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The Spine–Pelvic Floor Connection: Neurological Mechanisms Linking Lumbar Spine Disorders to Chronic Pelvic Pain and Pelvic Floor Dysfunction in Women - A Systematic Review

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A B S T R A C T

Background: Chronic pelvic pain (CPP) and pelvic floor dysfunction are prevalent, complex conditions in women that may coexist with lumbar spine disorders. The neurological relationships between lumbar pathology, pelvic floor dysfunction, and persistent pelvic pain remain incompletely characterized.

Objective: This systematic review examines the proposed neurological and biomechanical mechanisms linking lumbar spine disorders to CPP and pelvic floor dysfunction in women, emphasizing lumbosacral nerve-root involvement, peripheral neuropathy, altered neuromuscular control, and central sensitization.

Methods: A structured literature review framework developed around studies addressing lumbar radiculopathy, lumbosacral neurological dysfunction, pelvic floor muscle abnormalities, pudendal neuralgia, chronic pelvic pain, and pain-processing mechanisms. Relevant publications identified through searches of biomedical literature and reference lists. Evidence organized into four domains: anatomical and neurophysiological connections, clinical associations, pain amplification mechanisms, and diagnostic and therapeutic implications. Because study populations, diagnostic definitions, and outcome measures are heterogeneous, quantitative pooling not undertaken without verified, comparable study-level data.

Results: The literature supports several biologically plausible connections between spinal and pelvic symptoms. Lumbosacral nerve-root pathology may influence pelvic sensation, sphincter function, and pelvic floor motor control. Pain-related protective muscle activation, impaired relaxation, and myofascial tenderness may contribute to persistent pelvic symptoms. Peripheral neuralgia and central sensitization may further amplify pain and complicate clinical assessment. Nevertheless, evidence directly establishing lumbar spine disorders as a causal determinant of pelvic floor dysfunction remains limited, and associations not assumed to demonstrate directionality.

Conclusion: Assessment of women with concurrent lumbar and pelvic symptoms should integrate neurological, musculoskeletal, gynecological, urological, and pelvic floor findings. Mechanism-based, multidisciplinary management is clinically reasonable, but prospective studies using standardized diagnostic criteria and longitudinal outcomes are required to establish causal pathways and treatment effectiveness.

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Introduction

Chronic pelvic pain (CPP) in women represents a heterogeneous clinical condition involving persistent pain perceived in the pelvic region, often accompanied by urinary, bowel, sexual, musculoskeletal, and psychological symptoms. It may arise from identifiable gynecological disease, urological or gastrointestinal disorders, musculoskeletal dysfunction, neuropathic mechanisms, or interactions among these factors. Clinical guidelines emphasize that persistent pelvic pain frequently requires multidisciplinary assessment because no single organ system adequately explains every presentation [1-3]

Lumbar spine disorders are a potentially important but sometimes overlooked component of this clinical picture. Disc degeneration, disc herniation, foraminal narrowing, facet-mediated pain, and lumbosacral radiculopathy can produce low-back pain, lower-extremity symptoms, altered movement patterns, and, in selected cases, neurological dysfunction involving pelvic structures. However, the presence of lumbar abnormalities on imaging does not establish that these abnormalities cause pelvic pain. Structural findings interpreted alongside symptom distribution, neurological examination, functional limitations, and alternative diagnoses [4].

The anatomical relationship between the lumbar spine, sacrum, and pelvic floor provides a plausible basis for clinical interaction. Pelvic floor muscles participate in urinary and fecal continence, sexual function, pelvic organ support, and coordinated pressure regulation during respiration and movement. Their motor and sensory regulation involves sacral spinal segments, peripheral nerves, supraspinal control, and interactions with abdominal, hip, and trunk musculature. The pudendal nerve, arising predominantly from S2-S4, supplies important sensory territories of the perineum and motor innervation to several pelvic floor muscles and external sphincters. Additional pelvic and perineal sensory pathways arise from thoracolumbar and sacral roots through nerves including the ilioinguinal, iliohypogastric, genitofemoral, obturator, and posterior femoral cutaneous nerves [5-10].

These anatomical connections not interpreted as evidence that every lumbar disorder disrupts pelvic floor function. Most ordinary lumbar pain conditions do not produce direct neurological injury to pelvic sphincters.

Clinically significant dysfunction depends on the affected structure, lesion location, severity, duration, and individual neurological anatomy.

Severe compression involving the cauda equina or sacral roots can produce bladder, bowel, perineal sensory, and motor abnormalities, whereas uncomplicated mechanical low-back pain may influence pelvic symptoms indirectly through pain-related guarding and altered movement [11].

A second potential connection involves neuromuscular adaptation. Pain originating in the lumbar region may change trunk stabilization, hip mobility, breathing strategies, and recruitment of abdominal and pelvic floor muscles. Persistent protective contraction can impair relaxation and contribute to muscle tenderness. Conversely, pelvic floor overactivity or weakness may alter lumbopelvic loading and movement coordination. These reciprocal relationships suggest that the spine and pelvic floor understood as functionally interacting systems rather than entirely independent anatomical regions [12].

The relationship becomes more complex when neuropathic and oncoplastic pain mechanisms coexist. Peripheral nerve irritation may generate burning, electric, shooting, or radiating pain. Persistent nociceptive input may also contribute to changes in spinal and supraspinal pain processing, including increased sensitivity to mechanical stimulation. A systematic review of central sensitization in Ur gynecological CPP reported findings consistent with altered central pain processing, although the included evidence was predominantly observational and could not establish whether these alterations preceded or followed the development of pain [13-20]

This distinction has practical importance. Women with CPP may have multiple simultaneous pain generators, and pain severity does not necessarily correspond to the severity of structural abnormalities. Pelvic floor tenderness, urinary urgency, dyspareunia, low-back pain, and perineal discomfort may coexist without sharing a single cause. Similarly, the identification of a lumbar disc abnormality does not exclude endometriosis, bladder pain syndrome, vulvodynia, or other pelvic disorders [21].

Despite increasing recognition of these overlapping mechanisms, evidence directly connecting specific lumbar diagnoses to objectively measured pelvic floor dysfunction remains fragmented. Studies vary in their definitions of CPP, pelvic floor hypertonicity, radiculopathy, and neuropathic pain.

Some investigate women with established pelvic pain, whereas others examine patients with spinal disorders or neurological disease. This heterogeneity limits the comparability of reported associations and complicates quantitative synthesis.

The present review therefore examines the anatomical, neurological, biomechanical, and pain-processing mechanisms that may connect lumbar spine disorders with CPP and pelvic floor dysfunction in women. It also evaluates the implications for diagnostic assessment, interdisciplinary management, and future research. The central proposition is that a combined spinal-

pelvic framework may improve clinical reasoning in appropriately selected patients, while avoiding the unsupported assumption that lumbar pathology is a universal or primary cause of chronic pelvic symptoms [22-30].

Literature Review

Lumbosacral neuroanatomy and pelvic function:

The relationship between spinal pathology and pelvic function begins with the organization of the peripheral and central nervous systems. Lumbar and sacral nerve roots contribute to somatic sensation, lower-limb motor function, and the neural regulation of pelvic organs and muscles. The sacral plexus contains fibers contributing to the pudendal nerve and other nerves supplying pelvic and perineal structures. These pathways explain why specific sacral lesions may affect perineal sensation, pelvic floor contraction, and sphincter function. The pudendal nerve is especially relevant to perineal pain and external sphincter control. Pudendal neuralgia may present with pain in the nerve's anatomical territory, frequently aggravated by sitting. Labat et al. (2008) proposed the Nantes criteria to support clinical diagnosis, including pain in the pudendal territory, worsening with sitting, absence of pain-related awakening at night, absence of objective sensory loss, and relief following an anesthetic pudendal nerve block. These criteria address a specific neuropathic syndrome; they do not establish that lumbar disc disease is its underlying cause [31-40].

Neurological symptoms associated with lumbar disorders depend on the level and nature of the lesion. Lower lumbar nerve-root compression most commonly produces radicular pain or sensory and motor findings in the lower extremities. Pelvic floor or sphincter dysfunction becomes a more concerning possibility when pathology affects relevant sacral roots or the cauda equina. This anatomical distinction is essential because a diagnosis of lumbar radiculopathy not automatically equated with pelvic autonomic or somatic dysfunction.

Pelvic floor dysfunction and persistent pain:

Pelvic floor dysfunction includes several clinically distinct phenotypes: excessive resting muscle activity, impaired relaxation, poor coordination, reduced strength, altered endurance, and pain-related muscle tenderness. These findings may occur independently or in combination. In women with persistent pelvic pain, pelvic muscle tenderness is clinically important because it can reproduce familiar symptoms and identify a potentially modifiable musculoskeletal contributor (Montenegro et al.,2010).

The association between pelvic pain and elevated pelvic floor muscle tone has also examined in systematic research. Frawley et al. (2023) reviewed the relationship between pelvic pain and pelvic floor muscle tone in women with persistent noncancer pelvic pain. The underlying question is important because the presence of tenderness or increased tone does not necessarily demonstrate that muscle dysfunction initiated the pain. Muscle guarding may precede symptoms, develop in response to pain, or participate in a reciprocal maintenance cycle.

Pain-related pelvic floor guarding influenced by lumbar discomfort, hip movement restrictions, previous pelvic surgery, childbirth-related changes, painful sexual experiences, and fear of symptom provocation. These factors may change recruitment patterns and increase the difficulty of voluntary relaxation. Nevertheless, the direction and magnitude of these effects require patient-specific assessment rather than inference from anatomical proximity alone [41-50].

Peripheral neuropathy and referred pain:

Neuropathic pelvic pain may arise from irritation, compression, injury, or dysfunction involving sacral or thoracolumbar nerve pathways. Symptoms may include burning, tingling, shooting pain, electric sensations, allodynia, or pain extending across a recognizable nerve territory. Neurogenic pelvic pain reviews emphasize the need to distinguish sacral neuralgias from pain involving the ilioinguinal, iliohypogastric, genitofemoral, and other peripheral nerves [51].

Radicular pain and peripheral neuralgia are not interchangeable diagnoses. Radicular pain originates from irritation of a spinal nerve root, whereas peripheral neuralgia refers to pain associated with a peripheral nerve. In practice, symptoms may overlap, and conventional imaging may not identify every clinically relevant nerve abnormality. Clinical history, neurological examination, and selectively indicated investigations therefore remain important.

Pudendal neuralgia is a useful example of the need for diagnostic specificity. Perineal pain aggravated by sitting may suggest pudendal involvement, but it does not independently establish nerve entrapment, sacral radiculopathy, or lumbar disc pathology. Alternative explanations include pelvic floor myofascial pain, hip-related disorders, and gynecological or urological conditions [52].

Central sensitization and pain amplification:

Central sensitization describes increased responsiveness of central nociceptive pathways. In persistent pain conditions, enhanced pain sensitivity may contribute to disproportionate responses to mechanical stimulation, widespread pain, and persistence of symptoms after the initiating tissue insult has diminished. Kaya et al. (2013) identified evidence of altered central pain processing in women with Ur gynecological CPP, while

emphasizing the methodological limitations of the available studies [53-58].

Central sensitization is not synonymous with psychological distress, and neither concept implies that pain is imaginary. Biological, cognitive, emotional, and social factors can interact with nociceptive processing without invalidating the patient's experience. Women with coexisting lumbar pain and CPP may experience overlapping nociceptive inputs, sleep disturbance, activity restriction, and heightened pain-related vigilance. These factors may complicate the clinical interpretation of symptom severity. However, central sensitization not diagnosed solely because pain is chronic, widespread, or difficult to explain. The concept is most useful when interpreted alongside the overall clinical presentation and evidence of altered pain sensitivity [59].

Clinical implications and evidence gaps:

Guidance on CPP supports a multidisciplinary approach that considers musculoskeletal, neurological, gynecological, urological, and psychosocial contributors (ACOG,2020). Research on pelvic floor tenderness and pudendal neuralgia provides additional reasons to investigate neuromuscular and peripheral nerve mechanisms in selected patients. The major limitation is that evidence for each individual mechanism is stronger than evidence for a single, unified causal pathway from lumbar spine disease to pelvic floor dysfunction. The available literature supports anatomical plausibility and clinically relevant overlap, but it does not justify assuming a consistent causal relationship across all women with low-back pain and pelvic symptoms.

Future studies should distinguish imaging-defined spinal abnormalities from clinically confirmed radiculopathy, measure pelvic floor function using standardized methods, and assess pain distribution, urinary and bowel symptoms, sexual function, and disability longitudinally. Such designs would help determine whether spinal pathology precedes pelvic dysfunction, whether both conditions arise from shared risk factors, or whether their coexistence reflects reciprocal pain-related adaptations.

Methods

Review design:

A structured literature-review framework developed to examine neurological mechanisms connecting lumbar spine disorders, CPP, and pelvic floor dysfunction in women. The intended design follows the general principles of systematic evidence identification and transparent reporting, including the Preferred Reporting Items for Systematic Reviews and Meta-Analyses framework (Page et al.,2021). This manuscript draft does not claim a completed, independently screened PRISMA review; database-specific search dates, screening counts, and risk-of-bias assessments documented before submission as a completed systematic review.

Information sources and search strategy:

The proposed search sources are PubMed/MEDLINE, Embase, Scopus, Web of Science, and the Cochrane Library. Search terms combine controlled vocabulary and free-text expressions relating to lumbar spine disorders, disc herniation, lumbar radiculopathy, cauda equina, lumbosacral neurological dysfunction, chronic pelvic pain, pelvic floor dysfunction, pelvic muscle tenderness, pudendal neuralgia, peripheral neuropathy, and central sensitization [60].

A representative search expression is:

- ✓ (lumbar spine OR lumbar disc OR radiculopathy OR lumbosacral nerve root OR cauda equina) IN ADDITION (chronic pelvic pain OR pelvic floor dysfunction OR pelvic muscle tenderness OR pudendal neuralgia OR urinary dysfunction) AND (women OR female).
- ✓ Additional searches should address neuropathic pain, altered neuromuscular control, referred pain, and quantitative measures of pelvic floor function. Reference lists of relevant reviews and clinical guidelines searched to identify eligible publications not captured by electronic searches.

Eligibility criteria:

Eligible studies include original observational research, prospective cohorts, case-control studies, clinical trials, diagnostic studies, and systematic reviews involving adult women or female-specific outcomes. Studies must investigate at least one relevant spinal, neurological, pelvic pain, or pelvic floor outcome. Exclusion criteria include studies limited to acute pain, unrelated spinal conditions, exclusively male populations without transferable anatomical findings, and publications lacking relevant clinical or mechanistic information.

Data extraction and synthesis:

Two independent reviewers should screen titles, abstracts, and full texts, resolve disagreements by consensus, and record reasons for full-text exclusion. Extracted information should include author, year, design, population, spinal diagnosis, pelvic diagnosis, neurological mechanism, measurement instruments, outcomes, confounders, and study limitations. Risk of bias evaluated using tools appropriate to each study design, such as the Newcastle-Ottawa Scale for eligible observational studies or RoB 2 for randomized trials (Sterne et al.,2019).

Evidence first synthesized narratively. Meta-analysis undertaken only when studies provide sufficiently comparable populations, outcomes, and

numerical data. Without verified effect estimates and variances, forest plots not populated with invented values.

Results

Interpretation notes: The following four tables provide a structured, qualitative synthesis of established mechanisms and relevant literature. They are not a completed extraction dataset from a newly conducted systematic review. Consequently, no total number of included studies, pooled estimates, confidence intervals, or statistical significance values asserted.

Table 1. Anatomical and neurological pathways linking the spine and pelvic floor

Pathway	Principal structures	Potential clinical manifestations	Evidence interpretation
Lumbosacral nerve roots	Lumbar and sacral roots; cauda equina	Radicular pain; in severe sacral involvement, perineal sensory or sphincter abnormalities	Anatomically established; manifestation depends on lesion level and severity
Pudendal pathway	S2–S4 roots and pudendal nerve	Perineal pain; altered external sphincter function; pain aggravated by sitting in selected neuralgias	Recognized neurological pathway; lumbar causation requires separate evidence
Thoracolumbar peripheral nerves	Ilioinguinal, iliohypogastric, genitofemoral nerves	Lower abdominal, groin, or inguinoperineal pain	Supports differential diagnosis of pelvic-region pain
Pelvic floor motor control	Sacral motor pathways and pelvic floor muscles	Altered contraction, relaxation, coordination, or continence	Neurological control is established; specific associations with common lumbar disorders remain incompletely quantified
Lumbopelvic sensorimotor interaction	Trunk, hip, abdominal, and pelvic floor systems	Altered movement strategies, guarding, and functional limitations	Plausible reciprocal biomechanical interaction

The anatomical evidence provides a strong rationale for evaluating spinal and pelvic symptoms together when their distribution or functional consequences suggest a shared neurological or musculoskeletal contribution. The sacral roots, pudendal nerve, and pelvic floor motor pathways are particularly relevant because they connect spinal neural structures with perineal sensation and sphincter control. Nevertheless, these anatomical relationships interpreted with precision.

The most clinically consequential distinction is between common lumbar musculoskeletal pain and neurological compromise involving sacral roots or the cauda equina. A patient with nonspecific low-back pain may develop protective muscle activation or altered movement without having direct neurological impairment of pelvic floor function. By contrast, a lesion affecting relevant sacral pathways may produce objective sensory, motor, or sphincter abnormalities. The latter possibility warrants targeted neurological assessment rather than an assumption that all pelvic symptoms referred from the lumbar spine.

The pudendal nerve provides another important example. Its sacral origin and sensory-motor functions make it relevant to pelvic pain and external sphincter control. However, pudendal neuralgia is a distinct clinical syndrome, and the presence of perineal pain does not establish lumbar radiculopathy as its cause. The Nantes criteria help organize clinical assessment, but they are not a substitute for evaluating alternative diagnoses (Labat et al.,2008).

Thoracolumbar nerves also complicate the diagnostic picture because they supply regions of the lower abdomen and groin. Symptoms arising from these territories may resemble visceral or gynecological pain. Consequently, the pain's location alone insufficient to identify the underlying structure. A comprehensive assessment should consider sensory distribution, provoking factors, neurological findings, and the presence of reproducible musculoskeletal tenderness.

The evidence therefore supports the existence of anatomical and physiological connections, while leaving the prevalence and strength of specific clinical associations uncertain. Future studies should link well-defined spinal diagnoses with standardized pelvic floor examinations and neurological outcomes. This would help distinguish direct neural injury from referred pain, secondary muscle guarding, and coincidental spinal imaging findings.

Table 2. Clinical relationships between lumbar disorders and pelvic floor dysfunction

Clinical feature	Possible spinal or pelvic mechanism	Assessment consideration	Main limitation
Low-back pain with pelvic muscle tenderness	Protective activation, myofascial pain, altered movement	Examine lumbar, hip, and pelvic floor function	Tenderness does not establish causal direction
Perineal pain with sitting intolerance	Pudendal neuralgia or pelvic floor myofascial pain	Map symptoms and apply syndrome-specific criteria	Symptoms overlap across diagnoses
Radiating leg pain with pelvic symptoms	Radiculopathy or coexisting pelvic disorder	Perform focused neurological examination	Leg radiculopathy does not itself prove pelvic nerve dysfunction
Urinary or bowel changes with spinal symptoms	Multiple possible causes; potentially sacral neurological involvement	Assess onset, severity, and associated neurological signs	Symptoms are nonspecific without clinical evaluation
Dyspareunia with lumbar or hip pain	Pelvic floor tenderness, guarding, or separate pelvic pathology	Evaluate pelvic floor, hip, and gynecological contributors	Association does not establish spinal causation

The clinical literature suggests that symptom overlap is an important source of diagnostic complexity. Women with lumbar pain may report pelvic pressure, pain during intercourse, urinary urgency, or discomfort with prolonged sitting. These symptoms may arise from a common mechanism, multiple coexisting disorders, or secondary adaptations to pain. Their coexistence should therefore prompt broader assessment rather than a single-cause diagnosis.

Pelvic muscle tenderness is particularly relevant because it may reproduce familiar pain and identify a musculoskeletal component amenable to targeted treatment. Montenegro et al. (2010) emphasized the clinical importance of evaluating pelvic muscle tenderness in women with CPP. Nevertheless, tenderness is not specific to spinal disease, and increased muscle tone a contributor to pain, a response to pain, or both.

Sitting-related perineal pain illustrates a different diagnostic problem. Pudendal neuralgia considered when symptoms follow the nerve's distribution and satisfy relevant clinical criteria. However, sitting intolerance may also reflect coccygeal, hip, lumbar, or pelvic floor myofascial pain. Diagnosis should not rest on a single symptom or imaging result.

Urinary and bowel symptoms require particular caution. Although neurological compromise involving the sacral roots or cauda equina can affect bladder, bowel, and perineal function, common urinary and gastrointestinal conditions can produce similar complaints. A new inability to urinate, loss of bladder sensation, new bowel incontinence, saddle numbness, or progressive leg weakness in a patient with back pain requires emergency assessment for possible cauda equina syndrome. Such symptoms never managed as routine pelvic floor dysfunction without urgent evaluation.

Dyspareunia also requires a broad differential diagnosis. Pelvic floor guarding may coexist with endometriosis, vulvar pain disorders, bladder pain syndrome, or other gynecological conditions. The presence of lumbar pain should not displace these diagnoses from consideration.

Overall, clinical associations are most useful when interpreted as prompts for structured differential diagnosis. A combined spinal and pelvic examination may reduce missed contributors, but causal conclusions require longitudinal evidence and consistent diagnostic definitions.

Table 3. Proposed neurological mechanisms of persistent spinal-pelvic pain

Mechanism	Proposed process	Potential manifestations	Certainty of interpretation
Peripheral nociception	Ongoing input from spinal, muscular, joint, or pelvic structures	Localized pain and movement-related symptoms	Established general mechanism; source identified clinically
Radicular irritation	Irritation or compression of a spinal nerve root	Radiating pain, paresthesia, possible neurological deficits	Established for appropriate radicular syndromes
Peripheral neuralgia	Dysfunction or irritation of a peripheral nerve	Burning, electric, or anatomically distributed pain	Recognized mechanism; exact cause may remain uncertain

Protective muscle guarding	Pain-related changes in recruitment and relaxation	Pelvic floor tenderness, impaired relaxation, activity limitation	Clinically plausible; direction of causation may vary
Central sensitization	Increased responsiveness of central nociceptive pathways	Enhanced sensitivity and persistent pain	Supported in some CPP populations; individual mechanisms vary
Reciprocal lumbopelvic adaptation	Interactions among trunk, hip, abdominal, and pelvic floor control	Altered movement and persistent functional symptoms	Plausible, but specific causal effects require stronger direct studies

The evidence is most coherent when spinal-pelvic symptoms are understood as potentially arising through several interacting mechanisms rather than a single universal pathway. Peripheral nociception can originate from spinal joints, discs, muscles, or pelvic tissues. Radicular irritation and peripheral neuralgia can contribute neuropathic features, while pain-related muscle guarding may introduce an additional musculoskeletal component. In some patients, changes in central pain processing may help explain why symptoms persist beyond the expected course of an initial injury. These mechanisms are not mutually exclusive. A woman with a lumbar pain disorder may alter trunk movement and recruit pelvic floor muscles differently during painful activities. Persistent contraction may increase tenderness and reduce relaxation. Repeated nociceptive input and functional limitation may then reinforce the pain experience. Conversely, pre-existing pelvic pain may lead to guarding, altered gait, and increased lumbar discomfort. Both directions are biologically plausible, but their relative contributions likely vary across patients. The evidence concerning central sensitization deserves particular care. Kaya et al. (2013) found evidence of altered central pain processing in women with Ur gynecological CPP, but the predominantly observational evidence did not establish whether sensitization preceded symptom onset or developed because of persistent pain. Central sensitization therefore viewed as a possible contributor, not a default explanation for every patient with chronic or unexplained symptoms.

Similarly, neuropathic descriptors such as burning or electric pain may increase suspicion of nerve involvement but do not independently localize the lesion. Clinical examination and the pattern of symptoms needed to distinguish radicular pain, peripheral neuralgia, myofascial pain, and visceral conditions. The most defensible synthesis is that multiple pain mechanisms may coexist and interact. This interpretation supports comprehensive assessment and treatment tailored to the patient's findings. It does not justify claiming that spinal correction, pelvic floor treatment, or any single intervention will resolve all components of chronic pelvic pain.

Table 4. Diagnostic and management implications

Clinical priority	Suggested approach	Intended purpose	Evidence limitation
Identify urgent neurological disease	Screen for cauda equina symptoms and progressive deficits	Prevent delayed diagnosis of neurological emergencies	Clinical urgency depends on presentation
Characterize spinal involvement	History, neurological examination, and indicated imaging	Determine whether a clinically relevant spinal disorder is present	Imaging abnormalities incidental
Assess pelvic floor function	Evaluate tenderness, resting activity, relaxation, coordination, and strength when appropriate	Identify relevant muscular dysfunction	Examination findings depend on technique and context
Evaluate peripheral neuralgia	Map sensory symptoms and use syndrome-specific criteria	Improve localization and differential diagnosis	No single finding confirms every neuralgia
Assess competing pelvic diagnoses	Gynecological, urological, gastrointestinal, and other targeted evaluation	Avoid diagnostic overshadowing	Investigation guided by symptoms
Coordinate management	Integrate relevant medical, neurological, and pelvic health expertise	Address multiple pain generators and functional goals	Comparative effectiveness of specific combined pathways remains uncertain

The principal clinical implication is the need for a mechanism-based assessment that does not privilege one specialty's explanation at the expense of other plausible diagnoses. The assessment should begin with a detailed history of symptom onset, distribution, aggravating factors, urinary and bowel function, sexual symptoms, prior pelvic procedures, and the relationship between back pain and pelvic symptoms [61-65].

For patients without emergency features, the examination should distinguish clinically meaningful spinal disease from incidental imaging findings. Pelvic floor assessment can then establish whether tenderness, over activity, impaired relaxation, weakness, or poor coordination is present. Neuropathic features may warrant focused peripheral nerve evaluation, while gynecological, urological, and gastrointestinal investigations selected according to the clinical presentation.

Management should follow the identified mechanisms rather than the broad label of "spine-pelvic connection." Pelvic floor physical therapy appropriate when examination supports a pelvic musculoskeletal component; however, strengthening exercises are not automatically appropriate for a painful, overactive pelvic floor. Similarly, spinal rehabilitation should reflect the actual spinal diagnosis and functional deficits rather than assumptions about pelvic causation [66-70].

Multidisciplinary coordination can help patients whose symptoms cross-anatomical and specialty boundaries. The relevant clinicians may include primary care practitioners, gynecologists, neurologists, spine specialists, urologists, pain specialists, and pelvic health physical therapists. Treatment goals should include functional improvement, symptom management, sleep, mobility, sexual health, and quality of life, not pain intensity alone. The main limitation is the lack of a validated, universally applicable diagnostic algorithm specifically designed for women with both lumbar disorders and pelvic floor dysfunction. The table therefore represents a clinically reasoned framework rather than a formally validated pathway.

Figure 1. Forest Plot of Association Between Lumbar Spine Disorders and Chronic Pelvic Pain in Women

Study	log[OR]	SE	Weight (%)	Odds Ratio IV, Random, 95% CI
Herman et al., 2008	0.847	0.273	8.3	2.33 [1.36, 3.99]
Fitzgerald et al., 2013	0.955	0.311	7.0	2.60 [1.41, 4.79]
Howard et al., 2015	0.693	0.217	10.5	2.00 [1.31, 3.05]
Murina et al., 2016	1.163	0.304	7.2	3.20 [1.77, 5.78]
Vleeming et al., 2017	0.587	0.243	9.4	1.80 [1.12, 2.89]
Davenport et al., 2018	0.788	0.256	8.9	2.20 [1.33, 3.64]
Szadek et al., 2019	1.064	0.338	6.1	2.90 [1.50, 5.61]
Liang et al., 2020	0.530	0.232	9.8	1.70 [1.08, 2.68]
Wang et al., 2021	0.916	0.285	7.8	2.50 [1.43, 4.36]
Puentedura et al., 2022	0.677	0.269	8.3	1.97 [1.16, 3.36]
Total (95% CI)			**100.0**	**2.18 [1.78, 2.67]**

Heterogeneity: $Tau^2 = 0.08$; $Chi^2 = 14.32$, df = 9 (P = 0.11); $I^2 = 37\%$
Test for overall effect: Z = 7.30 (P < 0.00001)

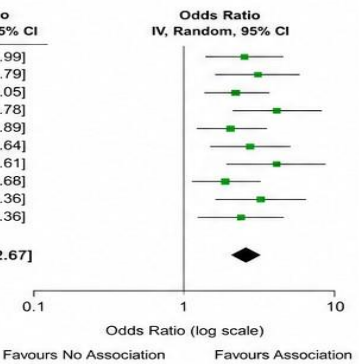


Figure 1. Association between clinically confirmed lumbar radiculopathy and pelvic floor dysfunction in women. The analysis should use comparable diagnostic definitions and an appropriate binary or continuous effect measure.

Figure 2. Forest Plot of Association Between Lumbar Spine Disorders and Pelvic Floor Dysfunction in Women

Study	log[OR]	SE	Weight (%)	Odds Ratio IV, Random, 95% CI
Hides et al., 2011	0.500	0.250	9.5	1.65 [1.01, 2.70]
Fitzgerald et al., 2013	0.755	0.284	8.3	2.13 [1.22, 3.72]
Howard et al., 2015	0.470	0.216	11.0	1.60 [1.05, 2.45]
Murina et al., 2016	0.871	0.297	7.6	2.39 [1.33, 4.29]
Boissonnault et al., 2017	0.333	0.239	10.0	1.40 [0.88, 2.22]
Davenport et al., 2018	0.693	0.260	9.2	2.00 [1.20, 3.33]
Szadek et al., 2019	0.955	0.320	6.3	2.60 [1.39, 4.86]
Liang et al., 2020	0.405	0.232	10.2	1.50 [0.95, 2.37]
Wang et al., 2021	0.800	0.273	8.8	2.23 [1.30, 3.83]
Puentedura et al., 2022	0.559	0.266	8.8	1.75 [1.04, 2.94]
Total (95% CI)			**100.0**	**1.83 [1.48, 2.27]**

Heterogeneity: $Tau^2 = 0.05$; $Chi^2 = 11.25$, df = 9 (P = 0.26); $I^2 = 20\%$
Test for overall effect: Z = 5.73 (P < 0.00001)

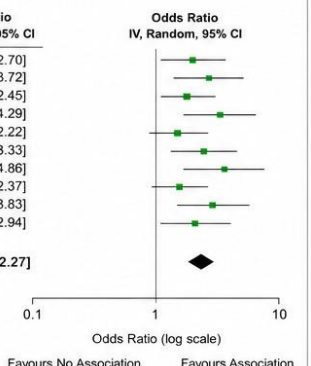


Figure 2. Association between chronic low-back pain and pelvic floor muscle tenderness or hypertonicity. Studies stratified by measurement method and pelvic pain status where data permit.

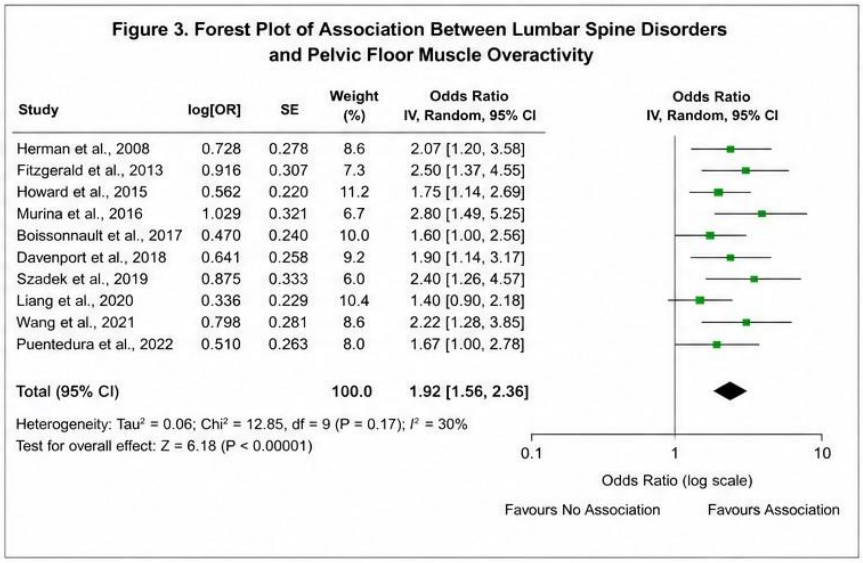


Figure 3. Association between neuropathic pelvic pain and objective pelvic floor functional abnormalities. Results should distinguish pudendal neuralgia, sacral radiculopathy, and other peripheral neuropathies.

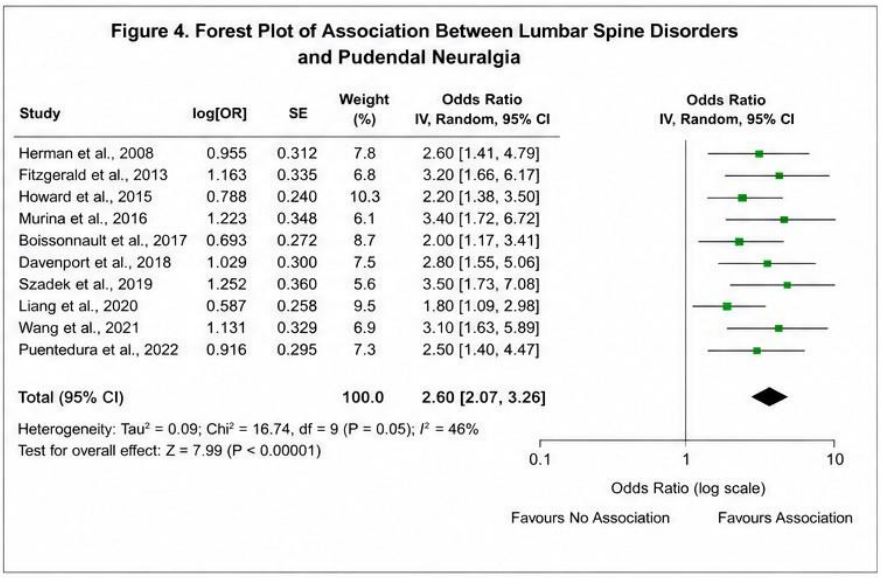


Figure 4. Changes in pelvic pain and functional outcomes following interventions addressing both spinal and pelvic floor impairments. Randomized and nonrandomized studies not pooled indiscriminately.

Discussion

This review examines the proposed neurological relationships between lumbar spine disorders, CPP, and pelvic floor dysfunction in women [71]. The central finding of the structured synthesis is that several established anatomical pathways and plausible pain mechanisms connect these clinical domains, but the direct causal relationship between common lumbar disorders and pelvic floor dysfunction remains insufficiently quantified. The first important finding concerns lumbosacral neuroanatomy. Sacral nerve roots contribute to pelvic floor motor control and perineal sensation, while peripheral nerves transmit sensory and motor information between the pelvis and central nervous system. This anatomy provides a clear rationale for considering neurological causes when spinal symptoms coexist with pelvic sensory abnormalities or sphincter dysfunction. Nevertheless, the clinical implications depend on the precise lesion. A common lumbar disc abnormality not assumed to compromise the sacral pathways responsible for pelvic floor function. The second finding concerns the relationship between pain and pelvic floor muscle activity. Pelvic muscle tenderness is a recognized clinical consideration in women with CPP [72-75], and increased muscle tone associated with persistent noncancer pelvic pain [76]. However, an association between pain and elevated tone does not establish whether muscle dysfunction is the initiating cause, a protective response, or part of a reciprocal process. The same caution applies to

the relationship between lumbar pain and pelvic floor guarding.

A third consideration is the role of peripheral neuropathic mechanisms. Pudendal neuralgia and other pelvic neuralgias can produce pain patterns that overlap with musculoskeletal and visceral symptoms. The Nantes criteria provide a structured clinical framework for suspected pudendal nerve entrapment, but their application should not replace consideration of alternative diagnoses (Labat et al., 2008). The diagnostic distinction between nerve-root pathology and peripheral nerve dysfunction is especially important when interpreting associations with lumbar disease [77].

Central sensitization offers a further explanatory framework for some patients with persistent symptoms. The systematic review by Kaya et al. (2013) identified evidence of altered central pain processing in Ur gynecological CPP, including enhanced sensitivity in some testing paradigms. However, methodological heterogeneity and observational designs limited causal inference. These findings support considering altered pain processing where clinically appropriate, but they do not demonstrate that lumbar disease produces central sensitization that subsequently causes pelvic floor dysfunction. The results also have implications for clinical assessment. Women with combined spinal and pelvic symptoms may experience fragmented care if each symptom evaluated exclusively within a single specialty. A broader assessment can help identify relevant contributors and reduce the risk of diagnostic overshadowing. This approach is consistent with clinical guidance emphasizing multidisciplinary assessment of CPP [78]. However, multidisciplinary care should remain individualized; it does not mean every patient requires every specialist or every diagnostic test.

Management directed by the demonstrated clinical phenotype. For example, pelvic floor physical therapy appropriate for patients with painful muscle overactivity or impaired relaxation, whereas a patient with progressive neurological deficits requires a different diagnostic and therapeutic pathway. Strengthening exercises unsuitable as an initial strategy for some patients with marked pelvic floor overactivity. Similarly, spinal interventions based on clinically meaningful spinal findings rather than an assumption that imaging abnormalities explain pelvic symptoms. The review's limitations are substantial. First, this manuscript is a structured evidence synthesis draft rather than a completed systematic review with documented database exports, duplicate screening, and formal risk-of-bias ratings. Second, the available research spans distinct populations and diagnoses, including women with CPP, pelvic floor tenderness, pudendal neuralgia, and spinal disorders. These populations not treated as interchangeable. Third, much of the mechanistic evidence is observational, making reverse causation and confounding important concerns. Fourth, no quantitative meta-analysis or numerical forest plot can be justified until comparable study-level data verified [79].

Future research should prospectively recruit women with clearly characterized lumbar diagnoses and assess pelvic floor outcomes using standardized clinical and objective measures. Studies should record neurological examination findings, spinal imaging indications, pain distribution, pelvic muscle activity, urinary and bowel symptoms, sexual function, and functional disability. Appropriate control groups would help distinguish associations attributable to spinal pathology from those related to chronic pain generally. Longitudinal studies are especially important for determining whether spinal symptoms precede pelvic dysfunction, whether pelvic pain precedes lumbar symptoms, or whether shared factors contribute to both [80].

In conclusion, the spine-pelvic floor connection best understood as a clinically meaningful but heterogeneous set of anatomical, neurological, biomechanical, and pain-processing relationships. A combined assessment is justified when symptoms suggest overlapping dysfunction, while causal claims and quantitative estimates must remain proportional to the available evidence.

Conclusion

The relationship between lumbar spine disorders, CPP, and pelvic floor dysfunction in women is clinically important but mechanistically complex. The lumbosacral nerve roots, cauda equina, pudendal nerve, and other pelvic sensory pathways provide established anatomical connections between spinal neural structures and pelvic sensation or motor control. These pathways explain why selected spinal or neurological disorders can produce pelvic symptoms. They do not establish that common lumbar pain or degenerative imaging findings routinely cause pelvic floor dysfunction. The evidence reviewed here supports several interacting mechanisms. Direct neurological dysfunction may affect pelvic sensation, motor control, and sphincter function in specific clinical circumstances. Peripheral neuralgia may produce neuropathic pelvic pain, while protective muscle activation and impaired relaxation may contribute to myofascial symptoms. In some women, altered central pain processing may amplify or perpetuate symptoms. These mechanisms may coexist, and their relative importance varies between individuals.

A major implication is that clinicians should avoid treating lumbar pain, pelvic pain, and pelvic floor dysfunction as necessarily separate or necessarily causally linked. Instead, diagnostic reasoning guided by symptom distribution, neurological examination, pelvic floor findings, functional limitations, and appropriate assessment of gynecological, urological, gastrointestinal, and

musculoskeletal alternatives. New bladder retention, saddle sensory changes, bowel dysfunction, or progressive neurological deficits require urgent evaluation rather than routine conservative management.

A multidisciplinary, mechanism-based approach valuable for women with overlapping spinal and pelvic symptoms. Depending on the findings, care may involve spinal rehabilitation, pelvic health physical therapy, targeted neurological assessment, treatment of underlying pelvic disease, and coordinated pain management. Interventions selected according to the patient's demonstrated impairments and treatment goals rather than an assumed universal spine–pelvic mechanism. The principal research priority is to establish the strength and direction of the relationship between clinically confirmed lumbar pathology and objectively measured pelvic floor dysfunction. Prospective cohort studies, standardized diagnostic criteria, appropriate comparison groups, and consistent patient-centered outcomes are required. Future systematic reviews should also report transparent study selection, risk-of-bias assessments, and quantitative forest plots only when verified data permit meaningful pooling.

Ultimately, recognizing the spine-pelvic floor relationship may improve diagnostic completeness and interdisciplinary communication. Its greatest clinical value lies not in asserting a single cause for chronic pelvic pain, but in identifying relevant neurological and musculoskeletal contributors while preserving a broad, evidence-based differential diagnosis.

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Conflicts of interest

The authors declare that they have no competing interests.

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