Chapter 11: Psychological models of pain

This chapter describes the definition and understanding of pain, for which some neurophysiological background is provided; the predominant psychotherapeutic models, their integration and the evidence base; and a brief note on philosophical contribution to psychological model development**.** It introduces important models that are further developed in chapters within this section, and refers to several chapters in other sections that draw, implicitly or explicitly, on these psychological models.

Definition of pain

"An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." (IASP Task Force on Taxonomy 1994).

This definition of pain is discussed in much more detail in chapter 33, but remains an important starting point for any consideration of the psychology of pain. It represented a significant advance on earlier definitions that referred primarily to sensation or perception (Auvray et al. 2010), and only to emotional components, if at all, as part of a response. Here emotion is identified as integral to the experience, as envisaged in the paradigm-changing gate control model of pain (Melzack and Wall 1965), described below in **Neurophysiology of pain**.

A note accompanying the definition gave equal importance to sensory and emotional components; acknowledged the unreliable relationship between pain and tissue damage; and recognized pain experience in those who lack language, human and non-human. But cognitive, social, and behavioral dimensions are missing, despite evidence for them in various animals (Langford et al. 2010; Low 2013; Mogil 2015; Sneddon 2011); the primacy given to verbal report overshadows reliable behavioral signs (Williams 2002; Hadjistavropoulos et al. 2011); and pain in the absence of tissue damage or disease is assigned to undefined "psychological reasons". The last statement reintroduces a psychosomatic model that is neither required nor helpful: known pain processes explain the generation and modulation of pain within the nervous system without lesion or pathology. Adding an undefined psychosomatic basis for pain reintroduces dualistic processes into an integrated understanding of pain. Patrick Wall (1994), one of the authors of the gate control theory, proposed in characteristically robust terms three reasons why pain cannot be satisfactorily explained by reference to observable pathology:

> "First, I find it unwise arrogance to believe that our present techniques of diagnosis are capable of detecting all relevant forms of peripheral pathology. Second, we are now beginning to realise ... that a peripheral event may trigger long lasting changes in the spinal cord and brain ... This means that overt peripheral pathology is capable of initiating a cascade of changes which may persist in the central nervous system long after peripheral pathology has disappeared. Third, we are now beginning to discover that sensory systems are not dedicated and hard wired but are normally held in a stable state by elaborate dynamic control mechanisms. The rules of the physiology of these control mechanisms allows them to be pushed outside their normal working range in which state they will oscillate or fire continuously." (p. 4)

Many pains, from mild and momentary to severe and prolonged, originate from an identifiable cause, often external. Pain is a warning of actual or impending injury, and after injury a constant or repeated reminder to take care of the injury. These processes are essential for survival, since they maximize chances of healing and recovery (Williams 2002, Williams 20176, Walters 1994). Injuries that become infected or infested, broken bones which further disintegrate through continued use, repeated encounters with noxious plants or animals that are not remembered and avoided: all lower chances of survival. So it is reasonable to look for causes of injury when pain is felt. What is unreasonable is to deny pain when no such cause can be found, and yet such statements are ubiquitous in medical texts and teaching, design of medical services, and decisions made about patients and their treatment (Donaldson 2009). Psychiatry and psychology have speculatively filled the apparent gap in explanation with concepts such as "hysteria" or faulty pre-pain personality (Pilowsky and Spence 1975), now formulated as psychosomatic pain in clinical and empirical work, despite the dearth of theory or evidence (Williams and Johnson 2011; Crombez et al. 2009; Merskey 2009)(see chapter 13). Both in medical and in commonsense or lay understanding of pain, people with pain but no corresponding physical signs may be suspected of imagining, exaggerating, or fabricating it, and of malingering or seeking benefits without being entitled through genuine illness or disability (Eccleston et al. 1997; Patients' Association 2010; Williams 2002).

Neurophysiology of pain: the basis for psychological models

The gate control theory (Melzack and Wall 1965) proposed an integration at the first synapse in the dorsal horn of the spinal cord of fast and slow conducted input from peripheral nerve endings and input descending from the brain and representing central control over peripheral input: "The model suggests that psychological factors such as past experience, attention and

emotion influence pain response and perception by acting on the gate control system" (Melzack and Wall 1965: p978). Initially, this control was conceptualized as inhibitory but the proliferation of pain research which resulted from their model found far more excitatory than inhibitory mechanisms. These mechanisms could not only amplify pain, but changes from the dorsal horn of the spinal cord onwards could produce afferent signals in the pain system from peripheral stimulation – such as light touch, or mild warmth - not normally associated with pain experience.

Research inspired by the gate control model focused on the periphery and on processing at the spinal cord level, often using restrained animals with acute pain and evoked pain responses. It was challenging to translate to humans with chronic pain, manifest distress, and spontaneous behaviors. The development of brain imaging techniques allowed some insight into processing of pain in the brain, subject to the shortcomings of models available for interpretation (Tracey and Bushnell 2009). This established correlation between processing in particular networks and subjective pain rating (Coghill et al. 2003), and the prominence of emotional processing even of expected experimental stimuli in people with chronic pain (Baliki et al. 2006), In brief, the signature of pain in the brain (Tracey and Bushnell 2009) bore out the definition as a sensory and emotional experience, with impact on cognitive and emotional function even in non-human animals (Low 2013).

It is useful to distinguish between the immediate pain experienced when tissue is damaged, and that prompts rapid escape, from the persistence of pain during healing. An evolutionary understanding is helpful but almost entirely neglected in the field of pain (Williams 2016). Evident even in invertebrates with a fairly simple nervous system (Walters 1994), nociception is generally unmodulated and the response is often reflexive. From that point, the inflammatory response and other systemic changes increase sensitivity of the area around the injury, prompting wound care and adjustments to movement (such as guarding or limping) and reduction of usual activities to minimize disturbance during healing (Walters 1994; Woolf 2010). The wounded animal – best described in mammals – conserves resources, losing interest in eating, exploration and pursuing social and sexual contact, while resting and sleeping more (Wall 1979; Williams 2016). In normal recovery, as healing proceeds and pain recedes, the individual becomes more active and loses the hypersensitivity and the changes to movement and activity that are also behavioral signs of pain.

In chronic pain, by contrast, the behavioral changes persist, as does pain. The injury (such as from trauma or surgery), if any, has healed; there may be ongoing disease such as rheumatoid

arthritis or endometriosis but the relationship of pain to pathology is very variable; or there may be neither injury nor pathology detected but the pain starts and continues without identifiable cause. One further possibility is neuropathic pain, when nerve tissue is damaged and multiple changes within the nervous system, peripheral, central, or both, generate pain on the slightest stimulus or spontaneously (Woolf 2010). It is chronic pain in particular that is often judged in medical or lay settings to lack a biological basis, and that has provided fertile ground for psychological models of pain .

The psychological models described and discussed here are the behavioral model, the cognitive and combined cognitive-behavioral models, and third wave models (see also chapters 12 and 35). All are implicitly or explicitly compared with what is often referred to as the 'medical model', an oversimplified and concrete account of pain as described above, that recognises tissue damage or pathology as the basis of pain, and the complaint of pain in the absence of identifiable tissue damage as suspect: as discussed above, this fails to integrate the contribution of the brain to the experience of pain.

The behavioral model

The foundation of psychology in chronic pain lies in the operant behavioral model developed by Fordyce (1976; Main et al. 2014), drawing on clinical experience in one of the earliest multidisciplinary pain clinics. If there was no longer a peripheral driver of pain (on the basis of medical assessment), his model focused on behaviors that indicated pain, verbal and nonverbal, from limping, guarding, or sighing to taking analgesics, soliciting social support, and seeking medical help. Fordyce explicitly rejected assumptions of covert psychological motivation as the explanation for pain, and took a neutral and compassionate stance towards chronic pain patients. Rest, attention, medication, but also recognition of progress were commonly used reinforcers.

Fordyce emphasized the distinction between "respondent pain", where behavior was a response to onset or exacerbation of pain, often acute-on-chronic pain (that is, acute exacerbation of chronic pain), and "operant pain" where the timing and frequency of behavior in relation to its consequences appeared to confirm that those consequences controlled the behavior. A common example of respondent knee pain would be evidenced by limping when under particular strain; of operant knee pain, of limping only when sympathetic others were present. Pain behaviors were addressed because

they were accessible (Fordyce et al. 1968), not through any presumption of cause. Fordyce was careful to engage with the wider context of the patient's life, to share the model with the patient, and to involve the patient's family members, workmates or employers in behavioral change. Alongside the reduction of pain behaviors, he stressed the importance of increasing well behaviors, again using operant methods, based on the patient's choice of leisure and other activities.

In experimental settings, where the pain has little or no threat value, and social relationships are constrained by design, it is possible to manipulate some pain-related behaviors, particularly verbal ones, by systematic positive or negative reinforcement: the model predicts behaviors well. In clinical settings many other variables apply and it is difficult to isolate or observe behavioral contingencies. Despite this, and the objections of Fordyce and colleagues (Fordyce et al. 1968), behavioral explanations were adopted into psychiatric practice and often subsumed into adverse moral judgements of patients. Rewarding behavioral consequences, such as attention, sympathy, and relief from unwanted duties were assumed to govern behavior, rather than observed, and were therefore reversed in order to change the behavior. While this is not a failure of the model itself, it was clear that contingencies alone could not adequately explain behavior in chronic pain, in clinical settings or in the patients' own environments. Just as in the conceptualisation of anxiety in the broader field of psychology, behavioral theories gave way to cognitive theory.

Cognitive and cognitive-behavioral models

In contrast to the behavioral model, there is no original authoritative statement of principles or practice for the cognitive model, nor has one emerged with time. Cognitive models were (like behavioral models) extrapolated from mainstream psychology, but piecemeal and predominantly from treatment studies rather than from experimental study of normal behavior. Early models drew heavily on cognitive processes of attention control, and on treatment for stress (Turk et al. 1983); subsequently, information processing theories in mainstream psychology directed attention to beliefs and predictions (not least about behavioral choices and their consequences), and to the beliefs about pain and the ways of coping with which they were associated (Turk and Okifuji 2002).

Other than by self-report, there was little access to cognitive processes in clinical settings: tests for implicit cognitive processes used in experimental settings were impractical. Studies of people with pain focused on a variety of cognitive processes by self-report: control and

self-efficacy; coping (Turk et al. 1983; Keefe et al. 1992). In clinical settings, behavior such as avoiding use of a painful although functioning limb, or turning every conversation to the hopeless situation of having chronic pain, remained the target for clinical intervention, now indirectly by addressing cognitive content (such as beliefs about pain) and processes (such as pessimistic thinking about pain). Many treatment trials (Morley and Williams 2006) refer to a book by Turk and colleagues (1983) in which the cognitive-behavioral model is briefly summarized:

> "Behavioral change is a reflection of the intimate interrelationships among the patient's cognitive structures (schemata, beliefs), cognitive processes (automatic thoughts, internal dialogue, images), interpersonal behaviors, and resulting intrapersonal and interpersonal consequences" (p. 5).

Above all, the individual's interpretation of his or her situation and of the world around, and its implications for mood, particularly depression, underpinned the targeting in treatment of the beliefs of the person with pain. Some even used cognitive concepts to explain all behavioral phenomena (Ciccone and Grzesiak 1984). In clinical settings, the language of "distorted" cognitions, "excessive" disability was widely used, as if correct adjustment to pain could be specified and shaped by changing patients' beliefs. This was not borne out by cognitive interventions (Turner and Jensen 1993), or by experimental tests of cognitive strategies such as distraction and use of imagery (Fernandez and Turk 1989), both of which bodies of work showed at best weak effects, despite patients' accounts of frequent use of the strategies. The use of the term 'coping' for the strategies people used to try to sustain a reasonable lifestyle despite pain was generally unhelpful. The notion that strategies could in themselves be adaptive or maladaptive without reference to context was widespread, although some asked questions such as "Coping with what?" (Keefe et al. 1992).

A highly influential model that arose from the integration of cognitive and behavioral models was the "fear-avoidance" cycle of Vlaeyen and colleagues (1995; Vlaeyen and Linton 2000), most recently critically reformulated (Crombez et al. 2012) in the light of evidence and theory. The original version remains the foundation of psychological understanding of pain in pain medicine. It postulated that the individual in pain *either* had little fear and therefore confronted the pain and found that it did not prevent activity, nor did activity worsen the pain

or the original injury (if any), *or* s/he was fearful, with catastrophic interpretation of pain as implying serious damage or disease, therefore rested and avoided activity, so becoming more disabled by disuse and depression. These in turn produced more pain with less and less activity. Despite its testability, very few attempts were made to challenge or to assemble evidence for the model; its strength lay in its roots in established work on phobia and in its apparent fit with patients seen in the clinic. It was rapidly adopted as an explanatory framework, and disuse and depression, in particular, became targets of treatment in their own right, as did catastrophic thinking (appraising everything pain-related in a very negative way), consistent with cognitive targets of therapy.

As with the cognitive component of pain, there is no coherent theory of emotion, and much work originates in pragmatic clinical settings. While Vlaeyen's model focused on fear, often used as if synonymous with anxiety or catastrophizing despite some important differences, people with chronic pain often seemed depressed, although because of the prevalence of theories of psychogenic pain they were not always willing to discuss it with doctors (Eccleston et al. 1997; see chapter 13). Standard diagnostic criteria and questionnaires risked overestimating depression by attributing physical symptoms, such as sleep disturbance, to mood alone (Pincus and Williams 1999). At the same time, pessimism and hopelessness in patients undermined attempts to treat them, so important to identify. Importing pathological constructs such as phobia and major depression from mainstream mental health has, in general, been unhelpful, and efforts to build a normal psychology of pain are welcomed (Eccleston 2011).

The third wave

The "third wave" is a collective term for diverse treatments arising from what is claimed as a common philosophy, but is hard to represent as such other than their recognition as suffering as part of life rather than something that can be avoided or vanquished. The treatments may be provided together or separately. The most prominent in the field of pain and based far more in clinical settings and directed towards treatment, rather than in experimental settings exploring the nature of pain psychology, are Acceptance and Commitment Therapy (ACT: Hayes 2004) and Mindfulness (McCracken and Vowles 2014). The roots of ACT are in behavioral theory of language, recognizing the shortcomings of attempting to teach the "right" cognitive strategies and coping approaches (McCracken 2009), and building on some

elements of cognitive behavioral therapy while rejecting others, particularly implicit and explicit tenets of cognitive therapy that involve controlling thought content. In particular, unwelcome thoughts are accepted as thoughts, and recognised as possibly mistaken and therefore to be observed but not necessarily followed. The extent of difference from cognitive and behavioral therapy is disputed (Hofmann and Asmundson 2008). There is a focus on accepting pain (rather than trying to avoid it or fight it), disengaging and disinvesting in thoughts and emotions associated with pain, and acting according to personal values (McCracken 2009), the individual discovering for him or herself what works best. This may seem to require a wise homunculus who controls actions and responses, but this is rarely addressed. It is also an individualist philosophy, a contrast to some Eastern philosophies to which it refers. Although acceptance of pain emerged as a heuristic construct (Eccleston and McCracken 2003) that was relatively easy to share with patients and those around them (although sometimes mistaken for defeated resignation in the face of pain), current theory emphasizes psychological flexibility rather than acceptance (McCracken and Morley 2014). Unfortunately, psychological flexibility, defined as the capacity to persist in or change behavior in relation to thoughts and feelings, in relation to the situation, and in relation to personal goals and values (McCracken and Morley 2014), has so far resisted quantification, and its component parts are assessed by rather transparent self-report.

Mindfulness was first used in chronic pain in the 1980s (Kabat-Zinn 1982) but not again (in the research literature) until it was well-established in mainstream psychology (Teasdale et al. 2002). Contributions to its development have come from academic settings, including neuroscience (Zeidan et al. 2012), and from Buddhist philosophy and practice (Burch and Penman 2013; Carson et al. 2005). Mindfulness emphasizes being in the present, rather than in regrets over the past or anxieties about the future, and fully in the present, encompassing awareness using all senses and recognizing the many experiences other than pain. Daily meditation is for some practitioners an essential element to cultivate mindfulness, and it is encouraged in patients in mindfulness-based treatments for pain (Burch and Penman 2013).

Problems with evidence

Much of the presumed support for each of the models above consists of reports of efficacy of the practice in clinical settings in improving pain, mood, and disability. However, meta-

analyses of cognitive behavioral and behavioral therapy have yielded successively smaller effects with each update (Williams et al. 2012; Morley et al. 2013), while early meta-analyses of ACT trials look promising (Veehof et al. 2011). The apparently diminishing effects occur for a complex combination of reasons, many methodological but some related to healthcare systems in which trials are run. More and bigger compound trials are unlikely to provide a definitive estimate of efficacy (Morley et al. 2013), nor is treatment efficacy a demonstration that treatment outcome is attributable to the specific techniques of treatment rather than to the nonspecific effects of contact with therapeutic services and personnel (Wampold 2005). Assertions that one treatment type might suit one kind of patient better than another, and that each will be the best for someone, may be wishful thinking and have no theoretical basis. Attempts to type patients at entry to treatment have been superficial and unlikely to succeed (Morley and Williams 2006). On the other hand, trials of single methods of treatment with selected patients, such as behavioral exposure to feared activities (Vlaeyen et al. 2012), or mindfulness methods for modulation of pain (Zeidan et al. 2012) show impressive results of a clinically relevant magnitude, and suggest that we are not realizing the potential of psychological methods. The field seems to be in stasis, with diverse research groups pursuing patient selection, treatment refinement, or treatment delivery.

Even if we were to design a more potent intervention, difficulties remain in conceptualization and measurement of outcome. Constructs which were reasonably well-founded in the field of psychological disorders are adopted and adapted in pain, particularly in clinical settings, in ways which do disservice to the theories and to patients: the reformulation of some behavioral methods to incorporate moral values, as described above, is a strong example. Further, psychologists' weakness for believing that any quality or experience can be measured by self-report on a questionnaire that presumes a simple linear measurable underlying construct, by no means true (Michell 2009), made for a proliferation of measures which, whatever their claimed domain, often overlapped considerably in content and produced unsurprising correlations interpreted as association of the two domains, such as (lack of) confidence in activity and disability. There is little evidence that people can accurately report their thinking or emotional processes, or their behavior, and yet the assumption underpins most clinical (Morley and Williams 2006) and many experimental studies. Any face-to-face assessment, such as of pain, is a social event (Schiavenato and Craig 2010), not a 'read-out' of a stable internal state. Self-report is a behavior, and like any

behavior, may serve a function, consciously or not, for the individual in pain in that particular context (Williams 2002): self-presentation as needy, or brave, or deserving of some specific consideration or exception.

While the neuroscience and experimental psychology fields have contributed substantially to our understanding (Bushnell et al. 2013; Legrain et al. 2009), much human research is essentially trivial, involving pain inflicted briefly and unthreateningly on healthy and willing participants, volunteer or paid. This usually has little to tell us about clinical pain that is threatening, unpredictable, unwanted and unrewarded (Eccleston and Crombez 1999; Moore et al. 2013; Wiech et al. 2010). Research on people with problematic pain produces far more complex and less newsworthy results than, for instance, an assertion such as that social rejection and physical pain (both minor and in healthy volunteers) are essentially similar (Eisenberger et al. 2003). At first the area lacked any theory, but coherent frameworks have been built around findings, in this case using evolutionary understanding (Chester et al. 2012) in which all emotions trace their roots to pain (Walters 1994), and a neural systems model (chapter 15). Nevertheless, Iannetti et al. (2013) demonstrated serious weaknesses in the thesis: problems of logical error (reverse inference) and loose interpretation of neuroimaging evidence.

Philosophical observations on pain

Philosophical and religious understandings of pain are represented in clinical discourses and practices. Dualism has dogged the study and understanding of pain, and persists in language, concepts, and the therapeutic options and their delivery to people with pain (Kleinman et al. (1992, p. 10).

Philosophical discussions of pain often invoke evidence on whether non-human animals feel pain (Dawkins 2015; Rose 2002; chapter 16), or whether humans feel pain without full consciousness. It is important to note that pain is evident in behavior and brain processing of unconscious patients (Arif-Rahu and Grap 2010; Schnakers et al. 2012), and that arguments about the completeness and functioning of the nervous system were used to deny – and so fail to treat - pain in infants and in people with cognitive impairment (such as dementia), despite behavioral evidence to the contrary (Hadjistavropoulos et al. 2011; Fitzgerald and McIntosh 1989; Rodkey and Pillai Riddell 2013) (see chapter 18). Craig (1997) argues persuasively

that pain has been poorly understood in many writings on consciousness, and concepts of consciousness misleadingly used in pain studies.

Conclusions

Despite the predominance of clinical concerns in driving research on pain, treatment failure is common, and availability of psychological methods alone or integrated with other treatment fall so far short of need (Breivik et al. 2006; Von Korff 2013) that there is no possibility of adequate provision for all those whose lives are affected by chronic pain. Understanding of pain needs to change in both lay and health care fields. Psychology of pain has largely developed without adequate reference to somatic experience (Eccleston 2016), to biological underpinnings, or to the social nature of humans, and has sought solutions before defining the problems. There is not yet any truly integrated bio-psycho-social model: most which claim that title are predominantly psychological. Arguably, to integrate with the biological and the social, psychology needs to adopt a framework, such as that provided by evolutionary theory, that relates behaviors common in pain across animals to its original functions, and to possible mismatches of those functions with the current environment (Williams 2016).

References

Arif-Rahu, M., Grap. M.J. (2010) "Facial expression and pain in the critically ill non-communicative patient: State of science review," *Intensive Critical Care Nursing* 26(6) 343–352.

Auvray, M., Myin, E., Spence, C. (2010) "The sensory-discriminative and affective-motivational aspects of pain," *Neuroscience and Biobehavioral Reviews* 34 214-223.

Baliki, M.N., Chialvo, D.R., Geha, P.Y., Levy, R.M., Harden, R.N., Parrish, T.B., Apkarian, A.V. (2006) "Chronic pain and the emotional brain: specific brain activity associated with spontaneous fluctuations of intensity of chronic back pain," *Journal of Neuroscience* 26 12165–12173.

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* 10 287–333.

Burch, V., Penman, D. (2013) "*Mindfulness for health: a practical guide to relieving pain, reducing stress and restoring wellbeing*," London: Piatkus.

Bushnell, M.C., Čeko, M., Low, L.A. (2013) "Cognitive and emotional control of pain and its disruption in chronic pain," *Nature Neuroscience Reviews* 14 502-511.

Carson, J.W., Keefe, F.J., Lynch, T.R., Carson, K.M., Goli, V., Fras, A.M., Thorp, S.R. (2005) "Loving-kindness meditation for chronic low back pain: results from a pilot trial," *Journal of Holistic Nursing* 23(3) 287-304.

Chester, D.S., Pond, R.S., Richman, S.B., DeWall, C.N. (2012) "The optimal calibration hypothesis: how life history modulates the brain's social pain network," *Frontiers in Evolutionary Neuroscience* 4 article 10.

Ciccone, D.S., Grzesiak, R.C. (1984) "Cognitive dimensions of chronic pain," *Social Science and Medicine* 19 1339-1345.

Coghill, R.C., McHaffie, J.G., Yen, Y.-F. (2003) "Neural correlates of interindividual differences in the subjective experience of pain," *Proceedings of the National Academy of Sciences* 100 8538-8542.

Craig, K.D. (1997) "Implications of concepts of consciousness for understanding pain behavior and the definition of pain," *Pain Research and Management* 2(2) 111-117.

Crombez, G., Beirens, K., Van Damme, S., Eccleston, C., Fontaine, J. (2009) "The unbearable lightness of somatization: a systematic review of the concept of somatization in empirical studies of pain," *Pain* 145 31–35.

Crombez, G., Eccleston, C., Van Damme, S., Vlaeyen, J.W.S., Karoly, P. (2012) "The fear avoidance model of chronic pain: the next generation," *Clinical Journal of Pain* 28 475-483.

Dawkins, M. (2015) "Animal welfare and the paradox of animal consciousness," *Advances in the Study of Behavior* 47 5-37

Donaldson, L. (2009) *150 years of the Annual Report of the Chief Medical Officer: On the State of Public Health 2008* (pp.32–39). London: Department of Health. http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/documents/digitalasset/dh_096233.pdf (Accessed 5 October 2015).

Eccleston, C. (2011) "A normal psychology of chronic pain," *The Psychologist* 26 422-425.

Eccleston, C. (2016). “Embodied: the psychology of physical sensation”. Oxford: Oxford University Press.

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

Eccleston, C., McCracken, L.M. (2003) “Coping or acceptance: what to do about chronic pain?” *Pain* 105 197-204.

Eccleston, C., Williams, A., Stainton Rogers, W. (1997) “Patients and professionals understandings of the causes of chronic pain: blame, responsibility and identity protection,” *Social Science and Medicine* 45 699–709.

Eisenberger, N.I., Lieberman, M.D., Williams, K.D. (2003) “Does rejection hurt? An fMRI study of social exclusion,” *Science* 302 290-292.

Fernandez, E., Turk, D.C. (1989) “The utility of cognitive coping strategies for altering pain perception: a meta-analysis,” *Pain* 38 123-135.

Fitzgerald, M., McIntosh, N. (1989) “Pain and analgesia in the newborn,” *Archives of Disease in Childhood* 64 441-443.

Fordyce, W.E. (1976) “Behavioral methods for chronic pain and illness,” St Louis, Missouri: C.V. Mosby and Co.

Fordyce, W.E., Fowler, R.S., Lehmann, J.F., DeLateur, B.J. (1968) “Some implications of learning on problems of chronic pain,” *Journal of Chronic Diseases* 21 179-190.

Hadjistavropoulos, T., Breau, L. & Craig, K.D. (2011), Pain assessment in adults and children with limited ability to communicate. In D.C. Turk & R. Melzack (Eds.) *Handbook of pain assessment (3rd Edition),* New York: Guilford Press.

Hayes, S.C. (2004) “Acceptance and commitment therapy, relational frame theory, and the third wave of behavioral and cognitive therapies,” *Behavioral Therapy* 35 639-665.

Hofmann, S.G., Asmundson, G.J.G. (2008) “Acceptance and mindfulness-based therapy: new wave or old hat?” *Clinical Psychology Review* 8 1-16.

Iannetti, G.D., Salomons, T.V., Moayedi, M., Mouraux, M., Davis, K.D. (2013) “Beyond metaphor: contrasting mechanisms of social and physical pain,” *Trends in Cognitive Sciences* 17(8): 371–378

IASP Task Force on Taxonomy, Merskey H., Bogduk N. (eds.) (1994), *Classification of Chronic Pain, 2nd Edition*, Seattle: IASP Press. http://www.iasp-pain.org/Taxonomy#Pain

Kabat-Zinn, J. (1982) “An outpatient program in behavioral medicine for chronic pain patients based on the practice of mindfulness meditation: theoretical considerations and preliminary results,” *General Hospital Psychiatry*:4(1) 43-47.

Keefe, F.J., Beaupré, P.M., Gil, K.M., Rumble, M.E., Aspnes, A.K. (2002) “Group therapy with patients with chronic pain,” in: Turk, D.C., Gatchel, R.J., (eds) *Psychological approaches to pain management: A practitioner's handbook. 2nd ed*., New York: Guilford Press.

Keefe, F.J., Salley, A.N., and Lefebvre, J.C. (1992) “Coping with pain: conceptual concerns and future directions,” *Pain* 51 131-134.

Kleinman, A., Brodwin, P.E., Good, B.J., Good, M-J.DelV. (1992) “Pain as human experience: an introduction,” in M.J.DelV. Good, P.E. Brodwin, B.J. Good, and A. Kleinman

(eds.), *Pain as human experience: an anthropological perspective*, Berkeley: University of California Press.

Langford, D.J., Tuttle, A.H., Brown, K., Deschenes, S., Fischer, D.B., Mutso, A., Root, K.C., Sotocinal, S.G., Stem, M.A., Mogil, J.S., Sternberg, W.F. (2010) "Social approach to pain in laboratory mice," *Social Neuroscience* 5(2) 163-170.

Legrain, V., Van Damme, S., Eccleston, C., Davis, K.D., Seminowicz, D.A., Crombez, G. (2009) "A neurocognitive model of attention to pain: behavioral and neuroimaging evidence," *Pain* 144 230-232.

Low, L.A. (2013) "The impact of pain upon cognition: what have rodent studies told us?" *Pain* 154 2603-2605.

Main, C.J., Keefe, F.J., Jensen, M.P., Vlaeyen, J.W.S., Vowles, K.E. (eds.) (2014), *Fordyce's Behavioral Methods for Chronic Pain and Illness: republished with invited commentaries*, Philadelphia, US: Lippincott Williams & Wilkins.

McCracken, L.M. (2009) Contextual cognitive-behavioral therapy for chronic pain, Seattle: IASP Press.

McCracken, L., Morley, S.J. (2014) "The psychological flexibility model: a basis for integration and progress in psychological approaches to chronic pain management," *Journal of Pain* 15(3) 221-34.

McCracken, L.M., Vowles, K.E. (2014) "Acceptance and commitment therapy and mindfulness for chronic pain: Model, process, and progress," *American Psychologist* 69(2) 178-87.

Melzack, R., Wall, P.D. (1965) "Pain mechanisms: a new theory," *Science* 150(3699) 971-979.

Merskey, H. (2009) "Somatization: or another God that failed," *Pain* 145 4-5.

Michell, J. (2009) "The psychometricians' fallacy: too clever by half," *British Journal of Mathematical and Statistical Psychology* 62 41–55

Mogil, J.S. (2015) "Social modulation of and by pain in humans and rodents," *Pain* 156(4) Suppl 1 S35-S41.

Moore, D.J., Keogh, E., Eccleston, C. (2013) "The effect of threat on attentional interruption by pain," *Pain* 154 82-88.

Morley, S., Williams, A.C.deC. (2006) "Editorial: RCTs of psychological treatments for chronic pain: progress and challenges," *Pain* 121 171-172.

Morley, S., Williams, A., Eccleston, C. (2013) "Examining the evidence of psychological treatments for chronic pain: time for a paradigm shift?" *Pain* 154 1929-1931.

Patients' Association (2010) "Public attitudes to pain," Patients' Association, UK.

Pilowsky, I., and Spence, N.D. (1975) "Patterns of illness behavior in patients with intractable pain," *Journal of Psychosomatic Research* 19 279-285.

Pincus, T., Williams, A. (1999) "Models and measurements of depression in chronic pain," *Journal of Psychosomatic Research* 47 211-219.

Rodkey, E.N., Pillai Riddell, R. (2013) "The infancy of infant pain research: the experimental origins of infant pain denial," *Journal of Pain* 14 338-350.

Rose, J.D. (2002) “The neurobehavioral nature of fishes and the question of awareness and pain,” *Reviews in Fisheries Science* 10(1) 1-38

Schiavenato, M., Craig, K.D. (2010) “Pain assessment as a social transaction: beyond the “gold standard”.” *Clinical Journal of Pain* 26(8) 667-676.

Schnakers, C., Chatelle, C., Demertzi, A., Majerus, S., Laureys, S. (2012). “What about pain in disorders of consciousness?” *AAPS Journal* 14 437-444.

Sneddon, L.U. (2011) “Pain perception in fish,” *Journal of Consciousness Studies* 18(9–10) 209–29

Teasdale, J.D., Segal, Z.V., Williams, J.M.G., Ridgeway, V.A., Soulsby, J.M. and Lau, M.A. (2000) “Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy,” *Journal of Consulting and Clinical Psychology 68* 615–623.

Tracey, I., Bushnell, M.C. (2009) “How neuroimaging studies have challenged us to rethink: is chronic pain a disease?” *Journal of Pain* 10 (11) 1113-1120.

Turk, D.C., Meichenbaum, D., and Genest, M. (1983) *Pain and behavioral medicine: a cognitive-behavioral perspective*, New York: The Guilford Press.

Turk, D.C., Okifuji, A. (2002) “Psychological factors in chronic pain: evolution and revolution,” *Journal of Consulting and Clinical Psychology* 70 678-690.

Turner, J.A., and Jensen, M.P. (1993) “Efficacy of cognitive therapy for chronic low back pain,” *Pain* 52 169-177.

Veehof, M.M., Oskam, M-J., Schreurs, K.M.G., Bohlmeijer, E.T. (2011) “Acceptance-based interventions for the treatment of chronic pain: a systematic review and meta-analysis,” *Pain* 152 533-542.

Vlaeyen, J.W.S., Kole-Snijders, A.M.J., Boeren, R.G.B., and 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. and Linton, S.J. (2000) “Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art,” *Pain* 85 317–332.

Vlaeyen, J.W.S., Morley, S., Linton, S., Boersma, K., de Jong, J. (2012) *Pain-Related Fear: Exposure-based Treatment of Chronic Pain*, Seattle: IASP press.

Von Korff, M. (2013) “Healthcare for chronic pain: overuse, underuse, and treatment needs,” *Medical Care* 51(10) 857-858.

Wall, P.D. “Introduction” in P.D. Wall and R. Melzack (eds.) (1994) *Textbook of Pain (3rd edition)*, Edinburgh: Churchill Livingstone.

Wall, P.D. (1979) “On the relation of injury to pain,” *Pain* 6 253-264.

Walters, E.T. (1994) “Injury-related behavior and neuronal plasticity: an evolutionary perspective on sensitization, hyperalgesia and analgesia,” *International Review of Neurobiology* 36 325-426.

Wampold, B.E. (2005) “Establishing specificity in psychotherapy scientifically: design and evidence issues,” *Clinical Psychology Science and Practice* 12 194–197.

Wiech, K., Lin, C.S., Brodersen, K.H., Bingel, U., Ploner, M., and Tracey, I. (2010) “Anterior insula integrates information about salience into perceptual decisions about pain,” *Journal of Neuroscience* 30(48) 16324-16331.

Williams, A.C.deC. (2002) “Facial expression of pain: an evolutionary account,” *Behaviour and Brain Sciences* 25 439-488.

Williams, A.C.deC (2016) “The evolution of pain-related behavior,” *Pain* 157(4) 788-90.

Williams, A.C.deC., Eccleston, C., Morley, S. (2012) “Psychological therapies for the management of chronic pain (excluding headache) in adults,” *Cochrane Database of Systematic Reviews* Issue 11. Art. No.: CD007407.

Williams, A.C.deC., Johnson, M. (2011) “Persistent pain: not a medically unexplained symptom.” *British Journal of General Practice* 61(591) 638-639.

Woolf, C.J. (2010) “What is this thing called pain?” *Journal of Clinical Investigation* 120 (11) 3742-3744.

Zeidan, F., Grant, J.A., Brown, C.A., McHaffie, J.G., Coghill, R.C. (2012) “Mindfulness meditation-related pain relief: evidence for unique brain mechanisms in the regulation of pain,” *Neuroscience Letters* 520 165-173.