Changing Paradigms for Chronic Pelvic Pain

A Report from the Chronic Pelvic Pain/Chronic Prostatitis Scientific Workshop, October 19–21, 2005, Baltimore, MD

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The field of prostatitis research is rapidly changing focus. It is shifting away from the biomedical model of medical/surgical therapy to the biopsychosocial model, which might hold the key to understanding the pathogenesis of and developing successful treatments for these chronic conditions. This article reviews the "new pathways to discovery" that arose from the Chronic Pelvic Pain/Chronic Prostatitis Scientific Workshop, held October 19–21, 2005, in Baltimore, Maryland.

Understanding Chronic Prostatitis/Chronic Pelvic Pain Syndrome

Richard Berger from the University of Washington (co-chair with Michel Pontari of Temple University) noted that since Ramon Gutierrez stated in 1913 that all prostatitis was undoubtedly caused by infection, little progress has been made in understanding chronic male pelvic pain. The purpose of the workshop was to develop alternative ways of thinking about this common and frustrating problem. Pain, the hallmark of chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), is a complex phenomenon. From the time of Descartes, most physicians have thought of pain as a simple stimulus response; a certain noxious stimulus produces the same pain in all people. Although Descartes' concept still seems intuitively correct, pain research has shown much more complexity. Changes in the central and peripheral nervous system in response to or in anticipation of pain alter the organism's perception of the stimulus. People do not experience the same pain after similar noxious stimuli. In the nervous system, neurotransmitters, cytokines, and neuropeptides change character and concentration, genes are activated, and new axon connections are made. Responses to stimuli might undergo long-term augmentation, and some neural activity leading to pain might occur spontaneously. Indeed it has been shown that men with CPPS have pain sensitization to heat and mechanical stimuli, meaning they experience stimuli in the pelvis more intensely than men without CPPS. There is also evidence that the men with pain who have a tendency to view adverse events as catastrophic are more likely to have more intense or a longer course for pain. "Prostatitis" must be viewed as more than an organ-specific disease. This important meeting was convened to examine and attempt to understand the roles of the prostate, pelvis,

bladder, the peripheral nervous system, central nervous systems (CNS), the psyche, and the social milieu in CPPS, an essential step toward improving treatment.

Failure of the Biomedical Model

The traditional biomedical treatment model that has been used during the last century has failed many patients. Using the biomedical model, we attempted to understand the etiology and pathogenesis of a disease process and then direct specific, usually organ-specific, medical and surgical therapy to rectify the problem. We focused our attention on the organ where the patient or physician assumed the pathology or pain to reside: in the case of CP/CPPS, the prostate. We largely ignored the neurologic, endocrinologic, and psychosocial milieu in which pain occurred and the sequelae of pain in those same systems. Medical and surgical therapy has been largely empiric, providing 20% to 40% improvement rates and dismal cure rates, especially in patients with long-term symptoms.

Although our understanding of patients with chronic pelvic pain associated with CP and interstitial cystitis (IC) has increased significantly over the last decade, we still do not know the definite mechanisms or, more likely, multifactorial mechanisms that lead to the chronic pain and disability associated with these conditions. There is little sound scientific evidence for an effective therapeutic agent for CPPS. In acute bacterial prostatitis and chronic bacterial prostatitis, antibiotic therapy is almost by definition the treatment of choice. Antibiotic selection is based on culture and sensitivity results and the ability of antibiotics to penetrate the prostate. Daniel Shoskes (Cleveland Clinic) and J. Curtis Nickel (Queen's University) pointed out that, despite success rates of up to 60% in nonrandomized trials, no randomized placebo-controlled trial has shown antibiotics to be clinically effective. Practically all urologists can cite anecdotal evidence of antibiotic effectiveness. Many of these anecdotal observations might be due to a strong placebo effect, the anti-inflammatory actions (cytokine- and tumor necrosis factor–blocking effects) of quinolones, tetracyclines, and macrolides, or the inherently cyclical nature of the symptoms and the patients' tendency to seek treatment when symptoms are worst. Reports of successful therapy with antibiotics, α-blockers, anti-inflammatories, herbal preparations, or other pharmaceuticals are mainly anecdotal, and these agents have been very unimpressive when measured against a placebo in a well-designed clinical trial. A National Institutes of Health multicenter trial showed no benefit of combining antibiotics and α-blockers. Alternatively, the large clinical trials showing no effect of antibiotics were performed mainly on men who had previously failed antimicrobial therapy, and results could be different in men who had not already failed antimicrobial therapy. Studies of antibiotics in early or primary care patients need to be performed.

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Drs. Nickel and Shoskes offered suggestions as to promising agents that should be studied. These include agents with neuromodulatory effects (eg, amitriptyline and pregabalin), with immunomodulatory effects (eg, cyclosporin or mycophenolate mofetil HCl [CellCept; Roche Pharmaceuticals, Nutley, NJ]), with anti-oxidative effects (eg, quercetin), or α-blockers in men with recent onset of symptoms.

Neuropathic Components of Pain in CP/CPPS

The central problem in men with CP/CPPS is pain. The experience of pain and suffering necessarily involves the nervous system in producing or mediating these symptoms. Our understanding of the causes of CP/CPPS requires a working knowledge of how nerves respond to stimuli and the factors that influence a patient's interpretation of this stimulation.

The Evolution of Pain Management

A perspective on the past, present, and future of pain management was provided by John Loeser (University of Washington). Dr. Loeser described the hierarchy of the individual's pain response. The initiating event is a noxious stimulus, such as from trauma, infection, or peripheral inflammation. Nociception then leads to its conscious perception (pain). The perceived pain is modulated by factors in the periphery, the spinal cord, and the brain. This idea, that the neurologic response is not static but able to be modified by feedback, is a central idea of pain physiology, called the

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"gate theory." Pain can also be reported by the CNS in the absence of nociception and be spontaneously perceived, such as in phantom limb pain and pain after stroke. Whether CP/CPPS is another example has yet to be determined. The perception of pain can lead to suffering when combined with the patients' previous experiences and the meanings given to the pain. Finally, pain and suffering lead to pain behavior, which is the only way we know someone is in pain. Dr. Loeser pointed out that these relationships underscore the concept that pain is not just a manifestation of something "broken" in the body that needs to be fixed; it represents a human symptom requiring a multidisciplinary management team to effectively treat.

Comparison of Acute and Chronic Pain

Ursula Wesselmann (Johns Hopkins University) compared and contrasted acute and chronic pain states. Chronic visceral pain of the pelvis or urogenital floor often begins with an inflammatory event. For example, vulvodynia frequently begins with a yeast infection, and a urinary tract infection often immediately precedes the development of IC. The problem in patients with chronic visceral pain, such as CP/CPPS, is that the initial event, be it infectious or traumatic, sets up a pain experience that persists long after the initial stimulus is gone. It is known that tissue injury can lead to sensitization (ie, a decrease in the intensity of the stimulus needed to elicit a response by the nerve). For example, in patients with sensitized bladder nerves, a low volume of bladder filling that normally does not produce symptoms can generate urgency and pain. Sensitization can occur in both the peripheral nervous system and CNS. Central sensitization, such as that manifested by increased spinal cord levels of cFos, can occur when inflammation in one area, such as the uterus, is followed by inflammation in an area of overlapping innervation, such as the vagina. Mechanisms underlying this development of the chronic pain state, independent of the initiating event, include changes in gene expression in the CNS, such as expression of cFos, loss of inhibitory interneurons, establishment of aberrant excitatory synaptic connections, and long-term potentiation of response due to changes in sensitivity of nerve synapses. Some of the cellular responses responsible for sensitization include phosphorylation of receptors for neurotransmitters on the nerves, resulting in a decreased threshold for activation. Stress might also play a role in central sensitization: rat pups placed in a quieter environment develop less sensitization after the induction of paw inflammation than those in a more stressful environment.

Alterations in nerve receptors and neuroendocrine mediators noted in peripheral and central sensitization might provide a target for therapy for CP/CPPS.

Mediators of CNS Neural Plasticity

Margaret Vizzard (University of Vermont) described in more detail several of the candidate neurotransmitters responsible for central sensitization in chronic pain states. There is an increase in staining for substance P and calcitonin gene-related polypeptide (CGRP) in rat bladder afferent cells of the lumbosacral dorsal root ganglia and spinal cord after cyclophosphamide (CYP)-induced cystitis. Another mediator that has been implicated in pelvic inflammation is PACAP (pituitary adenylate cyclase–activating protein). PACAP is in the vasoactive intestinal polypeptide family of neuroendocrine mediators. In animal models of inflammation induced by CYP, there is also increased staining of PACAP in the same bladder afferent cells in the lumbosacral dorsal root ganglion and pelvic spinal levels. Intrathecal instillation of an antagonist for the PAC_1 receptor results in decreased bladder overactivity in the CYP model. Inflammation also increases messenger ribonucleic acid and subsequent protein expression of other neurotropic factors, including nerve growth factor (NGF). Use of a recombinant NGF-sequestering protein, REN1820, leads to significant reduction in the number and amplitude of nonvoiding contractions, as well as decreased voiding frequency in animals with CYP cystitis. Alterations in NGF also mediate the changes in micturition reflexes in cystitis and might provide a target for therapy for these changes.

The Neurophysiological Relationship of Pain and Sex

Richard Bodnar (Queens College, New York, NY) reviewed the role of gonadal hormones in pain physiology. There are marked gender differences in analgesic response. Men generally display greater levels of analgesia at lower doses of analgesics than women. This occurs with both opioid and nonopioid analgesics and in the analgesia associated with the stress response. These differences are greater for μ-opioid agonists relative to κ- and δ-opioid agonists. Hormones are involved in both the organization and activation of the CNS. The organizational role is the effect of

the hormones on development of the CNS. An example is that male rat pups castrated 1 day after birth display adult analgesic responses to morphine similar to those in adult females; conversely, female pups androgenized with testosterone exhibit responses similar to those in adult males. The periaqueductal gray (PAG) area, located near pain-inhibitory centers, is one brain area that is involved in the differential effects of organization by hormones; the PAG is also a site in the emotional motor system, a link between emotions and pain perception.

Visceral Pain

Gerald Gebhart (University of Iowa) discussed the differences between pain from visceral sites and that from somatic sites. Nociception in internal organs or visceral pain differs from somatic pain in several respects. Visceral pain is diffuse and poorly localized, and it is referred and not felt at the source. One of the anatomic reasons for these observations is that there is extensive arborization of sensory nerves in their spinal cord levels; nociception is felt not just at one level of the spinal cord but at several levels at once. These areas are also receiving input from other viscera and somatic structures at the same time, leading to a mechanism for modulation of the painful area on other areas, and vice versa. The input from nociceptors is also multimodal; input can include, for example, both mechanical and thermal afferents at the same time. Visceral pain can be produced by stimuli different than that necessary to produce somatic pain. Injury is not required for visceral pain: it can be induced by hollow organ dissension, traction on the mesentery, ischemia, and chemicals. Also, visceral pain is associated with emotional and autonomic responses not seen with somatic pain.

Dr. Gebhart also discussed mechanisms of central sensitization in visceral organs. After tissue injury, there develops an increase in the number of spinal levels that respond to a stimulus from a given site. There is also increased input from higher CNS centers that influence the spinal response to nociception. After colon inflammation, for example, there is increased output from the rostral ventral medulla. Importantly, the rostral ventral medulla is also influenced by emotion and cognition, providing another link between these factors and the spinal level of the noxious stimuli. There can also be changes in the type of sensory fibers that mediate a pain response. Normal visceral sensation is carried by myelinated Aδ fibers. However, with a noxious stimulus, previously silent unmyelinated C fibers can become activated, and activated C fibers might fire more vigorously. C fibers, so called because they respond to capsaicin, contain many sensory neuropeptides, many of which are associated with somatic pain, such as substance P and CGRP.

Summary

Ursula Wesselmann (Johns Hopkins University) summarized the crucial clinical questions that need to be addressed:

1. Is it possible to prevent modifications of the nociceptive system and the progression from an acute to a chronic pain syndrome?
2. Can we predict which patients are at risk to develop a chronic pain syndrome after an acute pain event?

These questions underscore the importance of prevention before a sensitization and chronic pain event develops. Chronic pain is very difficult to treat, given the complex nature of the response and the interaction of the physiologic and subsequent psychosocial changes that occur and that can help to perpetuate the condition.

Psychosocial Aspects of Pain in CP/CPPS

The Meaning of Pain

This is the first time a urology meeting devoted to CP/CPPS explored the important psychosocial dynamics in patients with these conditions. It is hoped that this will lead to evidence-based biopsychosocial therapies that might not cure but at least will ameliorate symptoms, decrease disability, and improve the quality of life of patients suffering from CP and IC. David B. Morris (University of Virginia) explored the meanings of pain to "the pained"—that is, to persons in pain. He argued that our understanding of pain beliefs should extend beyond our current focus on causation, duration, and catastrophe. We should not focus on the meaning of pain too narrowly, a focus that usually emphasizes cognitive (as distinct from emotional) experience. He argued that emotion is intrinsic to cognition when examining the relevance of pain to the person in pain. He further pointed out that pain patients understand and experience their pain within a cognitive "frame," a concept often unrecognized by the framer. This important address set the stage for further discussion of the relevance and significance of the psychosocial aspects of pain to patients with CP (and IC).

Cognition and Emotion in Pain Perception

M. Catherine Bushnell (McGill University) further explored the influence of cognition and emotion in pain perception. Psychophysical studies have shown that pain perception is altered both by attention to the pain and the mood of the patient, but the nature of the modulation differs in individuals. The degree of

attention to the pain alters primarily the perceived intensity of the pain, whereas mood alters primarily the unpleasantness associated with the pain. Brain imaging experiments are now being used to explore mechanisms underlying psychological modulation of pain. These human studies have shown that attention and distraction show modulation of pain-evoked activity in the thalamus and in several other cortical regions, including somatosensory cortex, anterior cingulate cortex (ACC), and insular cortex. Other regions, including PAG, parts of the ACC, and orbital frontal cortex, have been shown to be activated when subjects are distracted from pain, suggesting that these regions might be involved in the modulatory circuitry related to attention. The speaker noted that much less is known about the pathways involved in the emotional modulation of pain; however, negative emotional states enhance activity in limbic regions, such as the ACC and insular cortex. These basic psychophysical studies not only improve our understanding of pain modulation in conditions such as CP and IC but also point to potential therapeutic targets.

Depression and Catastrophizing in Chronic Pain States

Jennifer A. Haythornthwaite (Johns Hopkins University) noted that depression often accompanies persistent pain, including chronic pelvic pain, and depression rates in pain patients are higher than those seen in the general population. Prospective studies (including studies in CP and IC) indicate that persistent pain increases the risk for depression and also suggest that depression increases the risk for persistent pain. Further studies suggest that elevated rates of depression seen in chronic pelvic pain are related to the experience of pain, not the presence or absence of an organic cause. Depressive symptoms occur on a continuum of severity, and even mild symptoms influence pain, pain-related coping, and pain-related disability. One of the maladaptive pain coping strategies—catastrophizing—is consistently associated with depression, pain, and disability. In addition to magnifying pain, catastrophizing seems to galvanize internal resources (attention, emotion, and behavior) and stimulates pain behavior. The social responses activated by catastrophizing consist of negative, punishing responses that enhance rather than mitigate pain and depression. Urologists need to be cognizant of depression as a psychiatric syndrome and of catastrophizing as a maladaptive coping strategy that can have subtle or significant impacts on the CP or IC patient's pain, daily life, and outlook.

Pelvic Pain Is Sexual Pain

Julia R. Heiman (Indiana University) suggested that pelvic pain is sexual pain. Chronic pelvic pain connected with sexual functioning has traditionally been discussed in women with vaginismus and dyspareunia. It is now apparent that patients with pelvic pain show augmented genital sensory processing, hypervigilance for coital pain, and a selective attentional bias toward pain stimuli. Little attention has been paid to sexual symptoms in men with CP. Men with CP, when asked, have reported sexual problems of pain during or immediately after sexual intercourse (associated with ejaculation), decreased frequency of sexual activities, varying degrees of erectile dysfunction, and decreased libido. Recent studies have shown that men with CP report less sexual pleasure and satisfaction. Sexual functioning as a predictor of poor quality of life and disability must be explored in future studies.

Methodology and Outcome Measures in Pain Studies

Dennis C. Turk (University of Washington) discussed the problems in methodology and outcome measures in clinical trials evaluating pain and pain perception. He argued that comprehensive assessment of the person who experiences pain should address emotional functioning, physical functioning, both positive and negative effects of treatments, and the patient's overall appraisal of the benefits of any treatment initiated. He further argued that pain-related outcome measures need to be developed in conjunction with the groups in whom they are planned to be used. Target groups of patients with CP and IC need to be involved in the development of the content of any questionnaire. He further questioned the traditional approaches to treatment outcome studies, which focus primarily on statistical significance. These studies should probably be focused on whether the effects are clinically relevant or meaningful. The problem is determining whether a treatment is clinically meaningful. The criteria the patient might use to determine whether treatment made a meaningful difference in his or her life would likely consist of a balance between the clinical effects weighed against the cost, inconvenience, and adverse effects. The patient might be pleased with a small degree of amelioration of pain and improvement in quality of life, particularly if there were few negative side effects and little inconvenience, whereas the health care provider might be looking for a larger reduction in pain and improvement in quality of life but be willing to accept more inconvenience and a larger number of adverse effects than would the patient. There must be some agreement as to the level of improvement that is clinically meaningful, to define "a minimally

important difference." In patients with pain and poor quality of life, a responder analysis in which a percentage of patients achieve that level of importance should be reported.

New Directions in the Treatment of CP/CPPS

Treatment of Neuropathic Pain in CP/CPPS

Alan Cowan (Temple University School of Medicine) pointed out the inherent limitations of translating results from preclinical neuropharmacology studies to the patient. Newer medications, such as the anticonvulsant gabapentin and pregabalin, are becoming increasingly used for neuropathic pain. The efficacy and safety of pregabalin are presently being evaluated in CP/CPPS by the National Institutes of Health Chronic Prostatitis Collaborative Research Network. There is also the possibility that drugs that are currently used for irritable bowel syndrome (eg, tegaserod, a serotonin type 4 receptor partial agonist) might hold some potential use for treating CP/CPPS.

The efficacy and safety of pregabalin, a new anticonvulsant used for neuropathic pain, are presently being evaluated in CP/CPPS by the National Institutes of Health Chronic Prostatitis Collaborative Research Network.

Alternative Therapies

Alternative therapies are generally therapies that are unproven. Some alternative therapies have later become standard medical therapy. Some define alternative as therapy not following a strict medical model of finding "the cause" of the disease and treating it with pharmacotherapeutic drugs or surgery of proven effectiveness. Alternative therapies might be combined with proven medical therapies. Alternative therapies might be self-administered. Alternative therapies might also be programs that address the biopsychosocial model and that are standard in many pain clinics. In this model, it is thought necessary to address not only the biological aspects of disease but also the meanings of the disease to the patient and the social and support systems that are affected by and that might support the patients' wellness or sickness. The psychological, neurologic, and sociological aspects of CPPS pain are detailed elsewhere in this review. In some sense, all therapies for CPPS remain alternative, given that no therapy has been proven to be effective.

Barbara Shorter (Long Island University) detailed self-treatment with dietary therapy in patients with bladder symptoms diagnosed with IC. It is well recognized that CPPS patients also perform self- and physician-prescribed dietary therapy. Patients identified caffeine, citrus fruits and juices, tomatoes and tomato products, vinegar, and alcoholic beverages as items that exacerbated bladder symptoms the most. Foods helpful to symptoms were not identified.

David Wise (Stanford University) presented the paradigm and results of the "Stanford Protocol: Paradoxical Relaxation/Trigger Point Release for the Treatment of Prostatitis/CPPS." Dr. Wise proposes that a chronically contracted muscular pelvic floor is responsible for the varied symptoms present in this syndrome. With his technique of "paradoxical relaxation" to relax the pelvic floor and modify the tendency to tense the pelvic muscles, along with rehabilitation by deactivation of pelvic trigger points, he claims to achieve a 72% success rate in patients who failed other therapy. Properly controlled and comparative trials are needed.

John S. McDonald (University of California, Los Angeles) presented a theory of the etiology of pelvic pain based on pudendal neuropathy, also called "pudendal nerve entrapment." This syndrome is analogous to median nerve compression in the wrist and occurs when the pudendal nerve is compressed between the sacroiliac and sacrospinous ligaments in the pelvis. This nerve carries motor, sensory, and autonomic fibers and has great anatomic variability. Compression and injury therefore might occur in many ways and produce multiple and varied symptoms, ranging from sitting pain, urinary pain, defecation pain, vulvo-vestibulitis, perineal pain, prostatitis pain, and prostatodynia. This syndrome can be found either with or without neurologic signs, making it difficult to diagnose. Dr. McDonald reports that sitting pain relieved by standing or sitting on a toilet seat is the most reliable diagnostic parameter. He uses a neural multilevel treatment protocol using neuroleptic agents, antidepressants, and pudendal nerve blocks with local anesthetics and steroids and sees progressive improvement with increasing numbers of blocks. Controlled trials are not available. Stanley Antolak (University of Minneapolis) treated pudendal "entrapment" with a series of pudendal nerve perineural

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injections consisting of 0.25% bupivacaine and triamcinolone acetonide in men with CPPS. A series of three injections was necessary to achieve a response. The durability of the response was variable and might be only for a short duration, but the treatment would also be useful as a diagnostic maneuver. Better methods to test the function of the pudendal nerve are needed before the true frequency of this syndrome can be determined.

Table 1
New Pathways in the Understanding of Chronic Prostatitis/Chronic Pelvic Pain Syndrome

Future Directions in the Understanding of Etiology and Pathogenesis
Long-term effect of acute nociception
Sensitization of the peripheral and central nervous system
Immunological interaction
Psychosocial modulation
Future Directions in Therapy
Immunomodulatory therapies
Neuromodulatory therapies
Physical therapies
Psychosocial-based therapies

Psychosocial Therapy for CP/CPPS

Clinical researchers in the field of CP and IC are only now starting to accept the clinical observations and empiric studies that attest to the importance of psychosocial and environmental factors in these chronic pain syndromes. Judith A. Turner (University of Washington School of Medicine) argued that psychosocial therapies can help patients identify and change maladaptive, cognitive, and behavioral responses to pain. These treatments would involve instruction and practice in adaptive cognitive and behavioral responses. She further suggested that such treatments would be effective in reducing pain, suffering, and pain-related disability among patients with chronic pain problems, such as CP and IC. Certainly, randomized controlled trials have supported the efficacies of these cognitive-behavioral therapies for a variety of chronic pain problems, including low back pain and arthritis. However, research has not examined the effectiveness of these therapies for CP (or IC). Recent studies on both CP and IC are now providing data regarding the psychosocial dynamics of patients with these conditions. These data can be used to develop evidence-based psychosocial treatment protocols that can and should be assessed in prospective trials.

The Biopsychosocial Model: The Key to Successful Treatment?

The poor quality of life and significant disability associated with the chronic pain of CPPS turn out to

Main Points

- Traditional biomedical treatment for chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) has failed many patients; antibiotics, α-blockers, anti-inflammatories, herbal preparations, and other pharmaceuticals have been very unimpressive when measured against a placebo in a well-designed clinical trial.
- It is hoped that an understanding of the psychosocial dynamics in CP/CPPS patients will lead to evidence-based biopsychosocial therapies that might not cure but at least will ameliorate symptoms, decrease disability, and improve quality of life.
- Psychophysical studies have shown that pain perception is altered both by attention to the pain and the mood of the patient.
- Urologists need to be cognizant of depression as a psychiatric syndrome and of catastrophizing as a maladaptive coping strategy that can have subtle or significant impacts on the CP patient's pain, daily life, and outlook.
- Little attention has been paid to sexual symptoms in men with CP; sexual functioning as a predictor of poor quality of life and disability must be explored in future studies.
- Randomized controlled trials have supported the efficacy of cognitive-behavioral therapies for a variety of chronic pain problems, including low back pain and arthritis; research has not examined the effectiveness of these therapies for CP.
- Physical therapies may hold the key to amelioration of pain and disability in patients who have developed dysfunctional myofascial pelvic pain.

be much more complicated than the simple traditional model provided by infectious, neuropathic, or immunologic etiology. Many factors, such as genetics, personality, attitudes, social/cultural environment, coping strategies, perceptions of control, emotions, and behavior disturbances all interact with the chronic pain to determine the individual patient's adaptation to persistent pain. The CP research community must explore these dimensions in patients suffering from the disease, to provide solid data for the development of individualized evidence-based biopsychosocial treatment protocols. The traditional biomedical models must continue to be pursued by the scientific community because they hold the key to the initiation of the condition in susceptible individuals. For any treatment to be successful (or curative), the factors that initiate the inflammatory and neuropathic state or continue to create continued inflammation and/or peripheral nerve stimulation must be dealt with. However, in patients with long-standing pain, the psychosocial therapeutic model likely holds the key to long-term amelioration of pain (or at least increased tolerance of pain), decreased disability, and improved quality of life in patients suffering from CP/CPPS.