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Kinesiophobia and Work Disability in Fibromyalgia: Cognitive Mediation in a Population-Based Study

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Abstract

Background: Work disability in fibromyalgia is only partially explained by symptom severity, suggesting a relevant contribution of cognitive–behavioral mechanisms. **Objective:** This study aimed to determine whether kinesiophobia is associated with fibromyalgia impact and work-related disability, and to assess whether pain catastrophizing mediates these relationships within a hierarchical biopsychosocial framework. **Methods:** This cross-sectional study included 2,096 women with fibromyalgia recruited through a nationwide online survey. Participants completed validated instruments assessing fibromyalgia impact (FIQ), pain catastrophizing (PCS), depressive symptoms (PHQ-9), central sensitization (CSI), and kinesiophobia (Tampa Scale). Pain-related work disability was defined using the Graded Chronic Pain Scale–Revised (GCPS-R). Hierarchical logistic regression models identified factors independently associated with work disability. Mediation was tested using bootstrapped analyses (5,000 resamples). **Results**: Kinesiophobia demonstrated a robust independent association with work disability (OR 1.03; 95% CI 1.02–1.05) after adjustment for sociodemographic factors, clinical pain phenotype, systemic burden, pain severity, psychocognitive load, and medication burden. Other relevant contributors included pain severity (OR 1.96; 95% CI 1.70–2.27), psychocognitive burden (OR 1.35; 95% CI 1.15–1.58), use of benzodiazepines (OR 1.74; 95% CI 1.33–2.28), and opioid use (OR 1.29; 95% CI 1.06–1.56). Mediation analysis indicated a significant indirect effect of kinesiophobia on work disability through pain catastrophizing (β = 0.131; 95% CI 0.078–0.188). **Conclusion:** Kinesiophobia is a proximal determinant of work disability in fibromyalgia, exerting direct and cognitively mediated effects through pain catastrophizing, reinforcing the fear-avoidance framework and the need for psychologically informed rehabilitation.

Keywords: fibromyalgia; kinesiophobia; pain catastrophizing; work disability; rehabilitation; chronic pain

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1. Introduction

Fibromyalgia (FM) is a chronic pain syndrome with an estimated prevalence of 2–5%, predominantly affecting women of working age [1]. FM is marked by widespread musculoskeletal pain, fatigue, sleep disturbances, cognitive dysfunction, and affective symptoms like anxiety and depression. These symptoms cause significant functional impairment and reduce quality of life [2]. Classified as nociplastic pain, FM results from dysfunctions in central pain modulation, including nociceptive amplification, reduced descending inhibition, and persistent central sensitization and neuroinflammation [3,4], reflecting interactions between biological, psychological, and social factors.

Among psychological factors linked to disability in chronic pain, kinesiophobia stands out. It is an excessive fear of movement, driven by concerns about pain or injury. Evidence shows kinesiophobia relates to reduced physical performance, altered proprioception, and less muscular endurance, regardless of pain intensity. It may limit rehabilitation by reinforcing threat perception and facilitating transition from persistent pain to disability [5–7]. In chronic pain, kinesiophobia affects 50–70% of individuals and can stem from direct pain or social and observational learning [8,9]. In fibromyalgia, kinesiophobia often co-occurs with pain catastrophizing—a pattern marked by rumination, magnification, and helplessness [10]. Together, these factors increase hypervigilance and avoidance, perpetuating a cycle of inactivity, pain, and limitation [11]. These phenomena reflect neuroplastic processes underlying both adaptation and central sensitization [12,13]. Emotional symptoms and psychiatric diagnoses often coexist with chronic pain, but current evidence supports a cyclical, interdependent relationship rather than a direct causal one. Thus, kinesiophobia is a modifiable, clinically relevant factor [14].

Robust evidence shows that physical activity and therapeutic exercise promote adaptive neuroplasticity. They enhance descending pain modulation through somatosensory and neuroendocrine mechanisms [13,15,16]. International guidelines recommend physical exercise, combined with pain education and cognitive interventions, as a first-line non-pharmacological approach for fibromyalgia [17]. Clinical trials and meta-analyses demonstrate that multimodal programs consistently improve pain, physical function, and quality of life [18–20]. However, treatment responses remain heterogeneous. Biopsychosocial factors such as kinesiophobia, catastrophizing pain, depressive symptoms, and the degree of central sensitization strongly influence these outcomes. These factors may also amplify functional disability [21].

Despite recognition of the importance of these factors, key gaps remain regarding their relative and interdependent contributions to functional outcomes suggesting that systemic burden may increase vulnerability to maladaptive cognitive processing, which in turn influences behavioral avoidance and functional limitation. This study aimed to examine whether association between kinesiophobia and clinical outcomes (fibromyalgia impact and work-related disability), and to evaluate whether pain catastrophizing mediates these relationships within a hierarchical biopsychosocial framework. To address this, hierarchical regression models were applied to account for demographic, clinical, and symptom-related factors, medication load and mediation analyses were conducted to explore the extent to which maladaptive pain-related cognitions may contribute to this association.

2. Materials and Methods

2.1. Design Overview, Setting, and Participants

This cross-sectional study followed STROBE guidelines. All participants provided written informed consent. The Research Ethics Committee of Hospital de Clínicas de Porto Alegre (Institutional Review Board - IRB # 2023-0210) approved the protocol. The recruitment period for this study began on August 1, 2023, and ended on November 30, 2023.

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2.2. Recruitment, Inclusion, and Exclusion Criteria

Individuals volunteered after responding to announcements in public spaces or advertisements on websites such as Facebook and Craigslist. These advertisements were shared through the networks of the National Association of Fibromyalgia and Related Diseases (ANFIBRO). A live online session introduced the project and addressed public questions. A message explaining the purpose of the study was shared on social media to invite people to take part.

Interested individuals accessed the study through a provided link. After confirming their interest, they received an informed consent form. Upon signing, participants received a questionnaire about sociodemographic data and the American College of Rheumatology (ACR-2016) questionnaire [22], via the Research Electronic Data Capture platform - REDCap. The evaluation collected medical history and symptom details to confirm diagnoses. The research team managed recruitment by sending up to three messages per individual. If registration was still incomplete after ten days, an additional reminder was sent. After participants completed their registration, they did not receive any additional messages.

To participate, individuals had to be at least 18 years old, able to read, and have a confirmed fibromyalgia diagnosis according to the 2016 criteria set by the American College of Rheumatology [22]. The 2016 ACR criteria stipulate that: (i) generalized pain, defined as pain in at least 4 of 5 regions, must be present; (ii) symptoms must be persistent for at least 3 months; (iii) the Widespread Pain Index (WPI) must be ≥7 with a Symptom Severity Scale (SSS) score ≥5, or a WPI of 4–6 with an SSS score ≥9; and (iv) the diagnosis is valid regardless of other comorbid conditions, and the presence of fibromyalgia does not exclude additional clinically relevant illnesses. Consistent with these criteria, participants needed a combined WPI + SSS score ≥13. Participants were excluded if they didn't meet the 2016 ACR criteria or did not complete study questionnaires after three follow-up attempts.

2.3. Instruments and Assessment

After providing consent, participants completed the questionnaires via REDCap. To minimize misunderstanding, each item included accessible instructions. All instruments were validated for the Brazilian population, ensuring accurate and reliable measurement. The research team was trained to guide the recruitment process and instruct participants on how to respond to the study demands. Response monitoring was conducted continuously, and in cases of incomplete answers, up to three follow-up contacts were made to ensure correction. Weekly meetings were held to discuss any doubts that arose during the study, allowing for calibration among evaluators. Instructions for all procedures and assessment methods were included in a standardized instruction manual.

2.3.1. Primary and Secondary Outcomes

Primary and secondary outcomes: the primary outcome was the pain that limited daily activities or work on most days for at least three months. The secondary outcome was the impact of fibromyalgia symptoms on quality of life assessed by the Fibromyalgia Impact Questionnaire (FIQ).

2.3.2. Outcomes Measures (Dependent Variable)

a) The dependent variable was pain-related work disability, operationalized as a dichotomous outcome. Pain-related work disability was assessed using the Graded Chronic Pain Scale–Revised (GCPS-R), with item 6 specifically evaluating pain-related work limitations. Participants classified as grade 3 (high-impact chronic pain)—defined as pain that limited daily activities or work on most days for at least three months—were categorized as incapacitated due to pain, whereas those classified as grades 0–2 were categorized as not incapacitated. This binary variable was used as the dependent outcome in the logistic regression and mediation analyses.
b) The Fibromyalgia Impact Questionnaire (FIQ) was used to assess the impact of fibromyalgia on function, symptoms, and overall quality of life across 10 domains, with higher scores indicating greater disease burden (e.g., physical functioning, pain intensity, fatigue, stiffness, sleep quality,

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mood, work impairment, and ability to perform daily activities), with higher scores indicating greater disease burden. We used the version validated for the Brazilian population [23].

2.3.3. Covariate Measures

c) The Kinesiophobia assessed using the Tampa Scale for Kinesiophobia (TSK), a self-report measure of fear of movement and re-injury. The original version comprises 17 items rated on a 4-point Likert scale (1 = strongly disagree to 4 = strongly agree), yielding a total score ranging from 17 to 68, with higher scores indicating greater kinesiophobia. The TSK assesses maladaptive beliefs related to physical activity and injury vulnerability and has demonstrated good psychometric properties across chronic pain populations, including fibromyalgia. In this study, the total TSK score was analyzed as a continuous variable. It is the main interest factor [24].

d) The Symptom Severity Scale (SSS) was used to assess the severity of three core somatic symptom domains—fatigue, symptoms of cognitive dysfunction and waking unrefreshed—we used the three corresponding items from the Symptom Severity Scale (SSS) of the 2016 revisions of the American College of Rheumatology (ACR) fibromyalgia diagnostic criteria [22]. Each symptom was rated according to its severity over the past week using a 0–3 scale: 0 = no problems; 1 = slight or mild problems, generally intermittent; 2= moderate or considerable problems, often present and/or of moderate intensity; 3= severe, pervasive, continuous, or life-disturbing problems.

e) The Pain Catastrophizing Scale (PCS) was used to assess catastrophic thinking related to pain. It contains 13 items scored 0–4 (total 0–52) covering rumination, magnification, and helplessness domains [25].

f) The Patient Health Questionnaire-9 (PHQ-9) was used to evaluate depressive symptoms over the past 15 days. The scale includes nine DSM (Diagnostic and Statistical Manual of Mental Disorders) -based items scored 0–27; in Brazil, a cutoff ≥9 indicates clinically relevant depression [26].

g) The Central Sensitization Inventory – Brazilian Version (CSI) was used to screen for central sensitization symptoms. This 25-item scale (0–100) evaluates somatic and emotional symptoms commonly associated with central sensitization, including diffuse pain, fatigue, non-restorative sleep, cognitive difficulties, headaches, and urological complaints [27].

h) AUDIT-C was used to assess alcohol consumption patterns and identify risk related to alcohol use. It consists of three items scored 0–4 [28].

i) Sociodemographic and clinical covariates: sociodemographic and clinical covariates were collected using a standardized questionnaire and included age (years), educational attainment (years or highest level of formal schooling), body mass index (BMI) category, occupational status (unemployed; employed/student/self-employed; retired; receiving disability benefits), and fibromyalgia duration (years).

j) Medication use was categorized a priori into the following groups: (i) Antidepressants, including tricyclic antidepressants, dual-action antidepressants, selective serotonin reuptake inhibitors (SSRIs), and a combined tricyclic/dual-action indicator; (ii) Sleep-related medications, including melatonin, zolpidem, and benzodiazepines; and(iii) Analgesics, including non-opioid analgesics (nonsteroidal anti-inflammatory drugs, dipyrone, and acetaminophen), any opioid use (binary indicator), and specific opioid agents (codeine, morphine, oxycodone, methadone, and tramadol).

2.4. Efforts to Address Potential Sources of Bias

To minimize potential sources of bias, all study procedures were fully standardized through REDCap, ensuring identical administration, automated data capture, and removal of duplicate entries. Recruitment was broad and public, and all instruments used were validated for the 2,096 population, reducing measurement error. Sensitive items (e.g., trauma exposures) were self-

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administered online to limit bias to social desirability. The research team followed a structured operations manual, with weekly calibration meetings and continuous data-quality monitoring. Incomplete responses triggered up to three automated reminders to reduce attrition bias.

2.5. Statistical Analysis

Participants were stratified according to work disability status. Categorical variables were compared using χ^2 tests, and continuous variables using independent t-tests or Mann–Whitney U tests, as appropriate. To estimate independent associations with work disability, logistic regression models were performed. Variables associated with the outcome at $P < 0.20$ in univariable analyses, as well as those considered clinically relevant, were considered for multivariable modeling.

Multivariable analyses followed a hierarchical conceptual modeling strategy, based on a biopsychosocial framework in which variables were entered according to their theoretical proximity to the outcome. Distal variables included sociodemographic factors (age and education). Intermediate variables comprised clinical and treatment-related domains, operationalized through composite indices, including the pain severity index, psychopharmacological load index, and analgesic load index. Proximal variables reflected cognitive–behavioral mechanisms, specifically pain catastrophizing and kinesiophobia. Composite indices were constructed a priori to reduce dimensionality and capture clinically meaningful domains. The pain severity index reflected aggregated measures of pain intensity, frequency, and interference. The psychopharmacological load index represented the cumulative use of centrally acting medications, and the analgesic load index captured the total burden of analgesic use, including opioid and non-opioid medications. All indices were treated as continuous variables. Variables were entered sequentially according to hierarchical levels [29]. Variables retained at each level were kept in subsequent models regardless of statistical significance, to preserve theoretical consistency and avoid inappropriate adjustment for mediators. Odds ratio (OR) estimates for each exposure were obtained from the model corresponding to the stage at which the variable was first introduced, to avoid overadjustment and attenuation of effects due to the inclusion of potential mediators [30].

In the first stage, demographic variables were entered, representing distal factors that could directly or indirectly influence all subsequent hierarchical levels, consistent with hierarchical modeling approaches in clinical research [31]. Intermediate-level variables, represented by composite indices, were entered in the second stage. Proximal cognitive–behavioral variables were included in the final stage. Within each hierarchical block, variables were entered using a forward stepwise procedure [32]. Variables that were statistically significant at the time of block entry ($P < 0.05$) were retained in subsequent models, even if their significance decreased after the inclusion of later blocks, to preserve theoretically relevant associations [33]. At each stage, variable retention was also guided by stability of effect estimates, conceptual relevance, and contribution to overall model fit, with statistical significance considered a complementary criterion [34]. Model comparisons and hypothesis testing were based on likelihood ratio tests. Multicollinearity was assessed using variance inflation factors (VIF) and tolerance statistics [35]. Results are presented as odds ratios (OR) with 95% confidence intervals (CI) and p-values.

To test the study hypothesis, regression-based mediation analyses were conducted to examine whether kinesiophobia mediates the association between pain catastrophizing and clinical outcomes, including fibromyalgia impact (FIQ) and work disability for fibromyalgia impact (FIQ), linear regression models were used, and standardized coefficients (β) are reported. For work disability, logistic regression models were applied, and results are expressed as odds ratios (OR). All mediation models were adjusted for age, education, pain severity index, psychopharmacological load index, and analgesic load index, selected a priori based on their role within the hierarchical conceptual framework. Indirect effects were estimated using bootstrapping procedures with 5,000 resamples, and statistical significance was determined based on 95% confidence intervals that did not cross zero. All analyses were conducted using IBM SPSS Statistics (version 22) and R software (version 4.4.2). Statistical significance was set at $p < 0.05$ (two-tailed).

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3. Results

3.1. Sample Characteristics and Work Disability Status

From the publicly available online survey widely disseminated across national platforms, 4,200 individuals initiated the questionnaire. After identifying and removing repeated attempts by the same participant, 3,500 unique respondents remained. Among these, 2,498 (71.4%) completed all required instruments. A total of 301 respondents did not meet the fibromyalgia screening criteria and were excluded, yielding 2,197 eligible cases. Because only 101 men completed the full survey—too few for trustworthy analyses separating results by sex—these participants were excluded from analysis. The final analytical cohort therefore comprised 2,096 women, representing 49.9% of all individuals who initiated the survey and 83.9% of those who completed all questionnaires.

Table 1 summarizes the sociodemographic and clinical characteristics, comorbidities, medication use, and health care engagement indicators of the study sample, stratified by pain-related work disability. Overall, participants were predominantly middle-aged women with long-standing fibromyalgia, high rates of medical comorbidities, and extensive use of psychotropic and analgesic medications, reflecting a population with substantial clinical complexity.

Table 1. Sociodemographic and clinical characteristics, comorbidities, medication use, and health care engagement, stratified by pain-related work disability (n=2,096).

	Disability to work		
Variable	No (n=1,036)	Yes (n=1,060)	OR (95% CI)
Age (yrs)	50.09 (±10.98)	51.28 (±9.39)	1.01 (1.00–1.02)
Formal education (yrs)	14.58 (±5.38)	13.17 (±5.46)	0.95 (0.94–0.97)
Alcohol Use Disorders Identification Test Consume (AUDIT-C) (yes)	640 (61.8)	504 (47.5)	0.56 (0.48–0.66)
Race Black/Brown (yes)	184 (17.8)	190 (17.9)	1.01 (0.80–1.27)
Smoke (yes)	138 (43.0)	183 (57.0)	1.61 (1.25–2.07)
Professional status n (%)			
Unemployed (yes)	85 (29.4)	204 (70.6)	3.01 (2.29–3.95)
Employed/Student/etc (yes)	733 (64.9)	396 (35.1)	0.33 (0.28–0.39)
Retired (yes)	180 (49.7)	182 (50.3)	1.02 (0.80–1.29)
Disability benefits (yes)	38 (12.0)	278 (88.0)	9.60 (6.66–13.85)
Body mass index (BMI) n (%)			
Normal weight (BMI <18.5–24.9 kg/m²) (yes)	253 (54.9)	208 (45.1)	0.78 (0.63–0.97)
Overweight (BMI 25.0–29.9 kg/m²) (yes)	366 (49.7)	371 (50.3)	1.03 (0.85–1.25)
Obesity (BMI ≥ 30 kg/m²) (yes)	411 (46.4)	475 (53.6)	1.33 (1.10–1.61)
Chronic disease n (%)			
Hypertension (HAS) (yes)	615 (44.3)	773 (55.7)	1.58 (1.34–1.86)
Diabetes(yes)	287 (46.1)	336 (53.9)	1.37 (1.12–1.68)
Stroke(yes)	99 (45.4)	119 (54.6)	1.20 (0.88–1.64)
Asthma(yes)	9 (33.3)	18 (66.7)	2.00 (0.88–4.52)
Chronic Obstructive Pulmonary Disease (COPD) (yes)	172 (39.0)	269 (61.0)	1.73 (1.39–2.16)
Number chronic diseases	1.14 (1.14)	1.42 (1.14)	1.22 (1.14–1.30)
Psychotropic medication use n (%)			
Use of anticonvulsants(yes)	501 (43.5)	652 (56.5)	1.58 (1.34–1.86)

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	Use of tricyclic antidepressants(yes)	244 (42.7)	327 (57.3)	1.57 (1.28–1.93)
	Use of dual antidepressants(yes)	414 (47.8)	452 (52.2)	1.16 (0.98–1.38)
	Use of benzodiazepines(yes)	101 (30.5)	230 (69.5)	3.04 (2.36–3.91)
	Use of zolpidem(yes)	74 (41.1)	106 (58.9)	1.46 (1.05–2.03)
	Use of carbamazepine(yes)	21 (42.9)	28 (57.1)	1.33 (0.72–2.45)
	Use of gabapentin(yes)	490 (43.2)	645 (56.8)	1.60 (1.36–1.89)
	Use of selective serotonin reuptake inhibitors (SSRI) (yes)	170 (41.5)	240 (58.5)	1.49 (1.18–1.87)
	Use of melatonin(yes)	73 (45.1)	89 (54.9)	1.20 (0.86–1.68)
Opioid Analgesic use n (%)				
	Non-opioid ≥2	737 (47.4)	819 (52.6)	1.23 (1.05–1.45)
	Opioid use	648 (62.6)	985 (92.9)	7.80 (5.90–10.30)
	Codeine	272 (42.0)	376 (58.0)	1.55 (1.28–1.87)
	Tramadol	331 (40.8)	481 (59.2)	1.68 (1.40–2.02)
	Morphine	29 (27.4)	77 (72.6)	3.10 (1.95–4.93)
	Oxycodone	10 (29.4)	24 (70.6)	2.70 (1.22–5.98)
	Methadone	6 (18.2)	27 (81.8)	5.15 (2.04–12.99)
Number of opioids analgesics		0.60 (±0.76)	0.86 (±0.83)	
Number of non-opioid analgesics		1.14 (±0.94)	1.28 (±0.95)	
Physical activity		532 (50.3)	526 (49.7)	0.97 (0.83–1.14)

Data were presented as mean (SD) or n (%), odds ratio (OR) with confidence interval (CI 95%).

3.2. Clinical, Psychological, and Functional Differences According to Work Disability

Table 2 displays fibromyalgia severity, pain interference, psychological variables, central sensitization, psychiatric comorbidities, adverse life events, and functional outcomes according to pain-related work disability. Participants with work disability showed consistently higher symptom burden and worse psychological and functional profiles.

Table 2. Clinical, psychological, and functional characteristics stratified by pain-related work disability. Data were presented as mean (SD) or n (%), odds ratio with confidence interval (CI 95%) (n=2,096).

	Disability to work		
Variable	No (n=1,036)	Yes (n=1,060)	OR (95% CI)
Fibromyalgia - Symptom Severity and Diagnostic Criteria (ACR 2016)			
Widespread pain index (WPI)	10.28 (±3.19)	11.12 (±3.32)	1.08 (1.06–1.11)
Symptom Severity Score (SSS)	9.46 (±1.66)	10.09 (±1.42)	1.22 (1.16–1.29)
Fatigue	2.55 (±0.55)	2.76 (±0.46)	1.85 (1.60–2.14)
Waking unrefreshed	2.61 (±0.57)	2.75 (±0.47)	1.62 (1.40–1.88)
Cognitive dysfunction	2.30 (±0.69)	2.45 (±0.62)	1.38 (1.24–1.54)
Fibromyalgia Severity (FS) - (WPI score plus SSS)	19.74 (±3.80)	21.21 (±3.96)	1.10 (1.07–1.13)
Pain intensity (7 days)	7.59 (±1.60)	8.29 (±1.45)	1.32 (1.24–1.40)
Pain interference (activities)	7.38 (±2.27)	8.59 (±1.52)	1.41 (1.32–1.50)

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Tampa Scale for Kinesiophobia (TSK)	43.96 (±8.32)	47.99 (±7.48)	1.06 (1.05–1.07)
Central Sensitization Inventory (CSI)	67.17 (±11.86)	70.69 (±11.26)	1.02 (1.01–1.03)
Pain Catastrophizing Scale (PCS) – total score	34.98 (±10.51)	39.15 (±9.59)	1.04 (1.03–1.05)
PCS – Magnification	7.86 (±3.01)	8.84 (±2.75)	1.12 (1.08–1.16)
PCS – Helplessness	15.40 (±5.04)	17.66 (±4.59)	1.09 (1.07–1.12)
PCS – Rumination	11.72 (±3.20)	12.64 (±2.94)	1.08 (1.05–1.11)
Fibromyalgia Impact Questionnaire (FIQ)	67.71 (±12.98)	75.99 (±11.51)	1.06 (1.05–1.07)
Patient Health Questionnaire-9 (PHQ-9)	16.07 (±6.09)	18.68 (±5.77)	1.08 (1.06–1.09)
Duration of fibromyalgia (yrs.)	15.41 (±11.31)	14.98 (±11.86)	0.99 (0.98–1.01)
Age at onset of fibromyalgia (yrs.)	34.25 (±12.03)	29.89 (±11.09)	0.96 (0.95–0.97)
Pain-related comorbidities n (%)			
Coexisting nociceptive musculoskeletal pain (yes)	427 (40.8)	619 (59.2)	2.0 (1.68–2.38)
Coexisting neuropathic pain (yes)	292 (42.7)	392 (57.3)	1.78 (1.48–2.14)
Coexisting nociceptive + neuropathic pain (yes)	163 (39.6)	249 (60.4)	2.12 (1.65–2.73)
Physical activity (yes)	532 (50.3)	526 (49.7)	0.98 (0.83–1.15)
History of psychiatric diagnosis n (%)			
Psychiatric diagnosis (yes)	457 (44.5)	570 (55.5)	1.55 (1.32–1.83)
Major depression (yes)	363 (42.3)	496 (57.7)	1.74 (1.45–2.08)
Anxiety disorder (yes)	372 (45.2)	451 (54.8)	1.48 (1.23–1.77)
Bipolar disorder (yes)	80 (38.5)	128 (61.5)	1.65 (1.19–2.29)
Panic disorder (yes)	86 (37.1)	146 (62.9)	1.94 (1.42–2.66)
Post-traumatic stress disorder (PTSD) (yes)	48 (34.0)	93 (66.0)	2.05 (1.39–3.02)
Number psychiatric diagnoses	0.92 (±1.20)	1.24 (±1.37)	1.18 (1.10–1.26)
Life adverse events n (%)			
Persistent physical aggression (yes)	206 (41.4)	292 (58.6)	1.36 (1.09–1.69)
Persistent verbal aggression (yes)	358 (49.0)	372 (51.0)	1.08 (0.90–1.29)
Persistent emotional aggression (yes)	522 (48.5)	554 (51.5)	1.06 (0.90–1.25)
Sexual abuse (yes)	163 (44.7)	202 (55.3)	1.28 (1.00–1.63)
Cumulative number of abuse events	1.51 (±1.39)	1.60 (±1.45)	1.05 (1.00–1.10)
Onset after major event (yes)	522 (47.8)	570 (52.2)	1.18 (1.00–1.39)

3.3. Hierarchical Logistic Regression: Factors Independently Associated with Work Disability

A hierarchical logistic regression model was constructed to evaluate factors associated with pain-related disability. Sociodemographic variables were entered first, followed by systemic burden, pain severity, psychocognitive burden, medication load, and finally kinesiophobia, reflecting increasing conceptual proximity to disability. OR are presented at the step in which each variable entered the hierarchical model, allowing assessment of their independent contribution prior to

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adjustment for more proximal factors. Distal variables showed modest associations with disability. Systemic burden and pain severity were associated with disability at entry but were attenuated after inclusion of psychocognitive factors. Notably, kinesiophobia (TSK) entered at the final stage and demonstrated a robust independent association, supporting its role as a proximal determinant of disability.

Table 3. Continuous variables are presented as mean (SD), and categorical variables as n (%). Odds ratios (OR) with 95% confidence intervals are shown at the step in which each variable entered the hierarchical model.

Variable	Step	No disability	Disability	OR (95% CI)	p
Age (years)	1	50.10 (±10.99)	51.28 (±9.40)	1.01 (1.00–1.02)	<0.001
Smoking status	1	138 (13.3%)	183 (17.3%)	1.40 (1.10–1.79)	0.007
Alcohol use (AUDIT-C category)	1	640 (61.8%)	504 (47.5%)	0.56 (0.47–0.67)	<0.001
Pain-related comorbidities	2	1.60 (±0.75)	1.84 (±0.78)	1.39 (1.24–1.57)	<0.001
Fatigue	2	2.55 (±0.55)	2.76 (±0.45)	1.79 (1.48–2.18)	<0.001
Sleep disturbance (non-restorative sleep)	2	2.61 (±0.57)	2.75 (±0.47)	1.27 (1.05–1.53)	0.013
Systemic biopsychosocial burden index	3	-0.10 (0.61)	0.10 (0.61)	1.28 (1.08–1.51)	0.004
Pain severity index	4	-0.22 (0.76)	0.21 (0.66)	1.96 (1.70–2.27)	<0.001
Psychocognitive burden index	5	-0.18 (0.78)	0.17 (0.70)	1.35 (1.15–1.58)	<0.001
Psychopharmacological load index	6	1.30 (0.91)	1.64 (0.96)	1.25 (1.12–1.39)	<0.001
Analgesic load index	6	-0.12 (0.76)	0.12 (0.80)	1.10 (0.97–1.25)	0.140
Kinesiophobia - TSK	7	43.96 (±8.32)	47.99 (±7.48)	1.03 (1.02–1.05)	<0.001

Block 1 – Sociodemographic factors: Age (years), Smoking status, Alcohol use (AUDIT-C category). Block 2 – Clinical pain phenotype: Pain-related comorbidities, Fatigue, Sleep disturbance (non-restorative sleep). Block 3 – Systemic burden. Composite index representing biopsychosocial systemic burden: mean [z (cumulative abuse), z (central sensitization), z (number of chronic diseases)]. Block 4 – Pain severity. Composite index of pain burden: = mean [z(WPI), z (pain intensity in the last 7 days), z (pain interference with activities)]. Block 5 – Psychocognitive burden. Composite index of affective-cognitive burden: mean [z(PHQ-9), z (PCS total), z (cognitive symptoms)]. Block 6 – Medication burden. Medication-related indices: sum of anticonvulsants, any antidepressant, benzodiazepines, and zolpidem; analgesic load index = mean [z (number of non-opioid analgesics), z (number of opioid analgesics)]. Block 7 – Kinesiophobia (proximal): Tampa Scale for Kinesiophobia (TSK).

Interpretation: Effects are shown at the point of entry in the hierarchical model, allowing visualization of attenuation or persistence across blocks. Kinesiophobia remains a proximal determinant of disability.

3.4. Cognitive Mediation of the Relationship Between Kinesiophobia, Work Disability, and Disease Impact on Quality of Life

Figure 1 illustrates a partial mediation model in which kinesiophobia significantly mediates the association between pain catastrophizing and fibromyalgia impact (FIQ), with a remaining direct effect after adjustment for age, education, number of non-opioid analgesics, number of opioid analgesics, and medication burden (indices comprised the sum of anticonvulsants, any antidepressants, benzodiazepines, and zolpidem).

Figure 2 depicts a mediation model in which kinesiophobia fully mediates the association between pain catastrophizing and pain-related disability, as the direct effect is no longer significant after adjustment while the indirect effect remains significant. All estimates are adjusted for age, education, number of non-opioid analgesics, number of opioid analgesics, and medication burden (indices comprised the sum of anticonvulsants, any antidepressants, benzodiazepines, and zolpidem).

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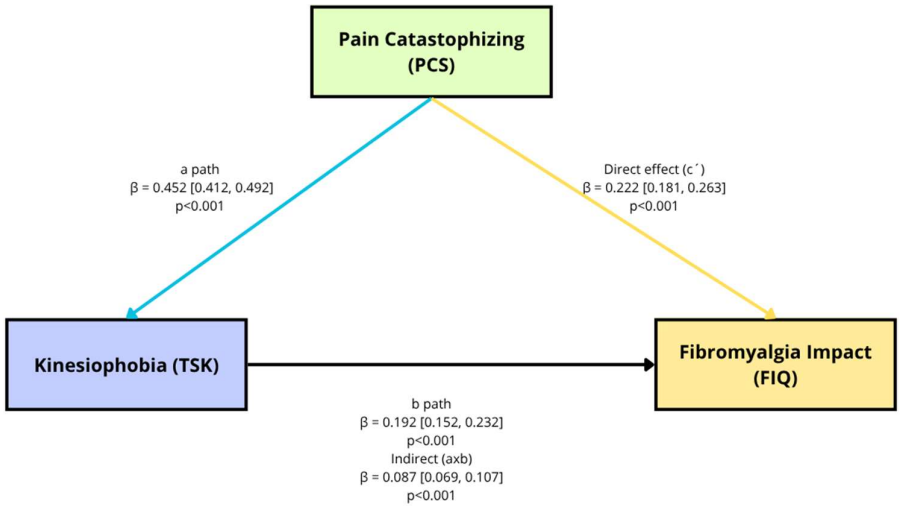

Figure 1. Mediation model examining the association between pain catastrophizing and fibromyalgia impact on quality of life, as assessed by the Fibromyalgia Impact Questionnaire (FIQ), through kinesiophobia. Path ***a*** represents the association between pain catastrophizing and kinesiophobia. Path ***b*** represents the association between kinesiophobia and fibromyalgia impact (FIQ). Path ***c'*** represents the direct effect of pain catastrophizing on fibromyalgia impact after accounting for the mediator. The indirect effect **(a × b)** represents the mediated pathway through kinesiophobia. Values displayed in the figure correspond to regression coefficients (β or OR), 95% confidence intervals (CI), and p-values derived from the fully adjusted model.

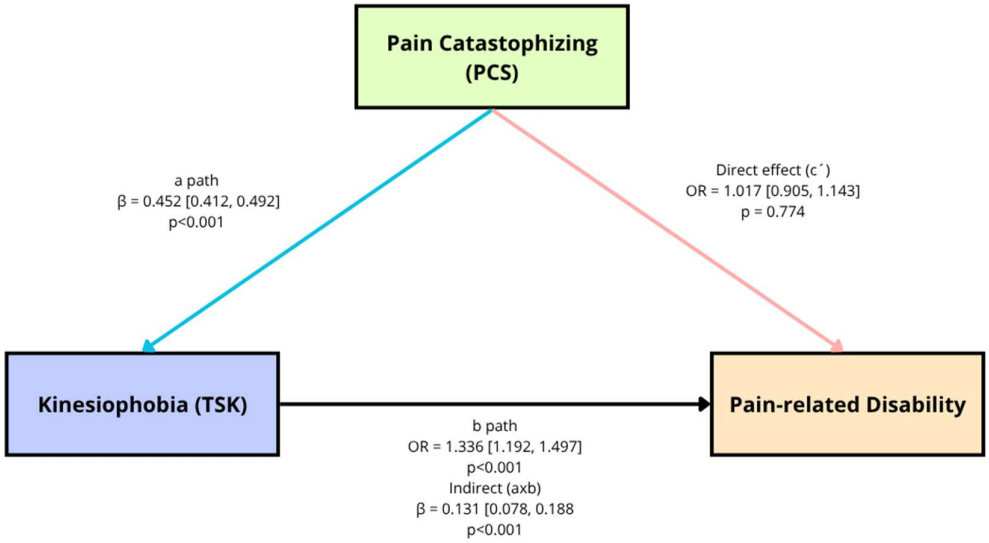

Figure 2. Mediation model examining the association between pain catastrophizing and pain-related disability through kinesiophobia. **Path *a*** represents the association between pain catastrophizing and kinesiophobia. **Path *b*** represents the association between kinesiophobia and pain-related disability. ***Path c'*** represents the direct

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effect of pain catastrophizing on pain-related disability after accounting for the mediator. The indirect effect ***(a × b)*** represents the mediated pathway through kinesiophobia. Values displayed in the figure correspond to regression coefficients (β or OR), 95% confidence intervals (CI), and p-values derived from the fully adjusted model.

4. Discussion

This study provides novel evidence that pain catastrophizing functions as a central cognitive mediator linking kinesiophobia to clinically relevant outcomes in fibromyalgia, within a hierarchical biopsychosocial framework that integrates overall pain burden and pharmacological load. While prior models have typically conceptualized catastrophizing as an antecedent of fear-avoidance, our findings support an alternative pathway in which fear of movement is associated with maladaptive cognitive processing, which in turn is linked to functional impairment. Consistent with this framework, kinesiophobia was significantly associated with pain catastrophizing, which in turn was associated with both fibromyalgia impact and work disability. Mediation analyses demonstrated that pain catastrophizing partially mediated the association between kinesiophobia and fibromyalgia impact and fully mediated its association with work disability. These associations remained robust after adjustment for sociodemographic factors, pain severity, and pharmacological burden, suggesting that cognitive processing of pain represents a key mechanism through which behavioral fear responses translate into functional impairment. This pattern aligns with the foundational fear-avoidance model proposed by Vlaeyen and Linton [36], in which fear-driven interpretations of pain escalate avoidance behaviors and amplify disability. These multivariable models further revealed that kinesiophobia emerged as an independent proximal determinant of work disability, even after accounting for systemic burden, psychocognitive factors, and medication use, reinforcing its central role in disability pathways. This supports the conceptual shift that cognitive mechanisms—especially catastrophic thinking—should be prioritized as therapeutic targets in individuals presenting with high movement-related fear.

This study indicates that kinesiophobia is associated with both work disability and reduced quality of life in women with fibromyalgia, and that this relationship is partly explained by pain catastrophizing. Although fear of movement showed a strong association with disability in unadjusted analyses, its effect was attenuated after adjustment for cognitive and psychological factors, suggesting that maladaptive pain-related cognitions represent an important intermediate mechanism. These findings are consistent with fear-avoidance models of chronic pain, in which fear of movement promotes behavioral avoidance and reinforces disability, particularly in conditions characterized by nociplastic pain [37,38]. These behavioral and cognitive associations align with functional neuroimaging findings demonstrating that fibromyalgia is characterized by altered connectivity in brain regions responsible for cognitive control, salience processing, and affective modulation. Resting-state studies have shown increased coupling between the dorsolateral prefrontal cortex (DLPFC) and the anterior cingulate cortex (ACC) [39], a configuration associated with heightened sensitivity to negative stimuli and reduced executive regulation mediated by the fronto-cingulate network [40]. Such alterations may contribute to interpreting pain as threatening, reinforcing hypervigilance, and amplifying catastrophic thinking [41,42]. This interpretation is further supported by evidence of enhanced salience attribution and abnormal top-down modulation in fibromyalgia, including increased functional connectivity within the insula and medial prefrontal cortex—networks implicated in maladaptive cognitive–emotional processing [43]. Taken together, these findings suggest that the cognitive–emotional mechanisms identified in the present study—particularly kinesiophobia and catastrophizing—are embedded within broader alterations in neural network functioning, providing convergent neurobiological support for their role in functional impairment.

Clinically, these findings suggest that kinesiophobia should not be conceptualized as an isolated therapeutic target, but rather as part of a broader cognitive–emotional framework that shapes functional outcomes in fibromyalgia. Within this framework, fear of movement interacts with

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maladaptive pain-related cognitions, particularly catastrophic thinking—to influence behavior, disability, and treatment response. Consequently, interventions focused exclusively on reducing fear of movement are unlikely to achieve meaningful or sustained improvements if catastrophic interpretations of pain are not concurrently addressed. This integrated perspective is supported by evidence indicating that psychological interventions yield greater clinical benefits when they simultaneously target both behavioral avoidance and maladaptive cognitive appraisals. Exposure-based and graded activity approaches appear to be more effective when combined with strategies aimed at modifying catastrophic thinking and pain-related beliefs. Moreover, contemporary evidence reinforces those multimodal approaches—especially those integrating pain education or cognitive-behavioral strategies with exercise-based therapies, producing the most consistent and clinically relevant improvements in function and pain outcomes [44]. This underscores that targeting isolated components of the fear-avoidance model may be insufficient, whereas interventions that address the interplay between cognition, emotion, and behavior are more likely to impact disability trajectories. Therefore, multimodal approaches integrating cognitive-behavioral strategies, pain neuroscience education, and graded exposure represent a more effective pathway to modifying disability trajectories. These findings reinforce the importance of psychologically informed pain management strategies in fibromyalgia care.

Also, these results showed that work disability and FIQ remained independently associated even after adjustment, indicating that the FIQ reflects a global disease impact encompassing physical function, fatigue, pain, and emotional distress. While work disability captures critical functional consequences of fibromyalgia, the FIQ represents a broader disease construct, overlapping with but not identical to disability [45]. Mediation analyses showed that kinesiophobia exerted both direct and indirect effects on FIQ through pain catastrophizing, suggesting that fear of movement contributes not only to specific disability outcomes but also to a substantial worsening of quality of life. In this context, the FIQ strengthens the clinical relevance of the findings and supports the interpretation that disability is embedded within overall disease severity rather than isolated symptoms.

The systemic burden index, which integrates cumulative abuse, central sensitization, and the number of chronic comorbidities, remained independently associated with work disability, although with reduced magnitude after inclusion of cognitive–emotional variables, indicating that disability in fibromyalgia arises in the context of a broader biopsychosocial load rather than from isolated symptom domains. Because central sensitization was not analyzed as an independent predictor—to avoid fragmentation of interrelated constructs and potential overadjustment—its contribution reflects a broader pattern of multisystem symptom amplification embedded within the systemic burden index. This approach acknowledges that central sensitization does not operate in isolation but rather interacts with cumulative life stressors and comorbidity load to shape symptom expression in fibromyalgia. However, once cognitive–emotional variables were introduced into the model, the influence of systemic burden—although still present—was attenuated, while kinesiophobia and pain catastrophizing showed stronger associations with functional outcomes. These proximal cognitive factors demonstrated a stronger explanatory weight for functional outcomes, suggesting that systemic biopsychosocial load provides the background vulnerability, whereas maladaptive cognitive processing influences the extent to which this vulnerability is expressed into work disability [46]. This interpretation aligns with models of sensitization-driven symptom persistence [47], while more accurately reflecting the hierarchical structure of the current findings. Thus, in this study, systemic burden contributes to the overall environment in which symptoms arise, but cognitive–emotional mechanisms—particularly kinesiophobia and catastrophizing—are the primary drivers that link nociplastic and multisystem load to functional impairment.

Work disability was independently associated with the use of anticonvulsants, benzodiazepines, and opioids; however, this association does not imply causality. Rather, medication use likely reflects greater disease severity and may serve as a proxy for treatment complexity and refractoriness. Several mechanisms may contribute to this association, including sedation, cognitive effects, and reduced alertness, which may further impair functional capacity. Additionally, long-term use of these

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medications may reflect patients with more severe symptoms and fewer non-pharmacological resources [48]. These patterns align with literature showing that high pharmacological load is often associated with greater centralized pain processing and psychological comorbidity [47]. Overall, these findings reinforce the interpretation that work disability is linked to complex clinical profiles rather than single factors and support the need for non-pharmacological, psychologically informed interventions.

4.1. Limitations

Several methodological considerations should be considered when interpreting these findings. *First*, the cross-sectional design precludes causal inference. Although mediation analyses were conducted within a theoretically guided hierarchical framework, they represent statistical mediation rather than temporal or mechanistic causation and therefore should be interpreted as hypothesis-generating rather than causal. *Second*, all measures were self-reported, which may introduce recall bias, mood-congruent reporting, and symptom amplification—particularly relevant in conditions characterized by cognitive load, affective dysregulation, and nociplastic pain. Nevertheless, the instruments used are validated, widely applied in fibromyalgia research, and capture clinically meaningful subjective experiences that are integral to the disorder. *Third*, the study relied on composite indices (systemic burden, pain severity, psychocognitive burden, and medication load), which were constructed a priori to reduce dimensionality, avoid multicollinearity, and reflect theoretically coherent clinical domains. While this approach enhances model stability and interpretability, it may obscure the contribution of individual components, particularly central sensitization or cumulative trauma, which are embedded within broader indices rather than examined as isolated predictors. The hierarchical modeling approach, combined with assessment of collinearity (VIF and tolerance), minimized redundancy, but residual overlap among symptom-based constructs is unavoidable in fibromyalgia. *Fourth*, the exclusive inclusion of women limits generalizability to men, who represented a small proportion of the initial sample. Online recruitment may also have introduced selection bias toward individuals with higher symptom burden, greater digital engagement, or stronger illness identification. However, the large national sample, internal consistency of indices, and robustness of multivariable effects across hierarchical blocks mitigate concerns regarding sampling heterogeneity and enhance the credibility of the observed associations. Finally, cognitive–emotional factors such as catastrophizing and kinesiophobia, which emerged as strong proximal determinants, may not only act as mechanisms linking symptoms to disability but may also influence self-report accuracy, threat appraisal, and interpretation of symptom severity. This dual role—mechanistic and perceptual—should be acknowledged, and future longitudinal or experimental studies incorporating behavioral and neurophysiological measures are needed to clarify directionality.

4.2. Conclusion

These findings show that kinesiophobia is a proximal determinant of work disability in fibromyalgia, with part of its effect mediated by pain catastrophizing. Cognitive–emotional mechanisms therefore play a central role in linking nociplastic pain to functional impairment, surpassing the contributions of pain severity, systemic burden, and medication load. Together, these results support the fear-avoidance framework and highlight the need for rehabilitation strategies that address maladaptive beliefs and promote adaptive engagement in activity.

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Informed Consent Statement: Signed informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request. All data are fully de-identified to protect participant confidentiality. Requests for data access can be directed to wcaumo@hcpa.edu.br..

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Abbreviations

ACR	American College of Rheumatology
AUDIT-C	Alcohol Use Disorders Identification Test – Consumption
BMI	Body Mass Index
COPD	Chronic Obstructive Pulmonary Disease
CSI	Central Sensitization Inventory
FIQ	Fibromyalgia Impact Questionnaire
FM	Fibromyalgia
FS	Fibromyalgia Severity (WPI + SSS)
HCPA	Hospital de Clínicas de Porto Alegre
ICF	Informed Consent Form

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IRB	Institutional Review Board
NPS	Numeric Pain Scale
NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
PCS	Pain Catastrophizing Scale
PHQ-9	Patient Health Questionnaire-9
PTSD	Post-Traumatic Stress Disorder
REDCap	Research Electronic Data Capture
SOC	Sense of Coherence
SSRI	Selective Serotonin Reuptake Inhibitor
SSS	Symptom Severity Scale
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
TSK	Tampa Scale for Kinesiophobia
UFRGS	Universidade Federal do Rio Grande do Sul
WPI	Widespread Pain Index

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