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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).

Not only are there differences in the pain medication treatment approaches most suitable for acute vs. chronic pain, but the brain itself and how pain is processed has been shown to change as a function of ongoing, persistent pain.

The Neuro-Signature of the Transition from Acute to Chronic Pain

Research is emerging to suggest that the way in which pain is processed by the brain changes as a function of pain duration, and critical differences between the brain in acute pain vs. chronic pain have been identified. In attempting to discover the explanatory pathway(s) underlying the transition from acute to chronic pain, researchers are hoping eventually to identify ways to interrupt this pathway and prevent pain chronicity from occurring. This line of research is particularly pertinent to pain resulting from some form of initial traumatic injury (as opposed to other degenerative conditions, such as arthritis, for example). However, given central sensitization (i.e., maladaptive neuroplasticity that heightens neurological pain processing and sensitivity) in chronic pain is also a factor underlying worsening outcomes for essentially all types of chronic pain, the findings of this research agenda may eventually hold widespread applicability (Apkarian et al., 2011; Woolf, 2011).

A promising line of laboratory-based research has attempted to gain insight into the transition from acute to chronic pain by comparing the brain activity of healthy individuals to those with chronic pain. One widely cited study implementing this approach utilized an experimental pain paradigm (specifically using a thermal pain stimulus) to examine the neurological response to the acute pain stimulus in the brains of healthy controls to those with a diagnosis of chronic low back pain (Baliki, Geha, Fields, & Apkarian, 2010). Results showed that brain activity was equivalent across these groups in areas of the brain associated with pain encoding and perception. Interestingly, however, activity in the bilateral nucleus accumbens (NAc)—an area of the brain responsible for both the encoding of the salience of the pain at stimulus onset (i.e., it signals *anticipation* of pain perception) and with the analgesia-related reward at stimulus offset—showed a disruption within the group of individuals with chronic low back pain. Specifically, this process was *reversed* in the individuals with chronic low back pain in that while healthy subjects were predicting a reward value at the cessation of the thermal pain causing stimulus, the NAc activity in the clinical sample showed they were *not* expecting reward at stimulus offset. Thus, the motivational value of analgesia was disrupted due to the presence of persistent pain. Further analysis showed that the reason for this disruption was a connective neural reorganization in the chronic pain group; in the healthy sample the NAc activity was mainly associated with the insula, whereas in the clinical sample the NAc was correlated with the medial prefrontal cortex (mPFC; a region that modulates emotional evaluations relative to the self). Moreover, the strength of this shift in connectivity in the clinical sample was in direct proportion to the amount of self-reported back pain intensity any given patient reported. Hence, both the chronicity as well as the intensity of the pain experienced were associated with brain reorganization (i.e., changes in areas of connectivity), notably within reward and motivational pathways in the context of chronic pain in this study.

In addition to chronic pain changing the way a pain stimulus is processed in specific pathways, research has also shown that living with persistent pain is associated with changes in brain processing in general, global (nonpain related) ways through disruption of critical homeostatic networks. In one seminal study comparing the brain activity of individuals with chronic low back pain to healthy controls while completing a minimally demanding visual attention task, no differences in performance or increases in brain activity were observed (Baliki, Geha, Apkarian, & Chialvo, 2008). However, marked differences in *decreased* activity were observed that correspond with a specific brain network, the default mode network, with the chronic pain group displaying reduced deactivation in several key areas of this region. This network represents the brain's activity in the resting state (i.e., in the absence of a subject doing anything) and a recent review of the literature reported this same reduced deactivation effect has been replicated across a number of chronic pain conditions (Apkarian et al., 2011). Research suggests that this disrupted default mode network becomes more pronounced as a function of chronic pain duration, with those individuals with a longer history of pain showing more disruption (Baliki et al., 2008). Importantly, in healthy individuals, the default mode network correlates negatively with activity in brain regions involved in attention and executive function, and an appropriate balance between these areas has been associated with memory function (Fornito, Harrison, Zalesky, & Simons, 2012). Thus, the findings of heightened default mode network activity in individuals with chronic pain may be a key reason why such individuals often report cognitive difficulties, such as trouble paying attention and declines in memory function.

Converging lines of research have demonstrated that chronic pain not only leads to changes in pain processing and associated connections, as well as general brain function (as described in the previous research on the default mode network) but there are also structural, anatomical brain changes that have been consistently observed in the presence of persistent, ongoing pain. Specifically, a loss in cortical regional gray matter volume has been found for a number of chronic pain conditions, including back pain (Schmidt-Wilcke et al., 2006), fibromyalgia (Luerding, Weigand, Bogdahn, & Schmidt-Wilcke, 2008), osteoarthritis of the knee (Rodriguez-Raecke, Niemeier, Ihle, Ruether, & May, 2009), and headache (Kim et al., 2008), among others. This loss in regional gray matter has been found to increase with longer duration of pain, higher intensity of pain, and as a function of the interaction between these factors (Apkarian et al., 2004; Geha et al., 2008; Kuchinad et al., 2007). Regional gray matter loss was also associated with cognitive declines in memory function in a sample of individuals with fibromyalgia pain (Luerding et al., 2008). Although the loss in gray matter volume may be an indicator of neuronal death, some research has found that these volume decreases are reversible when pain is relieved (Gwilym, Fillipini, Douaud, Carr, & Tracey, 2010; Obermann et al., 2009; Rodriguez-Raecke et al., 2009), suggesting that the observed structural changes in gray matter may more likely be caused by synaptic plasticity in these regions. The finding, however, that structural brain changes in the context of pain are reversible once adequate pain relief is achieved is certainly promising.

The exact timeline of the changes observed in the brain in acute vs. chronic pain has not been precisely identified, however, some research suggests that the neurological and psychological foundation for long-term pain is in place within hours of the initial injury (Carr & Goudas, 1999). Recent research is finding that the initial brain response to an inciting event (i.e., judgments of the intensity of the pain, quality, and meaning) potentially instigates a cascade of neurological changes that for some individuals sparks a gradual reorganization of the central nervous system, including structural changes, an increase in the number of pain receptors in the spinal cord, and a reduction in brain modulation processes (Apkarian, Baliki, & Farmer, 2013; Woolf & Salter, 2000). In one elegant study, the temporal causal relationship between injury, brain reorganization, and development of chronic pain (or recovery) was investigated by Apkarian and colleagues, who conducted brain imagining on individuals with subacute back pain, tracking them over a year to identify predictors of chronicity (Apkarian et al., 2013). Half of the sample went on to continue to experience the same magnitude of back pain 1 year later, and only these individuals (not those who recovered) showed a slow progression of gray matter density decreases. Findings showed that this loss of gray matter in the patients that went on to experience chronic pain was preceded by functional connectivity differences between the mPFC and NAc that were observable from the initial brain scans. This is consistent with the experimental research described above that identified this connectivity as a distinguishing feature of cortical reorganization in chronic pain (Baliki et al., 2010); what this study further demonstrated, however, was that the initial emotional response (as indicated by the mPFC-NAc disruption) represents a connectivity reorganization that ultimately may predict chronicity and also explain the structural changes observed as pain persists (Apkarian et al., 2013). Finally, this study also found that when an early pain medication treatment variable was included in the analyses along with the brain imaging data, results indicated that medication can play a protective role against pain chronification (Apkarian et al., 2013). The treatment parameter in isolation however, served no significant predictive function; the authors interpreted this finding as indicating that treatment outcome is contingent on the brain state. Although more research is needed to replicate and extend this finding, this research opens up exciting possibilities for the future in enhancing initial treatment that might prevent the transition from acute to chronic pain.

In sum, not all individuals who experience an acute injury go on to experience chronic pain; the correlates and predictors of who progresses to develop chronic pain and why this is the case are only beginning to be understood—we are at the tip of the iceberg. However, the evidence to date suggests that seemingly small differences in the initial brain state of the individual at the time of injury (and the period immediately following) can precipitate major differences in whether pain "heals" in the acute phase, or persists and becomes chronic. As discussed later in this chapter, the landscape for acute compared to chronic pain is vastly different not only at the level of neurological processing and cortical reorganization, but critically also in the cognitive, emotional, motivational, and behavioural response to pain. This, combined with our understanding of the theoretical models of the experience of pain, again provides a strong rationale for an interdisciplinary approach to chronic pain management that goes beyond pharmacopoeia.

The Human Experience (Bio*psycho*social)

The Emotional Impact of Chronic Pain

Chronic pain, for most people in most circumstances, is an aversive experience that typically elicits negative emotional and affective psychological responses. Thus, the bulk of the emotion research to date has focused on understanding the influence of negative emotions on pain and associated disability and rehabilitation. Due to their high comorbidity rates with chronic pain, particular attention has been devoted towards investigating the role of depression, anxiety, fear, and anger (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). More recent research has shifted towards understanding the role of positive emotions and resiliency, which may serve a protective function and buffer the stress associated with chronic pain. Psychosocial treatments for chronic pain target both negative *and* positive emotions to mitigate the impact of chronic pain on emotional functioning.

Depression

Rates of comorbid depressive disorders within chronic pain populations have been found to be approximately 40–50%, although this is likely an underestimate as depression often goes undiagnosed (and untreated) (Banks & Kerns, 1996; Dersh, Gatchel, Mayer, Polatin, & Temple, 2006; Romano & Turner, 1985). Not only is depression a prevalent comorbidity for people with chronic pain, but the combination is deadly. Lifetime prevalence of suicidal ideation in these individuals is approximately 20% and rates of suicide attempts are estimated to be between 5% and 14%, which translates to risk of death by suicide being at least doubled in individuals with chronic pain (Tang & Crane, 2006). Further, in one study depression was found to uniquely predict the degree to which pain interferes with daily activities and overall life satisfaction, even while controlling for demographic and key psychological variables (Day & Thorn, 2010). Research has also identified that poorer outcomes are associated with the treatment of pain when comorbid depression goes untreated (Shmuely, Baumgarten, Rovner, & Berlin, 2001). For improvement of engagement in valued activities, quality of life, and for optimal pain treatment, it appears screening for and treating depression is critical.

Anxiety

Although epidemiological data on the rates of anxiety in pain populations are limited, anxiety and fear about pain is a common experience. One large-scale study conducted within a fibromyalgia sample reported that approximately 44–51% of individuals endorsed substantial anxiety symptoms (Wolfe et al., 1990). Anxiety may be especially common in individuals where a definitive diagnosis identifying the "cause" of the pain has not been possible, leaving these individuals "not knowing why" they have pain and therefore worrying about what certain symptoms "might mean." For example, someone who has persistent daily headaches might become anxious that these are caused by an undetected brain tumor, and they might become fearful about what the future will hold (note that this example also demonstrates the very close proximity between cognitive interpretations of symptoms, i.e., pain = brain tumor, and an associated anxiety response).

Many individuals fear the pain will become worse, and anxiety-related fear of pain, fear of movement (kinesiophobia), and fear of re-injury is particularly debilitating (Vlaeyen & Linton, 2000). Some research has found that fear-related factors more accurately predict functional limitations than even pain severity, duration, or other biomedical factors (Crombez, Vlaeyen, & Heuts, 1999; Vlaeyen, Kole-Snijders, Rotteveel, Ruesink, & Heuts, 1995). Critically, catastrophic cognitions are theorized to engender this fear, and this then subsequently leads to increased avoidance of engaging in activities (Vlaeyen & Linton, 2000). As we will see in the next section, behavioral avoidance of activity and more time "resting on the couch" is associated with heightened pain, more disability, and lower return to work rates, all of which not only add to the heightened suffering of the individual, but place a substantial increased economic burden on the medical system (Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995; Vlaeyen et al., 1995).

Anger

Another critical emotion to target in treatment is anger. Anger and associated blame towards self, significant other, healthcare providers, employers, insurance companies, the person who caused the accident… There are a whole host of reasons that a person living with chronic pain might have to feel angry (ranging from slight irritation or frustration, up to fury). One study within a multidisciplinary pain clinic identified that approximately 98% of patients at the time of their intake assessment reported feeling some degree of anger, and for most (74%) this anger was directed toward themselves (Okifuji, Turk, & Curran, 1999). Healthcare providers were identified by Okifuji and colleagues (1999) as the second most frequent target of patients' anger (62%), and research by Burns et al. found that this may lead to patients reporting a weaker working alliance with their clinicians (Burns, Higdon, Mullen, Lansky, & Mei Wei, 1999). Given that working alliance is a strong predictor of treatment outcome in its own right (Lambert, 1992), a disrupted alliance will likely substantially lessen the probability of treatment success. Indeed, research across a number of different pain types has found a consistent relation between anger and worse pain treatment outcomes, including higher pain intensity ratings, longer pain duration, increased analgesic medication intake, and higher impaired functioning (Gatchel et al., 2007; Greenwood, Thurston, Rumble, Waters, & Keefe, 2003; Trost, Vangronsveld, Linton, Quartana, & Sullivan, 2012). Finally, anger, anxiety, and depression are closely related, likely in reciprocal relationships (Trost et al., 2012), and often co-occur.

Resilience

Although the preponderance of pain literature has focused on the maladaptive role of negative emotions, the potential buffering, protective role of resiliency, and positive emotions/affect have more recently begun to receive an upsurge of empirical attention. There are two complementary theories that are the most widely cited which attempt to explain how positive emotion may improve pain and coping: Fredrickson's "Broaden and Build" theory (Fredrickson, 2001) and Zautra and colleagues' dynamic model of affect (Zautra, Smith, Affleck, & Tennen, 2001). The Broaden and Build theory proposes that people with more

positive affect adapt more readily in the face of stress as they are able to broaden their outlook and build on their well-being that is already present, and in this way they are able to enhance their overall sense of well-being (Fredrickson, 2001). In support of this model, research suggests that positive emotions may foster recovery after pain flare-ups (Zautra et al., 2001), and one study found subjective happiness was associated with improved general health perception in individuals with low back pain (Takeyachi et al., 2003). In Zautra and colleagues' dynamic model of affect, they proposed a dynamic relationship between positive and negative affect such that during times of stress (e.g., when pain intensifies) the full range of affective experience collapses within a smaller, typically negative range. However, boosting affective complexity and the presence of positive emotions is hypothesized to be associated with decreased negative affective states, thereby functioning to preserve well-being during stressful times and build resilience (Zautra et al., 2001), and support for this hypothesis has been found (Davis, Thummala, & Zautra, 2014; Strand et al., 2006). Thus, both Fredrickson's notion of building on existing resources, and Zautra's theory of capitalizing on positive affect to dynamically counteract the negative affect associated with pain appear to be supported. One final potential protective factor worth mentioning is maintaining a sense of humor despite the pain. Humor and laughter are typically outward expressions of pleasant/positive emotions, and the use of humor has been shown to improve pain thresholds (Mahony, Burroughs, & Hieatt, 2001) and reduce pain intensity (Tse et al., 2010), and lead to the release of endorphins in the brain, a natural pain killer (Haig, 1988). Thus, appropriate use of humor in therapy might be a welcome addition for many reasons, including pain reduction!

The Behavioral Expression of Chronic Pain

The behavioral expression of chronic pain and the social ramifications of pain are difficult to distinguish, and although I describe them in separate sections here, as in all the other elements that coalesce in the experience of pain, they are closely interconnected. Spanning from the earliest investigations of the role of behavior in chronic pain adjustment, a key focus has been on understanding the influence of pain behaviors (Fordyce, 1976), which are functionally the communicative expression of pain. Pain behaviors can be verbal (e.g., statements such as, "I am hurting"), paraverbal (e.g., grunts, moans, sighs), and nonverbal (e.g., grimacing, wincing, resting, taking medication) (Fordyce, 1976; Sullivan, Adams, & Sullivan, 2004). Such behaviors may serve a protective, useful function in the short term when pain is acute by eliciting solicitous attention and responses, as well as assistance. However, when pain behavior engagement is maintained long term in the context of chronic pain, research has found that such behaviors predict reduced likelihood of return to work, higher compensation costs, and an increased number of lost work days, as well as increased self-reported disability (Prkachin, Schultz, & Hughes, 2007). Another study has found that decreases in guarding (i.e., protecting the site of pain by shielding the area to avoid it from being bumped etc.) and time spent resting were most strongly associated with improvement in self-reported disability during a multidisciplinary pain treatment (Jensen, Turner, & Romano, 2001). Along with individuals potentially lapsing into

unhelpful patterns of specific pain behaviors, there are also broader more general patterns of avoidance behavior that are maladaptive in the context of chronic pain.

Avoidance Behavior

Compelling support for a disability model based on fear and maladaptive behavioral avoidance activity patterns—The Fear Avoidance Model—has been found in reviews of the literature, with fear conceptualized as underlying avoidance behavior, leading to disuse and disability (Asmundson, Norton, & Vlaeyen, 2004; Vlaeyen & Linton, 2000). The functional "opposite" of avoidance is behavioral engagement. Reviews of the literature have shown that appropriately paced engagement in valued activities despite the pain correlates with a range of positive outcomes, including less pain intensity, depression, pain-related anxiety, lower levels of physical and psychosocial disability, and improved globally rated daily activity and overall emotional well-being (McCracken & Samuel, 2007; McCracken & Vowles, 2006; Thompson & McCracken, 2011). Thus, reducing behavioral avoidance and enhancing engagement in valued activities are key treatment targets in the management of chronic pain.

Sleep

Although perhaps not precisely or neatly falling under the category of "behavioral" aspects of chronic pain, disturbances in sleep are a commonly reported problem. Epidemiological data indicate that as many as 53 to 88% of individuals with chronic pain also suffer from sleep disorders (Smith, Perlis, Smith, Giles, & Carmody, 2000; Tang, Wright, & Salkovskis, 2007; Wilson, Eriksson, D'Eon, Mikail, & Emery, 2002). Even when potentially confounding factors such as depression, anxiety, and other medical problems are controlled, people with chronic pain are at a significantly higher risk for developing insomnia compared to people without pain (Taylor et al., 2007). Conversely, over 40% of people with insomnia also report chronic pain (Ohayon, 2005); hence the relation between pain and sleep problems appears to be reciprocal (Smith & Haythornthwaite, 2004). Further demonstration of this reciprocal nature is found in research which has shown that effectively treating a comorbid sleep problem improves pain outcomes (Khalid, Roehrs, Hudgel, & Roth, 2011). Moreover, anxiety and depression symptoms are closely correlated with pain and disturbed sleep; and targeting improved mood in treatment may improve both pain and sleep outcomes (O'Brien et al., 2010; Schrimpf et al., 2015). On the other hand, sleep is one of the greatest homeostatic affect modulators, so improving sleep will also likely improve mood and pain (Palmer & Alfano, 2016). Pre-sleep cognitive arousal and pain catastrophizing may represent additional useful treatment targets for combined pain and sleep problems, as a recent study found these factors to be powerful cognitive predictors of disturbed sleep symptoms in a clinical pain population (Byers, Lichstein, & Thorn, 2015). These reciprocal relations show that although we are discussing the various aspects of the chronic pain experience in "isolated" sections here, these domains are closely interconnected and any one element has the capacity to initiate a domino effect, eliciting (either adaptive or maladaptive) change in all the other components.

Cognitive Factors and Chronic Pain

Unfortunately, many individuals with chronic pain who do not have an identified pathology or medical diagnosis for their pain are often directly or indirectly told by healthcare professionals that the pain is "all in their head." Well, although, as we saw in the earlier section, research has confirmed that pain *is* processed in the brain, our consideration of the role cognitive factors play in pain here in this section is not the same as saying that the pain is "not real." It is the *exceedingly* small minority of individuals who might engage in malingering. Clinically it is most helpful to communicate that the pain *is* real, *and* that the way we think about it influences how "wide the gates are" (i.e., to paraphrase the Gate Control Theory) and how much pain we feel. In support of this, a plethora of research has shown that cognitive factors play a key maintaining role in prolonging negative emotions, initiating unhelpful behavioral responses, and perpetuating poor pain-related outcomes (Thorn, 2004).

Attention

At base, pain is an overriding stimulus that demands attentional processes to pay heed and to selectively attend to the pain at the cost of other information in the environment (Crombez, Van Ryckeghem, Eccleston, & van Damme, 2013; Eccleston & Crombez, 1999). Ontogenetically and evolutionarily pain serves a survival functional, claiming attention, leading to a fear-based urge to escape, and behavioral interruption. Within the context of ongoing persistent pain, the intrinsic role of attention in urging escape becomes maladaptive as in chronic pain there *is* no escape and so the fear-based urge is maintained due to lack of goal fulfilment. In this context, many individuals living with persistent pain develop a *hypervigilant* attention to the emotionally laden pain stimulus, or as otherwise described, a cognitive attentional bias toward noticing painful stimuli. As attention is a limited resource that is required for a number of cognitive functions, and that pain places a toll on attentional resources, it is not surprising that many people with chronic pain report difficulty concentrating, making decisions, and an array of other executive function problems (Eccleston & Crombez, 1999). Moreover, attention is the foundational, initial cognitive factor underlying the primary appraisal processes theorized to play a key role in models of stress and coping.

Primary Appraisals

As discussed earlier, chronic pain and the wider landscape of what living with chronic pain entails are often stressful. So along with that, models of stress point to specific cognitions that maintain the stress response and exacerbate pain. Just to briefly recap, in the Transactional Model of Stress and coping framework (Lazarus & Folkman, 1984), the stress response is initiated (or not) on the basis of the *meaning* given to the contextual stimulus (the "stressor") once it has entered the field of awareness. Specifically, Lazarus and Folkman (1984) described three primary appraisal judgment categories about the meaning of a potentially stress-inducing stimulus (e.g., pain): (1) *threat*, which is the evaluation that pain represents a danger that outweighs one's ability to cope; (2) *loss/harm*, where pain is viewed as damaging, and/or as a loss of some form, for example, loss in

the ability to work, engage in previously pleasurable activities etc.; or (3) *challenge*, where one perceives that he/she has the resources to cope with the pain. Of the three forms of primary appraisals, research has identified that threat appraisals are the most commonly reported within the context of chronic pain (Unruh & Richie, 1998). Threat appraisals and attentional processes are closely associated, and threat appraisals are theorized to be a key precipitating and maintaining factor in the development of hypervigilant attention to painful sensations (Crombez et al., 2013), leading to fear-avoidance beliefs that increase pain and disability, and negatively impact treatment and return to work rates (Pfingsten, Kroner-Herwig, Leibing, Kronshage, & Hildebrandt, 2000; Poiraudeau et al., 2006; Waddell, Newton, Henderson, Somerville, & Main, 1993). Further, beliefs that the feeling of hurt equals harm (i.e., pain equals damage) have been found to significantly predict physical dysfunction and poor adjustment to pain (Jensen et al., 1994). Loss appraisals in people with chronic pain have also been found to be frequently reported (Walker, Holloway, & Sofaer, 1999) and are theorized to lead to symptoms of depression, a sense of helplessness, and a reduction in adaptive coping behaviors (Thorn, 2004). Although research is limited on challenge appraisals in pain populations, one study found that only 14% of individuals appraised a recent painful experience as a challenge (Unruh & Richie, 1998).

Secondary Appraisals

Following closely on the heels of primary pain appraisals are ensuing secondary appraisals. Pain catastrophizing refers to an exaggerated negative mental set about actual or anticipated pain (Sullivan, Thorn, et al., 2001), and has variously been conceptualized as a primary appraisal, a secondary appraisal, and as a coping strategy (Thorn, 2004). Pain catastrophizing is by far the most researched and documented cognitive factor in the pain literature. A voluminous body of research has consistently demonstrated that it is a robust predictor of higher pain severity, disability, poorer social functioning, longer recovery times following surgery, greater healthcare utilization, and worse mood (e.g., depression and anxiety), above and beyond other factors such as disease severity, pain intensity, anxiety, and neuroticism (Day & Thorn, 2010; Drahovzal, Stewart, & Sullivan, 2006; Edwards, Cahalan, Mensing, Smith, & Haythornthwaite, 2011; Flor, Behle, & Birbaumer, 1993; Geisser, Robinson, Keefe, & Weiner, 1994; Keefe, Rumble, Scipio, Giordano, & Perri, 2004; Sullivan, Rodgers, & Kirsch, 2001; Sullivan, Thorn, et al., 2001). Pain catastrophizing is also theorized to be a primary precursor for exaggerated displays of pain behaviors, which serve a social communicative function in coping efforts to elicit solicitousness, empathy, and support from others in the environment (Sullivan, Thorn, et al., 2001). This theoretical framework has been called the Communal Coping Model of catastrophizing (Sullivan, Thorn, et al., 2001), and although available evidence is not entirely consistent with all its predictions (Sullivan, 2012; Tsui et al., 2012), a number of studies in both experimental and clinical samples has found support for many of its tenets (Giardino, Jensen, Turner, Ehde, & Cardenas, 2003; Holtzman & Delongis, 2007; Sullivan et al., 2004; Thibault et al., 2008; Tsui et al., 2012). Given the strong predictive role pain catastrophizing has been shown to play, over and above other factors, it represents a prime target for interventions aimed at improving pain coping, and in the next chapter I will

describe specific interventions that have been designed to target this powerful predictor of the experience of pain (Thorn, 2004).

Adaptive Secondary Appraisals

As noted above in the emotion literature, in the past several decades there has been an upsurge in research devoted towards understanding the potential protective role of positive psychological factors. In the context of cognitions, beliefs related to pain management self-efficacy—the conviction one can cope with, and manage pain—is one form of secondary appraisal that a large body of research has identified to play a protective role in fostering adaptive physical and psychological adjustment to pain (Rudy, Lieber, Boston, Gourley, & Baysal, 2003). Furthermore, enhanced self-efficacy has been found to be a critical mechanism underlying improvements in pain, disability, depression, and adjustment following pain treatment (Altmaier, Russell, Kao, Lehmann, & Weinstein, 1993; Jensen et al., 2001; Keefe, Rumble, et al., 2004). However, some research suggests that perceived control over the *effects* of pain, rather than the sensation of pain per se, may be most important in regards to facilitating better adjustment and less disability (Tan, Jensen, Robinson-Whelen, Thornby, & Monga, 2002).

More recently, positive psychology-oriented research efforts have focused on identifying the potential protective role of mindfulness and pain acceptance, and particularly promising results have been found. Kabat-Zinn (1990) defines mindfulness as "…the awareness that emerges through paying attention on purpose, in the present moment, and non-judgmentally to the unfolding of experience, moment by moment" (p. 145). In one study by Schutze and colleagues (Schutze, Rees, Preece, & Schutze, 2010), higher levels of mindfulness predicted lower levels of pain, negative affect, pain catastrophizing, fear of pain, pain hypervigilance, and functional disability. Acceptance of pain, defined as the willingness to experience pain and to continue to engage in activities despite the pain (McCracken, Vowles, & Eccleston, 2004), has been shown to be associated with an array of better outcomes, including less pain, depression, anxiety, physical, and psychological disability (McCracken & Eccleston, 2003). Finally, a model of psychological flexibility (described in greater detail in the next chapter) has recently been proposed as an integrated model of several positive psychology constructs that may play a protective role in the context of pain. Preliminary support for individual aspects of this multifaceted (predominantly cognitively oriented) psychological flexibility model has been found, although more research is still needed to elucidate these factors in relation to the experience of pain specifically (Hann & McCracken, 2014; McCracken, Vowles, & Zhao-O'Brien, 2010; Sturgeon, 2014).

The Wider Ramifications of Chronic Pain (Biopsycho*social*)

How Big is the Problem and Who is at Risk?

At a nomothetic social level, chronic pain is a pervasive, major health concern, and has been referred to as a public healthcare crisis (Darnall et al., 2016; IOM, 2011) with worldwide point prevalence estimates ranging between 2% and over

55% (Blyth et al., 2001; Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006; Catala et al., 2002; Elliott, Smith, Penny, Smith, & Chambers 1999; Eriksen, Jensen, Sjøgren, Ekholm, & Rasmussen, 2003; Harstall & Ospina, 2003; Moulin, Clark, Speechley, & Morley-Forster, 2002; Neville, Peleg, Singer, Sherf, & Shvartzman, 2008; Sjøgren, Ekholm, Peuckmann, & Grønbæk, 2009; Verhaak, Kerssens, Dekker, Sorbi, & Bensing, 1998). In the United States, it is estimated that chronic pain affects 116 million adults, and prevalence is on the rise (IOM, 2011). This figure translates into pain affecting more Americans than diabetes, heart disease (including both coronary heart disease and stroke), and cancer combined (The American Academy of Pain Medicine, 2016). Low back pain is the most common source of chronic pain (28%), followed by severe headache or migraine pain (14%), and neck pain (14%) (National Centers for Health Statistics, 2013). Persistent pain is not always a primary condition, however, and is often a secondary condition that further complicates and impedes treatment for a vast spectrum of injuries and diseases, such as postoperative recovery and treatment of malignancy. However, all of these prevalence findings may be considered a vast *under*estimate as it is also well established that pain is both underdiagnosed and undertreated (IOM, 2011).

Risk for the development of chronic pain is not evenly proportionate across demographic groups: pain discriminates. Thus, compounding the pervasive underdiagnosis and undertreatment of pain is the well-documented existence of fundamental health, treatment, and ethnicity disparities across a broad range of samples, settings, and types of pain (Tait & Chibnall, 2005). Prior research in regards to such disparities indicates that a number of intervening factors potentially influence the relationship between healthcare access, treatment, and outcome (Day & Thorn, 2010). Age is one such intervening factor and although the relationship between age and increased risk for pain has not always been found to be linear, most population-based research consistently shows that chronic pain disproportionately affects older individuals (Bergman et al., 2001; Blyth et al., 2001; Ng, Tsui, & Chan, 2002). In an extensive review of the available research on gender differences in pain, Unruh found that women are more likely than men to experience a variety of recurrent pain conditions and women report pain of greater intensity, frequency, and duration than men (Unruh, 1996).

While the relationship between pain and race is complex, most of the research to date has focused on the comparison between African Americans and White Americans, and considerable evidence suggests that African Americans report greater pain intensity in acute clinical pain and in a variety of chronic pain conditions (Breitbart et al., 1996; Chibnall, Tait, Andreson, & Hadler, 2005; Selim et al., 2001). However, it has been suggested that individuals from minority racial groups may suffer from more severe symptoms before seeking treatment and that this is consequently an important point to consider when examining research conducted in clinical samples (McCracken, Matthews, Tang, & Cuba, 2001). Further, a disproportionate number of African Americans live in rural areas and represent a higher percentage of individuals classified as low socioeconomic status (SES), and evidence suggests that documented racial differences may be explained by these factors rather than biological differences associated with race

per se (Day & Thorn, 2010; Kington & Smith, 1997; McIlvane, 2007; Williams & Collins, 1995). Taken together, the limited research on the experience of chronic pain in low SES and rural individuals suggests that annual income of less than US$25,000, no high school diploma, and rural residency are associated with a greater likelihood of having disabling chronic pain (Hoffman, Meier, & Council, 2002; Nguyen, Ugarte, Fuller, Haas, & Portnenoy, 2005; Portenoy, Ugarte, & Fuller, 2004). The overall impact is that people at the lowest end of the income gradient experience both intractable stressful circumstances and an impoverished lack of resources, which consequently combine to exacerbate their susceptibility to poor health and negative psychosocial indicants, and make them more vulnerable to experience chronic pain (Adler et al., 1994; Almeida, Neupert, Banks, & Serido, 2005).

Costs of Chronic Pain

Given the high prevalence rates of chronic pain it is not surprising that chronic pain is among the most common presenting complaints seen in medical practice, with some reports indicating pain accounts for more than 80% of physician visits (Gatchel, 2004). Low back pain has been reported as the second most frequent reason for visits to the physician (Hart, Deyo, & Cherkin, 1995), and headache pain alone accounts for 18 million physician visits per year in the United States (Schwartz, Stewart, Simon, & Lipton, 1998). Opioid analgesic medications are one of the most commonly prescribed treatments for chronic pain, but their misuse is now recognized as a healthcare crisis (IOM, 2011; NIH, 2011). The National Centers for Health Statistics, in their 2013 report, found a 300% increase in opioid analgesic consumption between 1999 and 2010, and death rates for poisoning involving opioid analgesics more than tripled between 2000 and 2010, resulting in over 16,000 opioid-involved overdose deaths in 2010 (National Centers for Health Statistics, 2013; NIH, 2011). Moreover, unrelieved pain is associated with longer hospital stays, increased rates of re-hospitalization, and often results in an inability of individuals to maintain health insurance (The American Academy of Pain Medicine, 2016), thereby further escalating the burden and cost chronic pain places on the healthcare system.

The direct and indirect economic costs of the widespread and debilitating nature of pain on society are astronomical. Most recent estimates in the United States reported that the total annual costs of pain ranged from $560 to $635 billion in 2010 constant dollars (Gaskin & Richard, 2012), an amount equivalent to approximately $2,000 annually for every person living in the US (IOM, 2011). Within this estimate, healthcare costs due to pain accounted for approximately $261 to $300 billion, and lost productivity costs (based on days of missed work, hours of work lost, and lower wages) due to pain ranged from $299 to $355 billion (Gaskin & Richard, 2012). Gaskin and Richard report these are conservative estimates as excluded from these figures are costs due to pain for nursing home residents, children, military personnel, and persons who are incarcerated (Gaskin & Richard, 2012). The costs of chronic pain are more than just financial, however, with chronic pain exacting a toll on every facet of life and functioning for the individual living in daily pain.

The Personal Social Cost

Focusing on just the macro-level societal and financial figures in isolation can detract from the human cost experienced at an everyday level for the individual and their friends, family, and loved ones living a life with chronic pain. The World Health Organization (WHO) reported findings from the IASP that one-third of people living with chronic pain are unable or less able to maintain an independent lifestyle due to the effects of the pain (WHO, 2004). Further, the WHO (2004) reported that approximately one-half to two-thirds of individuals with chronic pain are less able or unable to engage in physical exercise, enjoy normal sleep, attend to household chores, participate in social activities, drive a car, walk, or have sexual relations. Thus, the behavioral and social ramifications of pain are closely interconnected and extend across the entire spectrum of day-to-day living, from employment and level of independence, to the dynamic of interpersonal relationships, to what one can "do" in spare time or for hobbies.

A particular challenge for many individuals with worsening disability associated with chronic pain is that it negatively impacts one's ability to maintain gainful employment. Chronic pain has consistently been found to lead to higher rates of unemployment, which has a ripple on effect leading to downward socioeconomic drift (i.e., lower SES over time) and heightened associated stressors. Data collected within a primary care cohort study found that 13% of individuals with headache pain and 18% of individuals with back pain were unable to obtain or keep full-time employment over a 3-year period due to their pain (Stang, Von Korff, & Galer, 1998). Estimates from an Australian-based survey found that approximately 13% of those individuals who reported a chronic pain condition were unemployed, and over 17% were receiving disability benefit compensation (Blyth et al., 2001). Further, in this Australian-based study, higher levels of pain interference within those reporting chronic pain was associated with worse socioeconomic, health, and employment indicators (Blyth et al., 2001). Many people also change jobs due to pain (Magni, Caldieron, Rigatti-Luchini, & Merskey, 1990), which, depending on the type of change, may actually be beneficial. In a review of the literature, Teasell and Bombardier found that there is evidence to suggest that the availability of modified work or work autonomy is associated with less disability in people with chronic pain (Teasell & Bombardier, 2001). Thus, finding ways to enhance vocational rehabilitation in the context of chronic pain is critical.

Another co-occurring problem that often arises for individuals living with pain is that the pain (and its consequences) places a strain on relationships. Many individuals with chronic pain report pain to be an isolating experience and often feel their experience is not understood by family and friends. The WHO (2004) reported that one in four individuals living with chronic pain identify that relationships with a significant other, family, or friends are strained or broken due to the pain. Although the exact reasons for these deteriorations in relationships probably vary from person to person, it is likely that changes in what a person can or can't do any longer (i.e., possibly a companionable hike may have been a valued shared activity prior to the onset of pain

and the loss of being able to do this together due to the pain may lead to strain), financial stress, and/or potentially comorbid depression and anger may play a role. Research has found that higher rates of outward expressions of anger and hostility directed toward a spouse may detrimentally impact the spouse's mood and over time, lead to more punishing, critical spousal responses to the individual living with chronic pain (Burns, Johnson, Mahoney, Devine, & Pawl, 1996). Moreover, a recent study implementing ecological momentary assessment to examine concurrent and lagged effects found that patient-perceived spouse criticism and hostility predicted increased patient pain intensity (Burns et al., 2013). Interestingly in this study, it was found that spouse observation of patient pain behaviors may be a precursor to the criticism and hostility perceived by the patient (Burns et al., 2013). Based on these findings, some pain treatment approaches, as we will cover in the next chapter, have included client partners within the therapy, with promising results emerging as a consequence (Keefe, Blumenthal, et al., 2014).

Summary

Pain in different contexts can entail a range of responses; consider, for example, the pain of child birth as compared to the pain of a compound fracture—contextual differences have a substantial influence on the experience. Contemporary models of chronic pain are firmly rooted within a biopsychosocial perspective, recognizing the critical synergistic role of biological, psychological, emotional, social, and behavioral contextual factors. However despite this, even today, the biomedical model is the most dominant model *implemented* in healthcare and in the treatment of chronic pain, one only has to look at the statistics on the number of medical visits, surgical procedures, and opioid prescriptions to see this is the case. Healthcare providers who are exposed to the biomedical model in training, but receive little in the way of chronic pain curriculum, still approach the problem of chronic pain via focusing on assessment to find the "physical" peripheral pain generator, and then establish treatment to remove this "cause" (Thorn & Walker, 2011). And in conditions where it is not possible to remove the cause (i.e., as in the case of arthritis and many other chronic pain conditions), palliative approaches are implemented in the form of analgesic pain medication. However, in chronic pain conditions, often a specific pain generator cannot be identified, and palliative approaches to management often entail serious adverse side effects (Trescot et al., 2008). Thus, while the biomedical model may work well for acute pain, in the instance of chronic pain, this approach reinforces a passive patient role, as the individual searches for a "cure" (that likely may not exist). For treatment to be effective, the *person* experiencing the pain must be considered in a completely holistic sense—including the neurophysiological aspect, as well as emotions, behavior, cognitions, and context. Effective chronic pain treatments target not simply "the pain" as an unwanted, separate, yet often defining part of self, but a radical shift in perspective toward living a valued life, with pain and all.

References

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health, the challenge of the gradient. *American Psychologist*, *49*(1), 15–24.

Almeida, D. M., Neupert, S. D., Banks, S. R., & Serido, J. (2005). Do daily stress processes account for socioeconomic health disparities? *The Journals of Gerontology*, *60*, 34–39.

Altmaier, E. M., Russell, D. W., Kao, C. F., Lehmann, T. R., & Weinstein, J. N. (1993). Role of self-efficacy in rehabilitation outcome among chronic low back pain patients. *Journal of Counseling Psychology*, *40*(3), 335–339.

Apkarian, A. V., Baliki, M. N., & Farmer, M. A. (2013). Predicting transition to chronic pain. *Current Opinions in Neurology*, *26*(4), 360–367.

Apkarian, A. V., Baliki, M. N., & Geha, P. Y. (2009). Towards a theory of chronic pain. *Progress in Neurobiology*, *87*(2), 81–97.

Apkarian, A. V., Hashmi, J. A., & Baliki, M. N. (2011). Pain and the brain: Specificity and plasticity of the brain in clinical chronic pain. *Pain*, *152*(3 Suppl), S49–S64.

Apkarian, A. V., Sosa, Y., Sonty, S., Levy, R. M., Harden, R. N., Parrish, T. B., & Gitelman, D. R. (2004). Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *The Journal of Neuroscience 24*(46), 10410–10415.

Ashburn, M. A., & Staats, P. S. (1999). Management of chronic pain. *The Lancet*, *353*(9167), 1865–1869.

Asmundson, G. J., Norton, P., & Vlaeyen, J. W. (2004). Fear-avoidance models of chronic pain: An overview. In G. J. Asmundson, J. W. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 3–24). Oxford: Oxford University Press.

Baliki, M. N., Geha, P. Y., Apkarian, A. V., & Chialvo, D. R. (2008). Beyond feeling: Chronic pain hurts the brain, disrupting the default-mode network dynamics. *Journal of Neuroscience*, *28*, 1398–1403.

Baliki, M. N., Geha, P. Y., Fields, H. L., & Apkarian, A. V. (2010). Predicting value of pain and analgesia: Nucleus accumbens response to noxious stimuli changes in the presence of chronic pain. *Neuron*, *66*, 149–160.

Banks, S. M., & Kerns, R. D. (1996). Explaining high rates of depression in chronic pain: A diathesis-stress framework. *Psychological Bulletin*, *119*, 95–110.

Bantick, S. J., Wise, R. G., Ploghaus, A., Clare, S., Smith, A. M., & Tracey, I. (2002). Imaging how attention modulates pain in humans using functional MRI. *Brain*, *125*, 310–319.

Beecher, H. K. (1946). Pain in men wounded in battle. *Annals of Surgery*, *123*(1), 96–105.

Bergman, S., Herrstrom, P., Hogstrom, K., Petersson, I. F., Svensson, B., & Jacobsson, L. T. (2001). Chronic musculoskeletal pain, prevalence rates, and sociodemographic associations in a Swedish population study. *Journal of Rheumatology*, *28*(6), 1369–1377.

Blyth, F. M., March, L. M., Brnabic, A. J. M., Jorm, L. R., Williamson, M., & Cousins, M. J. (2001). Chronic pain in Australia: A prevalence study. *Pain*, *89*, 127–134.

Bracha, H. S. (2004). Freeze, flight, fight, fright, faint: Adaptationist perspectives on the acute stress response spectrum. *CNS Spectrums*, *9*(9), 679–685.

Breitbart, W., McDonald, M., Rosenfeld, B., Passik, S., Hewitt, D., Thaler, H., & Portenoy, R. (1996). Pain in ambulatory AIDS patients. *Pain*, *68*, 315–321.

Breivik, H., Collett, B., Ventafridda, V., Cohen, R., & Gallacher, D. (2006). Survey of chronic pain in Europe: Prevalence, impact on daily life, and treatment. *European Journal of Pain*, *10*(4), 287–333.

Brodal, P. (2010). *The central nervous system: Structure and function* (4th ed.). New York, NY: Oxford University Press.

Burns, J., Higdon, L., Mullen, J., Lansky, D., & Mei Wei, J. (1999). Relationships among patient hostility, anger expression, depression, and the working alliance in a work hardening program. *Annals of Behavioral Medicine*, *21*, 77–82.

Burns, J. W., Johnson, B. J., Mahoney, N., Devine, J., & Pawl, R. (1996). Anger management style, hostility and spouse responses: Gender differences in predictors of adjustment among chronic pain patients. *Pain*, *64*, 445–453.

Burns, J., Peterson, K. M., Smith, D. A., Keefe, F., Porter, L. S., Schuster, E., & Kinner, E. (2013). Temporal associations between spouse criticism/hostility and pain among patients with chronic pain: A within-couple daily diary study. *Pain*, *154*, 2715–2721.

Byers, H. D., Lichstein, K. L., & Thorn, B. E. (2015). Cognitive processes in comorbid poor sleep and chronic pain. *Journal of Behavioral Medicine*, *39*, 233–240.

Carr, D. B., & Goudas, L. C. (1999). Acute pain. *Lancet*, *353*, 2051–2058.

Catala, E., Reig, E., Artes, M., Aliaga, L., Lopez, J. S., & Segu, J. L. (2002). Prevalence of pain in the Spanish population: Telephone survey in 5000 homes. *European Journal of Pain*, *6*, 133–140.

Chibnall, J. T., Tait, R. C., Andreson, E. M., & Hadler, N. M. (2005). Race and socioeconomic differences in post-settlement outcomes for African American and Caucasian workers' compensation claimants with low back injuries. *Pain*, *114*, 462–472.

Crombez, G., Van Ryckeghem, D. M. L., Eccleston, C., & van Damme, S. (2013). Attentional bias to pain-related information: A meta-analysis. *Pain*, *154*, 497–510.

Crombez, G., Vlaeyen, J. W. S., & Heuts, P. (1999). Pain related fear is more disabling that pain itself: Evidence on the role of pain-related fear in chronic back pain disability. *Pain*, *80*, 329–339.

Darnall, B. D., Scheman, J., Davin, S., Burns, J. W., Murphy, J. L., Wilson, A. C., … Mackey, S. C. (2016). Pain psychology: A global needs assessment and national call to action. *Pain Medicine*, *17*, 250–263.

Davis, M. C., Thummala, K., & Zautra, A. J. (2014). Stress-related clinical pain and mood in women with chronic pain: Moderating effects of depression and positive mood induction. *Annals of Behavioral Medicine*, *48*, 61–70.

Day, M. A. (2017). Pain and its optimal management. In J. Dorrian, E. Thorsteinsson, M. Di Benedetto, K. Lane-Krebs, M. Day, A. Hutchinson, K. Sherman., *Health Psychology in Australia* (pp. 261–281). Port Melbourne: Cambridge University Press.

Day, M. A., Eyer, J., & Thorn, B.E. (2013). Therapeutic relaxation. In S. G. Hofmann (Ed.), *The Wiley Handbook of Cognitive Behavioral Therapy: A Complete Reference Guide. Volume 1: CBT General Strategies* (Vol. *1*, pp. 157–180). Oxford: Wiley-Blackwell.

Day, M. A., & Thorn, B. E. (2010). The relationship of demographic and psychosocial variables to pain-related outcomes in a rural chronic pain population. *Pain*, *151*(2), 467–474.

Dersh, J., Gatchel, R. J., Mayer, T. G., Polatin, P. B., & Temple, O. W. (2006). Prevalence of psychiatric disorders in patients in patients with chronic disabling occupational spinal disorders. *Spine*, *31*, 1156–1162.

Drahovzal, D., Stewart, S., & Sullivan, M. (2006). Tendency to catastrophize somatic sensations: Pain catastrophizing and anxiety sensitivity in predicting headache. *Cognitive Behaviour Therapy*, *35*(4), 226–235.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: A cognitive-affective model of the interruptive function of pain. *Psychological Bulletin*, *125*(3), 356–366.

Edwards, R. R., Cahalan, C., Mensing, G., Smith, M., & Haythornthwaite, J. A. (2011). Pain, catastrophizing, and depression in the rheumatic diseases. *Nature Reviews Rheumatology*, *7*(4), 216–224.

Elliott, A. M., Smith, B. H., Penny, K. I., Smith, W. C., & Chambers, W.A. (1999). The epidemiology of chronic pain in the community. *Lancet*, *354*, 1248–1252.

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science*, *196*(4286), 129–136.

Eriksen, J., Jensen, M. K., Sjøgren, P., Ekholm, O., & Rasmussen, N. K. (2003). Epidemiology of chronic non-malignant pain in Denmark. *Pain*, *106*, 221–228.

Flor, H., Behle, D. J., & Birbaumer, N. (1993). Assessment of pain-related cognitions in chronic pain patients. *Behaviour Research and Therapy*, *31*(1), 63–73.

Fordyce, W. E. (1976). *Behavioral methods for chronic pain and illness*. St. Louis, MO: Mosby.

Fornito, A., Harrison, B. J., Zalesky, A., & Simons, J. S. (2012). Competitive and cooperative dynamics of large-scale brain functional networks supporting recollection. *Proceedings of the National Academy of Sciences USA*, *109*(31), 12788–12793.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology. The broaden-and-build theory of positive emotions. *American Psychologist*, *56*(3), 218–226.

Gaskin, D. J., & Richard, P. (2012). The economic costs of pain in the United States. *The Journal of Pain*, *13*(8), 715–724.

Gatchel, R. J. (2004). Comorbidity of chronic pain and mental health: The biopsychosocial perspective. *American Psychologist*, *59*, 792–794.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, *133*(4), 581–624.

Geha, P. Y., Baliki, M. N., Harden, R. N., Bauer, W. R., Parrish, T. B., & Apkarian, A. V. (2008). The brain in chronic CRPS pain: Abnormal gray-white matter interactions in emotional and autonomic regions. *Neuron*, *60*, 570–581.

Geisser, M. E., Robinson, M. E., Keefe, F. J., & Weiner, M. L. (1994). Catastrophizing, depression and the sensory, affective and evaluative aspects of chronic pain. *Pain*, *59*(1), 79–83.

Giardino, N. D., Jensen, M., Turner, J. A., Ehde, D. M., & Cardenas, D. C. (2003). Social enviornment moderates the association between catastrophizing and pain among persons with spinal cord injury. *Pain*, *106*, 19–25.

Greenwood, K. A., Thurston, R., Rumble, M., Waters, S. J., & Keefe, F. J. (2003). Anger and persistent pain: Current status and future directions. *Pain*, *103*, 1–5.

Gwilym, S. E., Fillipini, N., Douaud, G., Carr, A. J., & Tracey, I. (2010). Thalamic atrophy associated with painful osteoarthritis of the hip is reversible after arthroplasty; a longitudinal voxel-based-morphometric study. *Arthritis and Rheumatology*, *62*, 2930–2940.

Haig, R. A. (1988). *The anatomy of humor*. Springfield, IL: Charles C. Thomas.

Hann, K. E. J., & McCracken, L. M. (2014). A systematic review of randomized controlled trials of acceptance and commitment therapy for adults with chronic pain: Outcome domains, design quality, and efficacy. *Journal of Contextual Behavioral Science*, *3*(4), 217–227.

Harstall, C., & Ospina, M. (2003). How prevalent is chronic pain? *Pain: Clinical Updates*, *11*, 1–4.

Hart, L. G., Deyo, R. A., & Cherkin, D. C. (1995). Physician office visits for low back pain. *Spine*, *20*, 11–19.

Hoffman, P., Meier, B., & Council, J. R. (2002). A comparison of chronic pain between an urban and rural population. *Journal of Community Health Nursing*, *19*, 213–224.

Holtzman, S., & Delongis, A. (2007). One day at a time: The impact of daily satisfaction with spouse responses on pain, negative affect and catastrophizing among individuals with rheumatoid arthritis. *Pain*, *131*, 202–213.

IASP Subcommittee on Taxonomy, C. o. c. p. (1986). Descriptions of chronic pain syndromes and definitions of pain terms. *Pain*, *3*, S1–S226.

IASP Taxonomy, I. T. F. o. (1994). *Classification of chronic pain*, 2nd ed. Seattle, WA: IASP Press.

IOM. (2011). *Relieving pain in America: A blueprint for transforming prevention, care, education, and research*. Washington, DC: The National Academics Press.

Jensen, M. C., Brantzawadzki, M. N., Obuchowski, N., Modic, M. T., Malkasian, D., & Ross, J. S. (1994). Magnetic-resonance-imaging of the lumbar spine in people without back pain. *New England Journal of Medicine*, *331*(2), 69–73.

Jensen, M., Turner, J. A., & Romano, J. M. (2001). Changes in beliefs, catastrophizing and coping are associated with improvement in multidisciplinary pain treatment. *Journal of Consulting and Clinical Psychology*, *69*, 655–662.

Jensen, M. P. (2010). A neuropsychological model of pain: Research and clinical implications. *Journal of Pain*, *11*, 2–12.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain and illness*. New York, NY: Delacourt.

Keefe, F. J., Blumenthal, J., Baucom, D., Affleck, G., Waugh, R., Caldwell, D. S., … Lefebvre, J. (2004). Effects of spouse-assisted coping skills training and exercise training in patients with osteoarthritic knee pain: A randomized controlled study. *Pain*, *110*(3), 539–549.

Keefe, F., Caldwell, D. S., Queen, K. T., Gil, K. M., Martinez, S., Crisson, J. E., … Nunley, J. (1987). Pain coping strategies in osteoarthritis patients. *Journal of Consulting and Clinical Psychology*, *55*, 208–212.

Keefe, F. J., Rumble, M. E., Scipio, C. D., Giordano, L. A., & Perri, L. M. (2004). Psychological aspects of persistent pain: Current state of the science. *Journal of Pain*, *5*(4), 195–211.

Khalid, I., Roehrs, T. A., Hudgel, D. W., & Roth, T. (2011). Continuous positive airway pressure in severe obstructive sleep apnea reduces pain sensitivity. *Sleep*, *34*(12), 1687–1691.

Kim, J. H., Suh, S. I., Seol, H. Y., Oh, K., Seo, W. K., Yu, S. W., … Koh, S. B. (2008). Regional grey matter changes in patients with migraine: A voxel-based morphometry study. *Cephalagia*, *28*, 598–604.

Kington, R. S., & Smith, J. P. (1997). Socioeconomic status and racial and ethnic differences in functional status associated with chronic diseases. *American Journal of Public Health*, *87*(5), 805–810.

Kuchinad, A., Schweinhardt, P., Seminowicz, D. A., Wood, P. B., Chizh, B. A., & Bushnell, M. C. (2007). Accelerated brain gray matter loss in fibromyalgia patients: premature aging of the brain? *The Journal of Neuroscience 27*(15), 4004–4007.

Lambert, M. J. (1992). Implications of outcome research for psychotherapy integration. In N. J. C. & M. R. Goldstein (Eds.), *Handbook of psychotherapy integration* (pp. 94–129). New York, NY: Basic Books.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York, NY: Springer.

Luerding, R., Weigand, T., Bogdahn, U., & Schmidt-Wilcke, T. (2008). Working memory performance is correlated with local brain morphology in the medial frontal and anterior cingulate cortex in fibromyalgia patients: Structural correlates of pain-cognition interaction. *Brain*, *131*, 3222–3231.

Magni, G., Caldieron, C., Rigatti-Luchini, S., & Merskey, H. (1990). Chronic musculoskeletal pain and depressive symptoms in the general population. An analysis of the 1st National Health and Nutrition Examination Survey data. *Pain*, *43*, 299–307.

Mahony, D. L., Burroughs, D. L., & Hieatt, A. C. (2001). The effects of laughter on discomfort thresholds: Does expectation become reality? *Journal of General Psychology*, *128*(2), 217–226.

McCracken, L. M., & Eccleston, C. (2003). Coping or acceptance: What to do about chronic pain? *Pain*, *105*, 197–204.

McCracken, L. M., Matthews, A. K., Tang, T. S., & Cuba, S. L. (2001). A comparison of blacks and whites seeking treatment for chronic pain. *The Clinical Journal of Pain*, *17*, 249–255.

McCracken, L. M., & Samuel, V. M. (2007). The role of avoidance, pacing, and other activity patterns in chronic pain. *Pain*, *130*, 119–125.

McCracken, L. M., & Vowles, K. E. (2006). Acceptance of chronic pain. *Current Pain and Headache Reports*, *10*(2), 90–94.

McCracken, L. M., Vowles, K. E., & Eccleston, C. (2004). The Chronic Pain Acceptance Questionnaire. *Pain*, *107*(1), 271–277.

McCracken, L. M., Vowles, K. E., & Zhao-O'Brien, J. (2010). Further development of an instrument to assess psychological flexibility in people with chronic pain. *Journal of Behavioural Medicine*, *33*, 346–354.

McEwan, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, *87*(3), 873–904.

McIlvane, J. M. (2007). Disentangling the effects of race and SES on arthritis-related symptoms, coping, and well-being in African American and White women. *Aging and Mental Health*, *11*(5), 556–569.

Melzack, R. (2001). Pain and the neuromatrix in the brain. *Journal of Dental Education*, *65*, 1378–1382.

Melzack, R. (2005). Evolution of the neuromatrix theory of pain. The Prithvi Raj Lecture: Presented at the third World Congress of World Institute of Pain, Barcelona 2004. *Pain Practice*, *5*, 85–94.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science*, *150*(699), 971–979.

Melzack, R., & Wall, P. D. (1982). *The challenge of pain*. New York, NY: Basic Books.

Moulin, D. E., Clark, A. J., Speechley, M., & Morley-Forster, P. K. (2002). Chronic pain in Canada-prevalence, treatment, impact and the role of opioid analgesia. *Pain Research and Management*, *7*, 179–184.

National Centers for Health Statistics. (2013). Health, United States, with special features on prescription drugs. 37th. Retrieved from http://www.cdc.gov/nchs/data/hus/hus13.pdf

Neville, A., Peleg, R., Singer, Y., Sherf, M., & Shvartzman, P. (2008). Chronic pain: A population-based study. *Israel Medical Association Journal*, *10*, 676–680.

Ng, K. F., Tsui, S. L., & Chan, W. S. (2002). Prevalence of common chronic pain in Hong Kong adults. *The Clinical Journal of Pain*, *18*, 275–281.

Nguyen, M., Ugarte, C., Fuller, I., Haas, G., & Portnenoy, R. (2005). Access to care for chronic pain: Racial and ethnic differences. *The Journal of Pain*, *6*, 301–314.

NIH. (2011). *Interagency pain research coordinating committee: National Pain Strategy*. Retrieved from https://iprcc.nih.gov/docs/DraftHHSNationalPainStrategy.pdf

Obermann, M., Nebel, K., Schumann, C., Holle, D., Gizewski, E. R., Maschke, M., … Katsarava, Z. (2009). Grey matter changes related to chronic posttraumatic headache. *Neurology*, *73*, 978–983.

O'Brien, E. M., Waxenberg, L. B., Atchison, J. W., Gremillion, H. A., Staud, R. M., McCrae, C. S., & Robinson, M. E. (2010). Negative mood mediates the effect of poor sleep on pain among chronic pain patients. *Clinical Journal of Pain*, *26*(4), 310–319.

Ohayon, M. M. (2005). Relationship between chronic painful physical condition and insomnia. *Journal of Psychiatric Research*, *39*, 151–159.

Okifuji, A., Turk, D. C., & Curran, S. L. (1999). Anger in chronic pain: Investigations of anger targets and intensity. *Journal of Psychosomatic Research*, *47*, 1–12.

Ossipov, M. H., Dussor, G. O., & Porreca, F. (2010). Central modulation of pain. *The Journal of Clinical Investigation*, *120*(11), 3779–3787.

Palmer, C. A., & Alfano, C. A. (2016). Sleep and emotion regulation: An organizing, integrative review. *Sleep Medicine Reviews* DOI: 10.1016/j.smrv.2015.12.006

Pfingsten, M., Kroner-Herwig, B., Leibing, E., Kronshage, U., & Hildebrandt, J. (2000). Validation of the German version of the Fear-Avoidance Beliefs Questionnaire (FABQ). *European Journal of Pain*, *4*, 259–266.

Poiraudeau, S., Rannou, F., Baron, G., Le Henanff, A., Coudeyre, E., Rozenberg, S., … Ravaud, P. (2006). Fear-avoidance beliefs about back pain in patients with subacute low back pain. *Pain*, *124*, 305–311.

Portenoy, R., Ugarte, C., & Fuller, I. (2004). Population-based survey of pain in the United States: Differences among White, African American, and Hispanic subjects. *The Journal of Pain*, *5*, 317–328.

Prkachin, K. M., Schultz, I. Z., & Hughes, E. (2007). Pain behavior and the development of pain-related disability: The importance of guarding. *Clinical Journal of Pain*, *23*, 270–277.

Purves, D., Augustine, G. J., Fitzpatrick, D., Katz, L. C., LaMantia, A., McNamara, J. O., & Williams, S. M. (2001). *Neuroscience* (2nd ed.). Sunderland, MA: Sinauer Associates.

Reynolds, D. V. (1969). Surgery in the rat during electrical analgesia induced by focal brain stimulation. *Science*, *164*(13), 1099–1109.

Rodriguez-Raecke, R., Niemeier, A., Ihle, K., Ruether, W., & May, A. (2009). Brain grey matter decrease in chronic pain is the consequence and not the cause of pain. *Journal of Neuroscience*, *29*, 13746–13750.

Romano, J., & Turner, J. A. (1985). Chronic pain and depression: Does the evidence support a relationship? *Psychological Bulletin*, *97*, 18–34.

Rudy, T. E., Lieber, S. J., Boston, J. R., Gourley, L. M., & Baysal, E. (2003). Psychosocial predictors of physical performance in disabled individuals with chronic pain. *Clinical Journal of Pain*, *19*, 18–30.

Schmidt-Wilcke, T., Leinisch, E., Ganssbauer, S., Draganski, B., Bogdahn, U., Altmeppen, J., & May, A. (2006). Affective components and intensity of pain correlate with structural differences in gray matter in chronic back pain patients. *Pain*, *125*(1–2), 89–97.

Schnitzler, A., & Ploner, M. (2000). Neurophysiology and functional neuroanatomy of pain perception. *Journal of Clinical Neurophysiology*, *17*(6), 592–603.

Schrimpf, M., Liegl, G., Boeckle, M., Leitner, A., Geisler, P., & Pieh, C. (2015). The effect of sleep deprivation on pain perception in health subjects: A meta-analysis. *Sleep Medicine*, *16*, 1313–1320.

Schutze, R., Rees, C., Preece, M., & Schutze, M. (2010). Low mindfulness predicts pain catastrophizing in a fear-avoidance model of chronic pain. *Pain*, *148*, 120–127.

Schwartz, B. S., Stewart, W. F., Simon, D., & Lipton, R. B. (1998). Epidemiology of tension-type headache. *Journal of the American Medical Association*, *279*(5), 381–383.

Selim, A. J., Fincke, G., Ren, X. S., Deyo, R. A., Lee, A., Skinner, K., & Kazis, L. (2001). Racial differences in the use of lumbar spine radiographs: Results from the Veterans Health Study. *Spine (Phila Pa 1976)*, *26*(12), 1364–1369.

Selye, H. (1956). *The stress of life*. New York, NY: McGraw-Hill.

Selye, H. (1973). The evolution of the stress concept. *American Scientist*, *61*, 692–699.

Shmuely, Y., Baumgarten, M., Rovner, B., & Berlin, J. (2001). Predictors of improvement in health-related quality of life among elderly patients with depression. *International Pyschogeriatrics*, *13*, 63–73.

Sjøgren, P., Ekholm, O., Peuckmann, V., & Grønbæk, M. (2009). Epidemiology of chronic pain in Denmark: An update. *European Journal of Pain*, *13*, 287–292.

Smith, M. T., & Haythornthwaite, J. A. (2004). How do sleep disturbance and chronic pain inter-relate? Insights from the longitudinal and cognitive-behavioral clinical trials literature. *Sleep Medicine Reviews*, *8*(2), 119–132.

Smith, M. T., Perlis, M. L., Smith, M. S., Giles, D. E., & Carmody, T. P. (2000). Sleep quality and presleep arousal in chronic pain. *Journal of Behavioral Medicine*, *23*(1), 1–13.

Stang, P., Von Korff, M., & Galer, B. S. (1998). Reduced labor force participation among primary care patients with headache. *Journal of General Internal Medicine*, *13*, 296–302.

Strand, E. B., Zautra, A. J., Thoresen, M., Ødegard, S., Uhlig, T., & Finset, A. (2006). Positive affect as a factor of resilience in the pain-negative affect relationship in patients with rheumatoid arthritis. *Journal of Psychosomatic Research*, *60*, 477–484.

Sturgeon, J. A. (2014). Psychological therapies for the management of chronic pain. *Psychology Research and Behavior Management*, *7*, 115–124.

Sullivan, M. J. L. (2012). The communal coping model of pain catastrophizing: Clinical and research implications. *Canadian Psychology*, *53*(1), 32–41.

Sullivan, M. J. L., Adams, H., Sullivan, M. E. (2004). Communicative dimensions of pain catastrophizing: Social cueing effects on pain behaviour and coping. *Pain*, *107*, 220–226.

Sullivan, M. J., Rodgers, W. M., & Kirsch, I. (2001). Catastrophizing, depression and expectancies for pain and emotional distress. *Pain*, *91*(1–2), 147–154.

Sullivan, M. J., Thorn, B., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain*, *17*(1), 52–64.

Tait, R. C., & Chibnall, J. T. (2005). Racial and ethnic disparities in the evaluation and treatment of pain: psychological perspectives. *Professional Psychology: Research and Practice*, *36*(6), 595–601.

Takeyachi, Y., Konno, S., Otani, K., Yamauchi, K., Takahashi, I., Suzukamo, Y., & Kikuchi, S. (2003). Correlation of low back pain with functional status, general health perception, social participation, subjective happiness and patient satisfaction. *Spine*, *28*(13), 1461–1467.

Tan, G., Jensen, M. P., Robinson-Whelen, S., Thornby, J. I., & Monga, T. (2002). Measuring control appraisals in chronic pain. *Journal of Pain*, *3*(5), 385–393.

Tang, N. K. Y., & Crane, C. (2006). Suicidality in chronic pain: A review of the prevalence, risk factors and psychological links. *Psychological Medicine*, *36*(5), 575–586.

Tang, N. K., Wright, K. J., & Salkovskis, P. M. (2007). Prevalence and correlates of clinical insomnia co-occurring with chronic back pain. *Journal of Sleep Research*, *16*, 85–95.

Taylor, D. J., Mallory, L. J., Lichstein, K. L., Durrence, H. H., Riedel, B. W., & Bush, A. J. (2007). Comorbidity of chronic insomnia with medical problems. *Sleep*, *30*, 213–218.

Teasell, R. W., & Bombardier, C. (2001). Employment-related factors in chronic pain and chronic pain disability. *Clinical Journal of Pain*, *17*(4), S39–S45.

The American Academy of Pain Medicine. (2016). AAPM facts and figures on pain. Retrieved from http://www.painmed.org/PatientCenter/Facts_on_Pain.aspx

Thibault, P., Loisel, P., Durand, M. J., & Sullivan, M. J. L. (2008). Psychological predictors of pain expression and activity intolerance in chronic pain patients. *Pain*, *139*, 47–54.

Thompson, M., & McCracken, L. M. (2011). Acceptance and related processes in adjustment to chronic pain. *Current Pain and Headache Reports*, *15*(2), 144–151.

Thorn, B. E. (2004). *Cognitive therapy for chronic pain: A step-by-step guide*. New York, NY: Guilford Press.

Thorn, B. E., & Walker, B. B. (2011). Chronic pain: Closing the gap between evidence and practice. In H. S. Friedman (Ed.), *The Oxford handbook of health psychology*. New York, NY: Oxford University Press, 375–393.

Trescot, A. M., Glaser, S. E., Hansen, H., Benyamin, R., Patel, S., & Manchikanti, L. (2008). Effectiveness of opioids in the treatment of chronic non-cancer pain. *Pain Physician*, *11*(2 Suppl), S181–S200.

Trost, Z., Vangronsveld, K., Linton, S. J., Quartana, P. J., & Sullivan, M. J. L. (2012). Cognitive dimensions of anger in chronic pain. *Pain*, *153*, 515–517.

Tse, M., Lo, A., Cheng, T., Chan, E., Chan, A., & Chung, H. (2010). Humor therapy: Relieving chronic pain and enhancing happiness for older adults. *Journal of Aging Research*, *2010*, 1–9.

Tsui, P., Day, M. A., Thorn, B. E., Rubin, N., Alexander, C., & Jones, R. (2012). The communal coping model of catastrophizing: Patient-health provider interactions. *Pain Medicine*, *13*, 66–79.

Unruh, A. M. (1996). Gender variations in clinical pain experience. *Pain*, *65*, 123–167.

Unruh, A. M., & Richie, J. A. (1998). Development of the pain appraisal inventory: Psychometric properties. *Pain*, *65*, 105–110.

Verhaak, P. F. M., Kerssens, J. J., Dekker, J., Sorbi, M. J., & Bensing, J. M. (1998). Prevalence of chronic benign pain disorder among adults: A review of the literature. *Pain*, *77*, 231–239.

Vlaeyen, J. W., Kole-Snijders, A. M., Boeren, R. G., & van Eek, H. (1995). Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain*, *62*, 363–372.

Vlaeyen, J. W. S., Kole-Snijders, A., Rotteveel, A., Ruesink, R., & Heuts, P. (1995). The role of fear of movement/(re)injury in pain disability. *Journal of Occupational Rehabilitation*, *5*, 235–252.

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art review. *Pain*, *85*, 317–332.

Waddell, G., Newton, M., Henderson, I., Somerville, D., & Main, C. J. (1993). A Fear Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*, *52*, 157–168.

Walker, J., Holloway, I., & Sofaer, B. (1999). In the system: The lived experience of chronic back pain from the perspectives of those seeking help from pain clinics. *Pain*, *80*, 621–628.

Williams, D. R., & Collins, C. (1995). US socioeconomic and racial differences in health: Patterns and explanations. *Annual Review Sociology*, *21*, 349–386.

Wilson, K. G., Eriksson, M. Y., D'Eon, J. L., Mikail, S. F., & Emery, P. C. (2002). Major depression and insomnia in chronic pain. *Clinical Journal of Pain*, *18*, 77–83.

Wolfe, F., Smythe, H. A., Yunus, M. B., Bennett, R. M., Bombardier, C., Goldenberg, D. L. … Sheon, R. P. (1990). The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis and Rheumatology*, *33*, 160–172.

Woolf, C. J. (2011). Central sensitization: Implications for the diagnosis and treatment of pain. *Pain*, *152*(3), S2–15.

Woolf, C. J., & Salter, M. W. (2000). Neuronal plasticity: Increasing the gain in pain. *Science*, *288*, 1765–1768.

World Health Organization (2004). World Health Organization supports global effort to relieve chronic pain. Retrieved from http://www.who.int/mediacentre/news/releases/2004/pr70/en/

Zautra, A. J., Smith, B., Affleck, G., & Tennen, H. (2001). Examinations of chronic pain and affect relationships: Applications of a dynamic model of affect. *Journal of Consulting and Clinical Psychology*, *69*, 786–795.