CHAPTER 1

Why Consider Cognitions When Treating Chronic Pain?

Our thoughts, often automatic and not necessarily obvious in our immediate consciousness, have a profound impact on both our short-term reactions and long-term adjustment to pain. Cognitive therapy focuses specifically on helping people recognize their thinking patterns so that they can change their relationship to the thought or belief. This process may involve changing the content of the thought to bring it more in line with reality, but it may also involve letting the thought pass as "just a thought." Cognitive therapy values and includes behavioral processes in its approach; the main difference between behavioral therapy and cognitive therapy is the conceptual rationale (and perhaps a more detailed focus on patient cognitions in cognitive therapy). Cognitive therapy is based on a cognitive model, which contends that our thoughts drive our emotions, our behavior, and, to some extent, our physical processes.

Why Cognitive Approaches Are Important in Pain Management

The interdisciplinary treatment of chronic pain has been the preferred method of treatment for several decades, with research to back its superiority over single-modality treatment (Dorflinger et al., 2014; Kamper et al., 2014; Turk et al., 2010). Historically, the first interdisciplinary pain clinics to include a psychological component in their treatment approach were based on the operant model of pain (Fordyce, 1976), and as such they were strongly behavioral. In a strict operant model, behavior is determined by reinforcement, and cognitions are less relevant to the prediction of behavior (Fordyce, Fowler, & DeLateur, 1968). Although the psychological treatment of chronic pain has since expanded to include the cognitive, its heritage is decidedly behavioral. Today's psychologically based interventions are grounded within a cognitive-behavioral model (Turk, Meichenbaum, & Genest, 1983) and do consider cognitive factors,

including appraisals, beliefs, and expectations, as well as ongoing cognitive processes, such as automatic thoughts and self-statements.

Although research has not yet identified the necessary and sufficient agents of change within cognitive-behavioral therapy (CBT), there is a wealth of research pointing to the importance of cognitions and adjustment to pain, in terms not only of perceived pain intensity, but also mood and functional ability (e.g., de Rooij et al., 2014). (See Chapter 3 for a more thorough discussion of the relevant research literature.)

For some patients, an unfortunate consequence of dealing with chronic pain may be that they come to develop a personal identity as "disabled chronic pain patients." Such persons continue to seek diagnoses and may submit to increasingly invasive medical cures, often being referred to multiple medical professionals, to no avail. They also take on a "sick person" role, equating chronic pain with disability. The paradox is that patients who accept their pain as a chronic condition have *lower* perceived pain levels, *less* pain-related distress and depression, *less* avoidance of activities, *lower* levels of disability, and *greater* daily function (McCracken, Barker, & Chilcot, 2014). "Acceptance" here is defined as recognizing that one has a chronic condition that cannot necessarily be cured, letting go of fruitless attempts to completely rid oneself of the pain, working toward living a satisfying life despite the pain, and not equating chronic pain with disability. In fact, it has been suggested that one of the main aims of CBT should be to facilitate patients' acceptance of their pain and, in doing so, to broaden their identity beyond that of a disabled chronic pain patient (Morley, Shapiro, & Biggs, 2004).

This goal, however, requires starting with a patient who may have been caught on the "conveyor belt" of repeated invasive biomedical approaches (surgery, multiple medications); moving her toward being an active collaborator in pain self-management strategies (aimed not at completely eliminating the pain but rather at increasing appropriately paced activity and learning skills to regulate her thoughts, emotions, and behaviors); and facilitating the ultimate goal of adopting a new identity as a person with pain. Obviously, individuals will vary widely in terms of their level of motivation and commitment to take on a very new approach. The typical patient, though, has been well steeped in a biomedical-only approach to dealing with health-related problems and thus may enter into CBT with little understanding of what is involved—or may assume that such approaches are only for those without "real" pain. Thus, it is probably unrealistic to expect typical patients with pain to adopt behavioral self-management strategies without also helping them to better understand how pain works in the brain, as well as helping them explore their own thoughts, attitudes, and beliefs as they relate to pain and themselves as persons with a chronic painful condition. Helping clients to become aware of and examine the thoughts, beliefs, and cognitive schemas that are shaping their coping attempts is an important step in empowering them to take on a new set of strategies and, ultimately, a new self-identity. Such cognitive motivational techniques are the essence of this book.

The father of operant treatment for chronic pain, Wilbert E. Fordyce, asserted that we must get patients with chronic pain to relinquish "ownership" of their pain, indicating that patients who "own" their pain have come to incorporate the pain/illness into their sense of personal identity (Fordyce, personal communication, October 22, 1999). This goal may be of immense therapeutic value because many sufferers *do* experience chronic pain as "their pain." Yet a key to successful treatment is the clinician's understanding and acknowledgment of each patient's pain experience. The challenge of cognitive therapy is to begin within the cognitive

and emotional framework of the patient and gradually invite the patient to enter into a different relationship with his pain. Regardless of the causes of the pain (and these causes are *always* multifaceted), it is the person's *experience* of pain that is key to cognitive therapy. As we will see, there is clear evidence that the patient's cognitive experience of pain is a better predictor of adjustment than any other variable.

Bear in mind that a cognitive approach to pain does not imply that a person's pain is not real. Many patients with chronic pain, upon being referred to a mental health practitioner, conclude that the physician believes their pain is psychogenic, functional, or psychologically based (in other words, not "real"). Indeed, when pain persists beyond the point at which an injury is declared to be healed, or when someone has pain but no biomedical etiology can be found, the patient is often assumed to be willfully exaggerating the pain, making it up to get out of something unpleasant or unconsciously "converting" a psychological issue into a physical one. Although some individuals knowingly fake their pain symptoms, persons characterized as malingerers or those with factitious disorder make up just a small percentage of the patients we are likely to see for pain management and cannot be reliably identified by any existing biomedical or psychological assessments (Fishbain, Cutler, Rosomoff, & Rosomoff, 1999; Howard, Kishino, Johnston, Worzer, & Gatchel, 2010). Nevertheless, for many individuals with chronic pain, these inferences only add insult to injury. An unfortunate related misconception is that patients willfully overreport the level of pain and distress they actually feel, and have more pain behaviors and greater dysfunction than are warranted by the physical evidence for the pain. There are many pejorative references made about "those kinds" of patients, such as "frequent flyers," "known to the system," "drug seekers," or even "FOS"—the "full of **it" diagnosis. My personal archenemy terms are "pillbilly," "houseplant," and "attention whore." As one might guess, being referred to by any of these labels is a recipe for promoting a combative rather than collaborative stance regarding managing the patient's illness.

When patients feel that the health care system has delegitimized their pain, they are less receptive to potential interventions by behavioral health practitioners. I have a favorite cartoon: a man sitting in a psychologist's office, who exclaims, "Of course the pain is in my head. It's a headache!" Pain is a perception, and like all perceptions, it is filtered through the brain. I tell my clients that, in a way, the pain *is* in their heads—not in the way that others have implied but because all pain, even the pain of a broken leg, is processed in the brain. Pain is perceived as pain because the brain interprets the stimulus as pain. Since the brain is the organ that processes cognitions and emotions, the brain is responsible for integrating sensory, cognitive, and emotional information as part of the interpretive process involved in one's overall experience of pain. The patient's cognitive and emotional experience of his pain *is* the reality. To really *do* cognitive therapy, you must successfully "get into the patient's head" as it relates to his pain.

There is an exploding knowledge base regarding the plasticity of the brain and descending pain inhibitory mechanisms in the brain; this edition of the book uses the brain and what we currently know about it as its linchpin for treatment rationale. However, as you will see as we proceed, we offer this information to the patient in a way that is jargon-free, understandable, and immediately useful. In the next part of this chapter, I summarize what is known about pain perception, also linking this knowledge to movement from the biomedical model of pain to the biopsychosocial model. Treatment Module 1 shows you how to put this information in patient-friendly terms.

The Central Nervous System and Pain

Nociceptive Pain

The ability to recognize pain is critically important to the survival of the organism. Pain motivates us to withdraw from potentially life-threatening stimuli. People born with a congenital insensitivity to pain actually have a reduced life expectancy, owing to their inability to perceive pain-related stimuli. Pain sensations coming from skin, muscles, or internal organs are part of the somatosensory system. Pain receptors are called "nociceptors," and instead of being specialized sensory organs, they are free nerve endings. Free nerve endings are the receiving ends of nerve tissue in the skin, muscles, or viscera. Free nerve endings can be stimulated by a variety of means, including intense mechanical, thermal, or electrical stimulation. When tissues are damaged, free nerve endings are also chemically stimulated by the release of chemicals from injured cells. This chemical stimulation is a complex process, and the release of chemicals from injured cells in turn increases the sensitivity of free nerve endings to other chemicals (a process called "chemical sensitization"). Once free nerve endings are stimulated, the message travels to the spinal cord via transmission fibers—axons. Whereas the free nerve endings are at the receiving ends of neurons, the axon carries the message from one end of the neuron to the other. At the level of the spinal cord, the first neuron in the message chain communicates with a second neuron via an electrochemical process that releases neurotransmitters. From the spinal cord, pain messages travel to the brain via several different potential pathways, and neurons along the way serve as relay stations in the transmission process.

Neuronal Plasticity

It is now clear that the morphology and physiology of the brain changes in response to the experience of pain itself. We call this phenomenon "neural plasticity"—the capacity of neurons to change their structure, their function, or even their chemical profile (Woolf & Salter, 2000). In the short term, brain-related pain processing can be altered (or "modulated") in a way that increases the sensitivity of neurons to even mild pain signals. As an example of short-term modulation of brain processes involved in pain perception, tissue injury causes the release of chemicals from injured cells, which in turn creates an increased sensitivity of the free nerve endings to other chemicals, thus producing a change in the way the nerve endings process a pain stimulus. Once the tissue is healed, the hypersensitivity *usually* returns to normal. Long-lasting alterations in neurons can also result from the experience of pain, and these changes are called "modifications." Modifications of the nervous system are more long-lasting; they include such structural changes as an increase in the number of pain receptors in the spinal cord following tissue damage and inflammation, and a reduction in brain inhibitory processes following nerve injury, as well as relative reductions and increases in gray matter and cell connectivity (May, 2011; Woolf & Salter, 2000). Short- and long-term neural plasticity may lead to conditions that have previously gone unexplained. For example, "allodynia" is a condition in which nonpainful stimulation (e.g., light touch) produces pain; "hyperalgesia" is a situation in which a mildly painful stimulus produces intense pain; and "referred pain" is the perception of pain spread to noninjured tissue (Covington, 2000; Iadarola & Caudle, 1997). Often these processes persist after the damaged tissue has healed. In addition, "neuropathic pain," or the sensation

of pain after injured nerve tissue has healed, is an illustration of the pathological alteration of the nervous system via the experience of pain. A good example of neuropathic pain is the long-lasting exquisite pain experienced by some people after a herpes zoster ("shingles") outbreak.

There is now good evidence that recurrent or chronic nociceptive input to the brain causes chemical and structural changes. Chronic pain is associated with *increases* in cortical activity in brain areas associated with the area of the body where pain is experienced and a shift in how the brain maps or represents these areas. Furthermore, in a longitudinal study examining the trajectory of acute to chronic pain, as pain duration increased, the relative activation in cortical activity in regions of the brain associated with emotion became greater than the cortical activity in the somatosensory cortex, which the authors cleverly referred to as "shape-shifting" (Hashmi et al., 2013). Numerous studies have now reported that pain chronicity produces a specific pattern of *decreased* gray matter in areas of the brain involved in the inhibition of pain (e.g., cingulate cortex, insula, temporal lobe, and frontal/prefrontal cortex; Apkarian et al., 2004; Kuchinad et al., 2007; Labus et al., 2014; May, 2011; Yang et al., 2013). It is important to note that this pattern of expansion and/or shrinkage is not randomly distributed, is linked to pain duration, and may represent neural changes due to the pain itself or changes due to the consequences of pain—but likely represent both changes (May, 2011).

The Gate Control/Neuromatrix Model of Pain

In their now famous gate control theory of pain, Ronald Melzack and Patrick Wall (1965, 1983) hypothesized that a gating mechanism in the spinal cord (specifically, the substantia gelatinosa of dorsal horn, which houses small interneurons that receive pain signals coming from the periphery and sends them on to the brain) can modulate the pain signals that ultimately reach the somatosensory cortex. (The somatosensory cortex is what we think of as the final destination of pain signals, where neurons are arranged into multiple maps of the body surface, each responding to a different kind of stimulation to a different part of the body [Holmes, 2016].) The revolutionary idea of the gate control model was that the central nervous system was not a mere receiver and transmitter of pain signals, but could decrease or increase the experience of pain by changing actual sensory input getting to the somatosensory cortex. Melzack and Wall also proposed that many areas of the central nervous system were involved in the experience of pain, rather than a single "pain center." They specifically implicated brain-mediated cognitive and affective factors as part of the neural process of pain perception. Updated to incorporate more recent research findings regarding brain processes, including the interaction of ascending and descending systems, the neuromatrix model of pain was later proposed as an extension of the gate control theory (Melzack, 1993, 1999).

The gate control theory opened the door for pain to be included within the biopsychosocial model of illness, and the integrated gate control/neuromatrix model provided an enhanced understanding of these processes (Melzack & Katz, 2013; Negm & MacIntyre, 2012). There is now strong evidence that a widespread network of neural loops involved in emotion and cognition (including such structures as the thalamus, limbic system, and various parts of the cerebral cortex) have connections to the somatosensory cortex, where pain is "mapped." These networks also send descending signals to the spinal cord gating mechanism, thus allowing for the transmission of more, or fewer, pain signals.

To give just a few examples of how cognitions and emotions are intimately involved in brain activity and the experience of pain, here are three relevant studies. In a study using functional magnetic resonance imaging (fMRI), researchers demonstrated that distinct areas of the brain are involved in pain processing (electric shock) versus pain anticipation (a light indicating the electric shock may or may not be coming) (Ploghaus et al., 1999). More importantly, although the level of brain activation in the regions associated with sensory pain processing remained stable across time, the level of activation in the more cognitive-emotional pain anticipation regions *increased* over time. In another experimental pain/fMRI study, when researchers manipulated the mood of pain-free participants, negative affect increased reports of pain intensity *and* was associated with more activity in the cingulate cortex and amygdala—both part of the limbic system (Berna et al., 2010; Yoshino et al., 2010). Furthermore, a clinical fMRI study demonstrated the association of higher scores on a psychological measure of harm avoidance with greater activity in the amygdala—part of the limbic system (Ziv, Tomer, Defrin, & Hendler, 2010).

Tissue Damage and Pain

As we now know, the experience of pain does not have a one-to-one correspondence with amount of tissue damage (which, importantly, does not mean the pain is not real). A wealth of research supports this fact. Many of the most common chronic pain problems (back pain, headache, fibromyalgia) reveal little physical pathology when assessed (Okifuji & Turk, 2015). A very important early research study comparing the spinal MRIs of patients *with* back pain to those of people *without* back pain reported that a large percentage of the people *without* any back pain showed significant disc abnormalities (Jensen et al., 1994). This finding has been replicated multiple times in back, hip, and knee studies (Blankenbaker et al., 2008; Borenstein et al., 2001; Carragee, Alamin, Miller, & Carragee, 2005; Jarvik et al., 2005; Link et al., 2003). Although tissue injury (or tissue healing) is still treated as if it is the best predictor of pain relief, it is not. As just one example of multiple studies dispelling this myth, in a study of workers with low back injuries, researchers found that depression, fear avoidance, and fear of movement (i.e., cognitive and affective variables) predicted 85% of the variance in recovery 6 months later, while actual physical pathology was a very poor predictor (George & Beneciuk, 2015).

Another common misconception is that acute injury *always* produces pain. If you break your leg, everyone expects you to be in pain. The fracture can be seen on the X-ray; it is quantifiable; it is therefore considered "real," and pain is seen as justified. Nevertheless, the relationship between acute injury and the experience of pain is not as automatic as you might think. For example, during World War II, many U.S. soldiers as well as local citizens were severely injured in a battle in Anzio, Italy. Frank Beecher, who was one of the medics there and later went on to become a pain researcher, observed that the meaning of the pain had a great deal to do with a person's experience of pain. Injury to the soldiers meant that they were going home, and many, even those with traumatic amputation of a limb, did not need pain medication. In contrast, the citizens of Anzio had no means of escape; with similar injuries, they experienced fierce pain and required a great deal of analgesic medicine (Beecher, 1959). This example holds personal relevance for me because my father was in General George Patton's artillery and, like many others, lost a limb at Anzio. Although he didn't remember being interviewed by Beecher, he did remember thinking, "This is my ticket home!"

It is now clear that cognitions, emotions, and pain experiences can actually change the way the brain processes input from pain receptors. In Chapters 2 and 3, I link what we know about the neural processing of pain to CBT approaches and the treatment approach covered in this book in particular.

Nomenclature Used for Defining, Diagnosing, and Treating Pain Disorders

Taxonomies of pain, pain diagnoses, and pain treatment methods are not covered in depth in this book. However, I provide some brief information below, as well as references to other resources.

Definitions of Pain

Consistent with the biopsychosocial model of pain, the International Association for the Study of Pain (IASP, 2014) defines "pain" as an unpleasant experience that accompanies both sensory and emotional modalities; may or may not be accompanied by identifiable tissue damage; and is influenced by multiple factors, including cognitive, affective, and environmental. Although the IASP does not provide definitions of "chronic pain," "acute pain," or "recurrent pain," since these terms are frequently used clinically and in the research literature, brief descriptions of these and other relevant terms are included in Table 1.1. See Turk and Okifuji (2010) for a fuller discussion of pain taxonomy.

TABLE 1.1. Common Pain Terms

- *Pain*: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP, 2012).
- *Acute pain*: Pain that is elicited by injury and activation of pain receptors (e.g., trauma, surgery, disease), usually lasts a short time, and remits when tissue is healed. Biomedical intervention is typically sought and often effective (Turk & Okifuji, 2010).
- *Chronic pain*: Pain that is often (but not always) elicited by an injury but worsened by factors removed from the original cause, usually lasts a long time, interferes with daily function, and is not explained by underlying pathology. Biomedical intervention is frequently sought and rarely effective (Turk & Okifuji, 2010).
- *Chronic pain versus acute pain*: A distinction commonly defined via arbitrary chronological demarcations (3 months, 6 months), or based on subjective notions of whether the pain extends beyond the expected healing period (Turk & Okifuji, 2010).

  Recurrent pain: Pain that is episodic (usually brief) but occurs across an extended time period, thereby sharing characteristics of both acute and chronic pain. Because the problems extend over a long period of time, social and behavioral factors may be more influential over illness behavior than over acute pain (Turk & Okifuji, 2010).
- *Pain behaviors*: Verbal or nonverbal actions that communicate discomfort (sighing, grimacing) or are used in an attempt to ameliorate pain (rubbing, using prosthetic devices; Prkachin & Craig, 1986).
- *Disability*: Restriction or loss of capacity to perform an activity in the normal manner (Turk & Okifuji, 2010). Note that *dysfunction*, characterized by disuse or lack of performance of a behavior, but not inability to perform a behavior, can result in eventual disability.

Pain Diagnoses

Until October 2015, patients with pain could receive diagnoses in two ways: via the diagnostic system of the *International Classification of Diseases* (ICD), which physicians use to classify physical health problems as well as mental disorders, or via the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), which psychiatrists and psychologists use to classify mental disorders. It was certainly possible for patients to receive ICD diagnoses of physical health problems as well as DSM diagnoses of mental disorders, and, clearly, comorbidity is not uncommon. Clinicians working with patients who have chronic pain can expect these individuals to carry either or both categories of diagnoses. In versions of the DSM prior to DSM-5, a diagnostic category of pain disorder was an option. DSM-5 does not have this category; the most closely related category would be somatic symptom disorder with predominant pain (American Psychiatric Association, 2013). The Health Insurance Portability and Accountability Act (HIPAA) has now mandated that ICD-10 (World Health Organization, 1992) be used for diagnostic coding of all services, which makes DSM-5 legally irrelevant (D. Bruns, personal communication, February 18, 2016). The ICD-10 diagnostic code for pain disorder with related psychological factors is F45.42.

Procedural Codes for Treatment

Under the Current Procedural Terminology (CPT) coding system, which provides reimbursement codes for mental health practitioners, activities that have been most frequently reimbursed include clinical interviews; psychological assessments; and individual, couple, and group therapy for patients diagnosed with mental disorders. In January 2002, six additional procedure codes, called the Health and Behavior (H&B) Codes, were put in place, and they now provide the means for behavioral health practitioners to work with patients who have physical health

TABLE 1.2. Health and Behavior Assessment and Intervention Reimbursement Codes under the CPT Coding System

- 96150: Initial health and behavior assessment (e.g., clinical interview focusing on pain condition, associated distress, perceived disability; pain-specific questionnaires).
- 96151: Reassessment of a previously assessed patient to determine the need for further treatment. May be conducted by a clinician other than the original assessor (e.g., interpretation of pain-related questionnaires and pain diaries, behavioral observations of patient–spouse or patient–partner interactions).
- 96152: Individual intervention sessions (can be weekly) to modify psychological, behavioral, cognitive, and social factors affecting the patient's physical health (e.g., individual cognitive therapy to modify the patient's motivation to engage in pain self-management behaviors).
- 96153: Group intervention sessions (two or more patients) to address biopsychosocial issues associated with physical health (e.g., group cognitive therapy to modify patient's belief systems regarding the cause, appropriate treatment of, and ability to self-manage pain).
- 96154: Intervention session with family and patient present (e.g., couple therapy to examine and change maladaptive interaction patterns promoting disability in the patient).
- 96155: Intervention session with family of patient, without the patient present (e.g., cognitive therapy with family members of patient during an invasive procedure).

problems but may not have mental illness diagnoses. These codes provide for assessment and intervention activities, including cognitive, behavioral, social, and psychophysiological procedures used for preventing, treating, or managing health problems. Table 1.2 provides a list of the H&B Codes. Medicare, Medicaid, and most private insurers now reimburse for H&B Codes, although at a lower rate per hour than with psychiatric codes. There is statewide as well as private insurer variability in the type of professional who is eligible to bill under the H&B Codes. For a detailed discussion of the H&B Codes, as well as excellent coverage of financially sustainable models of the practice of psychology in a medical setting, see Bruns, Kessler, and Van Dorsten (2014).

The Stress–Appraisal–Coping Model

The biopsychosocial model underscores the important interactions among biological, psychological, and social variables regarding illness and pain. Although it provides an important general reference point, it does not focus on cognitive mechanisms in sufficient detail to be used as an organizational guide for cognitive therapy. Cognitive therapy starts with conceptualizing the client's problems via the cognitive model. The cognitive model (Beck, 1976) is based on the understanding that patients' cognitions have an impact on other thoughts, emotions, behaviors, and even physiological processes. Lazarus and Folkman's (1984) transactional model of stress provides a good organizational framework for this cognitive treatment approach.

There is a huge literature regarding the impact of stress on the expression and course of many disorders, including chronic pain. In a nutshell, the stress–appraisal–coping model as applied to chronic pain suggests that patients' cognitions have a direct impact on their adjustment to chronic pain through their appraisal of the pain and related stressors, their beliefs about their ability to exert control over the pain situation, and their choice of coping options.

Treatment Rationale

Although the stress–appraisal–coping model provides a good conceptual/organizational structure for the treatment, I no longer use it as the main treatment *rationale* given to patients at the initiation of treatment. I now provide a greatly simplified version of the gate control/neuromatrix model as a rationale for treatment, and clients' responses have been overwhelmingly positive. For example, in a qualitative analysis of posttreatment interviews following group CBT compared to group education, participants in both conditions noted that learning about the gate control model was particularly useful to them as a way of helping them understand their pain experience (Day, Thorn, & Kapoor, 2011). Providing some educational context for why we will be focusing on thoughts and emotions, and tying it to what we know about brain processing of pain, simply makes sense to patients. They also wonder why they have never before been provided with this information! I expand on both the conceptual/organizational model (stress–coping model) and the treatment rationale in coming chapters.

Overview of This Book

Following this first chapter, Chapter 2 presents the stress–coping model as a conceptual/organizational model for the treatment and describes the gate control/neuromatrix model as the treatment rationale. Chapter 3 summarizes the research supporting the importance of cognitions in the experience of pain and provides the justification for targeting cognitive variables in our treatment methods. Chapter 4 provides guidance for the psychosocial assessment of chronic pain and underscores its relevance to the cognitive treatment approach. Some of the suggested assessment instruments are included in the Appendices. For others, the reader is guided to online resources. Part II of the book presents a 10-module manualized treatment approach for the application of cognitive therapy techniques for chronic pain. The treatment modules are preceded by an introductory chapter, which considers general therapeutic issues regarding the process of implementing cognitive therapy for pain management. Each treatment module includes case vignettes and excerpts of session transcripts to illustrate the actual therapeutic issues that arise and to help bring to life the therapeutic techniques used to deal with them. Each module also includes session outlines for therapist and client, a narrative summary of the session for the client, and worksheets/handouts, which can be reproduced and given to clients for their use.

Chapter Summary

In this chapter, I have introduced the rationale for providing a book focused on cognitive interventions for pain management. The neurophysiology of pain, and how it relates to the importance of thoughts and emotions, has been reviewed and linked to the biopsychosocial model. Current definitional, diagnostic, and treatment procedure nomenclature has also been reviewed. The organizational framework for the treatment approach covered in this book, the stress–appraisal–coping model of pain, has been introduced, and the treatment rationale, based on the gate control/neuromatrix model of pain, is noted as an important addition for the patient. Chapter 2 provides more details of the stress–coping model and the treatment rationale.

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