrast to some previous hypotheses that link family history to inflammatory pathways^{48,49} and the structural predisposition to spondylolisthesis⁵⁰, our analyses found no significant association between a family history of LBP and MCs or spondylolisthesis. MCs are generally regarded as markers of vertebral endplate inflammation and microfractures. As one possible explanation, the familial risk captured in this cohort is more closely related to mechanical and structural vulnerability (cartilage and disc wear) than to specific inflammatory endplate phenotypes. It is also possible that different MRI phenotypes reflect partly distinct pathophysiological pathways, or that limited statistical power reduced our ability to detect weaker associations for MCs and spondylolisthesis.

From a clinical perspective, although our findings do not establish causality, they show potential future applications. Family history of LBP, particularly the number of affected relatives and the presence of spine-related surgery in family members, may provide complementary information when characterizing risk profiles in individuals with LBP. Therefore, a more detailed family history assessment may be useful not simply as a binary yes/no variable, but as a graded marker of familial burden that captures both clinical vulnerability and structural susceptibility. For LBP patients with a confirmed family history, it's reasonable for clinicians to focus on both the disc and the progression of facet joint osteoarthritis in early intervention.

There are some limitations in this study: First, since participants were recruited for a back study, a selection bias may exist, as individuals with a family history might be more motivated to participate. For this reason, these findings cannot be directly generalized to the general population. Second, although participants provided detailed information about their relatives' back pain conditions at recruitment, these data were self-reported without independent medical verification, meaning recall bias and misclassification cannot be excluded. Third, the absence of detailed matching for family size (such as the number of siblings), specific household socioeconomic status or physical labor intensity (e.g., between siblings), may introduce residual confounding and limit the interpretability of the results, also limiting our ability to fully separate genetic from shared environmental effects that may contribute to poorer health outcomes and spinal degeneration. Fourth, as a cross-sectional study, we cannot establish a strict causal timeline. Fifth, this was an exploratory analysis involving multiple subgroup comparisons, which inevitably increases the probability of chance findings. Finally, some subgroups (e.g., surgery + family history) were limited by small sample sizes, resulting in borderline statistical significance. These associations require confirmation in future large-scale, independent prospective cohorts.

Conclusion

This study shows that family history of LBP is associated with LBP, lower quality of life and morphological abnormalities in the lumbar spine. The potential biological and environmental mechanisms through which family history is linked to health conditions warrant further longitudinal investigation.

Declarations

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Author Contribution

All authors contributed sufficiently to the work and accept public responsibility for the following: (1) conception and design (H.H, D.Y) (2) data acquisition (H.H, D.Y, L.S, R.H, C.D, F.Y) (3) analysis of data (H.H, D.Y) (4) drafting of the manuscript (H.H) (5) critical revision (H.H, D.Y, C.L, L.S, L.M, G.U, R.Y, C.D, F.Y, H.S) (6) obtaining funding (H.S) (7) administrative support (S.R, H.S) (8) supervision (C.L, H.S).

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

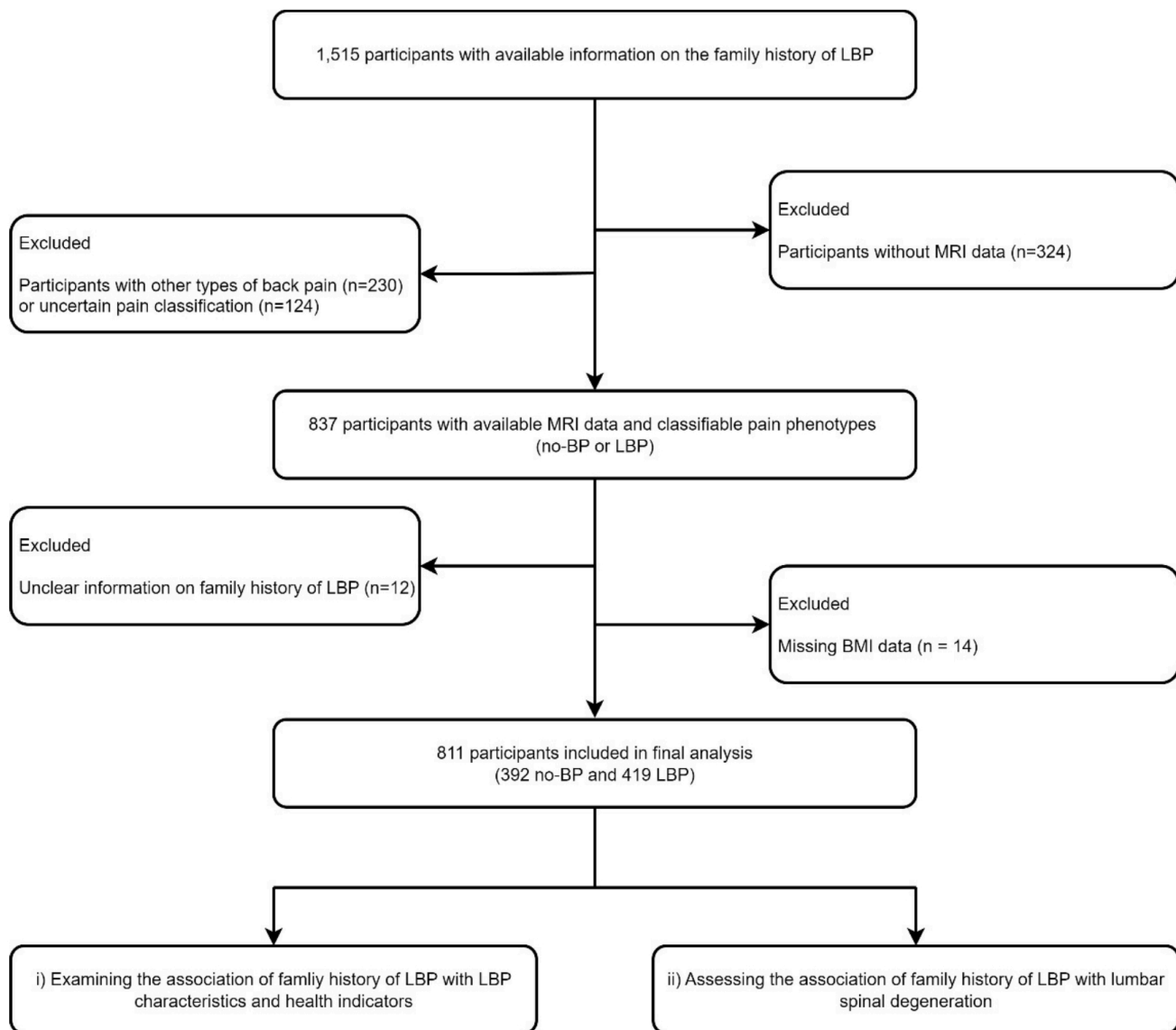


Figure 1

The flowchart depicting study recruitment. Abbreviations: MRI: magnetic resonance imaging, no-BP: no back pain, LBP: low back pain, BMI: body mass index