# Mindfulness-Based Cognitive Therapy for Chronic Pain

## A CLINICAL MANUAL AND GUIDE

Melissa A. Day

with website

WILEY Blackwell

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*To all people living with unremitting pain and those who are working with them to alleviate their suffering.*

# Contents

# Foreword

When I first heard about the proposal to develop a version of MBCT (Mindfulness-Based Cognitive Therapy) for patients living with chronic pain, I wondered whether acronyms had been confused, didn't they mean MBSR (Mindfulness-Based Stress Reduction)? Wasn't MBSR introduced to the world in the early 1980s through studies showing its impact on reducing chronic pain? Isn't chronic pain the "domain" of MBSR, whereas depression and anxiety are the "domain" of MBCT? These may have been quaint notions in the early days of the development and dissemination of MBSR and MBCT, but it is clear that they no longer apply. The participants who find their way into these interventions are often presenting with multiple diagnoses and, as has been amply demonstrated in the literature, pain and depression are often comorbid. We no longer have (if we ever did have) the conceptual luxury of segregating patients by diagnoses to treatments that address a singular problem. The answer instead, is to find the mechanisms that contribute to the perpetuation of symptoms and then find increasingly efficient and targeted ways of teaching patients how to address them.

This is exactly why Melissa Day's book outlining MBCT for chronic pain is so important. It represents a second generational format of the original MBCT framework that Mark Williams, John Teasdale, and I developed over 20 years ago. Marshaling psychological, neural, and social evidence, Day has identified internal and external drivers of the pain response and then modified the MBCT program to accommodate these elements. It actually reminds me of our own trajectory when we discussed how exactly to reconfigure the MBSR platform for patients who were recovering from a mood disorder.

This book will be embraced by clinicians who are interested in exploring Day's approach. Clear illustrations of how the central cognitive-behavioral therapy (CBT) and mindfulness components address pain amplification and maintenance are tied to specific sessions in which these elements are taught. In addition, the book is sensitive to and supports treatment integrity by emphasizing the importance of the therapist's own mindfulness practice, both as a way of knowing what is being taught "from the inside," but also to embody mindfulness more generally in ways that communicate grounding and presence, even if distressing experiences are present.

Finally, it is clear to see that this book is written with an intention toward service. The treatment manual outlines the eight-session structure and is supplemented by curricula for the therapist, handouts of class material, troubleshooting

tips, and a workbook for clients—practically a one-stop shop for delivering the therapy. This book will serve as a model for others who may be looking to modify existing mindfulness-based interventions for increasingly complex patient groups. For right now, it already provides a valuable template for helping patients learn how to change their relationship to chronic pain in meaningful and significant ways.

*Zindel V. Segal, PhD*
*The University of Toronto*

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## About the Companion Website

This book is accompanied by a companion website:

**www.wiley.com/go/day/mindfulness_based_cognitive_therapy**

The website includes:

- Appendices
- MP3 meditations
- Appendix A. Pre-Treatment Client Handouts
- Appendix B. Meditation Scripts
- Appendix C. MBCT for Chronic Pain Management: Daily Home Practice Record
- Appendix D. Example Program Completion Certificate
- Appendix E. Mindfulness-Based Cognitive Therapy Adherence Appropriateness and Quality Scale (MBCT-AAQS)
- Appendix F. The Brief 4-Session Clinical Manual
- Appendix G. Client Handouts for the 8-session Clinical Manual

# Introduction

Pain is symbiotic with the human condition, a universal experience. When pain persists and becomes chronic however, it can be the cause of intense and sometimes even relentless suffering. Chronic pain affects hundreds of millions of individuals worldwide and changes the entire landscape of a person's everyday existence: one's sense of self, relationships, employment and financial situation, hobbies … no aspect of experience is left untouched. All that is encompassed in people's thoughts, emotions and behavior, our entire phenomenal experience comes to fall within the landscape of ongoing pain. Chronic pain, by its very nature, is recalcitrant to traditional biomedical treatments consisting of medications and surgery alone. To address the pervasive landscape of chronic pain, psychological approaches—such as the Mindfulness-Based Cognitive Therapy (MBCT) approach we explore in this text—are incredibly beneficial across a range of pain types and target not just pain reduction, but also improved function, mood, quality of life and one's overall sense of well-being.

The majority of people living with persistent pain have seen an array of medical practitioners and most typically have a medical record bulging with various test results (some of which have led to various "conclusive" diagnoses along the way), have tried an armamentarium of pain medications, and many have had surgeries ultimately deemed "failures" as the pain persists and in some cases worsens. In the search to find some way to experience relief, most people living with persistent pain fall into the role of a passive recipient of biomedically driven healthcare. The approach described in this book, however, reverses that role, and places the person living with pain firmly and powerfully in the driver's seat: actively taking charge of managing their pain, suffering, and beyond that, their life. Thus, the MBCT for chronic pain approach is intended to be a complement, or in some cases an alternative to a traditional biomedical approach to pain. For most people, however, MBCT delivered as an integrated component within an interdisciplinary care team represents the ideal approach, and indeed, interdisciplinary treatment is considered the gold standard in chronic pain management (Ehde, Dillworth, & Turner, 2014).

There are a number of reasons as to *why* psychological approaches such as MBCT are effective for pain, although one primary, encompassing reason is that

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living with chronic pain is stressful and stress makes pain and suffering worse. If we target and improve stress management and coping skills, by default we also target the pain. Your client may say they are in pain but not stressed: well, psychological approaches such as MBCT can still help as they have been shown to enhance descending inhibition and modulation of pain, changing the way the brain processes pain itself. So if you are wondering who should be referred to such an approach as MBCT for chronic pain, the answer is anyone with chronic pain who wants to suffer less, and do more.

I see myself as both a scientist and a clinician, and initially I was hesitant in writing this book as I wanted to ensure that we first had a sufficiently large evidence base on MBCT for pain before facilitating the ready availability of its use. However, in the context of the relatively recent release of Segal, Williams, and Teasdale's excellent second edition of their MBCT for depression text, and the rapidly growing body of research over the past decade supporting its use for the treatment of an array of conditions, it is clear that MBCT is formed on a solid conceptual basis. Indeed, the widespread interest in mindfulness more broadly is growing at an exponential rate. In a recent review paper I wrote with my colleagues we reported that between 1990 and 2006 the number of published scientific articles on mindfulness went from fewer than 80 to over 600, and at the time we were writing that article there existed over 1,200 research articles in PubMed devoted to the topic (Day, Jensen, Ehde, & Thorn, 2014). We are now just at the beginning of witnessing the potential of MBCT for managing chronic pain, which is a particularly promising time.

My intention in writing this book is to provide a resource that is highly practical and of use to those of you who are clinicians (both experienced and in-training), researchers, or both, so that we can further our collective understanding and use of MBCT for chronic pain. Hence, this manual is intended to "bridge the gap" between researchers and clinicians, and I write the text from this perspective, as a true scientist practitioner. This book is not intended to spark a new and "trendy" revolution in therapy and research for chronic pain management. But primarily to provide a further treatment option for researchers to explore and for clinicians to use when it seems other available treatments aren't working or perhaps when they don't appeal to the client sitting in front of them. Just as we have multiple forms of antidepressant medication for depression in order to (hopefully) find the one class that best suits a given individual, so too do we need a range of psychological treatment options for chronic pain. MBCT represents another approach to pain management that may just reach that client whose pain is refractory to other treatment approaches.

My overarching aim is that in essence this book teaches the basics of *how to do*, or deliver, MBCT for chronic pain. And at base, in delivering MBCT and indeed any psychological approach for chronic pain, it is essential that the delivering clinician have a solid, in depth, core knowledge of pain, including pain theory, the neurophysiology of pain, the cognitive, emotional, behavioral, and societal correlates of living with chronic pain, as well as pain assessment and treatment. However, a recent pain psychology national needs assessment conducted in the USA identified that only 28% of graduate and postgraduate psychology training

programs include at least 11 hr of training in pain instruction, and more than one-third of the psychologists/therapists surveyed reported little or no education in treating pain (Darnall et al., 2016). Hence, if you feel unprepared to treat individuals with chronic pain, do not fear, you are not alone! Thus, Part I of this book is intended to provide a working knowledge base of pain and pain psychology and I provide references to additional learning resources throughout the book. I then transition into describing how this knowledge has informed the development and continued evolution of psychosocial approaches for chronic pain—the foundation upon which MBCT for chronic pain is built. I conclude Part I by introducing and describing the MBCT theoretical model as developed and subsequently applied to chronic pain.

Part II opens by providing an overview of the MBCT approach, which includes a description of the steps needed to prepare and "be ready" to deliver your first MBCT for chronic pain program. I suggest opportunities for further training in pain psychology and more specifically in delivering MBCT, describe practical considerations to address prior to starting up your first program, and include recommendations for tracking client progress to optimize outcomes and prevent premature drop-out and treatment failure. Then in Chapters 5 through 12, I provide step-by-step, detailed guidance on delivering the eight-session MBCT for chronic pain treatment. Each session includes a therapist outline as well as client handouts (also available for download for ease of distribution to your clients), and built in to each of the sessions are troubleshooting tips, illustrative case scenarios, and clinical experiences, as well as basic supervision so that you can enhance your delivery of this approach. Part II concludes with a number of suggested ways you can implement and adapt the MBCT for chronic pain manual for optimal use in your own clinical practice and research setting, along with some caveats and considerations for retaining treatment integrity when doing so.

A large number of online supplementary materials are included to further your learning and knowledge of the techniques and treatment structure, and to foster seamless implementation of this program in to your clinical or research setting. Pre-treatment client handouts to foster positive and realistic client expectations coming into treatment, meditation scripts for each meditation delivered along with downloadable MP3 guided audio files, session-related client handouts and meditation practice log record forms, a therapist fidelity monitoring form as well as a four-session version of the manual are all available for free download at the companion webpage. Additionally, I conclude the text with a number of other recommended excellent resources for continued and advanced learning opportunities.

This text brings together the efforts of innovative thinkers, both in the broad psychotherapy and pain literatures, to describe this fresh approach to traditional cognitive therapy that does hold so much promise for chronic pain management. The feedback I have received over time from clinicians I have trained in MBCT for pain is a resounding "Ahh, this is what I have been missing in my cognitive-behavioral therapy work." The integration of mindfulness into traditional cognitive-behavioral therapy adds a dimension of depth that resonates, feels genuine,

and provides a unique approach to shifting patients from unhelpful cognitive, emotional, and behavioral patterns into a way of being with their pain that allows them to adaptively live a life of meaning and value. I hope that in presenting this work—which truly rests on the shoulders of the groundbreaking work of many skilled scientist-practitioners in both the broad psychotherapy and pain literatures—that it will further our combined efforts to give people living with daily pain a way to live a meaningful life, with pain and all. May it be of benefit.

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# Part I

# Chronic Pain

# 1

# Defining Chronic Pain and its Territory

At some point in our lives nearly all of us have experienced pain. What I call "bare bones pain" is adaptive and is as essential to our everyday existence as being able to see, hear, touch, taste, and smell. Pain is our most profound teacher, claiming our attention, implanting itself in memory, readily recalled at a hint of danger. Rare individuals born with a congenital insensitivity to pain experience an abnormal amount of injuries and infections due to their inability to perceive and respond appropriately to painful stimuli and usually die young (Melzack & Wall, 1982). Most of the time when we experience pain, it naturally diminishes as the source (i.e., the injury in whatever form) heals. However, in some instances, pain persists beyond the normal or expected healing time, may arise with or without an identifiable "cause," is unamenable to traditional biomedical treatment options, and it becomes chronic. Along with the territory of chronic pain often comes depressed mood, stress, loss of gainful employment, relationship strain, and a host of other compounding circumstances—the pain is no longer "bare bones."

The International Association for the Study of Pain (IASP), the world's largest interdisciplinary forum devoted to science, clinical practice, and education in the field of pain defines pain as: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP Taxonomy, 1994, Part III, p. 3). Inclusion of the terms "unpleasant" and "emotional" in this definition clearly delineates psychology as integral in the experience of both acute and chronic pain. While there are a variety of taxonomies used to distinguish acute vs. chronic pain, the most common is a temporal profile. Depending on the type of pain and the various definitions, "chronic" is rather arbitrarily demarcated typically as pain experienced at least half of the days of the past 3 or 6 months (IASP Subcommittee on Taxonomy, 1986; NIH, 2011). For pain arising primarily from a specific injury, this 3- or 6-month time frame refers to the time that extends past the "normal" expected healing process from the initial injury (IASP Taxonomy, 1994); however, it often proves exceedingly difficult to determine the end of the healing process (Apkarian, Baliki, & Geha, 2009). Therefore, many have argued that such a taxonomy for classifying chronic pain is inadequate (Apkarian, Hashmi, & Baliki, 2011), and instead, some researchers have focused efforts on identifying brain maps and biomarkers for differentiating acute from chronic pain. However, one aspect from the various

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definitions that is now widely agreed upon is that chronic pain is inherently biopsychosocial in nature as opposed to simply a biomedical phenomenon that can be explained purely in terms of the amount of tissue damage (which was the popular view held right up until the 20th century). In using the MBCT approach to treat pain, it is first helpful to hold a working understanding of such historical perspectives, as well as to be familiar with (and to be able to explain to patients) the current knowledge base of each aspect that makes up the experience of pain: the biological, psychological or human experience, and social factors. Keeping in mind, though, that although these shared features of the experience of pain are common, in reality our experience of pain is deeply personal.

## A Historical Perspective of Pain

### The Biopsychosocial Model: Pain ≠ Just Broken Bones and Tissue Damage

Traditionally pain has been understood from a biomedical perspective that has equated the amount of pain experienced to the amount of underlying tissue damage in a 1:1 relationship. The biomedical model originated from the 17th century with Descartes' mind–body dualism philosophy, and dominated illness and pain conceptualization for almost 300 years, right up until approximately mid-way through the last century. Pain was described purely in reductionistic, mechanistic, physical terms and the brain was considered to play a passive, receptive role of pain signals; psychosocial factors were considered essentially irrelevant. However, Beecher, who served as a physician in the US Army during the Second World War, provided one of the most famous early documented examples of evidence refuting the biomedical perspective (Beecher, 1946). Of the civilians and soldiers that Beecher treated who had experienced compound fractures, penetrating wounds to the abdomen, lost limbs or other intensely painful injuries, Beecher noticed that the majority of the soldiers (as many as 75%) reported no to moderate pain, and required far less pain medication than the civilians with comparable injuries. Beecher documented that the differentiating factor seemed to be the *meaning* that the civilians and soldiers were attributing to the injury. To the soldiers, this was their ticket home—they were evacuated and returned to the US for recuperation; to the civilians on the other hand, they were to leave the hospital to return to their war-torn homeland, and to likely a loss of wages due to an inability to return to work.

Other research began to accumulate supporting Beecher's observations. As one eloquent research example, Jensen and colleagues (Jensen et al., 1994) conducted a magnetic resonance imaging (MRI) study examining the lumbar spines of asymptomatic individuals (i.e., people with no pain, or history of pain) and found that only 36% had normal intervertebral discs at all levels, while the firm majority (64%) had bulges of at least one (and typically more) lumbar disc. In another study, Keefe and colleagues demonstrated that coping strategies were more predictive of self-reported osteoarthritic knee pain than X-ray evidence of the disease (Keefe et al., 1987). Other everyday examples of where the level of injury doesn't necessarily map on to the amount of pain experienced include

when we see athletes playing through a game with a severe injury, maybe we hear on the news about a parent running through fire to rescue their child from a burning house, and yogis during deep meditation will not feel pain.

These observations and empirical findings, and a plethora of findings from other studies, called in to question the very foundation that the biomedical model was built upon, and clearly showed that "verifiable" tissue damage is a poor indicator of pain, and that the brain plays a dynamic, central role in pain processing and perception. Thus, mounting dissatisfaction with the biomedical models' account for illness and pain culminated in a tipping point when Engel (1977) formally challenged this prevailing conceptualization and proposed the integrated biopsychosocial model. The biopsychosocial model redefined illness (including but not specific to pain) as an entity not entirely subsumed under the biological sphere. Instead, manifest illness development, maintenance, and progression were viewed as the result of the convergence of a multitude of internal and external, biological, psychological, emotional, social, and behavioral influences. The shifted emphasis in Engel's approach—away from the purely physical realm—aligned perfectly with Melzack and Wall's (1965) "Gate Control Theory," and together these two models fueled a zeitgeist in the way pain was assessed and treated.

### The Neuromatrix Model of Pain

The Gate Control Theory—now known as the Neuromatrix Model of Pain—is often delivered as an educational component of psychological pain treatments (including MBCT, as you shall see) to convey the rationale to clients as to why psychological treatments work for *real* pain, so it is worth spending some time here to go over it in detail. In essence, this revolutionary theory proposed by Melzack and Wall was the first to formally hypothesize that the brain plays an active, dynamic role in the interpretive processes of the sensory experience of pain (Melzack, 2001, 2005; Melzack & Wall, 1965, 1982). This theory is in stark contrast to the biomedical conceptualization, where the brain was considered a passive recipient of pain signals from a peripheral pain generator (i.e., the identified "source" of injury/pain). The Gate Control Theory represented, for the first time, a conceptualization of pain that took into account the unique and highly interconnected role of neurophysiological pathways, thoughts, emotions, and behavior in determining the experience we call "pain." The original theory described how descending (inhibitory or excitatory) signals from the brain were the stimulus that opened or closed a gating mechanism in the spinal column, and that this mechanism ultimately controlled the amount of pain signals that could reach the brain. Specifically, the theory proposed that if the "gates" are narrowed or closed (i.e., if descending inhibitory signals from the brain predominate), fewer pain signals are processed in the brain and less pain is experienced; however, if the gates are wide open (i.e., if descending excitatory signals from the brain predominate) more pain signals are processed in the brain and the felt experience of pain is amplified.

The Gate Control Theory and the subsequent Neuromatrix Model paved the way for an ensuing body of neuroimaging research. Through the use of technology

such as functional MRI, studies have conclusively demonstrated that critical pain pathways travel through brain areas closely interconnected with cognitive and emotional activity (e.g., the thalamus, anterior cingulated cortex, and limbic system), and Melzack and Wall were the first to emphasize that this neuromatrix had the capacity to inhibit or enhance the sensory flow of painful stimuli. This important research on pain in the brain has demonstrated that psychological processes can actually shape the way painful stimuli are interpreted by the brain and thereby provides convincing evidence that psychological interventions for the treatment of chronic pain hold tremendous potential.

### Models of Stress

As I touched on in the Introduction, living with daily pain as a persistent companion is typically stressful, and stress in turn makes pain worse. Thus, an integral component in many pain treatments is learning to manage stress more effectively. Stress has become a popular term that is a catchphrase for a multiplicity of situations, pressures, and experiences—what one person experiences as stress though, another person might see as the environment in which to thrive. The term "stress" historically has origins in the field of physics, where it describes the force that produces a strain to bend or break an object; however, the way we typically use the word "stress" today was first coined by Seyle in the 1950s (Selye, 1956). Seyle was a pioneer in advancing our understanding of the physiological processes involved when animals are injured or placed under unusual or extreme conditions and he popularized use of the word "stress" to describe the nonspecific response (in mind and body) to any (internal or external) pressure or demand (Selye, 1956, 1973). This nonspecific stress response has since been identified to initiate through the action of the hypothalamic–pituitary–adrenal (HPA) axis and includes cognitive, emotional, physiological (including hormonal and immunological) sequalae, and the inciting factor in triggering this response was termed by Seyle as a "stressor" (Brodal, 2010; McEwan, 2007; Selye, 1956, 1973).

Evolutionarily, back in the days of the caveman, the stress response and the associated rush of adrenaline and other physiological changes served an adaptive, critical life-preserving function for facing off against often larger, faster, more powerful predators (i.e., the classic example of the "saber tooth tiger") where the options were to freeze, run, fight, or, as a last resort, play dead (Bracha, 2004). Unfortunately, however, this maintained function of the primitive brain lacks sophisticated differentiation ability and it is comparatively far less adaptive in the developed world today where this network is responsible for triggering essentially the same physiological response when you are not able to get a good cup of coffee. Further, Seyle observed that when the stress response is prolonged or we are exposed to unresolved stressors, this can lead to what he called "diseases of adaptation" where the once adaptive system breaks down over long periods of heightened elevation, and disease or illness ensues. Research has since confirmed that chronic stress leads to wide-ranging negative effects for the body (i.e., increases in blood pressure, blood sugar dysregulation, greater abdominal fat, hormone imbalances, reduced neurological and immune function, chronic

systemic inflammation, and reduced muscle strength) and has been linked to an enormous range of health conditions, including heart attack, stroke, respiratory disease, autoimmune conditions, depression, and chronic pain (Day, Eyer, & Thorn, 2013).

Sometimes not being able to get a good cup of coffee *is* enough to put us over the edge. As absurd as we know it is after the fact, in that moment, sometimes the smallest things can cause us to lose it. Taking this into account, a powerfully influential model in the evolution of biopsychosocial treatments for chronic pain was Lazarus and Folkman's (1984) Transactional Model of Stress. This model recognized that it is not always so much about the quality of the external stressor that matters, but equally important is the quality of the thought processes, judgments, or appraisals about what that stressor *means* to us at any given moment in time (Lazarus & Folkman, 1984). Lazarus and Folkman qualitatively differentiated among certain types of cognitions, considering them at varying levels, including immediate judgments in reaction to changes in the environment (termed primary appraisals, such as a threat, loss, or challenge), thought processes developed to guide choice of coping strategies (secondary appraisals), and more deeply held beliefs acquired over time. In Lazarus and Folkman's model, stress is the end result of something happening in the environment that is judged to tax or exceed our resources or ability to cope. Given that the very nature of living with chronic pain often becomes in and of itself a persistent stressor that "opens the gates" and makes the pain *worse*, it is no surprise that clinical pain researchers adopted Lazarus and Folkman's Transactional Model for refining the understanding and treatment of chronic pain, emphasizing that treatment can intervene at any of their proposed levels of cognition (Thorn, 2004).

## Neurophysiological Underpinnings (*Bio*psychosocial)

### Pain in the Brain

Extensive anatomical and electrophysiological data emerging from human and animal studies have converged to paint a comprehensive, reliable picture of the "biological" or neurological element of how pain is perceived and processed primarily in various regions of the brain (Jensen, 2010). Generally, pain perception (termed nociception) is conceptualized as a process that can be broken down into four (highly fluid and interconnected) elements: (1) *transduction*, the conversion of the painful stimuli detected by the pain receptors to an electrical message; (2) *transmission*, the process by which the electrical pain message is transmitted to the spinal column and brain; (3) *modulation*, the specific areas of the brain, including sensory, cognitive, and emotional processing areas, that are directly involved in descending signals that modulate the experience of pain; and (4) *perception*, the result of the "neuromatrix" of pain processing areas in the brain that process the pain signal, ultimately resulting in awareness of the experience of pain (Day, in press-b). Although some processing of pain signals does occur at the spinal cord level, the actual *experience* of pain is now widely understood to be the result of supraspinal (i.e., above the spine) neural activity.

Thus, at base, the specialized pain neuronal pathways stemming from the peripheral pain receptor to the cerebral cortex of the brain, termed the nociceptive system, comprises these four elements (Schnitzler & Ploner, 2000).

The first element, *transduction*, starts with pain receptors in the skin, muscles, and internal organs which are free nerve endings of neuronal cells that are called "nociceptors." Nociceptors are on the receiving end of pain-causing, noxious stimuli in the form of intense mechanical, thermal, electrical, or chemical stimulation. Nociceptors detect the noxious stimuli and convert the message to an electrical pain signal (transduction). This signal is transmitted to the axon, which are the thread-like fibers of the nerve cell. Two types of nerve fibers are involved in transmitting pain signals: (1) fast, myelinated axons for sharp, immediate pain; and (2) slow, nonmyelinated axons for chronic, dull, steady pain. At some point or another you may have accidently touched the stove or bumped the edge of the oven while removing a cake, and you likely recall an immediate sharp pain—this pain was transmitted by the fast, myelinated axons that are activated by strong physical pressure and temperature stimulation. This leads to a reflexive recoil of your hand away from the hot surface (the mechanism of which I will describe in more detail momentarily). Even after you ran your hand under cold water, you probably still felt a dull, more defuse type of pain in your hand afterwards—this is due to the slow, nonmyelinated fibers that are activated by the release of chemicals in the skin tissue when damaged. This slower pain serves a rehabilitative function in that it reminds us to protect the damaged body part. For either of these types of pain to elicit a behavioral (or cognitive/emotional) response however, the pain-related signal first needs to be carried along the axon to the spinal column (initial stage of *transmission*).

Once at the spinal column, the first level of pain processing occurs in neurons located in the dorsal horn (i.e., part of the gray matter towards the back of the spine), which respond specifically to the signals from the initial receiving nociceptors (Schnitzler & Ploner, 2000). The signal is transmitted across synapses from the nociceptor to the spine at the dorsal horn via an electrochemical process in which neurotransmitters are released and convey the message of the noxious stimuli. The axons of the neurons in the dorsal horn then cross the midline of the spine within one or two segments, and ascend to the brain via several partially independent pathways located within the spinothalamic tract. Neurons along this tract serve as relay stations conveying the noxious message and at all levels these ascending signals may be modulated by descending signals from the brain (this *modulation* process is described later in this section). However, for fast pain fiber types (the myelinated fibers), an "immediate" withdrawal from the pain stimulus is sometimes needed to minimize harm. Thus, in the example above where you might have accidently touched the side of the oven with your hand, rather than wait for the pain signal to be transmitted all the way to your brain, there is also a reflex mechanism processed at several synaptic links at the spinal column—termed the nociceptive flexion reflex pathway—which causes your hand to immediately recoil from the burning oven surface even prior to your brain processing the experience of pain (Purves et al., 2001).

The conscious *perception* of pain occurs when the pain signals are conveyed to various regions of the brain. If pain signals did not reach the brain, we would not

be aware of the experience of pain. Importantly, one of the first brain regions the ascending fibers of the spinothalamic tract project to is the medulla oblongata and the reticular formation; processing here affects consciousness (with the severest of pain causing unconsciousness), and cardiovascular and respiratory responses to pain. Other ascending fibers of the spinothalamic tract project to the thalamus, which acts as a relay station disseminating and projecting the pain signals to various distributed areas of the cortex in an extensive central network of pain processing (Schnitzler & Ploner, 2000). Research has consistently shown that the brain regions most closely linked to pain are the primary somatosensory cortex (sensory–discriminative aspects of pain), secondary somatosensory cortex (recognition, learning, and memory of painful experiences), limbic system (emotional processing of pain), and the anterior cingulate cortex (allocation of attentional resources to pain and processing of pain unpleasantness and motivational–motor aspects of pain), insula (involved in processing information about one's physical condition, autonomic reactions, and potentially in affective aspects of pain-related learning and memory), and the prefrontal cortex (general executive functions such as planning of complex responses to pain) (Bantick et al., 2002; Jensen, 2010; Schnitzler & Ploner, 2000).

In parallel to the processes and regions associated with the experience of pain are areas of the brain that are directly involved in descending signals and in centrally *modulating* the experience of pain. Therefore, the perception of pain is also a function of the degree of modulation concurrently present. These are termed "descending" modulatory pathways as they stem from areas of the brain that sit above where ascending pain pathways project from the spinal column to the brain. Descending modulation circuitry is proposed to arise from multiple cortical and subcortical areas of the brain (including the hypothalamus, amygdala, and the rostral anterior cingulate cortex) that feed in to the periaqueductal gray region (PAG), and with outputs from the PAG to the medulla (specifically the nucleus raphe magnus and the nucleus reticularis gigantocellularis located within the rostral ventromedial medulla) (Ossipov, Dussor, & Porreca, 2010). Activation of the PAG projects to the medulla and then to neurons in the spinal or medullary dorsal horns, thereby activating an opioid sensitive circuit that reduces pain (Ossipov et al., 2010). Studies since the 1970s have shown that electrical stimulation of the PAG leads to analgesia. In one early study, Reynolds found that electrical stimulation of the PAG caused profound analgesia so powerful that a laparotomy surgery could be performed in a fully conscious rat without observable signs of distress (Reynolds, 1969).

To this day, activation of this opioid sensitive circuit underlies the action of the most widely used pain-relieving drugs used in humans, including opiates, cannabinoids, nonsteroidal anti-inflammatories (NSAIDs), and serotonin/norepinephrine reuptake blockers (Ossipov et al., 2010). These advances in understanding of the central modulation of pain have led to substantially more effective pain management over the past several decades, especially for *acute* pain management (Ossipov et al., 2010). However, for *chronic* pain, the long-term use of pain-relieving drugs is often associated with minimal pain relief and substantial negative side-effects, including possible addiction, tolerance effects, constipation, rebound pain, impaired cognition, and nausea (Ashburn & Staats, 1999; Trescot et al., 2008).