

Yoga and Neuroimmune Modulation in Chronic Pelvic Pain Syndrome: Inflammatory Biomarkers, Central Sensitization, and Pain Phenotypes: A Narrative Review

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ABSTRACT

Background: Chronic pelvic pain syndrome (CPPS) is a heterogeneous, multisystem pain condition characterised by persistent pelvic pain with variable urogenital, bowel, sexual, and psychosocial symptoms. Accumulating evidence indicates that many patients exhibit neuroimmune activation (local and systemic inflammatory signalling) together with nociplastic features driven by central sensitization.

Aim: To synthesise mechanistic and clinical evidence on how yoga may alleviate CPPS through neuroimmune modulation—particularly inflammatory pathways and central sensitization—and to outline a phenotype-informed framework for future research and clinical translation.

Narrative Review Method: We conducted a narrative review of peer-reviewed human studies and key mechanistic literature indexed in PubMed, Scopus, Web of Science, and Google Scholar (2000–2025). Evidence was synthesised thematically across (i) CPPS phenotypes and pain mechanisms, (ii) inflammatory biomarkers and neuroimmune pathways, (iii) central sensitization and autonomic–HPA axis dysregulation, and (iv) clinical trials of yoga and closely related mind–body interventions in pelvic pain.

Key Findings: CPPS is associated with altered immune–inflammatory profiles (e.g., chemokines/cytokines, neurotrophins such as nerve growth factor, and oxidative stress markers) and with central pain amplification reflected in widespread hyperalgesia, symptom clustering, and comorbidity with chronic overlapping pain conditions. Yoga plausibly targets these mechanisms via vagal–autonomic regulation, reduced HPA-axis hyperarousal, improved affective regulation, and downstream suppression of pro-inflammatory transcriptional programs. Direct pelvic pain trials of yoga are limited but suggest improvements in pain intensity and quality of life; related evidence from urological pelvic pain, bladder pain syndrome, and central sensitivity syndromes supports benefits on stress reactivity, pain catastrophizing, sleep, and inflammatory signalling.

Clinical Implications: A phenotype-guided yoga prescription—mapping clinical presentation (visceral, pelvic floor myofascial, neuropathic, and nociplastic/centralized features) to targeted yoga components

(gentle asana, diaphragmatic breathing, relaxation, and meditation)—may optimize responsiveness and enable biomarker-informed monitoring.

Conclusion: Yoga has strong mechanistic plausibility as an adjunct therapy for CPPS by modulating neuroimmune activation and central sensitization. Robust, adequately powered trials integrating pain phenotyping, autonomic metrics, and inflammatory biomarkers are required to define responders and mechanisms of action.

KEYWORDS: Chronic pelvic pain syndrome; urological chronic pelvic pain; neuroimmune modulation; inflammation; cytokines; nerve growth factor; central sensitization; nociplastic pain; heart rate variability; yoga; pranayama; meditation.

ABBREVIATIONS

Abbreviation	Meaning
ANS	Autonomic nervous system
BPS/IC	Bladder pain syndrome / interstitial cystitis
CP/CPSP	Chronic prostatitis / chronic pelvic pain syndrome
CPPS	Chronic pelvic pain syndrome
CRP	C-reactive protein
CSI	Central Sensitization Inventory
CTRA	Conserved transcriptional response to adversity
HRV	Heart rate variability
HPA	Hypothalamic–pituitary–adrenal
IL	Interleukin
NGF	Nerve growth factor
NF-κB	Nuclear factor kappa-B
NRS	Numeric rating scale
PCS	Pain Catastrophizing Scale
PSS	Perceived Stress Scale
QOL	Quality of life
QST	Quantitative sensory testing
RCT	Randomized controlled trial
TNF-α	Tumor necrosis factor-alpha
UPOINT	Urinary, Psychosocial, Organ-specific, Infection, Neurologic/systemic, Tenderness phenotype domains
UCPPS	Urological chronic pelvic pain syndromes
VAS	Visual analogue scale

INTRODUCTION

Chronic pelvic pain syndrome (CPPS) is best understood as a clinical syndrome rather than a single-organ disease: pelvic pain persists for months, frequently co-occurring with urinary, bowel, sexual, and mood symptoms, and often lacks a unifying structural explanation. Contemporary guidance emphasises

multidisciplinary assessment and management because symptom drivers may arise from pelvic viscera, pelvic floor musculature, peripheral nerves, immune–inflammatory processes, and central pain amplification, with substantial overlap across gynecologic and urologic presentations [1,2].

In urology, the label CP/CPPS refers to chronic prostatitis–like symptoms without demonstrable infection (NIH category III) and is a prototypical model of urological chronic pelvic pain syndromes (UCPPS) [3]. In gynecology, chronic pelvic pain encompasses cyclical and non-cyclical pain conditions, pelvic floor myalgia, and chronic overlapping pain conditions (e.g., irritable bowel syndrome, fibromyalgia, migraine, temporomandibular disorders) that cluster with pelvic pain and predict higher disability [2,4,5]. The Multidisciplinary Approach to the Study of Chronic Pelvic Pain (MAPP) Research Network and other cohort initiatives have strengthened the view that many CPPS phenotypes represent systemic pain vulnerability states in which neuroimmune pathways and central sensitization interact with peripheral triggers [4,6].

Yoga is a mind–body discipline integrating physical postures (asana), breath regulation (pranayama), and meditation/relaxation practices. In chronic pain populations, yoga has demonstrated improvements in pain, function, and psychosocial outcomes, and mechanistic studies suggest effects on stress physiology, autonomic balance, inflammatory signalling, and pain-related cognitive–affective processes [7,8]. For CPPS, where neuroimmune activation and central sensitization contribute to symptom persistence and treatment resistance, yoga’s multi-component nature makes it a compelling adjunct therapy.

However, a major barrier to clinical translation is heterogeneity: patients differ in symptom clusters, pain mechanisms (nociceptive, neuropathic, nociplastic), comorbidities, and biological signatures. Phenotype-guided care frameworks—such as the UPOINT system for CP/CPPS—aim to stratify patients for mechanism-matched treatment [10,11]. Aligning yoga components with identifiable pelvic pain phenotypes and measurable neuroimmune endpoints could improve trial design and clarify who is most likely to benefit.

AIM AND OBJECTIVES

Aim

To synthesise evidence on yoga as a neuroimmune-modulating intervention for CPPS, with specific focus on inflammatory biomarkers, central sensitization, and clinical pain phenotypes.

Objectives

1. To summarise the clinical spectrum and phenotype frameworks relevant to CPPS.
2. To review peripheral inflammatory and immune pathways implicated in pelvic pain persistence.
3. To evaluate evidence linking central sensitization and autonomic–HPA axis dysregulation with CPPS symptom severity.
4. To map plausible mechanisms by which yoga may influence inflammatory signalling, autonomic regulation, and pain processing.
5. To appraise clinical evidence for yoga and closely related mind–body interventions in pelvic pain and UCPPS.
6. To propose a phenotype-guided yoga framework with candidate biomarkers and outcome measures for future trials.

METHODOLOGY (NARRATIVE REVIEW METHOD)

This narrative review was designed to integrate mechanistic and clinical evidence relevant to yoga and neuroimmune modulation in CPPS.

Data sources and search strategy: Searches were conducted across PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for literature published between January 2000 and December 2025. Search terms were combined using Boolean operators and adapted per database. A representative search string was: ("chronic pelvic pain" OR "chronic pelvic pain syndrome" OR "urological chronic pelvic pain" OR "CP/CPPS" OR "interstitial cystitis" OR "bladder pain syndrome") AND (yoga OR pranayama OR "mind body" OR meditation OR "yoga nidra" OR mindfulness) AND (inflammation OR cytokine* OR chemokine* OR "nerve growth factor" OR "central sensitization" OR nociplastic OR neuroimmune OR microglia OR "heart rate variability" OR cortisol).

Eligibility criteria: We included peer-reviewed human studies (RCTs, quasi-experimental studies, pilot/feasibility trials, observational studies) examining yoga or yoga-based practices in pelvic pain syndromes and/or reporting relevant outcomes (pain, QOL, psychosocial measures, autonomic markers, inflammatory biomarkers). We also included key mechanistic and clinical framework papers (phenotyping systems, biomarker studies, central sensitization literature) that contextualize CPPS neuroimmune mechanisms. Exclusion criteria were: animal-only studies (except when cited briefly for mechanistic plausibility), non-English articles without accessible translation, and publications lacking sufficient methodological details.

Study selection and synthesis: Titles and abstracts were screened for relevance, followed by full-text review for inclusion. Evidence was synthesised thematically, emphasising biological plausibility, consistency of findings, clinical relevance, and limitations. Where direct yoga trials in CPPS were scarce, we included closely related pelvic pain mind–body trials and mechanistic evidence from central sensitivity syndromes to support hypothesis generation.

MAIN BODY (THEMATIC SYNTHESIS)

1. Clinical Spectrum of CPPS and Urological Chronic Pelvic Pain Syndromes

CPPS is commonly operationalized as pelvic pain persisting for ≥ 3 –6 months, perceived in structures related to the pelvis, and associated with functional impairment; importantly, symptom persistence does not require an identifiable ongoing tissue injury [1,2]. In men, CP/CPPS accounts for the majority of prostatitis presentations and is characterised by pelvic/perineal pain, lower urinary tract symptoms, and sexual dysfunction, often fluctuating over time [3]. In women, chronic pelvic pain encompasses dysmenorrhea-related and non-cyclical pain, dyspareunia, bladder pain, bowel symptoms, and pelvic floor tenderness—frequently presenting as a chronic overlapping pain condition cluster [2,5].

Large observational cohorts (e.g., MAPP) demonstrate that patients with UCPPS frequently exhibit widespread symptoms beyond the pelvis, including fatigue, sleep disturbance, anxiety/depression, and pain at extra-pelvic sites, supporting the concept of systemic vulnerability and central pain amplification in subsets of patients [4,6]. Clinically, this matters because peripherally targeted treatments (antibiotics, anti-inflammatories, alpha-blockers) often show modest or inconsistent benefit when central sensitization and neuroimmune dysregulation predominate [12].

2. Pain Phenotypes and Patient Stratification

Phenotyping frameworks aim to reduce heterogeneity by grouping patients according to dominant symptom drivers. For male CP/CPPS, the NIH Chronic Prostatitis Symptom Index (NIH-CPSI) provides

a standardized symptom metric across pain, urinary symptoms, and QOL impact [13]. The UPOINT system further stratifies patients across domains—Urinary, Psychosocial, Organ-specific, Infection, Neurologic/systemic, and Tenderness—providing a mechanism-informed rationale for multimodal care [10,11]. Similar multidomain phenotyping has been applied to bladder pain syndrome/interstitial cystitis (BPS/IC) and female chronic pelvic pain, where pelvic floor myofascial pain, visceral hypersensitivity, neuropathic features, and centralized/nociplastic traits may co-exist.

From a mechanistic perspective, pelvic pain presentations can be conceptualised within three overlapping pain mechanisms: nociceptive (ongoing tissue inflammation/irritation), neuropathic (nerve injury/entrapment), and nociplastic (pain arising from altered nociception without clear tissue damage or somatosensory system lesion, closely aligned with central sensitization) [14,15]. In women with chronic pelvic pain, higher nociplastic burden is associated with greater pain severity and interference and with pelvic myofascial pain, supporting clinical relevance for central pain amplification assessment [16]. Tools such as the Central Sensitization Inventory (CSI) can help quantify symptom patterns consistent with central sensitivity syndromes, although it does not diagnose mechanism in isolation [9,17].

Phenotyping is also relevant for research: identifying subgroups with prominent inflammatory signatures versus those with predominant central sensitization may clarify yoga's mechanisms of action and improve signal detection in trials.

3. Peripheral Inflammatory Signalling and Candidate Biomarkers

Evidence from CP/CPSPS and UCSPS indicates that inflammatory signalling can be present even in the absence of overt infection. Studies of expressed prostatic secretions and seminal plasma have reported elevated cytokines and chemokines, including IL-8 and epithelial neutrophil-activating peptide-78 (ENA-78), implicating local immune activation and leukocyte recruitment [18]. Prospective work has suggested that seminal plasma chemokine profiles (including IL-8) may correlate with symptom patterns and could function as surrogate markers of inflammatory activity in CP/CPSPS [19]. Other reports have identified elevated cytokines in prostatic secretions and semen and support the concept of low-grade, persistent inflammatory signalling in subsets of patients [20].

Neurotrophins are a second biomarker class linking immune activation to pain. Nerve growth factor (NGF), a mediator of peripheral sensitization and neurogenic inflammation, has been shown to correlate with pain severity in CP/CPSPS and may represent a mechanistic bridge between inflammation and pain amplification [21]. NGF measured in prostatic fluid has been reported to track with symptom severity and treatment response, highlighting its candidate role as both biomarker and therapeutic target [22].

Oxidative stress is increasingly recognized as a contributor to chronic pelvic pain biology. Reviews synthesize evidence that reactive oxygen species, lipid peroxidation, and impaired antioxidant defense may contribute to tissue irritation, immune activation, and pain persistence in CP/CPSPS [23]. Earlier clinical studies reported oxidative stress markers in prostatic fluid, raising the possibility that redox imbalance is relevant in a subset of men with chronic pelvic pain [24]. These pathways intersect with inflammatory signalling through redox-sensitive transcription factors such as NF- κ B and with endothelial/urothelial barrier integrity—mechanisms also relevant to bladder pain syndromes.

4. Neurogenic Inflammation, Mast Cells, and the Immune–Neural Interface

Neurogenic inflammation describes a feed-forward loop in which peripheral nociceptor activation releases neuropeptides (e.g., substance P, CGRP) that increase vascular permeability and recruit immune cells, which in turn release mediators that further sensitize nociceptors. In pelvic pain, mast cells have received particular attention because they can amplify nociception through histamine, tryptase,

cytokines, and NGF release. Experimental and translational work supports mast cell involvement as a mediator of chronic pelvic pain and a contributor to immune–neural coupling in CP/CPPS [25].

Immunological reviews propose that autoimmunity, epithelial barrier disruption, microbiome-related triggers, and persistent immune activation can maintain a pro-inflammatory milieu within pelvic tissues, with downstream effects on peripheral and central pain pathways [14]. Clinically, these immune–neural loops may manifest as pelvic floor tenderness, allodynia, and symptom flares associated with stress and autonomic arousal.

5. Central Sensitization, Nociceptive Pain, and CNS Neuroinflammation

Central sensitization refers to increased responsiveness of nociceptive neurons in the central nervous system, leading to amplified pain from normal or subthreshold inputs. This phenomenon is a cornerstone concept in chronic pain mechanisms and has important diagnostic and therapeutic implications [26]. In pelvic pain, neuroimaging and experimental pain studies indicate altered central pain processing, including changes in brain structure/function and altered responses to noxious stimulation, suggesting that central changes can perpetuate pelvic pain even when peripheral drivers are weak or absent [27].

Nociceptive pain frameworks highlight that central amplification often co-occurs with fatigue, sleep disturbance, cognitive symptoms, and mood disorders—features that are frequently reported in UCPPS cohorts [4,14–16]. Mechanistically, neuroinflammation—glial activation and cytokine/chemokine signalling within the spinal cord and brain—can induce and maintain central sensitization. Comprehensive reviews describe how neuroimmune mediators and glial-derived factors modulate synaptic transmission and descending pain control, contributing to widespread chronic pain [28]. Human imaging evidence for glial activation in chronic pain conditions further supports neuroimmune contributions to persistent pain [29].

In CPPS, central sensitization may be reflected clinically by widespread pain sensitivity, symptom spread beyond the pelvis, and elevated CSI scores. These features are relevant for yoga because central sensitization is modifiable by interventions that reduce stress reactivity, improve sleep, and enhance descending inhibitory control.

6. Autonomic–HPA Axis Dysregulation and the Vagal Inflammatory Reflex

Chronic pelvic pain frequently fluctuates with stress, sleep disruption, and autonomic arousal, implicating the autonomic nervous system (ANS) and hypothalamic–pituitary–adrenal (HPA) axis as modulators of symptom severity. Central pain amplification models for pelvic pain include altered ANS balance and stress-axis activity as contributors to symptom persistence [27].

The concept of a vagal ‘inflammatory reflex’ provides a mechanistic template linking autonomic regulation to immune signalling: vagal activity can inhibit peripheral cytokine production through cholinergic pathways and modulate systemic inflammatory tone [30,31]. Heart rate variability (HRV), particularly vagally mediated indices, is widely used as a non-invasive proxy of autonomic flexibility and has established measurement standards [32]. Lower HRV is associated with stress vulnerability and may reflect reduced capacity to down-regulate inflammatory and pain responses.

Given that many pelvic pain presentations include heightened threat appraisal, catastrophizing, and anxiety/depressive symptoms, validated psychosocial tools (e.g., PSS, PCS, HADS) can complement mechanistic outcomes to capture yoga-responsive domains that influence central sensitization and pain coping [33–35].

7. Mechanistic Mapping: How Yoga May Influence Neuroimmune Pathways in CPPS

Yoga is not a single exposure but a package of interrelated practices. Mechanistically, it may influence

CPPS through several converging pathways:

1. Down-regulation of inflammatory signalling: RCT evidence shows yogic meditation and restorative yoga can shift leukocyte gene expression away from NF- κ B-linked pro-inflammatory programs under stress [36–38].
2. Autonomic rebalancing and stress-axis modulation: Yoga's emphasis on slow breathing, breath awareness, and relaxation may enhance vagal tone and reduce sympathetic overactivity. Through the inflammatory reflex, improved autonomic regulation may reduce cytokine output and neurogenic inflammation [30–32].
3. Central pain modulation: Yoga may support pain inhibition by reducing hypervigilance, threat appraisal, and pain catastrophizing while improving sleep and mood. Neurobiological models suggest that these cognitive–affective shifts can alter descending pain control and reduce central sensitization burden [26–28].
4. Neurotrophin and neuroplasticity pathways: Reviews of yoga and meditation biology report changes in neurotrophins such as brain-derived neurotrophic factor (BDNF) and improved stress resilience, which may be relevant to central neuroplasticity underlying chronic pain [39].
5. Pelvic floor and musculoskeletal mechanisms: Gentle asana emphasizing hip mobility, spinal decompression, and pelvic floor down-training may reduce myofascial trigger point activity and improve movement confidence. This may be particularly relevant in UPOINT Tenderness and Organ-specific domains.

Collectively, these pathways suggest that yoga could act as a ‘systems intervention’—simultaneously targeting immune signalling, autonomic regulation, and central pain amplification—making it theoretically suited to CPPS heterogeneity.

8. Clinical Evidence: Yoga and Mind–Body Interventions in Pelvic Pain

Direct clinical trials of yoga in CPPS remain limited but are emerging. In women with chronic pelvic pain, a controlled clinical study reported that a structured yoga program added to standard care improved pain and related outcomes compared with conventional management alone, suggesting feasibility and potential benefit of yoga-based pelvic pain rehabilitation [40]. Telehealth-delivered pelvic yoga is also being explored; feasibility work indicates that remote delivery can be acceptable and may improve pain interference and function, addressing access barriers common in pelvic pain care [41].

Because pelvic pain syndromes share neuroimmune and central sensitization mechanisms, evidence from adjacent UCPPS conditions is informative. For BPS/IC, mindfulness-based stress reduction (MBSR)—which shares attentional training and breath-focused practices with yoga—has been tested in randomized designs and associated with symptom improvements and reduced perceived stress [42]. Recent mechanistic pilot work has examined biologic correlates (e.g., microbiome or immune markers) alongside mind–body interventions in BPS/IC, supporting the feasibility of embedding biomarker endpoints within behavioural trials [43]. Additionally, CBT-based interventions for IC/BPS have demonstrated improvements in symptom burden and QOL, reinforcing the relevance of cognitive–affective mechanisms to pelvic pain outcomes and informing yoga trial design (e.g., integrating pain education and coping skills alongside practice) [44,45].

Yoga nidra and iRest-style guided meditation—closely aligned with yogic relaxation practices—have shown promise for persistent pain states, including improvements in pain interference and sleep, and may be particularly relevant to nociplastic phenotypes characterized by hyperarousal and sleep disturbance [46].

Overall, the current evidence base suggests that yoga and related mind–body approaches can improve clinically meaningful outcomes in pelvic pain, but larger, rigorously designed trials with mechanistic endpoints are necessary to confirm efficacy and to identify responder phenotypes.

9. Phenotype-Guided, Mechanism-Informed Yoga Framework for CPPS (Proposed)

A phenotype-guided yoga framework can be constructed by linking (a) dominant clinical phenotype, (b) hypothesised pathway, (c) yoga component, and (d) measurable endpoint. This approach aligns with UPOINT domain logic and with nociceptive/neuropathic/nociplastic conceptual models.

Key principles include:

- Start low and go slow: many patients exhibit hyperalgesia and fear of movement; gentle, non-provocative practice is essential.
- Prioritise breath and down-regulation for centralized phenotypes: slow diaphragmatic breathing, extended exhalation, and guided relaxation may be primary.
- Integrate pelvic floor down-training: cues emphasizing ‘release’ rather than strengthening can support pelvic floor hypertonicity phenotypes.
- Embed self-efficacy: brief education on pain mechanisms and pacing can reduce catastrophizing and improve adherence.

This mapping is summarised in Tables 1–4. Importantly, the framework is not prescriptive; it is intended as a testable hypothesis for mechanism-guided trials.

10. Research Gaps and Future Directions

Despite mechanistic plausibility, several gaps remain:

1. Limited pelvic pain–specific yoga RCTs: Most evidence is from small studies or from related pain conditions. Future trials should be adequately powered, multi-site, and include active comparators (e.g., stretching/education) to control for attention and movement.
2. Need for integrated phenotyping: Trials should incorporate UPOINT (or analogous multidomain frameworks), nociplastic screening (CSI, widespread pain indices), and pelvic floor examination to identify mechanistically coherent subgroups.
3. Biomarker integration: Candidate biomarkers include IL-8/chemokines, NGF, CRP, oxidative stress markers, and transcriptional signatures (e.g., NF-κB-related gene expression), as well as HRV and cortisol. Longitudinal sampling is needed to test mediation (biologic change → symptom change).
4. Outcomes beyond pain intensity: Pain interference, sexual function, sleep quality, and psychological flexibility may be more responsive and clinically meaningful than pain intensity alone. Standardization of core outcome sets for pelvic pain would enhance comparability.
5. Dose, adherence, and safety: Trials should report minutes/week, home practice adherence, and adverse events; restorative, non-provocative protocols are recommended for hyperalgesic patients.

Table 1. Illustrative CPPS clinical phenotypes, key features, and recommended outcome tools

Phenotype / domain	Clinical features (examples)	Candidate tools / endpoints
Visceral/urological (bladder or prostate dominant)	Suprapubic or perineal pain linked to bladder filling/voiding; urgency/frequency; pain with ejaculation; flares with irritants.	NIH-CPSI; O’Leary-Sant IC Symptom/Problem Index; voiding diary; pain NRS/VAS; urinary symptom scores.
Pelvic floor myofascial /	Pelvic floor hypertonicity;	Pelvic floor exam; myofascial

tenderness dominant	trigger points; pain with sitting/penetration; referred pain; dyspareunia; ‘tight’ pelvic floor.	pain rating; pain NRS; pain pressure thresholds/QST; functional scales; movement fear/catastrophizing.
Neuropathic features (pudendal/ilioinguinal etc.)	Burning, shooting pain; allodynia; dermatomal referral; provocation with nerve stretch; sensory changes.	Neuropathic pain questionnaires; QST; sensory mapping; painDETECT/DN4 (if used); analgesic response patterns.
Centralized / nociplastic (central sensitization) dominant	Widespread pain; fatigue; sleep disturbance; cognitive symptoms; multiple comorbid pain conditions; high pain interference.	CSI; widespread pain indices; PROMIS pain interference; sleep scales; HRV; stress measures (PSS); mood (HADS).
Psychosocial stress-reactive phenotype	High stress, anxiety/depression; catastrophizing; trauma history; symptom flares with stress; reduced coping resources.	PCS; PSS; HADS; psychological flexibility measures; HRV/cortisol; treatment expectancy and self-efficacy.
Inflammatory/immune-dominant signature (subset)	Evidence of pelvic inflammation (laboratory/clinical); symptom flares with immune triggers; co-existing inflammatory conditions.	CRP; cytokine/chemokine panels (e.g., IL-8); NGF; oxidative stress markers; transcriptional profiles (NF-κB-related genes).

Table 2. Mechanistic mapping: pathways → biomarkers → clinical links → yoga-related modulators

Pathway	Candidate biomarkers	Clinical relevance in CPPS	Yoga-linked modulators (hypothesised)
Chemokine/cytokine signalling and leukocyte recruitment	IL-8, ENA-78, TNF-α, IL-6, IL-10, CRP	Local inflammatory signalling in prostatic secretions/urine; symptom flares; pelvic tenderness; visceral hypersensitivity [18–21].	Down-regulation of pro-inflammatory gene expression (NF-κB-related); improved stress regulation; enhanced anti-inflammatory tone [36–38].
Neurotrophin-driven peripheral sensitization	NGF; BDNF (exploratory)	NGF correlates with pain severity and may contribute to neurogenic inflammation and peripheral sensitization	Reduced stress-driven neurotrophin dysregulation; improved affect regulation and pain coping; potential neuroplasticity effects

		[21,22].	[39].
Mast cell activation and neurogenic inflammation	Tryptase, histamine metabolites (context-dependent), NGF	Mast cells can amplify nociceptor activation and pain persistence; proposed in CP/CPPS mechanistic models [25].	Autonomic/vagal regulation and relaxation response; reduced stress reactivity and sympathetic drive (which can modulate immune cell activity).
Oxidative stress / redox imbalance	MDA, 8-isoprostane, SOD/GPx, total antioxidant capacity	Redox imbalance contributes to inflammation, tissue irritation, endothelial dysfunction, and pain persistence [23,24].	Improved antioxidant capacity via stress reduction and lifestyle co-benefits; reduced inflammatory transcriptional activity [38,39].
Central sensitization and CNS neuroinflammation	CSI (clinical proxy), QST indices; exploratory cytokines/chemokines in blood/CSF; imaging markers (research)	Central amplification contributes to widespread pain, high interference, and treatment resistance [26–29].	Reduced hyperarousal; improved sleep; decreased catastrophizing; enhanced descending inhibitory control; meditation/relaxation effects [26–28,46].
Autonomic–HPA axis dysregulation	HRV indices; salivary cortisol (awakening response); perceived stress scores	Stress-reactive symptom flares; impaired autonomic flexibility may perpetuate pain and inflammation [27,30–33].	Slow breathing and meditative practices enhance parasympathetic activity; normalization of stress physiology; improved interoception and coping [30–32,36].

Table 3. Selected human studies of yoga/mind–body interventions relevant to pelvic pain mechanisms

Author (Year)	Population / design	Intervention (dose)	Key outcomes	Main findings (brief)
Saxena et al. (2017) [40]	Women with chronic pelvic pain; controlled clinical study	Therapeutic yoga program added to conventional management; supervised + home practice (multi-week)	Pain, functional outcomes, QOL	Yoga adjunct associated with greater improvements in pain and QOL vs conventional management; limited by sample size and

				design.
Huang et al. (2025) [41]	Women with chronic pelvic pain; feasibility study (telehealth)	Remote pelvic yoga program; weekly sessions + home practice	Feasibility, pain interference, function	Tele-yoga feasible/acceptable; preliminary signals for improved function; needs powered RCT.
Kanter et al. (2016) [42]	BPS/IC; randomized trial (mindfulness-based program)	MBSR-style intervention (includes breath awareness and meditation practices)	Symptom indices, stress, QOL	Improved symptoms and stress vs control; demonstrates mind-body relevance in UCPPS.
Shatkin-Margolis et al. (2021) [43]	BPS/IC; pilot mechanistic study	Mind-body intervention with biomarker sampling	Microbiome/biologic correlates; symptoms	Feasibility of integrating biologic measures in behavioural UCPPS trials; exploratory findings.
McKernan et al. (2024) [44]	IC/BPS; randomized trial	CBT-based self-management program	Symptom severity, pain interference, QOL	CBT improved key outcomes; supports cognitive-affective targets also addressed by yoga.
Yu et al. (2022) [45]	IC/BPS; behavioral intervention study	CBT/self-management adjunct to standard care	Symptoms, stress-related outcomes	Improved symptom burden; highlights modifiable central mechanisms.
Barber et al. (2025) [46]	Persistent pain states; clinical trial (iRest/yoga nidra)	Guided yogic relaxation + home practice	Pain interference, sleep, mood	Reduced pain interference and improved sleep; relevant for nociplastic phenotypes.
Black et al. (2013) [36]	Stressed caregivers; randomized trial	Brief daily yogic meditation	Inflammation-related gene expression	Shifted transcriptome dynamics away from NF-κB pro-inflammatory signaling; mechanistic plausibility.
Bower et al. (2014) [37]	Cancer survivors with fatigue; RCT	Restorative Iyengar yoga (12 weeks)	Inflammation-related gene expression; fatigue	Reduced inflammation-related gene expression;

				supports anti-inflammatory effects in humans.
Gautam et al. (2020) [38]	Rheumatoid arthritis; controlled trial	Yoga-based lifestyle intervention (8 weeks)	Psycho-neuro-immune markers; QOL	Improved immune/inflammatory markers and QOL; supports broader neuroimmune modulation.
Saper et al. (2017) [47]	Chronic low back pain; RCT	Yoga vs physical therapy/education	Pain, function	Yoga improved pain/function; supportive evidence for chronic pain benefits applicable to pelvic pain models.

Table 4. Proposed phenotype-guided yoga prescription and candidate mechanistic endpoints for CPPS trials (hypothesis-generating)

Phenotype emphasis	Yoga components (examples)	Suggested dose (example)	Primary outcomes	Mechanistic endpoints
Centralized/nociplastic dominant	Diaphragmatic breathing (5–10 min); long-exhale pacing; guided relaxation/yoga nidra (15–20 min); mindfulness meditation; gentle floor-based mobility	30–45 min/day, 5–6 days/week for 8–12 weeks	Pain interference; sleep quality; stress; QOL	HRV (RMSSD); salivary cortisol; CSI; inflammatory gene expression (NF-κB-related, optional)
Pelvic floor tenderness/myofascial dominant	Gentle hip openers within comfort; supported child’s pose; supine bound-angle with support; cat–cow; pelvic floor ‘release’ cues; body scan	45 min/session, 3–5 days/week for 8–12 weeks	Pelvic pain NRS; dyspareunia; function	Trigger point tenderness ratings; pressure pain thresholds; HRV; PCS
Visceral/urological dominant (bladder/prostate)	Breath-focused relaxation; gentle spinal twists; restorative	30–45 min/session, 3–5 days/week for 8–12 weeks	NIH-CPSI / IC indices; urinary symptoms; pain	Inflammatory markers (e.g., IL-8 panel, CRP where feasible);

	postures; mindful walking; avoidance of strain/valsalva; hydration/bladder-friendly lifestyle education			NGF (exploratory); HRV
Stress-reactive psychosocial phenotype	Meditation and relaxation emphasis; pranayama for down-regulation; compassionate attention practices; brief journaling/self-efficacy prompts	20–30 min/day plus weekly group session	PSS; HADS; QOL; pain coping	HRV; cortisol; inflammatory markers optional
Mixed phenotype (common in practice)	Integrated sequence combining gentle asana + slow breathing + relaxation + brief meditation	45 min/session, 4–6 days/week	Composite responder outcome (pain + interference + QOL)	Pre-specified mediator model: HRV/cortisol and/or inflammatory panel → symptom change

DISCUSSION

This narrative review highlights CPPS as a neuroimmune and biopsychosocial pain condition in which peripheral inflammatory signalling, immune–neural coupling, and central sensitization frequently coexist. Evidence supporting inflammatory and neurotrophin involvement (e.g., IL-8/chemokines and NGF in pelvic secretions) suggests that ‘inflammation’ in CPPS is often subtle and dysregulated rather than overtly infectious, and may represent persistent immune activation that can sensitize nociceptors and drive neurogenic inflammation [18–22]. At the same time, cohort and neuroimaging evidence indicates that many patients exhibit centralized pain features, symptom spread, and altered CNS processing, consistent with nociplastic mechanisms [4,16,27].

Yoga is theoretically well positioned for such complexity because it targets multiple levels of the pain system. Breath-based and meditative components can influence autonomic regulation and stress physiology, offering a plausible route to reduce immune activation via the vagal inflammatory reflex [30–32]. Psychological mechanisms—reduced catastrophizing, improved mood and sleep, and greater self-efficacy—are also clinically critical because they shape pain perception and central sensitization dynamics [26–28,33–35]. Importantly, biologic studies demonstrate that yogic practices can modulate inflammatory gene expression patterns (including NF-κB-related transcription), providing mechanistic plausibility beyond symptom reporting [36–38].

However, the direct evidence base for yoga in CPPS is still limited. Pelvic pain–specific yoga studies are few, often small, and vary in protocol content and comparator selection [40,41]. This heterogeneity

mirrors a broader challenge in behavioural pain research: benefits may depend on patient phenotype, intervention dose, instructor expertise, and contextual factors (expectancy, group support). Therefore, negative or null findings in unstratified trials may reflect phenotypic dilution rather than lack of efficacy. A phenotype-guided approach offers a practical solution. For example, patients with prominent pelvic floor tenderness may respond best to gentle mobility, down-training, and relaxation components; patients with high nociplastic burden may need sustained breath-based down-regulation and sleep-focused yoga nidra; and patients with stronger inflammatory signatures may show biomarker shifts that precede symptomatic improvement. Such hypotheses can be tested using mediator analyses in trials that measure HRV, cortisol, and targeted inflammatory panels.

Safety and feasibility considerations are central to clinical adoption. Yoga for CPPS should avoid strain, deep end-range hip positions, strong abdominal pressure (Valsalva), and prolonged sitting postures that exacerbate symptoms. Restorative and trauma-informed delivery is advisable given the high prevalence of anxiety, prior pain-related trauma, and hypervigilance in pelvic pain populations. Telehealth delivery may expand access but requires careful instruction, modifications, and safety screening [41].

Overall, the most defensible conclusion is that yoga has mechanistic plausibility and preliminary clinical support as an adjunct to multidisciplinary CPPS management, but it requires rigorous, phenotype-informed trials to define its role, optimal protocol, and measurable biological mediators.

Broader chronic pain literature supports yoga as a safe adjunct for persistent pain: RCTs and meta-analyses in low back pain and fibromyalgia show improvements in pain-related function and coping [48–55]. Mechanistic trials report changes in stress physiology, HRV, and inflammatory biomarkers across populations, though protocols and effects vary [56–60]. Pelvic pain guidelines emphasise multimodal, patient-centred care and phenotyping, aligning conceptually with tailored yoga-based rehabilitation [1,61–68]. Emerging psychoneuroimmunology and chronic overlapping pain frameworks motivate integrating biomarker and central sensitivity endpoints in pelvic pain trials [69–80].

CONCLUSION

CPPS is increasingly recognized as a neuroimmune pain syndrome characterised by heterogeneity across inflammatory signalling, immune–neural coupling, and central sensitization. Yoga may alleviate CPPS through multi-level modulation of these pathways—supporting autonomic balance, reducing stress-axis activation, improving cognitive–affective pain regulation, and down-regulating pro-inflammatory transcriptional programs. Although pelvic pain–specific yoga evidence remains limited, existing trials and mechanistic literature justify further investigation. Future studies should integrate clinical phenotyping (e.g., UPOINT and nociplastic screening), standardized outcomes, and biomarker endpoints (HRV, cortisol, cytokines/chemokines, NGF) to identify responders and confirm mechanisms.

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