The effects of gender and coping on the perception of pain

Thesis presented by

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ABSTRACT

Human pain perception is now known to be mediated by the complex and dynamic interaction of biological and psychosocial systems. Research with both clinical and non-clinical populations has identified an array of factors which can influence pain, amongst which gender has become the focus of increased interest in recent years. However, although females generally seem to have lower experimental pain thresholds, report higher levels of pain and demonstrate lower pain tolerance than males, the pain research literature is characterised by conflicting findings regarding the direction, magnitude and robustness of such gender effects. Furthermore, gender differences may not occur equally with all types of noxious stimuli.

Investigating the impact of gender on pain is greatly complicated by the fact that gender in itself comprises both biological and psychological components. Gender-differentiated pain responses are therefore likely to involve physiological mechanisms such as the effects of gonadal hormones, as well as psychosocial determinants such as emotional responses and ways of coping. In this thesis, a series of controlled experiments was conducted to investigate the effects of gender and cognitive coping on cold pressor pain perception in healthy, pain-free individuals. The cold pressor paradigm was selected because relatively few previous studies have directly examined gender differences in this type of experimentally-induced pain. In light of potential fluctuations in female pain sensitivity as a function of hormonal status, cold pressor responses and the effectiveness of cognitive coping were also investigated in different phases of the menstrual cycle.

Gender differences in pain responses were evident here, but such differences occurred inconsistently across the series of experiments. Cognitive coping was found to have very limited impact overall, and no effects of menstrual phase were found on pain responses or on coping. These findings are discussed within a biopsychosocial framework of pain perception.

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1.1 Overview of introductory chapters

The purpose of the three introductory chapters of this thesis is to provide a general background to the role of psychology in pain research, and to contextualise the experiments reported here. This chapter includes an overview of some important early theoretical perspectives, and the subsequent progression through research towards a fuller understanding of the complexity of pain perception. The two subsequent chapters will review the empirical evidence for gender differences in pain responses, and the impact of cognitive coping on pain experiences respectively.

1.2 Introduction

Pain is an aversive phenomenon of proportions and complexity which warrants and requires ongoing scientific research and medical specialism. It is unlikely that the incidence and prevalence of pain can be accurately determined as an unknowable proportion of people will manage their pain privately, without recourse to professional healthcare. However, epidemiological research has provided some indication of the ubiquitousness of pain. For example, Wall and Jones (1991) estimated that approximately one in six adults in the U.S. is in pain at any given point in time. These authors have termed pain an epidemic, and "the most widespread and intractable of all health problems" (Wall & Jones, 1991, p.7). More recently, it has been estimated that every year in industrialised countries around 15-20% of the population experience acute pain, and some 25-30% have chronic pain conditions (Bonica & Loeser, 2001). Back pain, one of the commonest pain conditions in the general population, is estimated to affect up to 84% of adults at some time in their lives (Dionne, 1999), and has been categorised as the largest single cause of sickness absence from work in the U.S. (Fordyce, 1995). Although prevalence estimates vary substantially due to differences in survey methodology (such as the definition of pain severity used or the period of time studied), they clearly demonstrate overall that pain is a major public health problem which not only has negative impact on the lives of sufferers, but is also costly in terms of lost work time and demands on healthcare resources (Crombie, 1997).

1.3 The subjectivity of pain

The twentieth century saw substantial progress towards a fuller understanding of the physiological mechanisms underlying nociception, and consequent improvement of pharmacological pain management techniques. However, perhaps a more revolutionary

development has been recognition of the central importance of psychological factors in pain perception (Melzack & Wall, 1965). Acknowledgement of the influence of psychological factors was slow to emerge, despite long-documented observations that pain responses to a given noxious stimulus can vary greatly not only between individuals but also between different occasions in the same individual. However, if there is a single conclusion which resonates with the universal agreement of contemporary writers on the topic of pain, whether from empirical or philosophical disciplines, it is surely that pain is an inherently subjective experience. For example, Rey (1995) has stated that no two pains are ever identical, even those experienced at different times by the same person. According to Rey (1995), pain is so coloured by subjectivity that each experience is unique; factors such as previous experiences of similar pains, memory, and the state of mind when pain occurs can all modify the way pain is perceived and tolerated. Similarly, Morris (1993) asserted that pain is: "perhaps an archetype of subjectivity, felt only within the solitude of our individual minds" (p.14). The quote has an almost pessimistic tenor, but inasmuch as the individual mind is exemplified as the interpretative agent of pain perception, it also intimates that there is potential for the mind to modify such interpretation in a beneficial way and so to be therapeutically powerful. In the same volume, Morris has also argued that pain is never merely a sensation but always an experience interpreted by the brain and constructed by the mind. In this view, noxious stimuli are not passively received, but are interpreted within an idiosyncratic 'frame' comprising an array of factors, including appraisal of and attitudes to pain (and to health issues generally), affective states, beliefs and expectations. It has become increasingly clear that the fundamental subjectivity of pain is a product of an immensely complex and dynamic interaction of multiple psychological factors and physiological mechanisms. An ongoing challenge for pain research as a scientific discipline is the identification of systematic relationships within this intricate configuration which can be exploited to improve pain control and treatment.

1.4 The function of pain

The experience of pain is essential to human life, in that it provides vital warning signals of injury or pathology. This value is perhaps most starkly illustrated by the health problems which beset those rare individuals who are congenitally insensitive to pain. Lacking the 'alarm function' of painful feelings, they repeatedly sustain burns and

other injuries, and their life-expectancy is reduced because they fail to detect and respond to tissue damage and symptoms of illness (Sternbach, 1968). For such individuals the expedient detection and treatment of potentially life-threatening illnesses (e.g., peritonitis) is compromised by an absence of painful symptoms.

Evidently, the capacity to perceive pain is adaptive and advantageous in terms of survival. However, as Leriche (1939; cited in Melzack & Wall, 1996) noted, although the benefits of acute pain are evident in some instances, the value of persistent or chronic pain, occurring as it commonly does as a corollary of disease or a legacy of injury, is less clear. Unrelenting pain can have profoundly harmful effects on an individual's quality of life. For example, severe chronic pain can dominate conscious awareness, interfere with daily life and prevent sleep. In such situations, pain seems to have outlived its usefulness and is likely to have deleterious effects on both physical and psychological health, for example by reducing mobility and consequently physical condition, and promoting negative affective states (e.g., anxiety). When acute pain becomes chronic (persisting for more than six months), a circular relationship can develop between prolonged pain and negative affect (such as anxiety and dysphoria) and may trigger psychopathological conditions such as depression (Fishbain, Cutler, Rosomoff & Rosomoff, 1997). Conversely, pre-morbid personality traits or psychopathology may determine the level of psychological distress that chronic pain provokes (Merskey, 1999). Indeed, it has been argued that pain is never benign (Bonica, 1990) and that by suppressive action on immune function (which can permit increased tumour growth), and as a risk factor for suicide, in some circumstances pain can literally be a cause of death (Liebeskind, 1991).

1.5 The quest for pain control

Efforts to understand and control pain have been documented since earliest recorded human history (see Procacci & Maresca, 1998) and continue unabated. In antiquity, pain was commonly attributed to the presence of evil spirits in the body, and 'treatment' might involve exorcism rituals (Loeser, 2001). In the present day, with the accrued knowledge of the intervening centuries at our disposal, the search for dependable methods to alleviate the misery and suffering caused by unremitting pain remains an important research agenda. It has become clear that pain perception is highly complex, and while some questions have been answered, many more have emerged and continued

investigation is needed if pain is to be fully understood and controlled. In particular, while there has been considerable growth in knowledge of the physiological systems underlying pain perception, much remains unclear about the psychological factors involved. This may be at least partly because recognition of the significance of such factors is relatively recent.

In twenty-first century Western societies there is an implicit lack of acceptance of pain, a sense that technological advances should have rendered pain controllable, if not eradicable. Most of all, death should be pain-free, with no suffering involved. The medical approach to pain control has traditionally involved the administration of analgesics and anaesthetics; substances given to artificially induce a temporary state of painlessness. Plant-derived agents have been used to control pain in this manner for many centuries and remain important in contemporary pain pharmacology. For example, acetylsalicylic acid ('aspirin'), morphine and cocaine are still widely used as analgesic and anaesthetic agents. Recently, there has also been research into the analgesic potential of cannabinoid substances derived from cannabis sativa. Similar to opioid analgesics, cannabinoids closely resemble substances produced within the body and exert their effects via affinity with endogenous receptor sites in the brain (British Medical Association, 1997). Pharmacological advances in the last few decade have produced greatly improved drugs for pain control, with particular success in the control of acute pain. Nevertheless, research suggests that there are still many patients for whom pain control is incomplete (Bruster, Jarman, Bosanquet, Weston, Erens & Delblanco, 1994). For example, postoperative patients commonly report inadequate pain relief from pharmacological interventions (see Thomas, 1997).

Several key factors contribute to these difficulties in pain control. Firstly, because the experience of pain is not easy to verbalise (Scarry, 1985) patients may find it difficult to communicate the extent and characteristics of their pain experience to anyone else, including healthcare professionals. In this way, pain can be underestimated and the analgesic medication consequently prescribed may be insufficient. Secondly, despite recent advances in pain control, physicians may not necessarily have sufficient expertise in the specifics of pain management to ensure that patients obtain adequate pain relief (see Skevington, 1995). Thirdly, analgesics seem to be selectively effective for particular types of pain, e.g., morphine relieves post-operative pain effectively but not

neuropathic pain (Woolf, 2000). To complicate matters further, recent research suggests that certain analgesics may have variable effectiveness depending on the sex of the recipient (Miaskowski & Levine, 1999; Ciccone & Holdcroft, 2000; Holdcroft, 2003).

1.5.1 The need for alternatives to pharmacological pain control

Under certain circumstances, pharmacological pain control may be perceived as undesirable by the pain sufferer. In childbirth, for example, some women are determined to have a wholly 'natural' (i.e., drug-free) labour and delivery, others fear potential harm to themselves and/or their baby from pain control medication. Some individuals with persistent or chronic pain are also resistant to taking analgesics because they are afraid of developing increased tolerance or addiction to drugs (Skevington, 1995); a particularly common problem with opioids (Hawthorn & Redmond, 1998). In addition, drug therapy for pain can have undesirable side-effects, such as gastointestinal irritation and bleeding with aspirin (Melzack & Wall, 1996) or nausea, vomiting and respiratory depression with opioids (British Medical Association, 1997). Alternatives or adjuncts to pharmacological pain control are clearly needed by some pain sufferers and in this context the development of cognitive and behavioural techniques for pain management becomes necessary and worthwhile. To that end, identifying the psychological variables or processes with therapeutic utility can inform the design of cognitive techniques for coping with pain which can complement pharmacological pain management.

1.6 Difficulties defining and communicating pain

The preliminary research convention of clearly defining the construct under investigation at the outset presents problems when the subject of inquiry is pain. More than three decades ago, Sternbach (1968, p.1) defined pain thus: "This experience of pain, which most of us have had, is a subjective sensation which we can only imperfectly communicate to one another." The intervening years have seen many authors in the field of pain reiterate the essence of this statement. For example, Scarry (1985, p.4) stated: "pain comes unsharably into our midst as at once that which cannot be denied and that which cannot be confirmed."

Finding adequate language with which one individual can convey pain to another is problematic but crucially important in research and in the context of treatment.

Although virtually every adult human has first-hand knowledge of pain, it is an intrinsically private and subjective experience and so is difficult to communicate to others. Indeed, Scarry (1985) argued that the 'unsharability' of pain is partly due to its essential linguistic inexpressibility, and noted that extreme physical pain actively destroys language, eliciting in humans a reversion to the use of vocal sounds that precede language.

1.6.1 Early definitions of pain

The difficulty of communicating pain feeds into a related issue – defining pain. Many attempts have been made to produce a universally meaningful definition of pain, and long-running debate has ensued over whether pain constitutes an emotion or a sensation, or both. For example, Aristotle deemed pain a passion of the soul, an emotion opposite to pleasure, rather than a physical sensation (see Procacci & Maresca, 1998). The Aristotlean definition, although couched in ancient and unscientific terms, still has some pertinence today. Contemporary pain theory and research recognises not only that emotion is a component of painful experiences but also that cognition and affect are contributory factors in the pain perception process (Melzack & Wall, 1965). Marshall (1894; cited in Melzack & Wall, 1996), gave a description of pain that bestrode the sensation-emotion debate and seems to share some of the flavour of both ancient Greek and twenty-first century Western thinking; "pain is an emotional quality, or quale, that colours all sensory events" (p.161)

The origins of the word 'pain' have an emotional tone: from the Latin *'poena'* which means punishment or grief, and from the Greek *'poine'* meaning penalty (Hanks, 1987). A recent edition of the standard Oxford English dictionary provides three definitions of pain: "physical suffering or discomfort caused by illness or injury", "a feeling of marked discomfort in a particular part of the body" or "mental suffering or distress" (Pearsall, 1998). An implicit separation of the mental and physical is apparent from the fact that these are proffered as *alternative* definitions of pain. Wall (1999) noted that a question invariably asked whenever discussion of pain begins is: 'Do you mean mental or physical pain?' The question proceeds from the premise that these are two separate entities and illustrates a persistent dualism in the conceptualisation of pain. David Morris (1993) has dubbed this assumption 'The Myth of Two Pains'; notionally mental suffering is viewed as distinct from physical pain, the emotional pain of a broken heart

is held to be completely different from the physical pain of a broken leg. It becomes ever clearer that this dichotomy is false and that pain experiences are at once both physical and psychological, and under most circumstances inseparably so.

Melzack and Wall (1996) have also argued that the word 'pain' does not signify a unitary construct, but a *category* of widely varying and unique, multidimensional experiences with different causes. In their view, insufficient knowledge about the mechanisms of pain has also prevented precise definition but this situation may be rectified with further research. A related difficulty has been the elusiveness of a theoretical model which can explain the complex and variable phenomena of pain perception. The next section comprises description and critical appraisal of some important older models of pain, and their influence on the development of contemporary pain theory.

1.6.1.1 The Cartesian Theory of Pain

One of the most pervasive and enduring models of pain in the 20th century was founded on the concept that the body and the mind are wholly separate. The influence of this classical dualism on the study of pain derives from a theoretical framework proposed by Descartes in the mid-seventeenth century (see Melzack & Wall, 1996). In brief, Descartes believed that the universe was composed of two independent components; the physical ('res extensa') and the non-physical ('res cogitans'). Pain was considered a bodily sensation and pain transmission a mechanistic, hardwired system from the skin to the brain, with injury directly and instantly causing pain. Descartes likened the mechanism involved in sensory (and pain) transmission to ringing a bell; pulling on a bellrope directly and instantly causes the bell to ring, implying linear causality from stimulus to response. The chain of events conceptualised starts with a stimulus which causes a sensation in body, which is then followed by a perception in the mind. In this model, mental (psychological) states play no active part in the sequence, and are considered merely reactions to physical sensations, occurring in a separate (non-physical) realm.

That physical damage to body tissue causes pain, and that the intensity of the pain is proportional to the severity of the injury is intuitively plausible. Indeed, the model of pain mechanisms known as 'specificity theory' (which was the progeny of the Cartesian

theory of sensory perception) was founded on this assumption. It has since become abundantly clear that such simplistic accounts of pain perception are far from accurate; the link between tissue damage and pain is extremely variable. In fact, the mechanisms through which the experience of pain occurs are highly complex, and psychological factors have transpired to be of pivotal importance. However, Descartes' ideas exerted an enduring influence on the scientific study of pain, and echoes of the basic principles of the Cartesian model persisted into twentieth century pain theories (see Melzack & Wall, 1996).

Systematic research into pain perception did not begin until physiology was formalised as an experimental science in the nineteenth century; this involved the study of sensation and thus of pain. In parallel, the development of analgesic and anaesthetic drug therapies for pain moved forward significantly, especially in the isolation of morphine from opium. Physiological research generated two particularly important theories of pain during this period; specificity theory and pattern theory. Neither of these models of the mechanisms of pain perception included psychological factors, and both have been subsequently found to be flawed (Melzack & Wall, 1996). However, as both theories contributed significantly to the development of contemporary pain theory, they will be briefly outlined here.

1.6.1.2 Specificity Theory

The basis of specificity theory was that pain is transmitted via unique physiological mechanisms, both central and peripheral, which mediate pain separately from other senses. A specific pain system was hypothesised, within which the activation of dedicated pain receptors and transmitters in response to injury projected directly via the spine to a unitary 'pain centre' in the brain. In 1894, Von Frey (cited in Melzack & Wall, 1996) mapped out pain and touch spots and used histological study of skin to identify what he believed were specific nerve endings for each sensation; based on his findings and deduction he categorised four cutaneous modalities – touch, warmth, cold and pain.

Specificity theory espoused the Cartesian principles of direct, hard-wired transmission of pain through dedicated channels from skin to brain, and an invariable relationship between noxious stimulus and resultant pain. This model posited pain as a singular

sensation, separate from the other senses. Support for specificity theory was derived from animal vivisection studies, pain and touch were found to be dissociable by selective spinal cord surgery, which seemed to demonstrate their independence. Similar dissociations seen in humans with spinal disease or injury were also taken as evidence of the apparent modularity of pain and touch (see Loeser, 2001). Interestingly, recent research indicates that cortical representations of pain and touch may be closely located but separate, so there may be a dissociability between them but it is mediated at a central rather than peripheral level (Treede, Apkarian, Bromm, Greenspan & Lenz, 2000).

1.6.1.3 Pattern Theory

Physiological and anatomical study in the late nineteenth century also produced an alternative account of pain processing known as pattern theory (Goldscheider, 1894; in Melzack & Wall, 1996). According to pattern theory, pain results from excessive touch stimulation. In direct contrast to specificity theory, this model proposed that all sensory receptors are non-specific (i.e., capable of mediating pain or other senses, such as touch). The same receptors which mediate touch (for example) would produce pain if the stimulus was strong and repetitive. Pain would occur when neural activity reached a certain level through summation, and only intense stimuli would produce pain.

The central tenets of pattern theory were that stimulus intensity and summation are the key determinants of pain. Several versions of the theory were devised, with summation hypothesised variously as occurring at peripheral or central level, and via different neural mechanisms (see Melzack & Wall, 1996). Pattern theory could account for variation in sensory responses but posited that this was governed wholly by bottom-up factors, i.e., solely by characteristics of the stimulus.

1.6.1.4 Critique of dualistic pain theories

Although Eastern cultures embrace holistic approaches to health, the biomedical model, with its roots in Cartesian dualism, has been culturally persistent in the West. As lately as the mid-twentieth century, pain perception was still largely viewed as a hard-wired system in which psychological processes did not feature; specificity theory remained the medical mainstream perspective, and mind and body were treated as separate entities. However, it has become increasingly clear that the Cartesian model is fundamentally

inadequate as a theory of pain and there are a substantial number of pain phenomena which represent contradictory evidence against such rigid, dualistic models and point unmistakably towards fundamental interaction between psychological and physiological factors in pain perception.

There are numerous illustrations of the failure of dualistic models of pain. Firstly, the relationship between injury and pain is extremely variable. There are well-documented cases of episodic analgesia, severe or even life-threatening injury sustained without pain, or with pain experienced many hours later (e.g., Beecher, 1959) and conversely, of pain with no identifiable organic cause (Melzack & Wall, 1996). Over time it has become evident that the direct and proportionate stimulus-response relationship between noxious stimulation (or tissue damage) and pain experience which is specified in dualistic pain theory does not exist. Indeed, there is a crucial distinction to be made between nociception and pain. Nociception is the neural activation detectable in the peripheral nervous system in response to a noxious stimulus, such as physical injury, whereas pain is the experience that *may* ensue as a result of such an event. It has become clear that the two are not equivalent, are dissociable, and can be proportionately and temporally disparate. Secondly, and in direct opposition to pattern theories, innocuous stimuli can produce pain. For example, allodynia, in which even the gentlest touch causes excruciating pain, occurs in some patients with a form of neuralgia. Thirdly, the location of pain may be distant from the site of damage, thus individuals may experience referred pain, such as pain on the opposite side of the head from an aching tooth. Finally, pain can persist where there is no injury or long after healing, as in causalgia (severe burning pain which continues after tissue healing). Perhaps the most dramatic example of this last pain phenomenon is phantom limb pain, which is experienced in the space outside the body formerly occupied by an amputated limb (Jensen & Rasmussen, 1994).

Physiological research has revealed that specificity theory was inaccurate in several major respects. Notably, there seems to be *specialisation* in sensory neural fibres rather than true specificity. For example, cutaneous receptors have long been known to preferentially (but not solely) and with different liminal thresholds to particular stimuli, thus demonstrating specific *sensitivity* to stimulus modality (Sherrington, 1906; in Melzack & Wall, 1996). Likewise, some sensory neural fibres respond only to intense

stimuli, but do not necessarily or exclusively produce pain. Nor does a unitary 'pain centre' exist in the brain, in fact brain imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have demonstrated that multiple and diffuse cerebral cortical regions are involved in nociception (Treede, Kenshalo, Gracely & Jones, 1999; Price, 1999). The level of specialisation which has been found in receptor fibres is incompatible with pattern theory, as is the top-down modulation of pain perception by psychological factors which has been amply documented in the pain research literature throughout the second half of the twentieth century and beyond.

Clearly then, many aspects and types of pain are anomalous to both specificity and pattern theories. By the mid-twentieth century, these mechanistic models of pain were being challenged and found inadequate (Bonica, 1953) and more comprehensive approaches to pain and pain treatment finally began to take shape.

1.6.1.5 Progress in pain research and theory

Pain continues to provoke debate. Rey (1995) commented that "pain still has no clearly defined status; between being considered an emotion or a sensation, it has constantly been shunted between two equally unsatisfactory viewpoints" (p.6). However, the continued controversy regarding the definition of pain has not prevented significant advances in pain research and therapy; much is now known about how pain occurs and how it can be modified. Importantly, John Bonica (1917-1994) pioneered a multidisciplinary approach to the study and treatment of pain, which has brought far-reaching changes. Bonica strongly believed that both listening to patients (self-report of the first person experience of pain) and communication between the various professions involved in their treatment would effectively 'pool resources', increase understanding of pain, and lead to improved pain control methods. This collaborative approach led to recognition of the role of psychological factors in pain perception (International Association for the Study of Pain, 1994) and paved the way for the development of contemporary pain theory.

1.6.1.6 Gate Control Theory: A paradigm shift

In 1965, Ronald Melzack and Patrick Wall developed the first theoretical model of pain to conceptualise dynamic interaction between psychology and physiology in pain

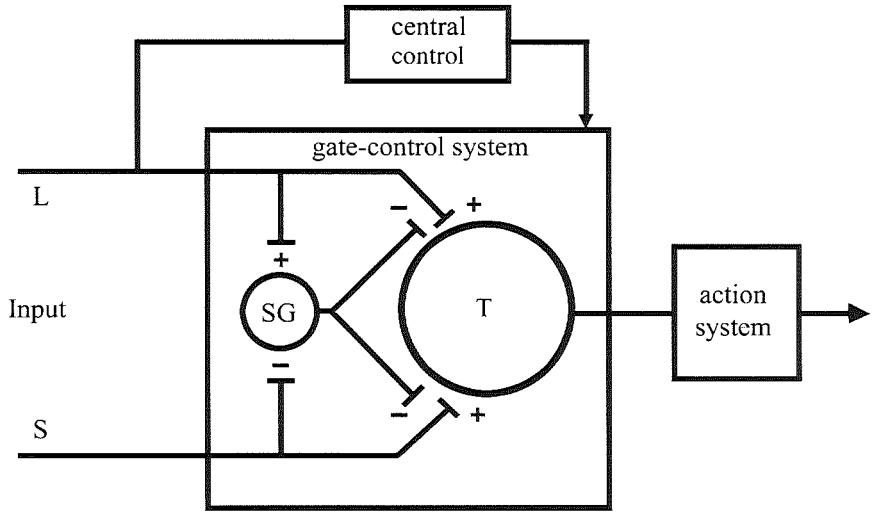
perception. Gate Control theory proposed psychological modulation of efferent pain inhibitory systems descending from higher regions of the central nervous system. The explanatory power of Gate Control emanates from its emphasis on a physiological basis for psychological influence on pain perception. The model was ground-breaking in its capacity to account for the highly variable relationship between tissue damage and pain, and in the recognition that pain perception is psychologically modifiable. A schematic diagram of Melzack and Wall's (1965) Gate Control model of pain perception can be found in Figure 1.1.

According to Melzack and Wall (1965), a neural 'gate' mechanism in the dorsal horn of the spinal column determines the extent to which pain is experienced in response to noxious stimuli (nociception). The gate metaphor signified that the flow of neural pain impulses is alterable, and that this determines the extent to which pain is perceived. The conceptual tenets of Gate Control theory owed something to both specificity and pattern theories in its proposition that pain transmission occurs as a pattern of excitation and inhibition of specialised neural fibres of different diameters, which mediate different types or qualities of pain (sharp, dull, fast, slow). However, Gate Control theory was revolutionary in the crucial role it assigned to descending influences from the higher central nervous system, governing the opening or closure of the neural gate. These CNS-generated influences include psychological factors such as past experience, attention, and emotion which influence pain response and perception via their action on the gate control system.

Gate Control theory reconceptualised pain as a *process*, rather than a simple sensation or signal (Kugelmann, 1997). According to Melzack and Wall (1965) there are interacting component systems (sensory-discriminative, motivational-affective and central control) which determine the pain experienced, and project to the motor system. Within this paradigm, pain perception is ascribed an essential multidimensionality both in its mechanisms and in the resultant experience. Refining the basic model in 1968, Melzack and Casey proposed three fundamental pain dimensions: sensory-discriminative, motivational-affective and cognitive-evaluative. These dimensions comprise parallel, interacting and simultaneously active systems in pain perception. According to Melzack and Casey's reformulation, output from transmission cells in the gate control system project to the sensory-discriminative system (via neospinothalamic fibres into the ventrobasal thalamus and somatosensory cortex) and motivational-

affective systems (via medial pathways into the reticular formation, medial thalamus and limbic system) which are mutually interactive with cognitive-evaluative systems in the higher CNS (termed 'central control processes'). Central control processes receive input from large fibre afferents and feed back to the gate control system, and to the sensory-discriminative and motivational-affective systems. The three systems interact and project to the motor system to govern action. Stated simply, Gate Control theory contends that both the process and the products of pain perception have powerful psychological components.

Figure 1.1: Schematic diagram of the Gate Control theory of pain (Melzack & Wall, 1965).



Note. 'L' input represents large-diameter, myelinated afferent fibres, 'S' input represents small-diameter afferents. These fibres project to the substantia gelatinosa (SG) – the proposed location of the 'gate'- and to transmission (T) cells which transmit locally to reflex circuits and to the higher CNS. Activity of 'L' fibres increases the inhibitory influence of SG on afferent terminals, whereas 'S' activation decreases it. 'Central control' represents higher CNS mechanisms which receive information from 'L' afferents and exert modulatory influence back into the gate control system.

Revisions to Gate Control theory have been necessary. For example, the original model differentiated only between large-diameter (facilitatory) and small-diameter (inhibitory) fibres, whereas it is now known that there are small-diameter, myelinated A-delta fibres and small-diameter, unmyelinated C-fibres as well as large-diameter A-beta fibres, all of which differ in functional specialisation. However, with refinements, Gate Control theory has remained the most useful explanatory framework for the complexity of pain perception (Wall, 1989), and each of the theoretical stages comprising the gate control mechanism has been supported by subsequent research (Melzack & Wall, 1996).

Gatchel and Weisberg (2000) recently stated that the Gate Control model still provides the most heuristic perspective of the wide range of pain phenomena encountered in medical settings.

1.6.1.7 The neuromatrix model: updated Gate Control

Proceeding from Gate Control theory, Melzack (1999) has proposed that the complex CNS mechanisms of pain comprise a neural network which he has termed the 'body-self neuromatrix'. This network integrates diffuse and parallel somatosensory, limbic and thalamocortical components which subserve the sensory-discriminative, cognitive-evaluative and affective-motivational aspects of pain conceptualised in Gate Control. A genetically-originated neural architecture is hypothesized in the neuromatrix model, which predicts a certain level of idiosyncrasy in pain responses (a 'neurosignature') and may predispose individuals towards developing chronic pain. According to Melzack, multiple inputs, in addition to somatosensory data, impinge on the neuromatrix and affect its output. These inputs include visual and sensory data which affect the cognitive interpretation of noxious stimuli, cognitive and emotional responses, and crucially, homeostatically-generated stress responses such as endocrine, autonomic, immune and opioid system activation. The neuromatrix model gives equal importance to genetic factors and the neuroendocrine mechanisms of psychological stress in the generation of pain experiences as it does to the neurophysiology of sensory perception.

Importantly for this thesis, the neuromatrix model may offer an explanation for gender differences in pain, at least in pain prevalence. Via action on cytokines, estrogen levels affect the release of cortisol, a hormone secreted during the normal stress response which acts on immune and opioid function to facilitate rapid response to threat. While cortisol is essential in a dangerous emergency, its continued secretion due to prolonged stress can adversely affect muscle, bone and neural structure, and can also suppress immune function. This may partly explain the greater prevalence of both autoimmune diseases and chronic painful illness in women compared to men. The individuality of the neurosignature in this model can also theoretically account for gender differences in pain sensitivity given the genetic and neuroendocrine system differences between males and females.

The neuromatrix theory reiterates the fundamental component structure of pain proposed in Gate Control, but provides a more comprehensive and integrated model of the variable experiences possible in response to noxious stimulation.

1.6.1.8 Contemporary definitions of pain

Recognition of psychological processes in pain perception has led to more flexible and comprehensive definitions of pain, notably that proposed by the taxonomy committee of IASP which stated that: "Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey & Bogduk, 1994, p.210).

Accompanying notes make clear the importance of psychological factors and of subjectivity. They state that pain is always subjective, and that individuals learn to use the word in childhood as a label for experiences of injury. The notes also assert that pain is a somatic sensation but also an emotional experience, can occur in the absence of any apparent tissue damage, and that nociceptive activity and pain are not equivalent.

The IASP definition is congruent with the tenets of Gate Control theory and has signified formal recognition that Cartesian-style models of pain are outdated and inadequate. Progressing from this working definition, pain research has broadened in scope. Investigation of the interactive effects of the many factors (both physiological and psychological) involved in the generation of pain is guiding the development of more effective treatment protocols. Often only partial answers to existing questions are found, and simultaneously new questions have emerged, such is the complexity of the pain perception system being unravelled.

Attempts to further refine the definition of pain to reflect the complexities involved continue. For example, Price (1999) has recently concluded that the IASP (1994) working definition of pain is confusing and not experiential enough. Price has proposed that pain is better defined as "a somatic perception containing (1) a bodily sensation with qualities like those reported during tissue-damaging stimulation, (2) an experienced threat associated with this sensation, and (3) a feeling of unpleasantness or other negative emotion based on this experienced threat" (Price, 1999, p.1). He has argued that this definition eliminates the previous requirement of an observable

association of the experiential aspects of pain with actual or potential tissue damage, which may not be demonstrable.

Whatever their remaining conceptual differences, contemporary definitions and models of pain have at least reached consensus regarding the integral role of an emotional component in pain experiences. Craig (1999) has asserted that emotional distress is the most "disruptive and undesirable" quality of pain and characterises it as the cause of suffering (p.331). Similarly, Melzack and Wall (1996) stated that "pain does not just have a sensory quality; it also has a strong negative affective quality that drives us into activity" (p.161). There can be little doubt of the importance of affective-motivational features of pain, which in addition to their psychological impact, provoke reflex actions and pain-responsive behaviours such as escape or avoidance. It has been argued that the affective-motivational features of pain may be clinically the most relevant (Willis, 1995) and assuredly to the sufferer this aspect of pain has enormous experiential significance.

However, the cognitive-evaluative component of pain may be of even greater importance as this psychological factor renders pain more than mere sensation - or even sensation with emotional qualities - it imparts meaning and idiosyncrasy. This higher-order analysis integrates and interprets the information produced by the other two components, exerting dynamic influence over them and over behavioural responses to pain. The cognitive–evaluative component is effectively a catalyst within pain perception, through which a series of neural impulses and neurochemical interactions become a highly individual and personal experience.

Although this thesis is primarily concerned with the psychological aspects of pain, these cannot be considered in isolation from the underlying neurophysiology of nociceptive processes. Indeed, the characteristics of the nervous system mechanisms involved may be pivotal to understanding the importance and complexity of psychological factors in pain. The functional neuroanatomy of pain perception, while still not fully understood, is germane to this discussion and will be described in the following sections.

1.7 The neurophysiology of pain perception

In broad terms, pain perception usually (but not exclusively) begins with the activation of afferent neural fibres in the periphery as a response to tissue injury, and the relay of impulses to transmission cells in the spinal cord. These in turn transmit both to local reflex circuits and to the brain. Neuronal facilitation and inhibition mechanisms at all synapses make enormously variable responses possible. Importantly, potentially noxious sensory stimuli are received and interpreted by an active nervous system which has been modified by past experience and is affected by current psychological states (such as anxiety); the resulting pain experience is contingent upon conditions within the nervous system. This section will therefore outline the main neurophysiological mechanisms underlying subcomponents of pain experiences at peripheral and central level.

1.7.1 Specialisation, not specificity

It is inaccurate to think of the neural mechanisms of pain as fixed. Indeed the distinction between specialisation and specificity is of crucial importance here, since it is the former and not the latter which characterises the neurophysiology of pain perception. Specificity implies that the components of the sensory system are hard-wired both receptively and productively; that neural structures respond exclusively to single types of stimuli and, in consequence, generate an invariable pain experience. In contrast, specialisation implies that sensory neural components respond preferentially and characteristically (but not exclusively) to certain types and levels of stimulation, but that these response patterns can be modulated by other sensory input or cognitive processes to produce varied experiences of pain, or none at all (Melzack & Wall, 1996). Specialisation has been found at both peripheral and central levels.

1.7.2 Peripheral nervous system mechanisms of pain perception

Sensory information, including the occurrence or imminence of injury, is transmitted to the central nervous system via modality-specialised primary afferent fibres in the periphery. Painful sensations are mainly projected to primary afferents by 'nociceptors', receptors which respond preferentially to noxious chemical, mechanical or thermal stimuli (Sherrington, 1906; in Melzack & Wall, 1996). However, it is important to note that nociceptors are not strictly 'pain receptors' as their activation in response to noxious stimuli will not necessarily result in pain. Much of the research into the

anatomy and function of peripheral neural mechanisms of nociception has focused on skin although somatosensory information also arrives from kinaesthetic and visceral sources (Price, 1999).

There are several kinds of nociceptive fibres, some of which are polymodal in that they respond to various stimulus modalities, while others have more specialised response properties. Two main types of nociceptor with distinct functional characteristics have been identified; unmyelinated C-fibres and myelinated A-fibres. The former are plentiful, relatively slow-conducting and mediate non-noxious thermal as well as nociceptive information. Most cutaneous afferents are C-fibre mechano-heat-sensitive nociceptors (CMHs) which can also be activated by chemical stimuli, and thus are polymodal. C-fibres are thought to mediate unpleasant, long-lasting (secondary) pain and the burning qualities of pain (Raja, Meyer, Ringkamp & Campbell, 1999).

By contrast, the activation of A-fibres is thought to elicit pricking pain, sharp sensations and aching pain. There are two main types of A-fibre, A-beta (Aβ) and A-delta (Aδ) both of which are myelinated, facilitating faster neural transmission than in C-fibres. Aβ nociceptors are thicker and conduct more slowly than Aδ fibres. Aδ fibres can be further distinguished, on the basis of their patterns of responsiveness to different stimuli, into Type I and Type II. Type I Aδ fibres are mechano-heat-sensitive nociceptors (AMHs) which are also strongly responsive to chemical stimuli (and so are polymodal) and respond to heat gradually. Less is known about Type II Aδ fibres, which are localised to hairy skin only, conduct more slowly than Type I, are largely insensitive to mechanical stimuli and are thought to evoke the initial (first) pain sensation to heat stimuli (Raja et al., 1999).

It is now clear that the different characteristics and properties of C-fibres and A-fibres subserve different aspects of communication about tissue states and pain between the peripheral and central nervous systems, but the important point for this discussion is that neural plasticity in primary afferents produces flexibility of function. It was initially proposed that activity in large-diameter Aβ fibres tended to 'close the gate' (i.e., inhibit nociceptive transmission) whereas Aδ and C-fibre activation facilitated nociceptive transmission (Melzack & Wall, 1965). However, under some conditions large-diameter fibres increase rather than inhibit pain perception. In addition, C-fibre function has

transpired to be rather complex, and not merely a slower 'back-up' system activated only if myelinated afferents are damaged. It is now known that while Aδ afferent nerve impulses rapidly signal to the CNS that an injury has occurred, the slower C-fibre impulses to the spinal cord produce lingering excitation that is thought to mediate tenderness, but which can also produce lasting after-effects, such as sensitisation in the central nervous system following nerve damage (Melzack & Wall, 1996). The peripheral nervous system is involved in antinociceptive as well as nociceptive action, for example, primary afferents in the periphery can be inhibited by endogenous analgesic (antinociceptive) mechanisms (e.g. endocannabinoid systems).

1.7.3 The dorsal horn and pain perception

The next stage in the transfer of information about noxious stimuli to the CNS is projection to the spinal dorsal horn, a region of key importance in pain perception. The complex neuroanatomy of the dorsal horn comprises a crucial junction of different components of the nervous system; the central terminals of primary sensory neurons, dorsal horn neurons and a complex network of CNS inputs and outputs. Some simple responses to nociception, such as the flexion reflex, are thought to be solely mediated at this level rather than involving supraspinal processing, but most pain responses involve higher CNS processes.

1.7.3.1 The laminar anatomy of the dorsal horn

In the spinal cord, white matter containing both afferent and efferent neural conduits between brain and spinal cord surrounds grey matter where synaptic activity in the spinal cord occurs, and which in turn lies around a central canal (Wall, 1989). The grey matter consists of a series of ten layers, or laminae, of which laminae I to VI comprise the dorsal horn (laminae VII to IX are in the ventral horn and lamina X immediately surrounds the central canal). Dendritic extensions between cells connect with axons in other laminae (Melzack & Wall, 1996). The laminae of the dorsal horn have neurochemical properties pertaining to pain modulation, as well as communicative connections with peripheral and central nervous system structures which are heavily implicated in pain perception.

Lamina I (the marginal layer) has many axonal projections to the thalamus forming the spinothalamic tract (STT), which is thought to be a key route of nociceptive information

between the spinal cord and the brain. Lamina V cells also project to the brain via the STT. Lamina II cells are predominantly interneurons which project intrasegmentally in the spinal cord and modulate lamina I and V cell activity. Lamina II has also been termed the 'substantia gelatinosa' (SG) and was the originally proposed location of Melzack and Wall's (1965) neural gate mechanism. Evidence of the involvement of the SG in pain attenuation comes from the high concentrations of endogenous inhibitory chemical agents, especially endogenous opioids such as enkephalins and dynorphins, found in this area (Melzack & Wall, 1996). C-fibres and A-delta fibres terminate in laminae I and II and V. Lamina V also receives input from interneurons in laminae I and II, and from A-beta (non-nociceptive) fibres, has a larger receptive field than either laminae I or II and so conveys much information about the characteristics of the noxious stimulus (e.g, location and intensity) to the CNS (Hawthorn & Redmond, 1998). The laminae run the full length of the spinal cord and dorsal horn laminae ultimately fuse into the medullary dorsal horn (Terman & Bonica, 2001).

1.7.3.2 Neurophysiology of the dorsal horn

Projecting neurons in the dorsal horn carry sensory information from the spinal cord up to brain areas which mediate perception, attention, learned behaviour, emotion and autonomic responses, and also exert descending influence on the excitation and inhibition patterns of neuronal activity in the dorsal horn (Melzack & Wall, 1996). Dorsal horn neurons demonstrate three main selective response characteristics (McMahon, 1994). Low threshold mechanoreceptors (LTM) respond to low-threshold innocuous afferent stimulation whereas nociceptive-specific (NS) cells, or nociceptors, respond preferentially to high-threshold noxious afferent inputs. Wide Dynamic Range (WDR) cells are activated by small, high threshold afferents as well as large, low-threshold, myelinated fibres and a range of stimulus intensities. A high proportion of the dorsal horn neurons which project to the brain to signal injury are WDR cells; far fewer are NS neurons.

However, crucially, neither the modality nor the responsiveness of dorsal horn neurons is fixed; under certain conditions NS cells may become WDR. Directly contradicting specificity (pain mediated solely by NS neurons) it now seems that WDR cells in this region firing at above a critical rate can also produce pain. Melzack and Wall (1996) have suggested that these proportional alterations in the functional identity of dorsal

horn cells may be analogous to different 'settings' of the gate control mechanism. The somatosensory system seems to have different operative states, governed by chemical and functional plasticity of primary sensory neurons in the dorsal horn and the brain, and sensory experiences are determined by these states and by shifts between them. Hence, patterns of excitation and inhibition in dorsal horn neurons contribute to the variable nature of pain experiences and can explain why a noxious stimulus does not always produce pain, and conversely an 'innocuous' stimulus may do so (Doubell, Mannion & Woolf, 1999).

Importantly for this discussion, the functional flexibility in dorsal horn neurons parallels that found in peripheral neural mechanisms and plays a key role in the dynamic responsiveness of the somatosensory system to sensory stimuli. The neurochemical involvement of SG neurons in endogenous pain inhibition also indicates that this region of the dorsal horn is particularly important in pain modulation.

1.7.4 Ascending neural pathways and pain perception

In response to noxious stimulation of peripheral afferents, groups of spinal neurons project upwards via neural pathways to the thalamus and the brainstem. Dorsal horn WDR and NS neurons project to the ventroposterior lateral (VPL) nucleus of the thalamus and medial thalamic structures. Neural pathways which appear to be of particular importance in pain processing are the spinothalamic tract (STT), the spinoreticular tract (SRT), the spinomesencephalic tract (SMT) and the spinohypothalamic tract (SHT) although there are also other connective routes (Terman & Bonica, 2001).

The STT, which originates predominantly in the dorsal horn of the spinal cord and projects directly to the thalamus, has long been considered the main ascending 'pain pathway' (Price, 1999). This neural conduit transmits nociceptive information to diffuse brain areas, including multiple thalamic regions, and sensory discrimination of the qualities of the stimulus occurs via spinothalamic connections. Lesions to the STT have been found to result in loss of both pain and temperature perception, which indicates that these modalities are linked in this pathway as well as in peripheral nociceptor responsivity (Craig & Dostrovsky, 1999). Clearly the ascending STT is not exclusively a pain pathway, as it transmits other sensory information (Treede et al, 1999). The

complex affective-motivational aspects of pain have been attributed to spinothalamic, spinohypothalamic, spinopontoamygdaloid, spinomesencephalic and spinoreticular pathways (Price, 1999).

1.7.5 Central nervous system and pain perception

Nociceptive information is relayed by ascending neural pathways to numerous, widespread brain areas which are functionally diverse and likely to subserve different facets of painful experiences and behavioural responses to them (Price, 1999). Nociceptive transmission becomes more diffuse as it travels higher in the CNS, and multiple brain areas become simultaneously active in the various components of pain perception (Guilbard, Bernard & Besson, 1994). According to Melzack and Casey (1968) the sensory-discriminative component of pain involves analysis of location, intensity, and duration of the noxious stimulus, the motivational-affective component relates to the unpleasantness and aversive drive of pain, and the cognitive-evaluative component involves anticipation, attention, suggestion and memory of past pains. Evidence of the interconnectedness of psychological and physiological mechanisms in pain comes from the identification of neural substrates of these component systems of pain perception within the CNS.

It is likely that virtually all pain experiences involve some higher CNS activity. Even simple reflex responses such as withdrawal which are mediated at spinal level can be influenced by cognition. Melzack and Wall (1996) provide an illustrative example: if a person grasps an expensive teacup which is too hot to hold, rather than simply dropping it they will usually try to put the cup down safely *then* nurse their hand. There have been many clear demonstrations of the impact of cognitive processes on pain perception (Weisenberg, 1998). For example, the threat of injury has been found to activate endogenous opioid antinociception, showing that cognitive processes produced in the CNS exert pain modulation effects even before pain begins. Descending impulses from the CNS related to expectation, attention and arousal have been also been found to increase dorsal horn nociceptor responses, adding to the perceived intensity of pain (Fields, 1992). Similarly, activation of pain modulatory regions of the brainstem (CNS-generated modulatory signal) have been found to exert facilitatory (as well as inhibitory) action on nociceptive transmission in dorsal horn neurons (Fields & Basbaum, 1999). This type of evidence that pain can be exacerbated, reduced and

perhaps even generated at CNS level, aside from the activation of primary afferents, is an important indicator of the power of psychological processes in pain perception.

The remainder of this section will provide a broad overview of the CNS structures now known to be involved in subcomponents of pain perception. The purpose of this is to describe the physiological framework supporting the psychological aspects of pain outlined in Gate Control theory. Similar to the peripheral nervous system, there seems to be specialisation in CNS pain processing rather than true specificity, with considerable functional overlap between brain areas. Notably, many CNS areas and structures exert both inhibitory and facilitatory influence on pain perception.

The spinal cord ascends and widens into the brainstem, a region with ascending neural projections to the cortex, descending projections to the spinal cord, and reciprocal connections to the limbic system. Three midbrain brainstem structures seem to be particularly important in pain perception: the periaqueductal grey matter (PAG), the reticular formation and the locus coeruleus (see Strong, Unruh, Wright & Baxter, 2002). The PAG, which is reciprocally connected to multiple levels of the nervous system, influences nociceptive, autonomic and motor systems (Strong et al., 2002). PAG projections to the dorsal horn exert both inhibitory and excitatory nociceptive transmission (Fields & Basbaum, 1994). The reticular formation of the medulla, pons and midbrain projects bidirectionally, to thalamic regions and the spinal cord, mediates motor, autonomic and sensory functions as well as aversive drive and has long been believed to have ascending influence on the motivational-affective aspects of pain (Melzack & Casey, 1968). Projections from the reticular formation, through the medial thalamus and hypothalamus, ascend to forebrain limbic areas involved in motivation and emotion (e.g., cingulate gyrus, hippocampus).

Various CNS regions are also known to exert descending influence on nociception. For example, the locus coeruleus is involved in the descending control of pain, via its inhibitory action on spinothalamic activity in the dorsal horn (Strong et al., 2002). CNS networks which involve many supraspinal but subcortical structures exert downward influence on pain perception. In particular, the periventricular hypothalamus, the PAG and the medial lower brainstem project through the dorsolateral funiculus of the spinal cord into the dorsal horns, mainly to the substantia gelatinosa. Electrical stimulation of

these structures has been found to cause analgesia in humans and other animals (Fields & Basbaum, 1999).

Before transmission to the cortex, afferent sensory and motor information is processed by the thalamus (Craig & Dostrovsky, 1999). This complex subcortical structure seems to contribute to many aspects of pain perception in a somewhat modular fashion. The ventrobasal thalamic nuclei mediate sensory-discriminative aspects of painful experiences (Chudler & Bonica, 1999), while other thalamic structures, such as the intralaminar thalamic nuclei, contribute to motor and affective responses to pain including aversive drive (motivational- affective component).

Noxious stimulation resulting in pain is extremely motivating and produces autonomic and emotional responses via the hypothalamus and the limbic system. The hypothalamus orchestrates autonomic and endocrine responses, and regulates internal somatic reactions to tissue damage and pain, while the limbic system mediates mood and incentive to act (Price, 1999). Pain modulatory networks in the CNS connect to limbic structures, such as the amygdala and hypothalamus, and to the brainstem. The amygdala is also thought to be involved in sensory discrimination of the intensity of pain. In this way, communication between neocortical regions and the limbic system interfaces sensory processing with emotion and motivation, and so prompts purposeful behaviours in response to pain.

Functional mapping of the cerebral cortical regions involved in pain perception is ongoing and has been mainly achieved through clinical studies of brain-damage or lesion and more recently, neural imaging technologies. Widespread electroencephalographic alterations and elevations in brain metabolism occur during pain, particularly in the frontal cortex . The main areas involved are the primary and secondary somatosensory cortex (SI and SII), infraparietal 7b, the anterior cingulate cortex (ACC), the insula, the prefrontal cortex, and the supplementary motor area. The ACC, which is involved in autonomic, motor and emotional behaviours, is also densely populated with opioid receptors and receives direct projections from thalamic nuclei involved in nociceptive processing (Devinsky, Morrell & Vogt, 1995). Research suggests that the ACC is of particular importance in the emotional aspects of pain (Rainville, Price, Carrier & Bushnell, 1997; Villemure & Bushnell, 2002). The affective

component of pain is thought to be mediated overall by the frontal-limbic network (Price, 1999), whereas parietal regions seem to be predominantly involved in sensory-discriminative aspects of pain perception, and infraparietal area 7b seems to be active in the integration of sensory and affective dimensions of pain.

Hence, although the sensory-discriminative, affective-motivational and cognitive-evaluative aspects of pain are intertwined within the overall experience, they do seem to have at least partially separable biological substrates within the CNS (Treede, Kenshalo, Gracely & Jones, 1999). For example, ascending transmission of the sensory and affective aspects of pain are predominantly mediated by the spinothalamic and spinoreticular pathways respectively. Selective lesions of the prefrontal cortex leave sensory pain sensitivity unchanged but seem to alter affective and evaluative reactions to it; pain is perceived but provokes neither emotion nor perception of threat (Price, 1999). Similarly, parietal or frontal lobe lesion can lead to 'pain asymbolia'; patients remain aware of the sensation and location of noxious stimulation, but neither withdraw from it nor complain, showing no aversive drive or negative emotion (Melzack & Wall, 1996). These dissociations indicate specialised neural substrates and a degree of modularity in the sensory-discriminative, affective-motivational and cognitive-evaluative aspects at the cortical level. They also demonstrate the key importance of the affective-motivational dimension in the aversiveness of pain; without a negative emotional response and the accompanying drive to be rid of it, sensory pain appears not to be accompanied by suffering or anguish and therefore to be tolerable. These findings may be especially pertinent to the research in this thesis. If the affective-motivational, cognitive-evaluative and sensory-discriminative aspects of pain are dissociable, coping styles or strategies which exert effects on affective-motivational and/or cognitive-evaluative states could selectively alter these aspects of pain experiences. However, generalisation from brain-injury and lesion studies is always compromised by the possibility of unusual cerebral neural architecture prior to damage. Whether the sensory and affective components of pain are separable under ordinary circumstances is not certain (Melzack & Wall, 1996; Eccleston & Crombez, 2000).

1.8 Measurement of pain

To evaluate the different aspects of pain, whether for clinical or research purposes, requires reliable methods of measurement. This presents a fundamental difficulty

because, as Turk and Melzack (1992) have stated by its very nature, pain can be assessed only indirectly. While there may be overt pain behaviours which are amenable to assessment (e.g., grimacing, guarding) these do not necessarily give accurate insight into the extent or nature of the pain experienced. Any measure of pain is, by definition, a *representation* of the experience of another individual, therefore pain assessment is an inferential process in which self-report is an essential component.

In experimental settings, pain threshold and pain tolerance are single-point measures of pain which are easy for participants to understand and provide. Pain threshold is the minimum amount or duration of stimulation that reliably evokes a report of pain in an individual, while pain tolerance is the length of time that a continuous noxious stimulus can be endured by an individual or the most intense noxious stimulus that can be tolerated (Miaskowski, 1999). Although the units of intensity or time thus obtained are commonly treated as objective measurements of pain, they are representations of subjective judgements about limited qualities of a sensation (e.g., just noticeably painful, intolerably painful).

Alterations in certain physiological parameters can be taken as indicators of physical and emotional responses to pain (e.g., cortical activation, heart rate, skin conductance, pupil dilation and stress hormone levels in body fluids) but in every case these are also potential indicators of responses to other stressors (such as fear or anxiety) rather than unique indices of pain. Furthermore, without an accompanying report of subjective pain, such correlates are meaningless. Clearly then, pain research must rely on self-report measures of pain from patients or participants in conjunction with observational data. Many useful types of ratings scales have been used to facilitate the self-report of pain, both with patients and experimental participants. Most of these are simple measures, such as visual analogue scales or Likert-type scales, which yield a single score or value of one dimension of pain, usually intensity or severity. Visual analogue scales (VAS) - usually a 100mm line with terminal anchors labelled with extreme values such as 'no pain' and 'worst pain possible' - are considered to be among the most useful simple pain rating methods because they are comprehensible to participants, easy to administer and score, and yield ratio-level data (Price, 1999). Reliability and validity of VAS has been established for measurement of experimentally-induced pain (Price, 1988) hence they have been much used in this context.

The importance of self-report pain data does not lessen the problems associated with obtaining it. Gracely (1999) reiterated the enduring difficulties surrounding the communication of pain to another person, stating, "one elusive goal in pain measurement is the assessment of pain sensitivity independent of pain labelling behaviour; that is the assessment of subjective pain without the biases which influence verbal report" (p.388). Such biases may stem from many sources, such as individual differences in use of language, in emotional expressivity, in stoicism or willingness to report pain. Although the validity and usefulness of self-report pain scales and pain observation methods has been queried (e.g., Craig, Prkachin & Gruneau, 1992), other reviewers such as Jensen (1997) have concluded that such methods do properly reflect the pain experiences they are used to assess.

Unidimensional measures of pain such as VAS, threshold and tolerance can only provide a restricted assessment of the complexity of pain experiences. However, a more comprehensive self-report measure is available to assess the cognitive-evaluative and affective-motivational, as well as sensory-discriminative aspects of pain. The McGill Pain Questionnaire (MPQ; Melzack, 1975) is a widely used multidimensional pain instrument which was developed using adjectives used by pain patients to describe their pain, and which includes separate measures of sensory and affective aspects of pain as well as a Likert-type scale and VAS to assess overall pain intensity. Reliability and validity of the MPQ have been established and it is available in different languages and forms, including a version for use with children.

1.9 Summary

To summarise, pain remains difficult to define, to measure and to control. However, considerable advances in knowledge during the last century have led to acknowledgement that pain is more than a simple sensation, and gradual recognition that pain perception is a highly complex function involving the interaction of both psychological and physiological mechanisms.

At the physiological level, it is now clear that there are flexible modulatory mechanisms in both the central and peripheral nervous systems which can exacerbate or inhibit the experience of pain. Responses to different stimulus modalities are mediated by receptor specialisation (rather than specificity) and the type and extent of pain elicited emerges

via patterns of neural excitation and inhibition. Neural plasticity of various kinds has been identified in key areas which mediate pain perception; in peripheral afferent neural mechanisms, dorsal horn nociceptive transmission structures, and in ascending and descending routes of neural communication between spine and brain.

The novel proposition of the groundbreaking Gate Control theory was that psychological factors modify these processes. It is now known that psychological states such as arousal, cognition and emotion, which are emergent properties of brain processes and subserved by particular cerebral regions, play a key role in the modulation of pain. Hence, descending influences from the brain modulate both spinal reflexes and upward sensory transmission from the spinal cord to the brain. Similarly, ascending messages to the brain can interact with descending influences, and a bidirectional loop of communication operating between the periphery, spinal cord and brain mediates the perception of pain.

As outlined above, the nervous system mechanisms underlying pain perception and the involvement of psychological processes and states in such mechanisms can account for both the complexity of pain experiences and for the instability of the relationship between noxious stimulation and pain. The functional flexibility which pervades the multiple mechanisms of pain perception can produce immensely variable experiences of pain even if the noxious stimulus remains the same, as well as substantial variation in pain sensitivity between individuals.

This chapter has outlined some of the history of contemporary pain theory and research, including the difficulties which have accompanied the development of current models and definitions, and has given an overview of how underlying neurophysiological mechanisms are likely to relate to the psychological and experiential aspects of pain. The interdependence of biological and psychological factors in human pain perception, as explained in Gate Control theory and the neuromatrix model, relates directly to the main focus of this thesis: gender differences in pain perception. In the context of these models, gender is a factor with both biological and psychosocial bases which may be an important determinant of pain perception (Wall, 1994; Berkley, 1997). The next chapter will discuss empirical evidence for differences in pain responses between males and

females, and outline the biological and psychosocial mechanisms which are likely to underlie such differences.

Chapter 2

Sex, Gender and Pain

2.1 Introduction

Perhaps the archetypal illustration of the interdependence of biological and psychological factors in pain perception is manifest in differences which are found between men and women. It appears that males and females may have significantly contrasting experiences of pain, and that females may be generally more pain sensitive (Fillingim & Maixner, 1995; Berkley, 2000). In humans, these differences are likely to arise through the interaction of physiological factors such as sex-specific anatomy and neuroendocrinology with psychosocial factors such as gender-related [1] attitudes, expectations and coping behaviours. The research reported in this thesis was designed to compare the experimental pain responses of healthy men and women, and to investigate the effects of contrasting types of cognitive coping instructions on such responses. To contextualise my own work, this chapter will provide an overview of previous research which has illustrated differences between male and female pain responses. Empirical evidence for gender differences in human clinical and experimental pain will be outlined, followed by a brief discussion of rodent research which has demonstrated sex differences in pain sensitivity. The review will focus mainly on human research, as although there is rodent literature which has indicated a biological basis to sex-differentiated pain responses, extrapolation from rodent to human populations may not be justifiable. Potential mechanisms underlying gender-differentiated pain responses will also be discussed, as will sex-differentiated responses to analgesic medication in humans and rodents.

Among the array of factors now known to have impact on pain perception, sex or gender differences have recently become more prominent. In the past, sex and gender comparisons were relatively scarce for several reasons. Firstly, females were generally omitted from biomedical research because of the potential confounding effect of menstrual or estrus cycle on findings. Secondly, the risk of damage to the offspring of participants in the early stages of (undetected) pregnancy contraindicated the inclusion of women in research such as drug trials (Fillingim, 2000).

[1] In the pain research literature some authors use the term 'sex' to refer to biologically-based distinctions between males and females and 'gender' to denote psychosocial differences (e.g., Robinson, Riley & Myers, 2000). Others use one of these terms only (see LeResche, 1999), and still others use both terms interchangeably (e.g., Giamberardino, 2000). While biological and psychosocial factors in pain perception are both pertinent to my own research, I do not consider the terms sex and gender synonymous (and therefore interchangeable) in this context. Similar to Robinson et al., (2000) I will use them as contextually appropriate throughout this thesis.

Finally, emphasis on sexual equality in recent years created a social trend to play down sex-related differences of any kind, although such political correctness seems of dubious value if it merely deflects research attention from important sex or gender-based differences with potential healthcare implications. While it is likely that recognition of gender differences in pain sensitivity was delayed by the exclusion of women from clinical trials of pain treatments, the imbalance is now being redressed. This has been reflected in increased research interest and the launch of a designated Special Interest Group within IASP devoted to Sex, Gender and Pain at the World Pain Congress in 1999.

2.2 Evidence of sex and gender effects on pain perception

There are varied sources of research data which provide evidence for sex and gender effects on pain perception, including clinical and epidemiological studies with human pain populations as well as experimental pain induction studies with pain-free individuals. Animal research, mainly with rodents, has also contributed to the investigation of sex effects in pain sensitivity and analgesic responses. In the last decade, a number of useful meta-analyses and reviews have summarised the accumulated findings from these research sources (Fillingim & Maixner, 1995; Unruh, 1996; Berkley, 1997; Ciccone & Holdcroft, 1999; Riley, Robinson, Wise, Myers & Fillingim, 1998).

2.2.1 Gender differences in clinical pain

According to Unruh (1996) "research suggests that the greater burden of pain may lie with women" (p.124). Empirical evidence support this conclusion, and the emergent picture is that females may be both more likely to experience pain and more pain-sensitive than males. However, the simplicity of Unruh's statement belies the difficulties involved in interpretation of the complex body of research literature which informs it.

Epidemiological research shows that the extent to which men and women experience pain differs, at least partly because of their differential morbidity from disease and propensity for accidental injury (Unruh, 1996). Reviewing gender differences in clinical pain, Unruh noted that a variety of painful illnesses afflict women more than men. It seems that women are more likely to suffer acute and non-fatal chronic disease (even exclusive of gender-specific illnesses such as reproductive disorders) whereas men

sustain more injuries and succumb to more life-threatening chronic disease. A recent study of pain prevalence in general medical practice reaffirmed this pattern; most patients who sought primary care for pain across the course of a year were female (60%), and virtually all of the most common pain diagnoses were more prevalent in women than men (Hasselstrom, Liu-Palmgren & Rasjo-Wraak, 2002). Although there are some painful illnesses which predominantly affect men, the proportion which are female-prevalent is far greater than those which are male-prevalent (LeResche, 1999). See Table 2.1, adapted from Berkley (1997).

Berkley (1997) has emphasised that the diversity of interacting factors which affect pain perception (and which may have differential impact for males and females) coupled with great variation in methods of inquiry, makes interpretation of the data on sex differences in endogenous pain very difficult. The large body of epidemiological research, from which the sex-prevalence of painful conditions are estimated, is particularly problematic as it encompasses many types of illness and disparate methodologies, measures of pain, populations and sources (see Merskey & Bogduk, 1994). It is also worth noting that since prevalence is calculated from the onset rate, number of episodic recurrences and the average episode duration, gender differences in prevalence can be due to elevation of any of these components. Despite these caveats, the epidemiological literature has provided useful information about painful conditions likely to affect men and women respectively.

Whether there are gender differences in persistent or chronic pain overall is not yet clear. However, a population-based survey of five common pain conditions (back pain, headache, abdominal pain, chest pain and temperomandibular pain) showed that women were more likely to report multiple pains than men, and that the prevalence of these pains was higher in women than men (Von Korff, Dworkin, LeResche & Kruger, 1988). Furthermore, various individual pain conditions seem to disproportionately affect women. For example, women are 2-3 times more likely to experience migraine and frequent recurrent headache than men (LeResche, 2000). Men and women also experience different types of headache and migraine, and cluster headaches (which are generally rarer than migraine or tension headaches) are the only type which seem to afflict men more than women (Holroyd & Lipchik, 2000). The prevalence of male headaches remains stable across adulthood, whereas for adult females it is considerably higher but declines in the mid-forties (LeResche, 2000). For musculoskeletal pain,

women generally report more intense and more frequent pain in more body locations than men, especially neck, shoulder, upper limb and hip pain, which may be partly due to the greater susceptibility of women to osteoarthritis, rheumatoid arthritis and fibromyalgia (Unruh, 1996). Predictably, the prevalence of joint pain generally increases with age in both sexes (LeResche, 2000). Sex-related differences in prevalence of back pain are smaller and less clear-cut (for example, the direction of sex-related differences seems to vary across vertebral regions). However, they are harder to interpret because epidemiological statistics include occupation-related back problems in both sexes, back pain associated with female reproductive processes, and the general increase of back pain with age. Multiple pains occurring as part of somatisation disorder or psychosomatic illness (i.e., with no known underlying physical origin and assumed primary psychological aetiology) are equally common in male and female children, but show higher female prevalence in adolescence and beyond (Unruh, 1996). Some studies indicate that females experience more pain than males arising from health care procedures, such as dentistry and surgery (Unruh, 1996), but other research has found no sex-related differences, for example in post-surgical pain (Feine, Morin & Lund, 1998).

Fewer epidemiological studies have examined abdominal pain than other types of pain. However, even with the exclusion of menstrual pain, women report more pain in this body region than men across the adult lifespan. The prevalence rate of abdominal pain declines with age for both sexes (LeResche, 2000). Although most of the studies conducted have nominally investigated gastro-intestinal pain, it is possible that referred pain with gynaecological origin may contribute to the higher levels of abdominal pain in women (Giamberardino, Berkley, Iezzi, de Bigontina and Vecchiet, 1997). Certainly, pathology in pelvic viscera may put women more at risk for referred pain in the muscle and skin of this area than their male counterparts.

Clinical data reveals that some diseases manifest themselves differently in men and women, hence painful symptoms may differ for males and females with the same illness. For example, the location and temporal characteristics of headache pain, and the pain experienced with cancer and multiple sclerosis have all been found to differ between the sexes. Unruh (1996) has pointed out that for certain diseases there are separate diagnostic criteria for men and women (e.g., IBS, appendicitis, migraine) and

furthermore there are sex-specific predictors and risk factors for some conditions (such as coronary artery disease). This strongly suggests a biological basis to male-female differences in clinical pain.

Table 2.1: Sex prevalence of painful disorders

Female prevalence	Male prevalence	No sex prevalence
migraine with aura	migraine without aura	acute tension headache
chronic tension headache	cluster headache	cluster-tic syndrome
post-dural puncture headache	post-traumatic headache	'jabs' and 'jolts' syndrome
hemicrania continua	SUNCT syndrome	secondary trigeminal neuralgia
cervicogenic headache	Raeder's paratrigeminal syndrome	neuralgia of nervus intermedius
tic douleureux	Pancoast tumour	painful opthalmoplegia
temperomandibular joint disorder	thromboanginitis obliterans	maxillary sinusitis
occipital neuralgia	brachial plexus avulsion	toothache; enamel defects
periapical periodontitis & abscess	pancreatic disease	toothache; pulpitis
atypical odontalgia	duodenal ulcer	cracked tooth syndrome
burning tongue	abdominal migraine	dry socket
carotidynia	lateral femoral cutaneous neuropathy	vagus nerve neuralgia
chronic paroxysmal hemicrania	post-herpetic neuralgia	stylohyoid process syndrome
temporal arteritis	hemophilic arthropothy	thoracic outlet syndrome
carpal tunnel syndrome	ankylosing spondylitis	brachial plexus tumours
Raynaud's disease		esophageal motility disorders
chilblains		chronic gastric ulcer
causalgia		Crohn's disease
reflex sympathetic dystrophy		diverticular colon disease
chronic venous insufficiency		colon carcinoma
fibromyalgia syndrome		familial Mediterranean fever
oesophagitis		hereditary coproporphyria
reflux oesophagitis with peptic ulcer		acute herpes zoster
slipping rib syndrome		burns
twelfth rib syndrome		
gallbladder disease		
post-cholecystectomy syndrome		
irritable bowel syndrome		
interstitial cystitis		
acute intermittent porphyria		
proctalgia fugax		
chronic constipation		
pyriformis syndrome		
peroneal muscular atrophy		
multiple sclerosis		
rheumatoid arthritis		
pain of psychological origin		

Adapted from Berkley (1997). Reprinted with the permission of Cambridge University Press.

In sum, reviews of the epidemiological and clinical research literature suggest that female gender is a risk factor for pain (LeResche, 2000). Women report more multiple pains in more widespread body regions than men, and of more types. It is also apparent

that in many cases the gender-specific prevalence of painful conditions varies with age, often with shifts seen around adolescence and the mid-to-late forties which may signal the impact of reproductive lifecycle (for women at least). Overall, women report more severe, more frequent and longer lasting endogenous pain experiences than males (Berkley & Holdcroft, 1999).

2.2.2 Gender differences in human experimental pain responses

Experimental psychophysical studies of pain perception in healthy humans generally indicate that females have lower pain thresholds, are more discriminative, report more pain and demonstrate lower pain tolerance than males (Fillingim & Maixner, 1995; Berkley, 1997). However, such gender differences in human experimental pain sensitivity are not always replicable and are likely to be affected by many factors. For example, the pain indices in which gender differences are found varies between studies and some of this inconsistency may be related to the variety of ways in which pain is produced in the laboratory. Most methods of experimental pain induction rely on the external application of noxious stimuli, such as heat, cold, ischaemia, mechanical pressure, electricity, and chemical substances (Fillingim, 2000). Experimental pain induction is often cutaneous, although muscle and viscera have also been used. In addition to wide variation in pain induction methodology, a range of different measures of pain response has been used, e.g., threshold, tolerance, numerical rating scales, verbal descriptor scales, physiological responses. Since this lack of homogeneity compromises comparison across studies to some extent, further research with consistent methodologies would help to clarify the relationships between gender and experimental pain sensitivity.

The temporal and qualitative characteristics of experimental pain vary according to the method of application used. For instance, thermal and electrical pain are relatively brief and are considered to have more sensory than affective qualities, whereas ischemic and noxious cold pain are longer-lasting and thought to provoke stronger affective responses (Rainville, Feine, Bushnell & Duncan, 1992). There are reasons to expect that gender might selectively affect the sensory and affective perception of pain (Fillingim & Maixner, 1995). For example, given their overall tendency towards greater emotional expressiveness (Skevington, 1995), it is feasible that women might rate painful experiences in more affective terms compared to men. Similarly, on the basis that

females show greater sensitivity to non-noxious stimuli than males (e.g., Rollman & Harris, 1987), they might also be more responsive to sensory aspects of pain. However, separate ratings of sensory and affective pain responses to noxious stimulation have not provided clear evidence for gender-specific perception of these pain dimensions (Fillingim & Maixner, 1995).

Nevertheless, gender does not seem to affect all types of experimental pain equally. Stronger and more replicable gender differences have been found for pressure pain and electrical pain than for thermal or ischaemic pain (Riley, Robinson, Wise, Myers & Fillingim, 1998). The finding that females are more sensitive to pressure pain than males seems to be the most consistent, and for electrical pain and cold pain most studies have also found enhanced female sensitivity relative to males (Fillingim & Maixner, 1995). Studies of gender differences in thermal pain sensitivity have produced conflicting results, and in many cases no gender differences have been discerned (e.g., Bush, Harkins, Harrington & Price, 1993). These inconsistencies may be methodological in origin, as some thermal pain studies have taken threshold or tolerance times as measures of pain response (e.g., Lautenbacher & Strian, 1991) while others have used signal detection or magnitude estimation (e.g., Clark & Goodman, 1974). However, it is notable that although some thermal pain studies have found no sex-related differences in pain perception, none has yet shown greater sensitivity in men than women (Riley et al., 1998). The most consistent gender differences observed in laboratory pain studies occur with induction methods which elicit deep, tonic pain (e.g., mechanical, cold) which resembles naturally-occurring pain such as headache or cramp (Fillingim & Maixner, 1995). Importantly, experimental pain responses show great interindividual variability, which is likely to contribute to discrepant findings across different studies (Rollman & Harris, 1987). Functional imaging research reinforces this inasmuch as brain activation patterns have been found to vary enormously between individuals receiving identical noxious stimulation, even when those tested are closely matched in demographic profile (Davis, Kwan, Crawley & Mikulis, 1998).

Whether sex-related differences in pain sensitivity are important has been the subject of debate. For example, Berkley (1995) questioned the practical significance of such differences since they are inconsistent, and are often small or fail to reach statistical significance. Certainly, gender differences in pain response seem at times to have

somewhat evanescent qualities. For example, in approximately one third of the thirty-four studies reviewed by Fillingim and Maixner (1995) no significant sex differences were found in experimental pain responses. However, in a more recent meta-analysis Riley et al. (1998) calculated and reported the magnitude of sex-related effects on pain sensitivity across various stimulus types, and concluded that they are substantial and important. Consistent with Fillingim and Maixner's (1995) conclusion that sex-related differences occur most reliably with pressure pain sensitivity, Riley and colleagues (1998) found the largest sex effects for this stimulus type. Females demonstrated lower pressure pain threshold and tolerance than males, with large mean effect sizes of .82 and .76 respectively. In a recent study, Chesterton, Barlas, Foster, Baxter and Wright (2003) replicated this gender difference and found that it remained consistent across multiple repeated measurements. A similar pattern was observed with electrical pain; women were found to have moderately lower threshold and tolerance than men but with medium effect sizes. Thermal heat pain studies also gave evidence for higher male pain thresholds but less consistently; the mean effect size across studies was in the medium range (.41), but there was greater variability in the effect sizes found in individual studies than for other types of stimulus. Riley et al. (1998) also noted that inadequate sample sizes in some of the studies they reviewed had provided insufficient power to detect sex differences in pain sensitivity (inflation of Type II error). According to Riley et al. (1998) the overall effect sizes for sex-related differences in experimental pain sensitivity were in the moderate to large range, and slightly larger for pain tolerance (.57) than for pain threshold (.55). However, it should be noted that the meta-analysis was conducted with unequal numbers of studies in each stimulus type; in the case of ischemic pain only one study was included. Furthermore, some stimulus modalities (notably cold pain) were not included. However, Myers, Robinson, Riley and Sheffield (2001) have recently reported moderate to large gender differences in cold pressor pain in the same direction as those reported by Riley et al. (1998).

The weight of evidence for the existence of sex-related differences in pain perception has grown in recent years, and such differences are increasingly considered to be of potential importance in clinical pain research and treatment development (Berkley & Holdcroft, 1999).

2.2.3 Sex differences in rodent experimental pain responses

Some evidence of sex-related differences in pain sensitivity has also been found in non-human species. The gender differences in human pain sensitivity observed in both clinical and experimental settings can only be meaningfully investigated and interpreted within a biopsychosocial context (Fillingim, 2000), i.e., taking account of social and psychological as well as biological factors. Research with laboratory animals (primarily rodents) is assumed to allow a 'cleaner' examination of sex–related differences in nociceptive processes, effectively highlighting physiological mechanisms. The converse of this rationale is that findings from rodent research are not necessarily generalisable to humans. However, since observable sex differences in rodent pain responses are taken as evidence of underlying biological mechanisms in a mammalian species and similar mechanisms are likely to be at least part of human pain perception, rodent research will be covered briefly here.

Research into rodent pain sensitivity uses a range of different methods of pain induction involving electrical, thermal, mechanical and chemical noxious stimuli (e.g., footshock, tail-flick, formalin injection), and a variety of behavioural responses (such as flinching, jumping, and paw-licking) are taken as indices of pain (Sternberg & Wachterman, 2000). Similar to the human research literature, the comparability of findings is compromised by the variety of pain induction methods used and of pain behaviours assessed in different studies.

The rodent research literature suggests that sex differences in pain sensitivity are more consistent for some noxious stimuli than for others. For example, female rats demonstrate greater sensitivity to electrically and chemically induced pain than males (Miaskowski, 1999). However, some studies using noxious heat have shown the reverse pattern, while others have discerned no significant sex differences in pain response (Sternberg & Wachterman, 2000). For instance, with both the tail-immersion (hot water) and tail-flick (radiant heat) test, males reflexively withdraw from the stimulus faster than females, while most studies using the hot-plate test indicate no sex differences in sensitivity to thermal heat pain (Mogil, Chesler, Wilson, Juraska, & Sternberg, 2000).

Although methodological variations between different laboratories could at least partially account for these conflicting findings, inconsistencies have occurred even within the same laboratory. As with humans, interindividual variability is likely to contribute to these mixed findings, but there is also some evidence for genetic factors in rodent pain responses (Mogil, Sternberg, Marek, Sadowski, Belknap & Liebeskind, 1996). Sex-differentiated pain sensitivity is found in certain rodent strains but not others, and analgesic responses seem to differ across strains, both of which strongly indicate that genetic factors are involved. Mogil (2000) has reported identification of a genetic locus associated with male-specific thermal heat sensitivity, and of another locus which is linked to female-specific variability in non-opioid stress-induced analgesia (SIA). Mogil contends that genotype interacts with sex to affect pain sensitivity in rodents, and has suggested that a similar interaction could occur in humans.

2.2.4 Experimental pain and the female reproductive cycle

The research discussed above shows good evidence that sex-related differences in pain sensitivity exist, but it is not yet clear why. Gonadal hormone secretion has been investigated as a potential moderator of pain sensitivity which is also likely to exert differential effects on the pain responses of males and females. In particular, there is some evidence for altered female pain sensitivity as a function of hormonal fluctuation across the reproductive cycle. This female-specific mechanism may contribute to sex-related differences in pain responses and might also help to explain the inconsistency of such differences. Comparison of experimental pain responses across different phases of the menstrual cycle has been used to investigate this possibility in humans and some similar studies have been conducted across rodent estrous cycle.

2.2.4.1 Menstrual cycle and human pain sensitivity

In one of the first systematic investigations of the impact of menstrual cycle on experimental pain sensitivity, Herren (1933) reported that pressure pain threshold was reduced in the premenstrual phase. However, subsequent research has not always reiterated this phasic effect and conflicting findings have emerged, with some of this variation seemingly related to the type of noxious stimulus applied (Fillingim & Ness, 2000a). For example, Procacci, Zoppi, Maresca and Romano (1974) found increased radiant heat pain sensitivity in the luteal phase (premenstrually) and decreased

sensitivity at menstruation. By contrast, Hapidou and Cantanzaro (1988) have demonstrated increased cold pressor pain sensitivity in the follicular (postmenstrual) phase. Furthermore, some studies have found no changes in pain sensitivity across menstrual phase for ischemic, pressure, electrical or cold pressor pain (Amodei & Nelson-Gray, 1989; Veith, Anderson, Slade, Thompson, Laugel & Getzlaf, 1987). Other researchers have found greater sensitivity and discrimination of radiant heat pain at ovulation, relative to premenstrual and post-menstrual phases (Goolkasian, 1980, 1983). Fillingim and colleagues (1997) found that ischemic pain sensitivity was reduced in the follicular phase compared to the ovulatory and luteal phases, but that sensitivity to thermal pain did not show significant alterations across the menstrual cycle. It certainly seems that menstrual phase does not affect sensitivity for all types of pain equally.

Menstrual cycle effects on experimental pain sensitivity may depend not only on the pain induction method used but also on the body site stimulated. For example, Giamberardino, Berkley, Iezzi, de Bigontina and Vecchiet (1997) found increased muscular and subcutaneous abdominal sensitivity to electrical pain around menstruation, compared to other menstrual phases. Similarly, Isselee, de Laat, Bogaerts and Lysens (2001) recently reported that pressure pain thresholds of masticatory and hand muscles were reduced perimenstrually (when sex steroid hormone levels are lowest). Bajaj, Arendt-Nielsen, Bajaj and Madsen (2001) have reported that heat pain threshold on the abdomen and pressure pain threshold on the lower back were lower around ovulation than at other phases of the menstrual cycle (premenstrual, menstrual, or luteal). Bajaj and colleagues suggest that periovulatory elevations in estrogen and luteinising hormone may cause peripheral and central sensitisation, which leads to hypersensitivity in the abdomen and lower back at this point in the cycle. These somatic areas are presumed to be referral sites of menstrual pain, and these authors have argued that the release of a range of inflammatory substances during menstruation may function as a 'latent algogenic stimulus' which produces hyperexcitability in uterine nociception.

There is also some evidence that oral contraceptives (OCs), which create an artificially steady state in sex steroid hormone levels, may mask menstrual phase effects on pain perception. For example, Hapidou and Rollman (1998) found that normally menstruating women showed an increased number of tender points on the body during the follicular phase, whereas OC users showed no cyclic changes in somatic tenderness

(see also Goolkasian, 1980). This may be because many oral contraceptives exert their effects by suppressing the mid-cycle LH surge and thus preventing ovulation (Ferin, Jewelewicz & Warren, 1993). In this way, OC users should not be subject to the heightened pain sensitivity which has previously been found to accompany the periovulatory elevation of estrogen and LH in normal (non-OC) cycles (e.g., Bajaj et al., 2001). However, at least one recent study has found similar menstrual cycle effects on pain sensitivity both with and without OC use (Isselee et al., 2001).

In a recent meta-analysis, Riley, Robinson, Wise and Price (1999) concluded that overall females exhibit greatest sensitivity to experimentally-induced pain in the luteal phase of the menstrual cycle. Higher pain threshold and pain tolerance seems to occur in the follicular phase (with effect sizes ranging from small to moderate) for most stimuli (i.e., pressure pain, thermal heat pain, ischemic muscle pain and cold pressor pain) except electrical pain, which shows the opposite pattern. Methodological differences between studies (such as varied pain induction techniques and methods of menstrual phase definition) coupled with small sample sizes may be largely responsible for the divergent pattern of results that has emerged from the human research literature. Nevertheless, Riley et al., (1999) contend that the effects of menstrual cycle on female pain sensitivity are too large to ignore, and are likely to partially (though not wholly) explain sex-related differences in pain sensitivity overall.

2.2.4.2 Estrous cycle and rodent pain sensitivity

Similar to the human research, some estrous phase comparisons with rodents have shown cyclic variation in pain responses, but inconsistencies abound. For example, Drury and Gold, (1978: in Gatchel & Turk, 1999) reported that female hormone levels affected response to electrical flinch–threshold. Tail-flick latency (withdrawal from noxious heat) has also been found to alter across the rodent estrous cycle (Kepler, Kest, Kiefel Cooper & Bodnar, 1989). However, there have been conflicting findings regarding the phase of highest pain sensitivity. For example, Martinez-Gomez, Cruz, Salas, Husdson and Pacheco (1994) concluded that rodent pain sensitivity increases in metestrus (high progesterone but low estrogen) and estrus (low estrogen and progesterone) compared to proestrus (both hormones peak) and diestrus (estrogen rising, progesterone falling). Others have found that female rats are less pain sensitive (i.e., demonstrate higher pain thresholds) in proestrus and estrus relative to metestrus

and diestrus (Bradshaw, Temple, Wood & Berkley, 1999). The strongest findings indicate that pain sensitivity peaks in late proestrus and early estrus, when estrogen and progesterone are in transition from peak to low levels (see Fillingim & Ness, 2000b). Similar to humans, reproductive cycle phase in rodents seems to affect pain sensitivity but further research is needed to clarify the effects and hormonal mechanisms involved.

2.2.5 Gender differences in human analgesia

If sex-related differences in pain sensitivity exist, one practical consideration is in terms of male and female differences in analgesia. For example, a number of patient-controlled analgesia (PCA) studies have shown that men generally tend to take up more opioids than women post-operatively, whereas other PCA studies have found no sex differences in analgesic uptake (see Miaskowski & Levine, 1999, for review). Greater uptake suggests that opioid analgesia is less effective for men than for women. However, strictly speaking, these findings demonstrate gender-differentiated *use* of opioid analgesia but do not permit firm conclusions regarding sex-differentiated efficacy of opioids. Many factors other than its effectiveness can influence the amount of a drug an individual is willing to self-administer, such as attitudes to drugs generally or anxiety about the addictiveness of opiates.

Most PCA studies have involved mu-opioids, but other types of opioid may also be more beneficial for females than males. For example, kappa-opioids (e.g., pentazocine, nalbuphine and butorphanol) have been found to provide better pain relief for females than males (Gear, Miaskowski, Gordon, Paul, Heller & Levine, 1999). However, it seems that gender differences in responses to analgesics may be dependent on drug type. For example, ibuprofen has been found to significantly reduce experimental pain in men but not women (Walker & Carmody, 1998). A significant hindrance to identifying optimal analgesics for men and women respectively has been that, prior to the last decade, women were excluded from clinical drug trials. Ironically, this meant that not only was it impossible to determine whether analgesics were differentially effective for men and women, but also that drugs which provide better analgesia for women than men (such as kappa-opioids) were tested only on all-male samples. If male-only trials indicated weak analgesic effects, trials of such drugs were discontinued without ever testing their effectiveness for women.

Fortunately, the relative dearth of gender comparisons in analgesic effectiveness is now being redressed with the inclusion of female groups in clinical drug trials (see Holdcroft, 2002). Investigation of the influence of gonadal hormones on human analgesia is also needed; animal studies have indicated co-localisation of opioid and gonadal steroid receptors in several brain regions, and interaction between gonadal hormones and neurotransmitters involved in opioid analgesia (Kest, Sarton & Dahan, 2000). At present, it seems that opioids may generally provide better analgesia for women than for men, although the mechanisms underlying this difference remain unclear (Fillingim, 2002).

2.2.6 Sex differences in rodent analgesia

Although there has been more rodent than human research into the impact of sex on analgesic responses, the accumulated literature is still relatively limited and the findings are far from equivocal. Sex-related differences in antinociceptive responses have been found with certain opioid agonists in some studies but not others and the direction of such differences, when found, seems to vary with the substance used and the type of noxious stimulus employed (Miaskowski, Gear & Levine, 2000).

Most studies have examined the effects of opioid agonists such as morphine (which act on mu receptors) on rodent responses to noxious stimuli (see Miaskowski et al., 2000). Greater antinociception has been found with morphine and with alfentanil in males compared to females (e.g., Cicero, Nock & Meyer, 1997; Miaskowski et al., 2000). However, some research has found no sex differences in morphine responses in rats (e.g., Ali, Sharif & Elkadi, 1995). The antinociceptive effects of selective mu-opioid agonists were also found to be more effective for male rats with thermal heat pain, but not with electrical pain. Selective delta-opioid agonists also seem more beneficial to males than females, but only at high dosages, and for heat pain (Bartok & Craft, 1997).

Similar to the human research, sex differences in analgesia in the rat also seem to vary by drug substance. Whereas most rodent studies have found enhanced opioid antinociception (greater pain reduction) in males relative to females, some other analgesic agents are more effective for females than males. For example, males show greater cocaine-induced analgesia, whereas females show greater analgesia in response to nicotine (Craft & Milholland, 1998). This suggests sex-related differences in

physiological pain-inhibition systems other than opioid mechanisms (Sternberg & Wachterman, 2000).

There may also be a genetic component to sex differences in rodent responses to analgesics. Mogil, Chesler, Wilson, Juraska and Sternberg (2000) investigated sex differences in heat pain responses and analgesic response to morphine between outbred strains of rats and mice. They found that male rodents generally demonstrated greater morphine antinociception than females, but also that the existence, strength and direction of sex differences in pain responses and in analgesic response to morphine was strain-dependent. This was taken as clear evidence of the influence of genotype on thermal nociceptive sensitivity and of strain effects on morphine antinociception. Interestingly, estrous phase did not affect nociceptive sensitivity, although there was some evidence of interaction between genotype and estrous phase in morphine antinociception in female mice (Mogil et al., 2000).

Rodent opioid antinociception also seems to be affected by sex steroid hormones (Miaskowski, Gear & Levine, 2000). Evidence has come from studies of exogenous manipulation of hormone levels as well as research into the effects of estrous cycle on analgesic effectiveness. Some studies have shown that gonadectomised male and female rats show greater morphine antinoception than intact ones, and that ovariectomised female rats show greater morphine analgesia than intact males (see Fillingim & Ness, 2000). There has been some evidence of altered sensitivity to morphine in female rats across estrus phase (e.g., Kepler, Kest, Cooper, Kiefel & Bodnar, 1989), but it is not yet clear in which phase morphine is most effective (Miaskowski et al., 2000). Generally, the rodent research indicates that elevated estrogen, either alone or with elevated progesterone, is associated with reduced analgesic responses to opioids (Fillingim & Ness, 2000b).

Taken together, the experimental research literature on pain responses in humans and rodents indicates that females are generally more sensitive to noxious stimuli than males, may also benefit less from some analgesics and may experience alterations in pain sensitivity associated with reproductive cycle. These factors may partly account for the greater susceptibility of women to pain which is documented in the epidemiological and clinical literature (Fillingim & Maixner, 1995). In light of this, an important task for

pain research is to ascertain what the underlying mechanisms of enhanced pain sensitivity in females may be.

2.3 Potential mechanisms of gender differences in human pain responses

A broad division is often made between psychosocial factors in pain responses (such as gender-role expectancies, beliefs, attitudes, affective states and coping style) and biological factors (such as genetically-mediated neurological and anatomical characteristics, and gonadal hormones). The dichotomy is an artificial one - it is more realistic to think of these factors as enmeshed and mutually interactive - but for research purposes separate evaluation of biological and psychosocial factors may help to tease apart their respective impact on pain responses. Berkley and Holdcroft (1999) have argued that psychosocial factors, such as the greater willingness of women to report pain and seek medical help, affect sex-differentiated pain prevalence rates but many of the factors which govern gender-differentiated pain sensitivity are likely to have a biological basis. Research has substantiated this assertion. For example, Ellermeier and Westphal (1995) found that gender differences in pain sensitivity were demonstrable using an autonomic indicator of pain (outside voluntary control). In their study, females showed greater pupil dilation during pressure pain than males, which was interpreted as physiological evidence for gender differences in sensory and/or affective pain mediated by biological mechanisms. Similarly, functional imaging research has shown that, although the same brain areas are activated in males and females during noxious heat stimulation (including prefrontal cortex, insula and thalamus), females not only rate the stimulus as more intense than males but also demonstrate significantly greater activation in some of these brain regions (Paulson, Minoshima, Morrow & Casey, 1998; Casey, 1999, 2000). Evidently, sex-differentiated pain processing is detectable at central nervous system level.

The next section will outline the putative biological mechanisms underlying gender differences in pain perception, and will be followed by an overview of psychosocial factors which are likely to contribute to such differences.

2.3.1 Biological factors

The relative hypersensitivity to pain found in women echoes sex-related differences observed in other sensory modalities. For instance, the sensory detection thresholds of

women are also lower than those of men in the gustatory, olfactory, auditory and, importantly, the tactile modality (Aloisi, 2000). This suggests a biologically-mediated perceptual disparity between the sexes (Berkley, 2000), and perhaps indicates an innate basis for female hyperalgesia. In support of this hypothesis, Guinsberg, Peres, Almeida, Balda, Berenguel, Tonelotto and Kopelman (2000) found that newborn girls show more facial expression of pain than newborn boys during and just after a painful procedure, a sex-related difference observed before any possible effects of culture or learning and hence presumed to be biologically-mediated. However, most studies of pain responses in children have not found sex-related differences (e.g., Meier, Berde, Di Canzio, Zurakowski & Sethna, 1999) or have found differences in behaviours such as crying, but none in subjective pain or physiological correlates of pain (e.g., Bournaki, 1997). Furthermore, while some research has found that girls demonstrate more pain behaviours than boys during the same painful procedure (e.g., Fowler-Kerry & Day, 1993), others have found the opposite pattern (e.g., Gruneau & Craig, 1987). The fact that gender differences in pain responses are neither consistent nor clearly evident from birth shows that a generalised sensory disparity between males and females is not the singular cause of such differences; multiple and interactive underlying mechanisms are more likely. Generally, there seem to be less consistent gender differences in pain sensitivity in childhood than in adulthood, which suggests that factors linked to maturation are involved.

2.3.1.1 Reproductive biology

Sex-related differences in pain sensitivity seem to be closely connected with reproductive biology. For example, the painful manifestations of many diseases change across reproductive life stages and alterations in experimental pain sensitivity associated with puberty, pregnancy, menstrual cycle phase and hormonal medication have been observed in humans and other animals (Fillingim & Maixner, 1995; Berkley, 1997; Fillingim et al. 1998).

There are many reproductively-linked physiological differences between males and females which are likely to contribute to gender differences in pain. For example, stark differences in male and female reproductive anatomy lead to sex-specific pains in the pelvic region (Wesselman & Burnett, 1999) and women experience more frequent pain from pelvic viscera than men (Bonica, 1990). Although painful conditions specific to male anatomy do occur, such as testicular pain caused by trauma or infection, male pain

in general is far more likely to be acute and treatable, and men do not seem to suffer regular pain in the absence of pathology (see Unruh, 2002). According to Berkley (1997) the female pelvic region, which has a more complex internal anatomy than that of the male, is particularly likely to sustain trauma or disease, and thus to be a site or source of female pain. Berkley (1997) has argued specifically that the vagina and uterus constitute a conduit into the body for injury and/or potential pathogens (e.g., tampons, sexual intercourse, medical examination, pregnancy, parturition). Injury or disease of pelvic organs, which are heavily innervated by C-fibres, can result in peripheral and central sensitisation, and this in turn can produce referred pain and hyperalgesia. In addition, women experience alterations in digestion, metabolism, urinary function, cardiovascular function and temperature regulation associated with their reproductive lifecycle (Berkley & Holdcroft, 1999). These sex-specific physiological effects may bear not only on the sex prevalence of painful illnesses but also on the effectiveness of therapeutic measures, for instance by altering the pharmacokinetics and pharmacodynamics of analgesic medications.

There are certainly various painful experiences unique to women which are caused by reproductive processes or diseases. Labour pain, post-partum pain and pain due to pelvic inflammatory disease are examples of female-specific pain arising from reproductive viscera (Giamberardino, 2000). Other causes of female-specific pain include miscarriage, ectopic pregnancy or pathology of reproductive organs such as uterine or ovarian cancers (Unruh, 1996). Aside from these various forms of pain with pathological origins, many adult females are likely to experience pain linked to normal reproductive processes, such as pre-menstrual breast tenderness, dysmenhorrhea and perimenstrual headache. Moderate to severe pain associated with menstruation (dysmenorrhea) is an extremely prevalent form of recurrent pain for many women of reproductive age (Unruh, 1996). It has been estimated that by late adolescence over 70% of girls experience dysmenhorrhea, which is generally most severe at that age (Andersch & Milsom, 1982). Dysmenhorrhea, which is believed to be due to prostaglandin action increasing uterine contractility and sensitising nerve endings (but is not associated with pelvic abnormality) can be very intense for some women. Such recurrent visceral pain can be accompanied by hyperalgesia in referral areas. For example, Giamberardino, Berkley, Iezzi, de Bigontina and Vecchiet (1997) found that electrical pain threshold in abdominal muscle was reduced perimenstrually and raised in

the luteal phase of the menstrual cycle, and that this effect was more pronounced in dysmenhorrheic women. Subsequent studies have demonstrated that this perimenstrual reduction in pain threshold in muscle is greater in long-term dysmenhorrheics than in women who had only experienced this type of recurrent pain for a few years, indicating a sensitisation proportional to the duration of previous painful experience (see Giamberardino, 2000).

Such repetitive pain experiences could cause a female-specific form of central sensitisation in both the spinal cord and brain, leading to a long-term increase in female pain sensitivity (Berkley, 1997). Prolonged or recurrent painful experiences can alter neural receptive fields and firing responses (Melzack & Wall, 1996) and subsequently ambiguous stimuli may be interpreted as painful and noxious stimuli experienced as more intense. Recent research indicates sex-differentiated patterns of neural response to noxious stimuli which may contribute to such sensitisation. For example, greater temporal summation of mechanical pain (Fillingim, Maixner, Kincaid & Silva, 1998) and heat pain (Sarlani & Greenspan, 2001) has been found in women than in men. Increased temporal summation involves CNS-mediated upregulation of neuronal excitability in the dorsal horn nociceptors, and may be involved in the greater female risk of developing of pathological chronic pain (Staud, Vierck, Cannon, Mauderli & Price, 2001). In addition, functional neuronatomy research has indicated that there may be sex-specificity both in neurological layout and in patterns of recovery from injury, in both sympathetic and central nervous systems (see Berkley, 1997). For example, structural differences between males and females have been found in brain regions directly associated with reproduction (Breedlove, 1994) and gonadal hormones seem to be involved in neural plasticity of the sympathetic nervous system following injury (Demotes-Mainard, Vernier & Vincent, 1993).

2.3.1.2 Gonadal hormones

An aspect of reproductive system function which may have particular importance to sex-related differences in pain perception is the secretion of gonadal steroid hormones. Gonadal hormones have profound effects on CNS function throughout life, affecting neuronal structure through genomic action on DNA, as well as directly affecting neuronal firing patterns in multiple brain regions (see Aloisi, 2000). Research with human and rodent populations has indicated that alterations in gonadal steroid hormone

levels have widespread effects on the neurotransmitter function involved in nociceptive processing at both peripheral and central nervous system sites. For example, estrogen and progesterone affect the receptive fields and other response properties of certain neuronal groups, including primary afferent pathways from the periphery into the dorsal horn of spinal cord (see Bradley & Alarcon, 2000). Gonadal hormone levels also influence many CNS mechanisms involved in pain transmission, for example affecting levels of neuromodulators which are involved in nociceptive processes such as substance P, GABA, glutamate, dopamine, serotonin and norepinephrine (Fillingim & Ness, 2000a). There is also a close association between the autonomic nervous system (ANS) and the endocrine system, and there is some recent evidence to suggest both sex differences and menstrual cycle alterations in ANS activity (see Naliboff, Heitkemper, Chang & Mayer, 2000).

In addition, reciprocal interaction between endogenous opioids and gonadal hormones may influence both baseline pain sensitivity and responses to analgesics through lowered endogenous pain inhibition and altered receptor activity (Holdcroft, 1997). The interaction of hormones and opioid systems in pain modulation has been empirically demonstrated. For example, recent animal research has shown that estrogen can affect pain sensitivity by influencing the secretion of enkephalin, an important opioid component of endogenous pain inhibition in the spinal dorsal horn (Amandusson, Hallbeck, Hallbeck, Hermanson & Blomqvist, 1999). Overall, it seems that sex steroid hormone levels affect multiple inhibitory and excitatory neural systems and so influence both nociception and antinociception.

Estrogen, progesterone and testosterone are functionally active in both males and females, but the relative levels of these gonadal hormones and the patterns of fluctuation in their secretion across the lifespan differ greatly between the sexes. In particular, post-puberty there are far more frequent and dynamic changes in sex steroid hormone levels in the female body than the male. For females, large shifts in estrogen and progesterone secretion occur at several key stages: puberty, pregnancy, menopause, and senescence. In addition, there are radical alterations in female hormone levels within each menstrual cycle during the reproductive years. For males, although puberty itself features dramatic hormonal shifts, there is relative hormonal stability post-puberty until testosterone levels begin to decrease as a function of ageing (Berkley & Holdcroft, 1999). According

to Berkley (1997), these substantial sex differences in the pace and extremity of hormonal fluctuation affect male and female pain perception in adulthood.

Alterations in the sex prevalence of painful disorders across the lifespan probably signify the impact of changing hormone levels (LeResche, 1999). Support for this hypothesis comes from the fact that gender differences in pain prevalence mainly emerge around adolescence (Von Korff, Dworkin, LeResche & Kruger, 1988) and differences in male and female pain sensitivity become more evident post-puberty (Rollman, Lautenbacher & Jones, 2000). Notable examples are migraine and temporomandibular pain (both much more commonly experienced by females than males) which show a sharp rise in incidence around puberty (LeResche, 1999). Similarly, the prevalence of migraine for women decreases post-menopausally, but still remains higher than for men (Unruh, 1996).

Less is known about the effects of sex steroid hormones on pain sensitivity in males than in females, but there has been some evidence that testosterone is inversely related to pain (see Fillingim, 2001). Angina pain becomes more prevalent in men as testosterone secretion declines with age (Berkley, 1997) and testosterone therapy significantly reduces pain and limitations on physical activity (English, Steeds, Jones & Diver, 2000). However, there are also contradictory findings such as the typical onset of male cluster headaches at puberty (when testosterone increases) and the decrease of headache and abdominal pain in older men (Berkley & Holdcroft, 1999). Consistent with their lower hormonal lability, men seem to experience fewer alterations in pain sensitivity across the lifespan than women (Berkley, 1997).

The female reproductive cycle in humans and other mammals is a function of hypothalamic-pituitary-ovarian interaction and is characterised by hormonal fluctuation. Several lines of evidence suggest that hormonal status may be a particularly important factor in female pain perception (Miaskowski, 1999). For example, pain sensitivity has been found to alter across reproductive cycle phase in both human and rodent females. Moreover, rodent research has shown that hormonal alterations, both exogenous (e.g. ovariectomy) and endogenous (e.g., estrous cycle) affect endogenous analgesic responses such as stress-induced analgesia (SIA). In addition, as outlined earlier in this

chapter, hormonal conditions appear to influence the effectiveness of certain analgesics (Fillingim & Ness, 2000).

According to Berkley (1997) the fluctuation of hormones during the female ovarian cycle profoundly influences pain perception. For example, many functional gastro-intestinal (GI) disorders, including irritable bowel syndrome (IBS), are more prevalent in females than males after puberty, manifest different symptom patterns in women and men (Lee, Schulson, Mayer, Chang & Naliboff, 1999) and seem to be particularly affected by cyclic alterations in endogenous hormone levels. Fluctuation of estrogen and progesterone across the menstrual cycle affects GI function and pain, such as transit time and IBS-related abdominal pain. In addition, women report more GI symptoms during menses than in other phases of the cycle, and such symptoms are rated as more painful by IBS sufferers than by controls, which suggests an enhancement of abdominal sensitivity when hormone levels fall perimenstrually (Heitkemper & Jarrett, 1992). However, it is not yet clear whether menstrual cycle affects IBS symptoms by effects on gut motility (via autonomic nervous system) or interaction with endogenous opioid systems (Naliboff, Heitkemper, Chang & Mayer, 2000).

Holdcroft (1997) has proposed that interaction of female-specific hormone secretion with endogenous opioid systems affects both pain perception and analgesic responses. Some research supports this contention. For example, there are female-specific endogenous analgesic mechanisms, which are hormone-sensitive and involve particular CNS pathways and neurotransmitters (Fillingim & Ness, 2000). One of these is pregnancy-induced analgesia (PIA) which is seen in many species including humans, and usually peaks just before parturition. PIA can be artificially produced by simulation of the hormonal milieu of pregnancy, can be reversed by opioid antagonists and involves selective activation of opioid receptors (Gintzler, 1980; Dawson-Basoa & Gintzler, 1996). The other is vagino-cervical stimulation-produced analgesia (VSPA), which has been observed in humans (Whipple & Komisaruk, 1985) and rodents (Komisaruk & Wallman, 1977) and can be manipulated by artificial modulation of sex steroid hormones and adrenergic agonists. Both of these mechanisms demonstrate the interaction of hormones with neurotransmitters in a female-specific form of endogenous analgesia.

2.3.1.3 Estrogen, progesterone and pain

Research has demonstrated that estrogen and progesterone do affect pain responses, but has not conclusively shown what the exact effects of these hormones are. For example, depletion of estrogen may lead to an increase in some types of pain. The occurrence of most female migraines coincides with the radical fall in estrogen which precedes menstruation (see Holroyd & Lipchik, 2000). Although the exact mechanisms are unknown, the drop in estrogen might trigger migraine through effects on neurotransmitter levels (which could alter vascular function at cerebral level) and/or through impact on endogenous opioid systems. In addition, female joint pain and vaginal pain are both found to increase when estrogen levels fall post-menopausally Confusingly, there is also recent evidence that estrogen has pronociceptive impact (Fillingim, 2001). For example, the incidence of abdominal pain, migraine and tension headaches decreases in post-menopausal women (Berkley & Holdcroft, 1999). Furthermore, estrogen therapy has been associated with enhanced clinical pain of several types, including back pain (Musgrave, Vogt, Nevitt & Cauley, 2001), temperomandibular pain (LeResche, Saunders, Von Korff, Barlow & Dworkin, 1997) and orofacial pain (Wise, Riley & Robinson, 2001).

Similarly, there is conflicting evidence concerning the effects of progesterone. Some research indicates that progesterone may be associated with analgesia. For example, certain painful conditions (e.g., migraine) have been found to improve or disappear during periods of elevated progesterone secretion such as pregnancy, the mid-luteal menstrual phase or lactation (Giamberardino, 2000). Furthermore, some anaesthetics (e.g., alphaxalone) have a progesterone base, which adds weight to the proposition that this hormone may attenuate pain (Berkley & Holdcroft, 1999).

Other studies suggest progesterone may contribute to increased pain sensitivity. Clinical studies have found increases in pain report in postmenopausal women on Hormone Replacement Therapy (e.g., Wise et al., 2000) and a recent study found that such women have lower thresholds and tolerance to experimental thermal pain than women not on HRT or men (Fillingim & Edwards, 2001). HRT, which is used to treat the negative physical consequences of menopause, exerts its effects by artificially increasing estrogen and progestin levels. The few investigations to date suggest that women taking HRT may become more pain-sensitive and more prone to pain. However,

it is worth noting that some of the pains which worsen post-menopausally, such as joint pain and back pain, will be at least partly attributable to mechanical damage - the 'wear and tear' of ageing - which makes it difficult to isolate the effects of declining hormone levels. Similarly, the effects of the supplementary hormones on pain are not easy to assess, since the women for whom HRT is prescribed are, by definition, experiencing a natural decline in sex steroid hormones but are also likely to be increasingly at risk of age-related painful illness (e.g., arthritis).

2.3.2 Psychosocial factors

While it seems clear that biology lays down a physiological framework for sex-differentiated pain perception, there is growing consensus that psychological factors are also powerful determinants of male and female pain experiences. According to Berkley (2000), while the greater vulnerability and sensitivity to pain seen in women seems to have a strong biological basis, the variability and inconsistency of gender differences in pain responses is probably due to the influence of psychosocial factors. The remainder of this chapter will briefly discuss the impact of various psychological and social variables on pain, with particular reference to their potential contribution to gender differences in pain responses. The subsequent chapter will provide a detailed discussion of coping with pain, the psychosocial mechanism which, together with gender, is the focus of the research reported in this thesis.

2.3.2.1 Emotion and pain

That emotion is both a contributory factor and an experiential component of pain is well-established and the potential for negative emotion (such as anxiety) to worsen pain has been empirically demonstrated (Melzack & Wall, 1996). For example, experimentally-induced negative affective states have been shown to exacerbate pain responses (Cornwall & Donderi, 1988). Anxiety is considered particularly relevant to acute pain (Chapman & Turner, 1990) as it provokes a range of physiological responses and has been found to increase pain perception (Bonica, 1990). The greater propensity of females to experience mood disorders generally, and to respond to pain with more negative emotional responses than their male counterparts, may therefore be linked to gender differences in pain perception (Robinson, Riley & Myers, 2000). For example, some research suggests greater female anxiety and fear of potentially painful experiences such as dentistry (see Rollman, Lautenbacher & Jones, 2000). In addition, female chronic pain patients tend to score higher on measures of anxiety and depression

than their male counterparts (Jensen, Nygren, Gamberale, Goldie & Westerholm, 1994). However, such findings could reflect a stronger tendency for women to express emotion generally rather than gender differences in negative affect per se.

2.3.2.2 Hypervigilance and pain

Rollman (1998) has proposed that a propensity to be hypervigilant to pain might be an even more important determinant of the enhanced pain sensitivity found in women than biological factors. Hypervigilance is a generalised pattern of heightened focus and responsiveness to internal or external discomfort which involves perceptual, affective and cognitive processes (Rollman et al., 2000) and is likely to affect pain report. There is some evidence that women appraise bodily feelings and describe aversive sensations differently than men, and that women report more intense, numerous and frequent somatic symptoms (Barsky, Peekna & Borus, 2001). On this basis, the fact that women consult health-care services for pain more than men (Skevington, 1995) may be a function of many interacting factors such as higher female morbidity for painful diseases, generally enhanced somatic awareness in women relative to men and gender-related models of illness behaviour.

2.3.2.3 Gender-normative influence

The state of being male or female in human society is not simply a matter of biological inheritance, it is also equivalent to belonging to a culturally-defined group. Indeed, gender has been called a 'scheme for the social categorisation of individuals' (Sherif, 1982). As such, there are likely to be broad expectations of differences in male and female behaviour when in pain, and perhaps consequent gender-related differences in coping with pain overall. According to gender schema theory (Bem, 1981) cultural norms for the sexes make it likely that men and women will be motivated to respond differentially to stressors, including painful experiences. Gender differences in human pain responses are therefore likely to be partly attributable to reporting and/or response biases arising from psychosocial processes (such as learnt behaviours). Research bears this out; men and women seem to differ in their beliefs, opinions and attitudes about pain, including those regarding the impact of gender on pain. For example, Bendelow (1993) investigated perceptions of pain, gender and social beliefs about health, illness and pain characteristics, using questionnaires and interviews, and found that more women than men thought that anxiety, fear and depression affected their pain

perception. Interestingly, only half as many men as women completed the questionnaire items which assessed this, perhaps indicating a male bias away from considering, or expressing their thoughts about emotion and pain. The study also confirmed that differential socialisation of males and females in childhood may affect perception of and reaction to pain in adulthood; males reported they felt encultured to be stoic in the face of pain (Bendelow, 1993). Research also suggests that females may acquire greater awareness of pain through social modelling processes within families (Koutanji, Pearce & Oakley, 1998).

Gender differences in pain behaviours which reflect gender-specific socialisation, such as willingness to disclose pain, will affect not only epidemiological statistics of pain prevalence but also self-report in clinical or experimental settings (LeResche, 1999). It seems as if such gender-role influence may exert stronger (or perhaps qualitatively different) effects on men than on women. For example, Levine and DeSimone (1991) found that men reported lower experimental pain ratings to a female researcher than to a male researcher, whereas the responses of women did not differ according to the gender of the researcher. This effect is not universal, as some studies have found that gender differences in pain responses are unaffected by experimenter gender (e.g., Feine, Bushnell, Miron & Duncan., 1991). However, it does seem that men might be especially prone to behaviour which is consistent with perceived gender roles and that this precludes male candour about pain even if the researcher is not female. Unruh, Ritchie and Merskey (1999) found men were significantly less likely than women to report to a male interviewer that they had looked for comfort, cried or moaned during recent pain.

Gender differences in attitudes to pain may promote unrealistic expectations. For example, some research has shown that males and females differ in anticipatory estimates of pain but not in report of actual pain experienced (Fowler-Kerry & Lander, 1991). Furthermore, assumptions about gender and pain may affect not only the pain perception of the individual but also the beliefs (and consequently the actions) of those who treat their pain. For instance, it is commonly believed that because pain often accompanies reproductive processes such as menstruation and childbirth, women are somehow biologically equipped to cope better with pain than men (Bendelow, 2000). In one study, two-thirds of female participants and one-third of males expressed this view (Bendelow, 1993). There has been evidence of this type of bias in healthcare

professionals; one study found that most nurses held opinions about gender differences in pain sensitivity which led them to assess the pain of men and women in their care differentially (McCaffery & Ferrell, 1992). There may be particular negative ramifications for women in pain, such as a propensity to undertreat female pain; an ironic possibility in light of the apparent likelihood that women experience more pain than men. Indeed, several studies have shown that some physicians and nurses prescribe and administer less and weaker analgesic medication to women than to men (e.g., Beyer, DeGood, Ashley, & Russell, 1983).

Men and women do seem to differ in their pain-related cognition and emotion. For example, while women tend to be more worried and irritated by pain, men are more likely to respond emotionally to pain with embarrassment (Klonoff, Landrine & Brown, 1993). Such responses are likely to affect how they appraise and cope with pain (Unruh, 1996). Coping style can significantly affect responses to pain, and there is some evidence that males and females cope differently. Some studies have indicated that coping style shows early in life and tends to be congruent with traditionally-perceived gender roles. For example, school age girls have reported that they cry, moan or seek comfort when in pain, whereas boys reported they distract themselves (cognitively or behaviourally) and engage in more problem-solving than girls (e.g., Reid, Gilbert & McGrath, 1994; cited in Unruh, 1996). This is directly relevant to one of the main research objectives of this thesis, which is to investigate the relative effectiveness of different types of coping for men and women. A more detailed discussion of the existent research into coping with pain will constitute the next chapter.

2.4 Summary

Several key points emerge from this selective review of research into sex and gender-related differences in pain. Firstly, sex-related differences in pain perception are well-documented overall but conflicting findings abound in both the human and rodent literature. In both human and animal research, methodological variation has contributed to inconsistencies in findings and made it very difficult to generalise or conclude from any single part of the literature. Nevertheless, sex-related differences in pain responses have been replicated in rodents as well as humans, which advances the proposition that such differences are at least partly biologically based.

While there is convincing evidence for a biological infrastructure to gender differences in human pain responses, this is not fully explanatory. It is becoming evident that sex-related differences in pain have multiple and interactive underlying causes. Anatomical and physiological differences between males and females affect neural pain mediation mechanisms, and in humans at least, concurrent psychological processes affect pain perception as well as motivation and behaviour in the context of pain. It follows that any gender differences in pain responses will be a product of the interaction of these factors.

On the basis of existing knowledge, the situation for women with regard to pain perception appears to be particularly complex. A body of evidence is emerging which suggests that women are likely to experience more occurrences and recurrences of pain across a lifetime than men, are more susceptible to painful illnesses, and are likely to experience pain associated with reproductive processes. It also seems females may also be more pain–sensitive than their male counterparts, due to neurophysiological and hormonal factors. Recent research, with humans and with rodents, has indicated an important role for gonadal hormones in pain perception. It seems that sex-steroid hormones exert multiple peripheral and central effects which can modulate both nociceptive processing and analgesic responses. Male gonadal hormone levels in healthy adulthood remain very stable compared to those of females, which reduces opportunities for investigation of their effects on male pain perception. Paradoxically, while it is widely believed that women are biologically adapted to cope with pain, they may generally manifest certain cognitive and perceptual characteristics which exacerbate pain. It appears that for men, the experience of pain across the lifetime is less likely and less severe than for women. However, this may not accurately represent the actual pain experiences of men, as they generally tend to express emotion, and probably pain, less readily than women at least in part due to gender-role normative pressure to be 'brave' or stoic. Such pressure could affect the candour of male pain report whether they are reporting to a man or a woman.

The psychosocial factors involved in gender-differentiated pain responses which have been briefly discussed above suggest that that both sex-specific pain reporting style and coping tendencies are part of self-reported and behavioural responses to pain. One of the primary objectives of my own research was to test the relative effectiveness of different

forms of cognitive coping instructions for men and women in an experimental pain paradigm. Accordingly, Chapter 3 will draw on the substantial body of research into the influence of coping strategies on pain responses - with particular reference to gender - to provide a context for the experiments in this thesis.

Chapter 3

Coping with Pain

3.1 Introduction

As the principles of Gate Control theory predicted, and subsequent research has verified, the generation of pain experiences is only loosely associated with tissue damage. Presently, the most credible perspective is an integrative and interactive biopsychosocial configuration, in which psychological and social as well as physiological factors are powerful, interlinked determinants of pain. The theoretical importance of the affective-motivational and cognitive-evaluative dimensions of pain perception is further actualised in the strong influence of these factors on the ways in which individuals cope with pain in real life. It seems that coping with pain is particularly a function of idiosyncratic psychological characteristics of the individual such as their attitudes, beliefs and emotional states, as well as exogenous factors such as the circumstances and nature of the noxious stimulus. Furthermore, research indicates that certain methods of coping can directly affect the level of pain experienced, both negatively and positively (Boothby, Thorn, Stroud & Jensen, 1999). For example, coping can alter both the perception of pain and autonomic responses during noxious stimulation (Thompson, 1981; Weisenberg, Schwarzwald & Tepper, 1985). Coping strategy or style can also directly exacerbate or attenuate the level of acute pain experienced during painful medical procedures (Weisenberg, 1999). Ways of coping may also impact on pain perception more indirectly (or at least more gradually) by improving or worsening adjustment to living with chronic pain (Skevington, 1995).

This chapter provides an overview of research into coping with pain, including the types of coping strategy commonly used to deal with pain, and the efforts which have been made to ascertain which of these are most beneficial. The role of attention in coping strategies is also discussed, as is evidence for gender differences in coping with pain. A selective abstraction of the existent research on coping with pain is made with pertinence to the experimental work in this thesis, and as such, is not intended as a comprehensive review of the extensive coping literature.

3.2 Definition and theory of coping

In general parlance, coping is dealing effectively with something difficult. Within psychology more specific definitions of coping have been generated, often derived from the study of stress. For example, coping has been defined as: "cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing

or exceeding the resources of the person" (Lazarus & Folkman, 1984, p.141). Coping has also been termed purposeful effort to manage or vitiate the negative impact of stress (Jensen, Turner, Romano & Karoly, 1991).

Theoretical perspectives on coping developed in the context of stress research can be appropriately applied to pain, which is considered a universal form of stress (Gatchel & Turk, 1996). One such theory is Lazarus and Folkman's (1984) transactional model of stress, which posits that 'dispositional variables' (such as personality, social roles and biological factors) in conjunction with an ongoing series of appraisals, affect how people react to and cope with stressors. In this model, primary appraisal processes lead to categorisation of a potential stressor as either irrelevant, benign or stressful. If it is appraised as stressful, further evaluation will adjudge it a threat, a challenge, or likely to involve harm or loss, and this will in turn elicit emotional responses. Appraisals of potential harm are likely to trigger negative emotion such as anxiety or fear, whereas challenge appraisals might evoke positive emotion such as excitement. According to Lazarus and Folkman (1984) secondary appraisals comprise an individual's beliefs about coping methods and their likely effectiveness. Coping responses follow, which in turn will have far-reaching effects on many aspects of everyday life, such as level of activity, physical condition, morale and social functioning (Boothby et al., 1999).

In Lazarus and Folkman's model then, a noxious stimulus will be stressful only if it is initially appraised as unmanageable, and the emotions engendered during appraisal affect whether and what type of coping efforts are made. A key point for pain coping is that noxious stimulation deriving from whatever source (whether endogenous or exogenous) is not received by a passive organism. More realistically, each person has idiosyncratic - and to some extent alterable - psychological and social characteristics which are a product of their biological inheritance and their learning experiences, and which will affect both their perception of pain and their responses to it. Individual differences will clearly have great impact both on pain and on coping, therefore the utility of different coping strategies will depend on many factors and is likely to vary between individuals, as well as between situations.

The meaning attributed to pain is also likely to affect individual attitudes to coping. For example, acute pain experienced during dental treatment carries very different meaning

to chronic pain due to disease. Although both of these forms of pain may elicit emotional responses such as anxiety and fear, in the former these are likely to be anticipatory and temporary whereas in the latter they may become as chronic as the pain itself, to such an extent that they become psychopathological (see Strong et al., 2002). Furthermore, because repetitive experiences of acute pain can produce neurophysiological alterations leading to persistent pain after healing (Hawthorn & Redmond, 1998), chronic pain can develop in the absence of disease, yet the sufferer may continue to interpret hurt as harm (Cipher & Fernandez, 1997). Transient pain (e.g., experimental pain) has temporal parameters and/or rewards which contribute to its more positive connotations. For example, the dental patient anticipates short-term discomfort from a procedure that will benefit their oral health, whereas the chronic pain sufferer has no time limit by which to rationalise their coping, and no health benefits to construe.

Hence, when researchers examine the effects of coping strategies for pain in a laboratory setting, as has been done in the experiments reported in this thesis, there is both advantage and limitation in the known meaning of the pain involved. The advantage is a methodological one; standardised experimental pain induction with safety assurances and informed consent can be fairly assumed to have similar meaning for all participants. The limitation is one of ecological validity; findings from experimental pain induction with healthy, pain-free individuals may not be generalisable to clinical or chronic pain patients for whom the meaning of pain is likely to be infinitely more complex and varied, and inevitably more negative.

3.3 Cognition, coping and pain

Recognition of the importance of psychological factors in pain has promoted research into cognitive processes in the context of coping with pain. Weisenberg (1999) has stated that cognitive approaches are concerned with the way the person perceives, interprets and relates to the pain "rather than elimination of the pain per se" (p.345). This somewhat underplays the dynamic modulatory involvement of cognition in the perception of pain as posited in Gate Control theory. However, even if cognitive coping does simply render pain less distressing and/or more manageable rather than actually reducing or abolishing it, continued efforts to refine cognitive strategies would nevertheless have intrinsic value. In fact, there is now a considerable body of literature

which attests that cognition clearly can affect pain perception, both as a factor in the inception of pain, and as a therapeutic component of pain treatment. Furthermore, recent functional imaging research has indicated that the modulation of pain processing by cognitive mechanisms involves activation and probably interaction of specific brain regions (see Petrovic & Ingvar, 2002). The cognitive processes involved in pain and coping include attention, imagery, expectation, memory and schemata (LeResche, 1999).

A diverse collection of strategies for coping with pain has accumulated, many of which involve psychological processes. Some examples are: ignoring pain, prayer, relaxation, controlled breathing, hypnosis, analgesic medication, ingestion of alcohol, physical exercise, bed rest, and a variety of cognitive strategies including various methods of distraction. Cognitive methods, which involve attentional processes and may also incorporate imagery and/or internal dialogue, aim to directly modify thought in order to attenuate pain (Tan, 1982). The last decade has seen an increase in the application of cognitive coping strategies to many kinds of painful health conditions and medical procedures, including low back pain, rheumatoid arthritis, cancer pain, bone marrow aspiration and lumbar puncture, neck and shoulder pain, headache and abdominal pain in children, temporomandibular joint pain, limb pain, burns, and burning mouth syndrome (Weisenberg, 1999).

Various coping strategies develop spontaneously when people are in pain. Indeed, it seems improbable that any individual could fail to have psychological (as well as physical) reactions to feeling pain, which constitute instinctive attempts to cope with the experience. Spontaneous cognitive coping strategies reported during painful experiences include praying, reinterpreting pain, ignoring pain, distraction and positive appraisal (Boothby et al., 1999). However, the kind of spontaneous coping which occurs in response to pain is not always beneficial. For example, Chaves and Brown (1985) found that 44% of dental patients engaged in cognitive strategies such as attention diversion and coping self-statements during treatment, but that 37% of the sample catastrophized in some way during the dental procedure. Catastrophizing about pain is defined as an exaggerated negative orientation toward noxious stimuli (Sullivan, Bishop & Pivik, 1995). This type of cognition during noxious stimulation has been associated with intensified pain and greater distress (Sullivan, Thorn, Haythornthwaite, Keefe, Martin,

Bradley & Lefebvre, 2001), so these patients may have inadvertently exacerbated any pain experienced during their treatment. As we shall see, coping responses which involve exaggerated attention to pain (such as catastrophizing and hypervigilance) are generally disadvantageous and may be involved in gender differences in pain responses.

3.3.1 Classification of coping strategies

Various categorisation schemes have been proposed to group coping strategies into broad types. As these have often been produced via factor analysis of a particular coping measure, the resultant categories are necessarily derivative of the instrument used, but there is thematic overlap between different schemes. For example, Tan (1982) listed six types of coping: imaginative inattention (evocation of imagery incompatible with pain), imaginative transformation of pain (interpretation of the subjective experience as other than pain), imaginative transformation of context (acknowledgement of pain with transformation of setting), attention-diversion to external events (attentional focus on an aspect of the environment), attention-diversion to internal events (attentional focus on self-generated thoughts) and somatization (focus on painful area with detachment). In a similar typology, six groups of cognitive strategies specifically for coping with pain were identified by Fernandez and Turk (1989): external focus of attention, neutral imagery, dramatised coping, rhythmic cognitive activity, pain acknowledging and pleasant imagery.

Superordinate classifications have also been applied to coping typologies, often in the form of dichotomous groupings. For example, coping methods have been categorised as: confrontation, distancing, self-control, seeking social support, escape or avoidance, accepting responsibility, rational problem solving and positive reappraisal, and these can be further distinguished as either problem-focused or emotion-focused modes of coping (Folkman & Lazarus, 1980, 1985). Both these types of coping are thought likely to be used by most people (Weisenberg, 1999) but their relative usefulness may depend on the appraised controllability of the situation. For example, Forsythe and Compas (1987) found that problem-focused coping was associated with less distress in situations perceived as controllable, but with more distress in situations appraised as uncontrollable. The reverse pattern was found for emotion-focused coping.

Efforts to identify the most effective ways of coping with pain have led to dichotomous classifications of strategies as adaptive or maladaptive (e.g., Jensen, Turner, Romano &

Lawler, 1994). For example, passive strategies (e.g., rest and use of analgesics) have been termed maladaptive whereas more active coping (e.g., physical activity, ignoring pain, exercise) has been termed adaptive (Brown & Nicassio, 1987). However, since such classifications have often been based on correlational analysis of adjustment outcomes in chronic pain patients with various diagnoses, they may not be universally applicable and in particular may not be generalisable to acute pain (Haythornthwaite & Heinberg, 1999).

There are types of cognitive coping response which involve over-attending to pain and which might be invariably maladaptive, whether pain is acute or chronic. Catastrophizing, which involves a disproportionately negative mental set during actual or anticipated pain experience and exacerbates pain (Sullivan et al., 2001) is one such maladaptive cognitive tendency. Jensen et al., (1991) have argued that catastrophizing is more a type of primary appraisal than a coping response, which suggests a traitlike thinking style. Distorted thinking may develop in the context of persistent pain and could have far-reaching effects on both physical and psychological health. For example, cognitive biases associated with selective processing of pain-related information have been observed in chronic pain patients, and may negatively alter the way individuals think of themselves in terms of pain and illness (for review, see Pincus & Morley, 2001). Furthermore, such cognitive biases are not limited to chronic pain patients. For example, information-processing biases towards pain-related material have been found in pain-fearful but otherwise healthy individuals (e.g., Keogh, Ellery, Hunt & Hannent, 2001) which indicates that cognitive predispositions to attend preferentially towards pain-relevant material exist in pain-free populations. Although more is known about the impact of maladaptive coping on chronic pain than on acute or experimental pain (Jensen et al., 1991), an association between catastrophizing and increased pain has been found in healthy (pain-free) individuals as well as in various pain patient groups. For example, Crombez, Eccleston, Baeyens and Eelen (1998) found that pain-free individuals who experienced frequent catastrophic cognitions about pain were more fearful when threatened with intense pain than those who infrequently catastrophized about pain. Experimental research has also shown that healthy individuals who catastrophize during noxious stimulation report more pain that those who do not (Sullivan & Neish, 1998), show reduced tolerance for pain (Geisser, Robinson & Pickren, 1992) and are more distressed by pain (Sullivan et al., 1995). Seemingly,

catastrophizers are hypervigilant to somatic threat, have problems shifting focus away from pain and so attend disproportionately to the negative feelings evoked by noxious stimuli (Crombez et al., 1998). The processes underlying catastrophizing are as yet unclear but a tendency for pain to capture and hold attention seems to be a central feature.

3.3.2 Efficacy of coping methods

A substantial body of research has been conducted to assess whether cognitive coping techniques are generally useful in pain management, and to ascertain which, if any, are the most effective strategies. This has been made difficult, at least in part by the heterogeneity of assessment methods applied to both coping and outcome in the research literature (Boothby et al., 1999). There has been relatively little use of true experimental designs to examine the effect of coping strategies on pain; much of the research has been conducted with chronic pain, often using cross-sectional designs which assess coping summarily over a retrospective period of time (Haythornthwaite & Heinberg, 1999). However, some prospective studies have found evidence for beneficial effects of particular coping strategies, such as distraction (e.g., Affleck et al., 1992). In a recent meta-analytic review, Morley, Eccleston and Williams (1999) concluded that active psychological treatments based on cognitive-behavioural therapy effect beneficial change on a range of pain-related and coping outcomes.

Clearly, it is particularly important to establish the relative utility of different coping strategies for chronic pain patients, whose pain experiences are ongoing. However, relatively little research has examined the effect of coping strategies on acute pain responses. In light of the obvious differences between chronic and acute pain, and the difficulties associated with generalising from one domain to the other, it is important to study the utility of different coping methods in the context of both types of pain. Different coping strategies may be effective for short-lived pain (such as that caused by mild injuries or medical procedures) than those which are useful for chronic pain. Indeed, some coping strategies which are associated with negative effects on health in the long-term (e.g., passive avoidance, denial) may be beneficial as temporary strategies to cope with acute pain. For example, the use of avoidant coping strategies has been found to reduce anxiety levels about surgical pain, and to improve recovery rate

(Wilson, 1981). Investigating the effects of cognitive coping strategies in the context of experimentally-induced pain provides a useful test model for acute clinical pain.

There is empirical evidence that taught coping strategies can improve pain tolerance. For example, Meichenbaum and Turk (1976) developed a coping rationale with cognitive-behavioural principles which they termed Stress Inoculation Training (SIT). The method involved educational reconceptualisation of pain, acquisition of coping skills and practice of these skills. The coping strategies taught included deep breathing, relaxation, use of pleasant images, use of positive self-statements and self-reinforcement for having coped. Meichenbaum and Turk tested the effectiveness of SIT for experimentally-induced ischaemic pain and found that pain tolerance almost doubled, increasing from 17 minutes pre-training to 32 minutes post-training. In the laboratory, pain tolerance is generally the parameter which is most sensitive to the effects of coping strategies; less impact is evident on pain threshold or pain report (Arntz & Schmidt, 1991).

In a recent review of research into cognitive control of pain, Weisenberg (1999) has concluded that strategies which alter the way an individual attends to a pain stimulus can be used to increase pain tolerance. However, interestingly, Neumann, Kugler, Seelbach and Kruskemper (1997) have reported that even non-directive suggestions can raise pain tolerance. Participants either listened to general information about physiological and biochemical pain theory (non-directive suggestions) or suggestions that coping with pain is easy, that recall of effects of analgesic drugs is possible and that their body state was incompatible with pain (directive suggestions) or nothing (control condition) in the intertrial interval between two pressure pain inductions. Neither pain threshold nor perception of pain intensity differed across groups but pain tolerance increased in the second trial for those who were given non-directive suggestions. A possible explanation for this is the impact of individual differences: the directive suggestion given may have been incompatible with the coping style of some participants in that group, whereas non-directive suggestion left participants free to invoke their own choice of coping method.

The success of cognitive strategies seems to depend greatly on motivational factors (Weisenberg, 1994). Cognitive efforts to cope with pain are often discussed with

reference to self-efficacy, which has been defined as possession of relevant skills coupled with the belief that one is capable of applying them (Bandura, 1977). Self-efficacy beliefs are thought to determine the effort individuals will make and how long they will persist against aversive circumstances. For example, Jensen, Turner and Romano (1991) found that chronic pain patients beliefs in their capabilities were strongly related to their reported efforts to cope. High self-efficacy has also been associated with tolerance of higher levels of experimental pain (Weisenberg, Schwarzwald & Tepper, 1996). Similarly, Dolce, Doleys, Raczynski, Lossie, Poole and Smith (1986) found self-efficacy expectancies were the best predictor of cold-pressor tolerance. Litt (1988) also reported that changes in self-efficacy expectations predicted changes in cold pressor tolerance, and concluded that self-efficacy expectations relate to the importance of perceived control, in that those who benefitted most from a sense of control over pain were those most confident they could exert it.

There has been other evidence that issues of control are important in pain perception (Skevington, 1995). For example, locus-of-control (beliefs about control over life events attributed either internally to the self, or externally to sources outside the self) appears to be extremely salient to pain and coping. The construct has been applied to beliefs about control over pain, and a specific measure, the Beliefs in Pain Control Questionnaire (BCPQ; Skevington, 1990) has been designed to measure locus-of-control for pain. High scores on internal locus have been associated with less intense pain, less frequent pain and better coping (Skevington, 1990; Toomey, Mann, Abashian & Thompson-Pope, 1991). Seemingly those who perceive they have control over their own pain suffer less than those who believe control over their pain lies with others, or is a matter of chance.

Studies of patient-controlled analgesia (PCA) have demonstrated the positive effects of enhanced self-control over pain in a clinical context. Surgical patients who are able to self-regulate their post-operative analgesia demonstrate lower uptake of medication, and report less pain and distress (Thomas, 1997). Such self-regulation is attributed to a heightened sense of control over their own pain; these patients had tangible rather than merely perceived internal locus-of-control for pain, which seems to have directly affected their perception. Other configurations have also been noted in the relationship of control to pain. Interestingly, Keefe and colleagues (2001) found in their recent study

with rheumatoid arthritis patients that spiritual and religious coping was associated with enhanced ability to control and decrease pain. This type of coping can be categorised as passive with an external locus of control; i.e., faith in a higher power outside the self, and so by some accounts should be maladaptive (see Skevington, 1990). For these patients this was clearly not the case; their faith in forces outside themselves facilitated a better sense of control over their pain.

Thorn and Williams (1989) explored another aspect of perceived control using 'goal specification' (specifying a given time limit for the duration of the pain stimulus) within the cold pressor paradigm and found that varying the goal alters perceived pain intensity and pain tolerance. They found that asking participants to tolerate the stimulus for a given period of time, rather than to the limit of endurance, increased pain tolerance and the perceived effectiveness of cognitive strategies. In this situation it is likely that appraisal of a painful stimulus with known time parameters engenders a greater sense of control. If the time is unspecified, the individual presumably approaches the task wondering how long they will be able to bear it, and automatically experiences a level of uncertainty about their ability to cope. Such findings may be relevant to the disparity between coping with acute clinical pain and coping with chronic pain.

It has been suggested that while cognitive coping strategies are useful, there is no clear advantage of one strategy over another (e.g., Tan, 1982; Turk, Meichenbaum & Genest, 1983). On the basis of meta-analysis, Fernandez and Turk (1989) reported that cognitive coping strategies were generally more effective than no-treatment or expectancy control. However, these authors also concluded that overall, techniques using imagery were the most effective and those involving repetitive cognitions or acknowledgement of pain sensations were least effective. Fernandez and Turk interpreted these findings as plausible within a limited capacity model of attention; imagery techniques exert greater demands on attention, and provide greater distraction from pain than repetitive cognitions or acknowledging pain sensations. There may also be a physiological basis to the effectiveness of imaginal distraction. According to Melzack and Wall (1996), attention exerts modulating effects on pain perception via descending cortical influences on the gate mechanism in the dorsal horns of the spinal cord. Imagery is also known to involve neurophysiological activity in higher brain centres (Achterberg, 1984), which potentially exerts inhibitory effects on pain transmission in the same manner.

3.3.3 Attentional direction in coping with pain

Eccleston (1995) has argued that attention is a key factor in pain perception, and can be thought of as selective monitoring which filters the constant barrage of information flowing from both the external environment and internal body states. Distraction is a closely related process, whereby attention is shifted away from one source of information onto another. According to Eccleston, this is exactly what happens when pain impinges upon an individual; attention to ongoing matters is intruded upon and pain distracts them from the task at hand. Similarly, LeResche (1999) has suggested that perceptually pain is a somatic (rather than visual) image that captures or narrows attention. This is consistent with limited-capacity models of attention (e.g, Broadbent, 1958) which assert that, given competing stimuli, attention will become selective and this will lead to the exclusion or neglect of some of the input. Support for these propositions comes from evidence that the strong demands that pain makes on attentional resources disrupts performance on tasks which are attempted during pain (Crombez, Eccleston, Baeyens & Eelen, 1996).

Eccleston and Crombez (1999) have proposed that pain is able to interrupt other activities and thought processes because it has salience as a signal of threat to the organism; thus it takes precedence over other information and prompts an immediate response. These authors argued that pain is a signal with survival value and is therefore 'selected for action', i.e., pain interrupts other processing and demands attention because there is a biological predisposition to react to it. If, as Eccleston and Crombez suggest, we are biologically primed to respond to pain over other stimuli, distraction away from pain is maladaptive and should be unlikely. In fact, attention to pain seems to be a context-sensitive process, as demonstrated by instances of episodic analgesia such as those seen in battlefield injuries (Beecher, 1959; cited in Melzack & Wall, 1996) and the occurrence of stress-induced analgesia. In high-risk situations, where survival is likely to be enhanced by low (or absent) perception of pain, it seems that even severe tissue damage may not be accompanied by immediate pain and attention can remain focused on other, temporarily more pressing issues, such as escape from a dangerous environment. These examples obviously differ greatly from many less extreme situations in which pain may occur, but nevertheless demonstrate that in certain circumstances attention to pain is modifiable.

Attention is a pivotal component in many of the coping strategies used to manage pain (Cioffi, 1991, Eccleston, 1995; Eccleston & Crombez, 1999) and a broad distinction between coping methods can be made on the basis of the direction in which attention is engaged during pain. Avoidant coping strategies involve diversion of attention away from the noxious stimulus and/or one's responses to it, whereas non-avoidant (sometimes referred to as 'attentional' or 'focused') coping strategies involve attending to the stimulus and/or responses to it (Suls & Fletcher, 1985). Both avoidant and non-avoidant strategies can be used to cope with pain, but which of these approaches to coping is most useful overall has not yet become clear.

Distraction of attention away from pain in low risk circumstances is unlikely to be easy, but there is some evidence that it is both possible and useful. For example, distraction has been found to be a useful way to reduce children's distress during medical procedures (Kleiber & Harper, 1999). Many different variants of distraction strategy have been devised and used in attempts to lessen pain, ranging from simple types such as counting or other repetitive cognitions, to mathematical calculus or complex mental imagery. Characteristics of the distraction technique seem to be important determinants of effectiveness. For instance, McCaul and Malott (1984) proposed that the more 'attention-grabbing' a distraction is (e.g., novel or cognitively demanding), the more effective it will be as a coping strategy for pain. Reversing this principle, it is likely that characteristics of the pain experience will also determine the success of distraction; it should be easier to distract attention from mild pain than from severe pain. Consistent with this principle, McCaul and Malott (1984) have also reported that while distraction is indeed an effective pain coping strategy for low intensity stimuli, sensation redefinition is better for more intense pain. However, McCaul, Monson and Maki (1992) found no effect of various types of distraction on the self-reported distress of participants due to laboratory-induced pain.

Taken together, these findings partially support Eccleston and Crombez's (1999) assertions about the interruptive nature of pain, but also indicate that distraction can be a useful method of coping with some pain experiences. Eccleston (1995) has argued that distraction is more likely to be useful for acute pain than for chronic pain, whereas chronic pain might be better dealt with by monitoring rather than avoidance. Since pain is consumptive of central attentional resources, Eccleston contends that an adequate

distractor needs to be as attention-demanding as the painful stimulus, the pain short-lived and the strategy used consonant with the coping style of the individual.

If cognitive coping strategies can displace nociceptive processing to some degree by temporarily capturing attention, it follows that a distraction technique such as mental imagery would compete for and divert attention from the nociceptive stimulus whereas acknowledgement/focusing would involve maintaining attention to the pain. From this perspective, acknowledging or focusing attention on pain might seem counterintuitive and distraction confers more obvious benefits. However, avoidance of pain is not always advantageous. Meta-analyses of empirical research into the relative efficacy of avoidant and non-avoidant coping strategies for pain (Mullen & Suls, 1982; Suls and Fletcher, 1985) revealed that both types of coping strategy were better than no instructions in the short-term. However, Suls and Fletcher (1985) also found that avoidance was more useful than non-avoidance in the short term (i.e., within three days), whereas for pain persisting beyond two weeks duration focused attention (non-avoidance) seemed to be more effective. This 'time x strategy' hypothesis has been supported in several subsequent studies. For example, Holmes and Stevenson (1990) found greater adaptation (lower negative affect, pain severity and distress from bodily dysfunction and higher social activity) in chronic pain sufferers who used attentional strategies than those who were avoidant copers, and the reverse pattern for patients with recent-onset pain. However, Suls and Fletcher (1985) also reported that there was a notable exception to the 'time x strategy' pattern. One type of non-avoidant strategy - focus on the sensory aspects of pain rather than emotional or cognitive responses to it - was found to be superior to avoidance even in the short term. Leventhal, Brown, Scacham and Engquist (1979) proposed that potentially aversive experiences can be processed in two different ways; by attending to concrete, sensory information or by attending to emotional or threatening qualities. Attending to the discrete, sensory aspects of sensations is presumed to elicit 'neutral' perceptions of them which displace negative, emotional reactions. By contrast, if attention is focused on emotional responses to noxious sensations, this negatively 'colours' the interpretation of incoming sensory information, causing more distress. There has been some empirical support for these proposals. For example, non-emotional focus on somatic sensations has been found to have positive sequelae for coping with pain (Cioffi, 1991; Boothby et al., 1999).

Conflicting findings in recent research have further clouded the issue of whether avoidant or non-avoidant coping is more effective. For example, McCracken (1997) found that, contrary to the 'time x strategy' hypothesis, chronic low back pain patients who reported greater pain vigilance also reported greater pain intensity and more emotional distress. McCracken has concluded that attending to pain may magnify its perceived intensity, and a sort of hypervigilance may develop which facilitates prompt action to prevent pain worsening, and consequently the tendency to attend to pain is reinforced.

It may be the case that the appropriateness of focusing on pain depends on both the coping style of the individual and the situation in which pain occurs. For example, Logan, Baron and Kohout (1995) found that sensory focusing produced lower intensity pain for highly stressed dental patients (i.e., those who had a high desire for control but felt they had little control). Logan and colleagues suggest that the effectiveness of sensory focus found for this 'high desire/low felt' group was more related to their initial stress (high anxiety and anticipated pain) than to the type of pain involved. Other research has indicated that avoidance of pain can have negative effects for certain types of pain. For example, Hill (1993) found that phantom limb pain patients reported more severe pain and distress when they used distraction than when they focused on their pain. However, Affleck, Urrows, Tennen and Higgins (1992) reported that rheumatoid arthritis sufferers found distraction more effective for their pain. Clearly, differences in the characteristics and circumstances of the pain experiences are relevant in these examples. Distraction from the long-term and consistent chronic pain of arthritis seems an adaptive response since awareness of it confers no conceivable benefit.

Avoidant and non-avoidant coping may affect different aspects of pain experiences. For example, Cioffi and Holloway (1993) gave varied instructions prior to a cold pressor trial; to monitor pain, to suppress all thoughts about pain, or to think about something else (i.e., a distractor). Differential effects of the three types of strategy were in evidence after, but not during, the experimental pain induction. No significant variation in pain tolerance was found across the different coping strategies, but differences in recovery from pain were observed between the strategy groups. Those who had been instructed to monitor the pain recovered more quickly than those who had been instructed to suppress pain-related thoughts. A possible explanation for the Cioffi and Holloway findings is

that if attention is directed towards painful feelings, as in the monitoring/focusing condition, even small changes are likely to be noticed and so the onset of recovery may be more readily detected. In addition, the suppression strategy group rated an innocuous somatic stimulus after the cold pressor trial as more unpleasant than did the other groups suggesting that, in this experiment at least, avoidance may have had a lingering negative effect. These findings may relate to previous research into suppression of thoughts which has indicated that this can result in a 'rebound' effect, a negative aftermath from a type of avoidance (Wegner, 1994), and suggests that non-avoidant coping might facilitate quicker recovery than avoidance, at least for experimental pain. Overall, the inconsistent pattern of research findings to date does not justify judgement of avoidant coping as generally superior to non-avoidant coping, or vice versa. It may transpire that these two ways of directing attention are differentially suitable for certain types of pain, circumstances and individuals.

3.4 Gender and coping

One of the main objectives of the present programme of research is to investigate the differences in male and female responses to experimental pain induction. Previous research indicates that men and women do not react to stressors, including pain, in the same way. Stress management style, including the type of coping strategies used, seems to be different for males and females. For example, some research has indicated that men are more likely to use active, problem-focused coping methods whereas their female counterparts tend to utilise emotion-focused strategies, to express emotion, and to seek social support (e.g., Vingerhoets & Van Heck, 1990). Affleck and colleagues (1999) observed that female arthritis sufferers use more emotion-focused strategies than their male counterparts. These findings are concordant with the well-documented tendency for women to be more emotionally expressive than men (Skevington, 1995). Savedra, Gibbons, Tesler, Ward and Wegner (1982) found evidence that this tendency starts early in life; girls used more affective terms to describe their pain than boys. Finally, Robinson, Riley and Myers (2000) concluded that "men and women cope ... in ways that are consistent with dominant cultural gender role stereotypes" (p.51). They summarised the differences as reflecting a greater female tendency toward emotional and interpersonal coping, in contrast to a male preponderance to adopt a more autonomous, problem-solving approach.

According to Unruh (1996), males and females are likely to learn different coping strategies during their lives. From a psychosocial perspective, females may learn gradually as they mature physically that painful experiences related to their sex are likely to occur (and recur), hence pain and pain-related emotions can become idiosyncratic constructs within personal memory and expectations are engendered about female-specific pain. For example, although some women do not experience pain with menstruation, or headache pain (related to menstruation or otherwise), for many women these are regular pains they learn to cope with. Women may therefore come to expect pain in a way that men do not, and develop pain schemata which produce enhanced vigilance for painful feelings and heightened focus on pain sensations when they do occur (Turk & Rudy, 1992; Sullivan, Rouse, Bishop & Johnston, 1997). Furthermore, the model of female reproductive processes (e.g., childbirth) generally portrayed by the media, family and/or folklore is likely to feature pain.

Sociocultural learning processes in childhood may influence attitudes to pain and coping behaviours in adulthood. For example, infants learn how to behave 'appropriately' when in pain through the reinforcing responses of their parents or caregivers. If slight injuries are treated as serious by parents, or vice versa, this will influence children's attitudes and behaviours in response to pain. Learning how to respond to pain may also occur vicariously, through witnessing the pain responses of influential social models such as family members or other close associates. For example, the attitudes of parents towards pain tend to be acquired by their children and retained in adult life. People with more 'pain models' in their families (i.e., a greater number of family members with persistent pain or showing pain symptoms) have been found to report more frequent pain experiences (Edwards, Zeichner, Kuczmierczyk & Boczkowski, 1985). Such individuals would have had increased opportunity to learn pain behaviours through observation of salient models. This type of social learning within families may produce differences in the extent to which men and women cope by expressing pain to another person. Edwards et al. (1985) found that women reported more pain than men, and that the influence of familial pain models on frequency of pain report was stronger for females. Similarly, Fillingim, Edwards and Powell (2000) found that family history of pain was associated with increased pain complaints and enhanced experimental pain sensitivity in females but not males. Unfortunately, the relative numbers of male or female familial pain models to which the men and women in the

study were exposed was not reported; this would have allowed the interesting possibility of examining the effects of same-sex (gender-role specific) modelling of expressiveness of pain.

Some studies have indicated that women feel a greater need for a sense of control over pain than men (Liddell & Locker, 1997) but it is unclear whether there are significant sex differences in perceived control over it (Haythornthwaite et al., 1998). Other research has shown that some control-related issues such as exercising choice over how to cope with painful experiences may be more important to men than women. Rokke and al'Absi (1992) have found that women tolerated cold pressor pain better if they used a coping strategy consistent with their coping style, but choosing their own coping method conferred no particular benefit. In contrast, men demonstrated greater pain tolerance with choice of strategy than in any other coping condition.

It seems that women may generally have greater belief in the effectiveness of coping strategies for pain, and this in turn may affect the extent to which they try to use such strategies. For example, Williams and Keefe (1991) found that among chronic pain patients who perceived their pain as enduring and understandable, males gave lower ratings of their ability to decrease pain via coping strategies than females. Affleck, Urrows, Tennen and Higgins (1992) found that women had a larger repertoire of coping strategies for rheumatoid arthritis than men, and engaged in more coping activities each day. Similarly, in a study of gender effects on pain appraisal and coping, Unruh, Ritchie & Merskey (1999) found that women reported using greater number of coping strategies than men overall, and were more likely to resort to multiple strategies. Other studies have indicated that women use more analgesic medications but also try out more types of therapy and more combinations of approach, and seem to benefit more from cognitive therapies (Berkley & Holdcroft, 1999). It does seem that women may take a more open-ended approach to coping with pain than men, as shown by their greater propensity to try out more coping methods. This may be related to the fact that women experience more types of pain across a lifetime than men, trying different strategies for different types of pain may represent a heuristic basis for flexibility in coping. Overall, the few studies which have examined gender differences in coping specifically with pain accord with the general coping literature that women acquire and use a wider range of coping strategies than men.

Earlier in this chapter, the relative benefits of avoidant and non-avoidant coping with pain, and their differential usefulness according to the duration of pain were discussed. However, recent research has indicated that the relative benefits of avoidant and focused coping may not simply rest on the temporal qualities of pain. Gender differences in an innate tendency to attend to pain may be an important factor in the suitability of avoidant and non-avoidant coping for males and females. For example, women are thought to have a greater tendency to monitor both internal and external sources of threatening information than men (Miller, 1987) and also seem to catastrophize more about pain than their male counterparts (Sullivan et al., 1995). Women have also been found to show greater tendency towards cognitive processes which are conceptually similar to catastrophizing, such as hypervigilance (Rollman, 1998). Hypervigilance is a generalized cognitive and perceptual over-responsiveness to exogenous and endogenous discomfort, including but not restricted to pain (Lautenbacher & Rollman, 1993). It may be that a generally heightened awareness of somatic states in women subsumes these observed gender differences in cognitive style. Sullivan et al. (1995) found that, in particular, women scored higher than men on the tendency to ruminate and feel helpless when in pain, which corresponds to previous theory and research indicating an enhanced tendency in women towards contemplative and emotionally expressive responses to stress (e.g., Endler & Parker, 1994). These proclivities may have an impact on the efficacy of attentional coping strategies for women in pain. For example, Heynemann, Fremouw, Gano, Kirkland and Heiden (1990) have suggested that catastrophizing may hamper the ability to distract attention away from pain effectively.

The relative usefulness of avoidant and non-avoidant coping for males or females in pain has not been clearly established. Some studies indicate that focusing might be better than distraction for men, whereas others suggest that women may particularly benefit from focusing on pain. For example, Keogh, Hatton and Ellery (2000) found that the direction of attentional processes during experimental pain induction had different effects on male and female participants. Consistent with previous research, males were found to be more tolerant of cold pressor pain than females. However, focusing on pain positively affected (i.e., lowered) male subjective ratings of sensory pain, whereas female sensory pain ratings were unaffected by coping strategy. Keogh et al. (2000) concluded that, for men, focusing on pain may be a beneficial coping

strategy, but that it is unclear how attention might best be directed to help women cope successfully with pain (see also Keogh & Mansoor, 2001).

In contrast, Leventhal (1992) studied women in labour and concluded that focusing, with this type of pain at least, is better than distraction. However, the pain of labour has unique characteristics, such as increasing intensity and other qualitative changes as parturition approaches, which are likely to make distraction less viable, and focusing attention on the pain much more likely and probably more adaptive. Certainly prospective mothers will ultimately need to use their awareness of their own body sensations to co-ordinate voluntary muscular control with the involuntary waves of uterine muscle contraction which increase in frequency and intensity in the last stages of labour, so in this situation focusing may be particularly adaptive for women. As there were obviously no parallel male participants in Leventhal's study, these findings reveal something about the relative utility of focusing and avoidance for this particular type of female-specific pain, but do not broaden understanding of the interaction of gender with avoidant and non-avoidant coping.

Other recent research indicates that the method of non-avoidance may be important and that focusing on the sensory rather than emotional aspects of pain may be differentially beneficial to males and females. In a recent study, Keogh and Herdenfeldt (2002) found that for men, focusing on the sensory aspects of experimental pain raised their pain threshold and tolerance and reduced their sensory pain, when compared to focusing on their emotional responses to it. Women in this study reported higher affective pain when they used emotion-focused coping than when they focused on the sensory aspects of pain. A possible explanation for these findings is that for women, the use of emotion-focused coping reinforced an innate tendency to be more emotionally expressive, leading them to endorse the affective descriptors more than the sensory ones when they rated their experience of pain after the pain induction trial.

The research into the effects of attentional coping on experimental pain discussed above adds weight to a wide range of clinical observations that have shown females to be generally more pain-sensitive than males (Berkley, 1997). For example, women seem to have lower tolerance for cold pressor pain than men and there is some evidence that focusing on pain sensations may lessen pain for men but not for women, so it appears

that non-avoidant coping at least may be differentially suitable for males and females. It may be the case that certain approaches to coping with pain are broadly more beneficial to men than women (and vice versa), although Leventhal's (1992) findings suggest that this may depend on the type of pain in question. Given the many differences between artificially-induced pain and chronic pain, such effects would need to be investigated in clinical groups with pain occurring in both men and women, in order to establish whether they are limited to experimental pain. If gender-related patterns of response to experimental pain are replicable with pain patients, such findings might usefully contribute to the design of therapeutic pain interventions tailored for each gender.

3.5 Summary

The accumulated research into coping demonstrates that psychological processes can be usefully harnessed to cope with pain. However, while empirical support grows for the utility of psychologically-based pain management, it remains unclear which of the psychological factors involved in coping with pain are most important (Weisenberg, 1998). Similarly, no unequivocal conclusions can be drawn regarding which coping strategies are optimal. In particular, it is not clear whether it is better to direct attention towards pain or away from it. There has been a tendency to group coping strategies as simply adaptive or maladaptive, but this is an oversimplified classification since their effectiveness seems to contingent upon a number of factors including the gender of the individual in pain and the type of noxious stimulus applied. Further research is clearly needed to clarify the relationships between gender, coping and pain responses.

3.6 Summary of introductory chapters.

To recapitulate, it is now generally accepted that both psychological and physiological systems underlie human pain perception, and that sensory-discriminative, cognitive-evaluative and motivational-affective components are distinguishable and interactive within this process, as outlined in Gate Control theory. Examination of the accumulated pain research literature indicates that pain experiences are shaped by the interplay of an idiosyncratic array of situational and personal variables, and are therefore highly alterable. Furthermore, there is evidence that gender may be an important determinant of pain responses, and that both biological and psychological mechanisms underlie this interaction. Broadly, it seems that females are both more likely to experience pain than males and more sensitive to it. Multiple factors are likely to contribute to gender

differences in pain, including anatomy, physiology, social learning, cognition and emotion. There may also be differences in the way that men and women appraise and cope with pain which affect their respective pain experiences. Although research has shown that pain can be modulated by cognitive processes, which indicates that alterations to cognition can be therapeutically useful, it is not yet clear whether gender and cognition interact systematically in pain perception.

The focus of this thesis is the influence of gender on pain perception and pain coping. If, as growing evidence suggests, men and women differ not only in the extent and type of pain they experience across a lifetime, but also in how they respond to pain and to treatments for it, this offers potential for optimising coping methods on the basis of gender. However, both the pain and coping research literature have produced inconsistent findings with respect to gender differences. Further research is needed to ascertain whether gender differences in pain responses are robust and replicable with all types of noxious stimuli, and whether gender-specific coping approaches are needed. In light of this, a series of experiments was designed to examine the responses of healthy male and female adults to acute pain induced under standardised laboratory conditions, and to assess the impact of cognitive coping strategies on such responses. The cold pressor method of pain induction was chosen for this series of experiments, as relatively few studies have directly examined gender differences in responses to this type of noxious stimulus despite its resemblance to naturally-occurring pain.

Chapter 4

Experiment 1

Gender, Coping Style and Cold Pressor Pain

4.1 Introduction

Growing evidence from epidemiological, clinical and experimental pain research indicates marked and significant differences in the pain experiences of males and females (see Chapter 2). It is becoming clear that a range of biological, psychological and sociocultural differences between human males and females may affect their respective responses to pain at subjective, behavioural and physiological levels. It also seems that an important contribution to these contrasting experiences of pain may arise from disparity in the ways that males and females cope with pain (Unruh, 1996; Rollman, 1998). Recent research, which has directly examined the impact of gender on coping with clinical pain, has indicated important differences between males and females in their use of coping strategies for pain and in pain appraisal (Unruh et al., 1999). In particular, catastrophizing may be an important construct within coping which has special relevance to gender differences in pain. Research with various types of pain patients has shown that women are more likely to catastrophize than men (Jensen, Nygren, Gamberale, Goldie & Westerholm, 1994; Sullivan et al., 2001) and that in some cases catastrophizing is a mediator of gender effects on pain responses (e.g, Keefe, Lefebvre, Egert, Affleck, Sullivan & Caldwell, 2000). In addition to these clinical findings, gender differences in coping with experimental pain have been found (see Fillingim & Maixner, 1995) as has evidence that catastrophizing can be a mediator of gender effects in experimental pain (e.g, Sullivan, Tripp & Santor, 2000) but it is unclear whether these effects are robust and replicable.

This chapter reports the first of a series of empirical investigations of male and female pain responses using the cold pressor experimental pain induction with healthy adult volunteers. Relatively little research has examined gender differences in cold pressor pain (see Riley et al., 1998) or the importance of pain coping style with this type of experimental pain. Experiment 1 was therefore an exploratory investigation in which the primary research objective was to ascertain whether healthy males and females respond differently to cold pressor pain. In light of previous indications of gender-differentiated coping with naturally-occurring pain, further objectives were to explore the relationships between gender, pain coping style and experimental pain responses. Gender differences in pain responses were predicted, specifically that females would show greater pain sensitivity and report more pain than males. Females were expected to demonstrate lower pain threshold, lower pain tolerance and higher scores on self-

report pain measures than males. Differences in pain coping style were also expected between males and females; particularly that females would report more catastrophizing than males.

4.2 Experimental paradigm

There are a number of advantages to the cold pressor method of pain induction which was used in this series of experiments. According to Gracely (1994), an experimental pain stimulus should be easy to apply and produce a distinct pain sensation with rapid onset and offset. The cold pressor technique, in which a limb or extremity is immersed in very cold water, produces a continuous, severe pain which increases quickly but also decreases fairly rapidly after termination of stimulus contact. The recovery time (interval between termination of stimulus contact and cessation of pain) after exposure to cold pressor is approximately 10 minutes (Wolff, 1984), which offers potential for repeat testing within a single test session. This was important here, as repeated exposures within one test period would be necessary in several experiments in the series. In addition, the cold pressor technique has been widely used and does not cause tissue damage to participants. Face validity of the procedure is good; participants find it painful but also acceptable, which makes loss of participants through withdrawal less likely. This also reduces the risk of confounding affective pain responses with fear of the stimulus itself, which might occur with electrical pain, for example. The ecological validity of the cold pressor technique is good, as the pain elicited has a natural quality (Gracely, 1999). Tonic pain models such as cold pressor are considered to evoke an affective response which more closely resembles clinical pain than brief noxious stimuli such as electrical pain (Treede, Kenshalo, Gracely & Jones, 1999). Use of the cold pressor technique was approved by Goldsmiths College Ethics Committee.

The majority of cold pressor studies report the use of water chilled with ice to between 0 - 3°C, although some research with adults has used temperatures as high as 5°C (e.g., Rainville et al., 1992) or 7°C (e.g., Ahles, Blanchard & Leventhal, 1983). Some cold pressor studies with children have used water at 10°C (e.g., Fanurik, Seltzer, Roberts & Blount, 1993). Cold pressor produces a dull, diffuse, aching pain, which usually onsets after approximately 10-15 seconds and increases rapidly. Between 1 and 2 minutes after immersion, a tingling sensation in the fingers begins to accompany the aching pain. If immersion continues for more than 3 minutes a severe burning or smarting sensation

begins in the skin of the hand, which increases in severity until it becomes extremely uncomfortable. The pain of cold pressor at low temperatures such as 0-1°C usually peaks between 2 and 4 minutes and then drops down to a low-level throb accompanied by growing numbness. With continued exposure, a secondary stark rise in pain is perceived at around 10 minutes, but this pattern of pain differs from that exerted by water between 3-7°C. Since less exposure to cold pressor is taken to elicit lower intensity pain and longer exposure to produce more intense pain, variation of water temperature in different studies compromises comparisons of pain intensity between studies (Eccleston, 1995). In light of this, care was taken to ensure that all experiments in this series were conducted with the same cold pressor temperature (1°C) which ensured that all participants were exposed to a standardised stimulus.

It is usual for researchers to impose an upper time limit to cold pressor exposure, which is commonly set at either 2 or 5 minutes. This limit is not normally made explicit to participants before the procedure, except in studies which involve setting a tolerance goal as part of an experimental manipulation (e.g., Thorn & Williams, 1989). A 5 minute limit was imposed in all cold pressor exposures in the present series of experiments, but was not disclosed to participants.

4.3 Method

4.3.1 Design

A quasi-experimental design was used in Experiment 1. The single independent variable was gender, with two levels: male and female. The dependent variables were pain threshold, pain tolerance, pain intensity, sensory pain, affective pain and coping style. Measures of pain threshold and pain tolerance were obtained directly from the cold pressor task. Three additional pain indices obtained from the cold pressor were ratings of changes in pain severity between pain threshold and tolerance (Mild Pain, Moderate Pain, Severe Pain). Sensory pain, affective pain and pain intensity were self-report measures obtained from a pain questionnaire. Coping style was measured by questionnaire. In addition, questionnaires to measure state and trait anxiety were included since these variables can affect pain responses.

4.3.2 Participants

Standard exclusion criteria for cold pressor pain induction were implemented: individuals with previous experience of frostbite, Raynaud's disease or other circulatory disorders, or who suffered from cardiovascular disorders, hypertension, migraine or any chronic pain condition were excluded from the experiment. Individuals taking any medication (including antidepressants, etc.), who had taken alcohol or analgesics within the previous twelve hours, or had ingested caffeine within the previous three hours were also excluded. On this basis, 110 participants (55 females, 55 males) were recruited from the student and staff population at Goldsmiths College. The pain response data of 4 participants was lost through equipment failure, leaving a total sample of 106 (55 females, 51 males). Age range was 18–55 years (mean 24.1, SD 6.7). All participants were in good general health, and not currently in pain.

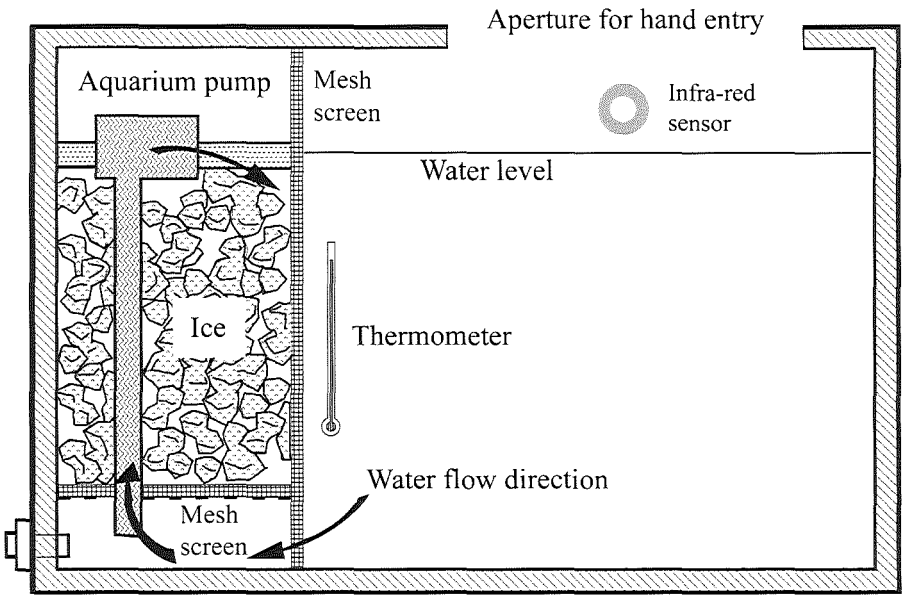
4.3.3 Pain Induction

4.3.3.1 Cold pressor apparatus

The cold pressor equipment comprised two custom-built, insulated tanks of 25-litre capacity. Tank 1, which was referred to throughout the experiment as 'the warm tank', contained tepid water, maintained at 31°C by an aquarium heater and circulated by an air pump. Tank 2, which was referred to as 'the cold tank' throughout, contained water chilled with ice to a steady temperature of 1°C. The ice was contained behind a mesh screen, in order to prevent contact with the skin of participants during cold pressor exposure. The cold water was circulated by an underwater pump to prevent local warming and maintain a constant temperature throughout the tank. A general laboratory, mercury-filled glass thermometer with a range of –10/110°C was suspended in the water of each tank to allow regular water temperature checks. The water depth was the same in both tanks. See Figure 4.1 for a technical diagram of the cold tank.

Infra-red sensors rebated into opposing walls of the cold tank triggered a time switch linked to a computer when a participant's hand broke the beam upon entry to the water, registering the start of a cold pressor trial. The infra-red beam was reinstated upon removal of the hand, triggering a second switch and registering the end of a cold pressor trial. A peripheral electronic switch box linked to the computer was activated by a series of buttons clearly marked with labels of ascending pain severity. Each button press recorded a time in seconds for a pain value on the scale.

Figure 4.1: Technical diagram of cold pressor apparatus



4.3.3.2 Cold pressor task

Participants immersed their non-dominant hand and forearm in the warm tank for 5 minutes prior to the cold pressor task, maintaining fingertip contact with the bottom of the tank throughout immersion. This is a previously established procedure which aims to standardise cutaneous temperature prior to cold pressor exposure (Wolff, 1984). During warm tank immersion participants were given clear, standardised instructions, both verbally and in written form, regarding the cold pressor procedure and the method of pain rating to be used. The exact instructions given are stated in Appendix 1.

Participants were shown the button-press apparatus for pain ratings at this time, a check for understanding was made and any questions arising were answered. Immediately after withdrawal from the warm tank, participants immersed their non-dominant hand and lower forearm in the cold tank, again maintaining fingertip contact with the bottom of the tank. They were required to indicate the point at which the cold sensations became painful and subsequent changes in pain level by pressing the appropriate buttons, and to keep their hand in the cold water to the limit of their tolerance. Unknown to participants, an upper limit of 300 seconds immersion was employed for all cold pressor exposures, beyond which instructions were given to withdraw from the cold tank.

4.3.3.3 Pain ratings

Pain threshold was operationally defined as time in seconds elapsed from the entry of the hand into the cold water to the first perception of a painful sensation. Participants indicated the onset of pain by pressing a button marked 'Just Noticeable Pain'. After pain threshold, participants pressed three more buttons to indicate changes in pain severity, labelled in ascending order; 'Mild Pain', 'Moderate Pain' and 'Severe Pain'. The pain change ratings thus obtained were measured in seconds. This system was designed and implemented with the aim of examining temporal changes in pain severity, in particular whether the pace at which cold pressor pain increases is different for males and females. Pain tolerance was operationally defined as time in seconds elapsed from the entry of the hand into the water to the point of withdrawal.

Immediately after withdrawal, the Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) was completed to rate the pain during the cold pressor immersion when it was at its worst, providing self-report measures of sensory pain and affective pain. The SF-MPQ was chosen because it is quickly administered, has good face validity and correlates highly with the long form McGill Pain Questionnaire (Melzack,1975), which has been used extensively in clinical and research applications, and has satisfactory reliability and validity (Melzack & Katz, 1992). In a recent study, Wright, Asmundson & McCreary (2001) have confirmed the factorial validity of the SF-MPQ as a measure of sensory and affective dimensions of pain. The SF-MPQ comprises 15 pain descriptors which refer to sensory and affective qualities of pain and are rated on a 4-point scale from 0 (*none*) to 3 (*severe*). A Pain Rating Index is calculated for both categories, ranging from 0-33 for sensory pain and 0-12 for affective pain. The SF-MPQ includes a rating of pain intensity on a 100mm VAS with terminal anchors labelled '*no pain*' and '*worst possible pain*', which provides ratio scale data (Melzack, 1987).

4.3.4 Additional questionnaires

Coping Strategies Questionnaire: Coping style was assessed using the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983). The CSQ is a 44-item self-report questionnaire which assesses the use of coping strategies when in pain, and the effectiveness of these strategies for control and reduction of pain. This measure was chosen because it comprises seven subscales which tap particular ways of coping with pain: reinterpreting painful sensations, catastrophizing, ignoring painful sensations,

praying and hoping, diverting attention, increasing behaviour, and coping self-statements. In addition, two 'effectiveness' items measure perceived ability to control and decrease pain through use of coping strategies. Satisfactory reliability and validity has been established for the CSQ (Rosenstiel & Keefe, 1983; Crisson & Keefe, 1988). CSQ items are rated on a 7-point Likert-type scale. Responses on the first 42 items range from 0 (*never do*) to 6 (*always do*). Scores for the subscales are derived by summing the six item scores in each. Total scores range from 0-36 and higher scores indicate greater use of coping strategy subtype. Mean subscale scores are calculated by dividing the subscale total by the number of items therein, giving a range of 0-6. The response format of the two effectiveness items ranges from 0 (*no control*) to 6 (*complete control*) and from 0 (*can't decrease it at all*) to 6 (*can decrease it completely*). The two effectiveness items are scored separately giving a range of 0-6 for each, with a higher score indicating greater self-rated ability to control and decrease pain by the use of coping strategies. Composite coping measures can be produced from the CSQ, combining subscales which measure related coping constructs, and can be used to examine the relationship of different types of coping with pain outcomes (Boothby, Thorn, Stroud & Jensen, 1999). If sample size is limited, analysis using coping composites can protect against the risk of Type I error which is a possibility with multiple analyses of individual coping subscales (Jensen et al., 1991).

State-Trait Anxiety Inventory: As anxiety can influence pain responses, and women often score more highly on measures of anxiety than men (Rollman, 2000), participants in Experiment 1 completed the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983). The STAI has been widely used in both research and clinical settings and comprises two scales: the STAI-S (20-items), which assesses current state anxiety, and the STAI-T (20-items), which assesses characteristic trait anxiety. Satisfactory psychometric properties have been demonstrated for the STAI scales (Spielberger et al., 1983). The response format of the state anxiety scale (STAI-S) is a 4-point numerical scale to indicate the intensity of current feelings in relation to each item-statement, ranging from 1 (*not at all*) to 4 (*very much so*). On the trait anxiety scale (STAI-T), a 4-point numerical scale is used to indicate the frequency of feelings in relation to each item-statement, ranging from 1 (*almost never*) to 4 (*almost always*). The range of scores for both forms of the STAI is 20-80, with higher scores indicating greater anxiety.

4.3.5 Procedure

Participants were first screened against the exclusion criteria outlined above and then completed coping style, state anxiety and trait anxiety measures. Participants were then shown the cold pressor apparatus and given a standardised explanation of the experimental procedure, including assurances regarding the safety of the procedure, notification of their right to withdraw, and assurances of confidentiality and anonymity in data analysis. Participants then underwent a single cold pressor exposure. All participants gave their informed consent in writing before the pain induction procedure, and were debriefed at the end of the experiment.

4.3.6 Statistical analysis

Tests of difference (independent-samples t-tests) were conducted to ascertain whether males and females differed in pain responses, coping style, age, state anxiety and trait anxiety. Correlational analyses were conducted on the same variables to investigate associations between them.

4.4 Results

4.4.1 Data Screening

All raw data were screened and examined for normality of distribution. Where variable distributions were found to be badly skewed, appropriate transformations were applied to improve normality, as recommended by Tabachnick and Fidell (1996). Accordingly, the following variables were logarithmically transformed: pain tolerance, state anxiety, trait anxiety, pain threshold. Square root transformation was applied to affective pain scores and to all coping subscale scores. Reflection and square root transformation was applied to pain intensity scores. Although statistical analysis was conducted on transformed data if this was performed, raw data means and standard deviations are presented throughout for clarity and ease of interpretation.

4.4.2 Gender differences in coping style, anxiety and age

Descriptive statistics for coping subscales, state anxiety, trait anxiety and age can be found in Table 4.1.

Table 4.1: Mean questionnaire scores and ages of males and females, Experiment 1 (standard deviations in parentheses).

Measure	Males	Females
Coping		
CSQ Rei	13.39 (8.04)	11.00 (8.74)
CSQ Cat	11.23 (6.91)	11.54 (6.11)
CSQ IgSe	16.47 (7.08)	14.31 (7.75)
CSQ PraH	11.86 (5.45)	13.09 (4.90)
CSQ Divat	15.74 (6.95)	16.05 (7.68)
CSQ IncBe	14.31 (5.58)	14.67 (6.38)
CSQ Css*	21.82 (6.00)	17.93 (6.41)
CSQ AbCo	3.59 (1.02)	3.56 (1.07)
CSQ AbDe	3.12 (1.05)	2.94 (1.03)
Anxiety		
STAI-S	35.82 (10.89)	36.82 (9.63)
STAI-T	44.10 (10.67)	43.50 (11.16)
Age	24.90 (7.30)	23.40 (6.04)
n	51	55

Key: CSQ Rei = Reinterpreting painful sensations, CSQ Cat = Catastrophizing, CSQ IgS = Ignoring sensations, CSQ PraH = Praying and hoping, CSQ DivAt = Diverting attention, CSQ IncBe = Increasing behaviour, CSQ Css = Coping self-statements, CSQ AbCo = Ability to control pain, CSQ AbDe = Ability to decrease pain, STAI-S = State anxiety, STAI-T = StateTrait Anxiety Inventory-Trait, * = $p<.05$, ** = $p<.01$.

Independent samples t-tests revealed a significant difference between males and females on the coping self-statements (CSQ Css) coping subscale (t (104) = 3.27, $p<.01$), with males reporting greater use of coping self-statements than females. However, this difference should be interpreted with caution, as when Bonferroni-style correction for multiple tests is applied this small difference becomes non-significant. No significant differences were found for any other coping subscale, state anxiety, trait anxiety age.

4.4.3 Gender differences in pain indices

Although all participants pressed the first button (Just Noticeable Pain) appropriately, observation indicated that a few participants may not have pressed all the remaining buttons (Mild Pain, Moderate Pain, Severe Pain) promptly to indicate subsequent changes in pain level. Delayed button pressing may therefore have confounded the pain change rating data to some extent. Accordingly, pain change descriptives are presented for interest in Appendix 2 but ratings 2-4 inclusive (Mild Pain, Moderate Pain and Severe Pain) were not included in the main statistical analysis.

Descriptive statistics for pain threshold, pain tolerance, sensory pain, affective pain and pain intensity can be found in Table 4.2.

Table 4.2: Mean pain index scores for males and females, Experiment 1 (standard deviations in parentheses).

Pain Index	Males	Females
Pain threshold	13.55 (10.24)	10.50 (7.22)
Pain tolerance*	183.95 (112.80)	84.21 (73.41)
Sensory pain *	12.90 (6.00)	15.94 (5.52)
Affective pain	2.22 (2.26)	3.00 (3.02)
Pain intensity *	6.34 (1.73)	6.96 (1.83)
n	51	55

* = p<.05, ** = p<.01

Independent samples t-tests were conducted on group differences for males and females on pain threshold, pain tolerance, sensory pain, affective pain, pain intensity (VAS). Significant gender differences were found in pain tolerance (t (104) = 5.57, p<.001; see Fig 4.2), sensory pain (t (104) =-2.71, p<.01; see Fig 4.3) and pain intensity (t (104) = 2.12, p<.05; see Fig 4.4). As predicted, compared to females, males had higher pain tolerance and reported lower sensory pain and pain intensity.

Figure 4.2: Mean pain tolerance times of males and females, Experiment 1.

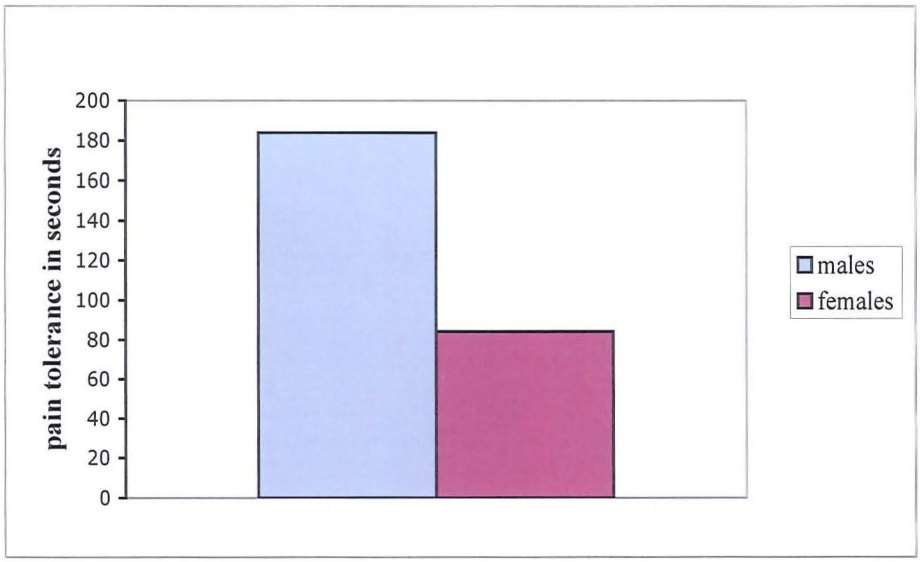


Figure 4.3: Mean sensory pain ratings of males and females, Experiment 1.

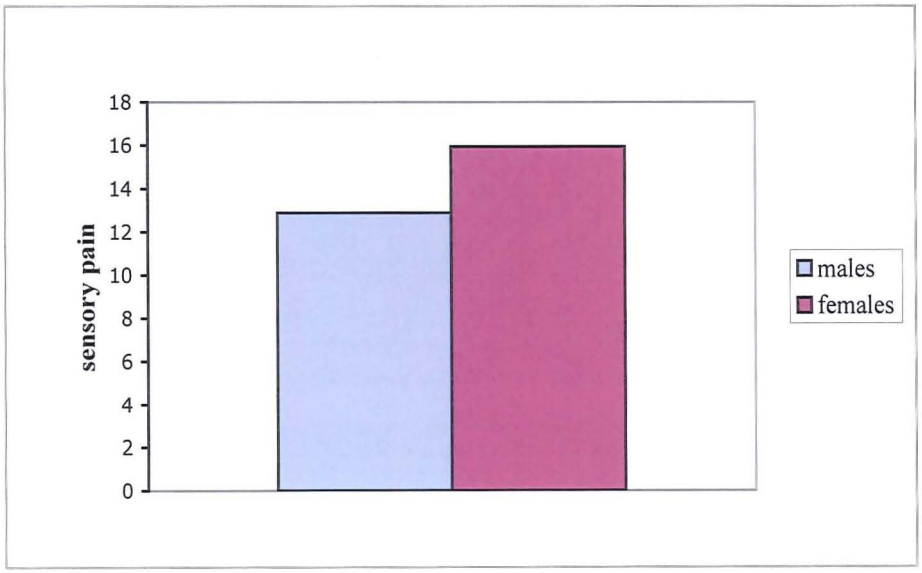
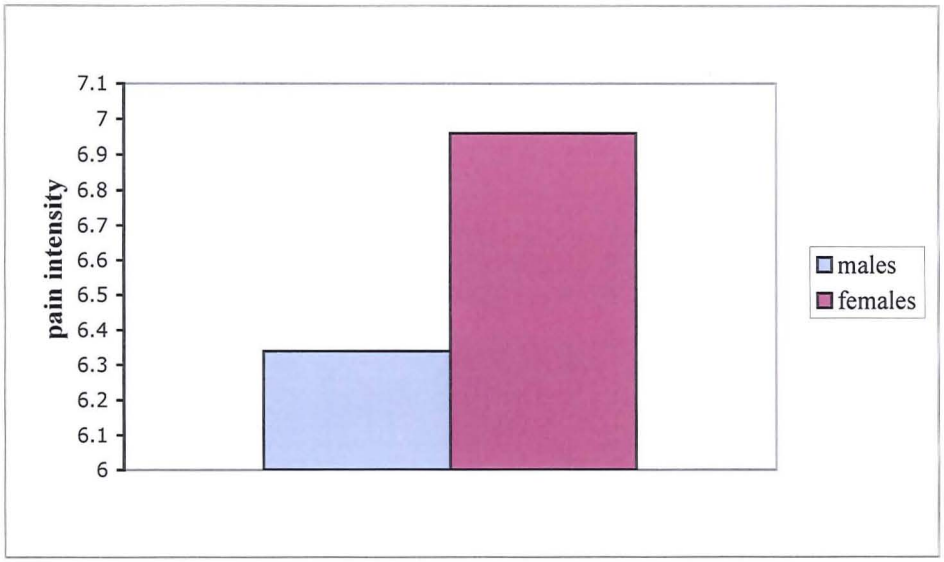


Figure 4.4: Mean pain intensity ratings of males and females, Experiment 1.



4.4.4 Correlation analysis

Pearson's correlations were conducted between pain indices, age and questionnaire scores for all participants (See Table 4.3). To establish whether there were gender differences in the pattern of correlations, the same analysis was repeated separately for males (see Table 4.4) and females (see Table 4.5).

Table 4.3: Correlations between questionnaire scores, pain indices and age for all participants, Experiment 1 (n = 106).

	Pain Threshold	Pain Tolerance	Sensory Pain	Affective Pain	Pain Intensity
Coping					
CSQ Rei	.104	**.243***	-.040	.007	.026
CSQ Cat	**-.209***	**-.194***	**.261****	.157	-.129
CSQ IgSe	.076	**.251****	-.071	.055	.173
CSQ PraH	-.104	**-.242***	.170	**.235***	-.033
CSQ DivAt	-.037	-.007	.097	.165	.016
CSQ IncBe	.014	.123	.064	.175	.140
CSQ Css	.109	**.226***	-.073	.054	.132
CSQ AbCo	-.097	.059	-.075	-.037	.145
CSQ AbDe	-.122	.039	-.078	.072	.038
Anxiety					
STAI-S	.044	.003	.097	.136	.124
STAI-T	.062	-.020	-.092	-.005	.004
Age	.052	.042	-.183	.033	.088

Key: CSQ Rei = Reinterpreting painful sensations, CSQ Cat = Catastrophizing, CSQ IgS = Ignoring sensations, CSQ PraH = Praying and hoping, CSQ DivAt = Diverting attention, CSQ IncBe = Increasing behaviour, CSQ Css = Coping self-statements, CSQ AbCo = Ability to control pain, CSQ AbDe = Ability to decrease pain, STAI-S = StateTrait Anxiety Inventory-State, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

For the whole sample, pain tolerance was positively associated with reinterpreting sensations, ignoring sensations and coping self-statements. This indicates that higher reported use of these methods of pain avoidance was associated with higher tolerance. Catastrophizing was positively correlated with sensory pain, and negatively correlated with pain tolerance and pain threshold. Praying/hoping was negatively correlated with pain tolerance, and there was also a small but significant positive correlation between praying/hoping and affective pain. These findings suggested that greater reported use of coping strategies which involve focus on pain (catastrophizing and praying/hoping) was associated with higher pain report and reduced threshold and tolerance. No significant associations were found between age, state anxiety or trait anxiety and the pain indices.

Table 4.4: Correlations between questionnaire scores, pain indices and age for males only, Experiment 1 (n = 51)

MALES	Pain Threshold	Pain Tolerance	Sensory Pain	Affective Pain	Pain Intensity
Coping					
CSQ Rei	-.016	.103	-.081	.009	-.020
CSQ Cat	-.204	-.259	**.332***	.259	-.151
CSQ IgSe	-.025	.179	-.128	-.037	.213
CSQ PraH	-.071	-.192	.027	.250	.028
CSQ DivAt	-.038	-.001	.041	.271	.058
CSQ IncBe	-.012	.113	.250	.245	.030
CSQ Css	.071	.053	.022	-.035	.017
CSQ AbCo	-.146	.226	-.115	**-.329***	.069
CSQ AbDe	-.122	.051	-.177	.003	.003
Anxiety					
STAI-S	.006	.127	.103	.279	.156
STAI-T	.129	-.117	.013	.164	-.007
Age	.064	.027	-.201	-.023	.047

Key: CSQ Rei = Reinterpreting painful sensations, CSQ Cat = Catastrophizing, CSQ IgS = Ignoring sensations, CSQ PraH = Praying and hoping, CSQ DivAt = Diverting attention, CSQ IncBe = Increasing behaviour, CSQ Css = Coping self-statements, CSQ AbCo = Ability to control pain, CSQ AbDe = Ability to decrease pain, STAI-S = StateTrait Anxiety Inventory-State, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p<.01

As can be seen from Table 4.4, when correlations were repeated for male participants only (n=51) a positive association between Catastrophizing and sensory pain and a negative association between Ability to Control pain and affective pain were found. However, the same analysis repeated for female participants (n=55) revealed no significant correlations.

Table 4.5: Correlations between questionnaire scores, pain indices and age for females only, Experiment 1 (n = 55)

FEMALES	Pain Threshold	Pain Tolerance	Sensory Pain	Affective Pain	Pain Intensity
Coping					
CSQ Rei	.251	.247	.041	.027	.007
CSQ Cat	-.196	-.133	.159	.031	-.177
CSQ IgSe	.174	.221	.048	.158	.074
CSQ PraH	.016	-.252	.163	.160	-.083
CSQ DivAt	.028	.006	.014	-.009	-.042
CSQ IncBe	.177	.233	-.127	.098	.152
CSQ Css	.090	.127	-.006	.177	.079
CSQ AbCo	-.021	.001	-.020	.213	.221
CSQ AbDe	-.107	-.035	.026	.028	.028
Anxiety					
STAI-S	.104	-.048	.065	.004	.131
STAI-T	-.011	.017	-.173	-.122	-.003
Age	.025	-.103	-.120	.144	.092

Key: CSQ Rei = Reinterpreting painful sensations, CSQ Cat = Catastrophizing, CSQ IgS = Ignoring sensations, CSQ PraH = Praying and hoping, CSQ DivAt = Diverting attention, CSQ IncBe = Increasing behaviour, CSQ Css = Coping self-statements, CSQ AbCo = Ability to control pain, CSQ AbDe = Ability to decrease pain, STAI-S = StateTrait Anxiety Inventory-State, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** =p< .01

4.4.5 Coping composites

Following Boothby, Thorn, Stroud and Jensen (1999) coping composites were produced from CSQ coping subscales. Based on the whole sample correlations observed here, two statistically-derived coping style composites were produced from subscales which were significantly correlated with pain tolerance, and which were also significantly intercorrelated. One composite was the sum of three CSQ subscales, which involved directing attention away from pain in various ways (reinterpreting sensations, ignoring sensations, coping self-statements) and was labelled 'Avoidant Coping Composite' (ACC).

The other composite was derived from the two CSQ subscales in which attention is directed towards pain (catastrophizing, praying and hoping) and was labelled 'Non-Avoidant Coping Composite' (NACC). The two coping style composites, ACC and NACC, were found to be correlated with pain tolerance to a similar degree and in the same direction as their component subscales (r (106) = .278, p<.01 and r (106) = -.248 , p<.05, respectively).

Independent-samples t-tests were conducted to establish whether males and females differed on the two coping composites. A significant difference was found for ACC (t (104) = 2.40, p<.05), indicating that males reported more avoidant coping than females (male mean = 51.69, SD = 16.59; female mean = 43.24, SD = 19.46). No gender difference was found for NACC.

Correlational analyses of coping composites with pain indices were therefore conducted separately for males and females. Similar to the analysis of individual coping subscales with pain indices, a different pattern of associations was found for males than for females. For males, NACC was positively associated with affective pain (r (51) = .29, p<.05), whereas for females, no significant associations were found.

4.5 Discussion

The aims of Experiment 1 were to ascertain whether there were differences in cold pressor pain responses between men and women, and whether coping style contributed to gender-differentiated pain responses.

As expected, gender differences were found on several pain indices. Women demonstrated lower tolerance for pain and reported more sensory pain and pain intensity than men. Pain threshold, for which gender differences are generally smaller than for pain tolerance, also showed a gender-based differentiation in the predicted direction (i.e., lower for females than males) but this did not reach significance. This pattern of findings has not been uncommon in previous research, and may be related to the effect size of gender differences in pain threshold which have been calculated as smaller than the effect size of gender differences in pain tolerance (Riley et al., 1998). However, with more than 50 per group in Experiment 1, this should have been adequate.

Unlike previous research, no differences in state or trait anxiety were found between men and women, nor were any associations found between either measure of anxiety and any pain index. Anxiety may not have been relevant in this experiment for a number of reasons. For example, mean STAI scores of this healthy volunteer sample were consistent with norms established with similar non-clinical groups of a similar age range (Spielberger et al., 1983). In other words, participants in the present experiment were not a highly anxious group, nor was there an experimental induction of anxiety. Furthermore, any anticipatory anxiety about the cold pressor task may have been allayed by knowledge that the sensations elicited would be both temporary and harmless.

Coping can have powerful impact on pain responses, and if, as previous research suggests, men and women differ in their pain coping (see Unruh et al., 1999) this factor may contribute to gender differences in pain responses. Very little evidence of relationships between coping style and pain responses was found in Experiment 1, except that males reported a greater tendency than females to engage in self-reassurance about coping with pain. In contrast to previous research (e.g, Sullivan et al., 2000; Keefe et al., 2001) no gender differences in catastrophizing were found in this experiment, nor was coping style or catastrophizing in particular found to mediate gender effects on pain responses. A possible reason for this disparity may be that many of the studies which have found such gender differences have done so using the Pain Catastrophizing Scale (PCS; Sullivan, Bishop & Pivik, 1995), a questionnaire specifically designed to measure the construct, whereas in the present experiment catastrophizing was measured by a subscale of the CSQ. Associations were found between catastrophizing and several pain indices in the present experiment (pain threshold, pain tolerance and sensory pain).

A self-reported coping style involving attention directed towards pain (NACC) was found to be associated with and lower pain tolerance, and a coping style involving more avoidant coping style (ACC) was associated with higher pain tolerance. However, this experiment has not provided evidence that the effects of gender and of coping style on pain responses are inter-related.

Experiment 1 provided some indication that coping style influences pain responses, but found little difference overall in the ways in which men and women report that they cope with pain, and did not find interactive effects of gender and coping. One possible reason for this is the lack of direct involvement of coping strategies in the experiment. Participants were not instructed to actively engage in coping strategies during the pain task. The assessment of coping made here was via a questionnaire completed before the cold pressor task, and as such constitutes a passive self-report measure of coping. The salience of coping was therefore arguably low for these healthy volunteers, certainly much lower than it would be for chronic pain patients. Different results may have been found with a comparison of coping strategies actually implemented during cold pressor exposure.

4.6 Summary

In summary, this exploratory study has confirmed that females are less able to tolerate cold pressor exposure and find it more painful than males; gender differences were found in both behavioural and self-report measures of pain which is consistent with previous research. Although evidence was found of associations between gender and pain as well as between coping style and pain, the fact that these relationships were independent of each other may have been due to a methodological limitation of this experiment, namely the manner in which coping style was assessed.

In light of these findings, Experiment 2 was designed to establish whether the gender differences in pain response found here would replicate, and to directly test the effect of coping style on pain responses using an experimental manipulation of coping strategy during cold pressor exposure.

Chapter 5

Experiment 2
Gender, Coping Strategies and Cold Pressor Pain I

5.1 Introduction

Despite an innate tendency for pain to capture attention (Eccleston & Crombez, 1996) pain sufferers commonly report that they attempt to 'take their mind off' pain. There has been some evidence that avoidant coping, such as distraction, is best when pain is short-term (e.g., Mullen & Suls, 1982; Farthing, Venturino & Brown, 1984), whereas non-avoidant coping is better if pain persists (Suls & Fletcher, 1985). However, as discussed in Chapter 3, the relative effectiveness of coping by directing attention towards or away from pain remains in debate (Cioffi, 1991, Suls & Wan, 1989). For example, Cioffi and Holloway (1993) have found that if attention is diverted away from experimental pain, it dissipates more slowly than if attention is focused on it, and recent research with cold pressor pain has indicated that avoidant and non-avoidant coping may be differentially effective for men and women (Keogh et al., 2000).

The design of Experiment 2 draws on both the findings and the limitations of Experiment 1, in which some evidence of differences in male and female coping style was found, specifically a greater self-reported tendency towards avoidant coping in males than females. Avoidant coping style was associated with higher pain tolerance in Experiment 1, whereas non-avoidant coping style was inversely related to pain tolerance.

Following up on the avoidant and non-avoidant coping style composites found to be important in Experiment 1, an avoidant and a non-avoidant strategy were selected for use in Experiment 2. Cioffi and Holloway (1993) treated suppression of thoughts about pain and distraction from pain as separable types of avoidant coping. However, it seems improbable that any individual in pain can keep their thoughts off pain *and* any other attentional target. It is more likely that to suppress thoughts about pain, attention must be diverted elsewhere - and the cognitive effort of trying to suppress thoughts about pain arguably constitutes distraction in itself. Distraction, rather than suppression, was therefore selected as the avoidant coping method in Experiment 2 and a no-treatment control group was not incorporated, as participants given no instructions were considered likely to engage spontaneous coping strategies (Chaves & Brown, 1985; Cioffi & Holloway, 1993). The coping techniques compared in Experiment 2 were focusing attention on the physical sensations elicited during noxious stimulation (non-avoidant strategy), and distracting attention away from painful sensations using

imaginal distraction (avoidant strategy). These techniques are referred to as non-avoidant and avoidant coping throughout the chapter. These strategies were selected for contrast in attentional direction but similarity in level of efficacy. Among avoidant techniques, imaginal distraction seems to be more effective than strategies such as repetitive cognitions or counting, and is among the most effective overall (Fernandez & Turk, 1989). Imagery is also a common component of the spontaneous coping methods used by pain patients (Turk, Meichenbaum & Genest, 1983). Similarly, focusing on the sensory qualities of pain rather than the emotional aspects of the pain experience is among the most effective methods of coping with short-term pain experiences (Suls & Fletcher, 1985), and has even been found to be superior to avoidance for acute pain in some studies (e.g., McCaul & Haugtvedt, 1982).

There were three primary research objectives in Experiment 2. The first of these was to establish whether the gender differences in cold pressor pain responses found in Experiment 1 would replicate. The second objective of Experiment 2 was to empirically test the relative effectiveness of avoidant and non-avoidant coping for cold pressor pain using an experimental manipulation of coping strategies during pain induction. It was reasoned that this would improve on the questionnaire assessment of coping style used in Experiment 1, which was conducted prior to (and thus out of context with) the pain that participants experienced during cold pressor exposure. By instructing participants to actively engage in coping strategies during cold pressor pain in Experiment 2, it was expected that coping would acquire greater salience for participants and that a direct comparison of the two types of coping would be facilitated. No interaction between gender and coping was found in Experiment 1, perhaps because pain coping instructions were not implemented during pain induction The coping manipulation would also facilitate the third objective, which was to examine the potential interaction of gender and coping in the context of cold pressor pain.

A secondary objective of Experiment 2 was to investigate potential gender differences in pain-related cognition and emotion. Previous research indicates that such differences are likely, and may contribute to gender differences in pain responses. Specifically, women seem to experience more fearful and anxious thoughts in anticipation of pain (Rollman, Lautenbacher & Jones, 2000) and also to express more negative emotion in response to pain (Robinson, Riley & Myers, 2000). Comments from participants in

Experiment 1, although not formally assessed, reiterated this pattern. For example, comments from numerous female participants revealed that before the cold pressor task they were 'dreading it' or 'couldn't stop thinking about how much it would hurt' or were fearful or worried about the forthcoming pain. Very few such comments were made by male participants in Experiment 1, which may indicate a difference both in the way males and females think and feel about pain and in their tendency to express such thoughts and feelings. Accordingly, assessments of pain-related cognition and emotion were incorporated in Experiment 2 and a measure of social desirability was also included.

It was expected that females would show greater sensitivity to cold pressor pain than males, specifically that women would demonstrate lower pain tolerance and report greater sensory pain and affective pain than men. It was expected that avoidant and non-avoidant coping might differentially affect the rate at which painful sensations dissipate after cold pressor exposure, specifically that pain recovery would be slower with avoidant coping. It was also predicted that avoidant and non-avoidant coping might differentially affect pain perception for males and females.

Women were expected to report more pain-relevant thoughts prior to cold pressor exposure (Thought Records) and more anticipatory fear of cold pressor than men. Gender differences were also expected in negative emotion and catastrophizing, specifically that women would report more negative emotion and catastrophize about pain more than men. It was expected that emotional states might vary as a function of coping strategy but the likely direction of such effects was not specified.

5.2 Method

5.2.1 Design

A mixed design was employed with gender as the between-groups factor (male vs. female) and coping strategy as the within-groups factor (avoidant vs. non-avoidant). The main dependent variables were pain threshold, pain tolerance, sensory pain, affective pain and pain intensity[1].

Pain recovery, operationally defined as time elapsed from withdrawal from cold pressor to cessation of pain, became a sixth dependent variable in Experiment 2. Additional variables were emotional state, catastrophizing, negative affect, trait anxiety, social desirability, expectations about cold pressor (Anticipatory Responses) and pain-related cognition prior to cold pressor exposure (Thought Records).

5.2.2 Participants

63 participants (32 male, 31 female) were recruited from the student and staff population at Goldsmiths College. The pain response data of 1 participant (male) was lost through equipment failure, 2 participants' data (1 male, 1 female) were removed because they reported no perception of pain threshold, and a further 7 participants (6 males, 1 female) were dropped from the analysis due to non-compliance with coping strategy instructions during one or both cold pressor trials. The resultant total number of participants was 53 (24 male, 29 female). Participant age range was 20-43 years (mean 28.7 years, SD = 6.12). All participants were in good general health, were not currently in pain or taking any medication, and had not consumed any alcohol or analgesics on the day of testing. The standard exclusion criteria for cold pressor were used, as outlined in the previous study. Participants were paid a nominal sum of £3.00 for their participation.

5.2.3 Thought Records

In order to assess potential differences between the pain-related cognition of males and females in Experiment 2, their thoughts immediately prior to cold pressor exposure were recorded. Participants were asked to verbalise their thoughts continuously for 5

[1] Although the pain change rating system in Experiment 1 was originally intended for use throughout the series of cold pressor experiments, the accuracy of some ratings was unclear in Experiment 1. Consequently, a simplified pain rating system with only two button presses was implemented in all subsequent experiments. The first button, labelled 'Just Noticeable Pain' was retained to provide a measure of pain threshold and the method of pain tolerance measurement remained unchanged. A second button, labelled 'No More Pain', provided an index of recovery time after withdrawal from the cold pressor.

minutes whilst alone in the laboratory, and were audiotaped. A similar procedure has been used by Sullivan, Rouse, Bishop & Johnston (1997) using a written thought record. While it is unlikely that any method will capture 100% of participant thoughts (a certain level of *ad hoc* self-censorship must be assumed) it was expected that the audiotape method would inhibit the free flow of thought less than the written method. The exact instructions given to participants regarding verbalisation of their thoughts appear in Appendix 3. Participants were assured of confidentiality and anonymity and informed that the audiotape would be heard only by researchers for the purposes of data coding and erased afterwards.

Loss of 15 participants' Thought Records occurred due to inaudible recorded speech levels or participant silence during the recording interval either due to misunderstanding of instructions or non-compliance. By definition, in aiming to capture the initial thoughts of participants as they approached the cold pressor pain induction for the first time, the Thought Record procedure could not be repeated for any participant. Thought Records were thus obtained for a total of 41 participants (18 males, 23 females).

Thought Records were coded independently by two judges (S.S., C.O.) who were blind to the experimental hypotheses and to participants' scores on any other measure. Verbalised thoughts were classed as 'pain related' if they referred to aspects of the forthcoming pain induction procedure (e.g., "The water looks very cold"), anticipatory emotions (e.g., "I'm feeling quite nervous about it putting my hand in there now"), anticipated sensations (e.g.,"I wonder how much it will hurt") or anticipated tolerance (e.g., "Don't know how long I can keep my hand in there"). Phrase structure based on speech cadence (i.e., where phrases or sentences sounded as if they were separated by periods) was used to unitize Thought Records. Participant scores thus obtained were the number of occurrences of pain-related thoughts during the 5 minute period.

5.2.4 Pain induction

5.2.4.1 Cold pressor apparatus and task

The cold pressor apparatus and task were the same as in Experiment 1.

5.2.4.2 Pain ratings

A simplified pain rating system with only two button presses (pain threshold and pain recovery) was implemented. The modified instructions given to participants appear in Appendix 4. As in Experiment 1, pain tolerance was measured via infra-red detection of entry to and withdrawal from the cold pressor. Self-report pain was assessed using the Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987).

5.2.5 Additional questionnaires

State-Trait Anxiety Inventory: The trait form of the State-Trait Anxiety Inventory (STAI-T; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983) was administered as in Experiment 1.

Pain Catastrophizing Scale: Catastrophizing was assessed using the Pain Catastrophizing Scale (PCS; Sullivan, Bishop & Pivik, 1995) and was administered before cold pressor exposure. The PCS was chosen over the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983) given its more specific focus on catastrophizing. The PCS is a 13-item self-report measure of catastrophic thinking associated with pain, which provides a measure of general tendency to catastrophize (total score) as well as three subscale measures: 'magnification' (tendency to exaggerate the threat value of pain stimuli); 'rumination' (tendency to increase attentional focus on pain-related thoughts) and 'helplessness' (tendency to adopt a helpless orientation to coping with painful situations). Item scores are on a 5-point scale from 0 (*not at all*) to 4 (*all the time*). Scoring range is 0-52 for general catastrophizing (total). Scoring ranges of the subscales are as follows: magnification 0-12, rumination 0-16, helplessness 0-24. Satisfactory reliability and validity has been demonstrated for the PCS (Sullivan et al., 1995).

Depression Anxiety Stress Scales: To assess potential differences in negative affectivity between males and females the short version of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) was administered. The DASS21 is

a self-report measure used to assess recent (over the past week) symptoms of depression, anxiety and stress. The response format is a 4-point scale where 0 = 'did not apply to me at all', 1 = 'applied to me to some degree or some part of the time', 2 = 'applied to me to a considerable degree or a good part of the time, 3 = 'applied to me very much or most of the time'. In the DASS21, there are 7 items in each subscale (depression, anxiety and stress). Total subscale scores on the DASS21 are doubled to give a range of scores between 0-42 for each subscale. The DASS has been shown to have satisfactory reliability and validity (Lovibond & Lovibond, 1995). The DASS21 was administered before cold pressor exposure.

Marlowe-Crowne Social Desirability Scale: In view of the potential impact of gender-role normative influence on male and female pain report (LeResche, 1999) the Marlowe-Crowne Social Desirability Scale (MCSDS; Crowne & Marlowe, 1960) was incorporated to ascertain whether men and women in Experiment 2 differed in social desirability. The MCSDS is a 33-item self-report questionnaire designed to assess the extent to which individual seek to present themselves in a favourable light. The MCSDS has a True/False response format, with 18 items scoring 1 when answered true and 0 when answered false and the remaining 15 items reverse-scored. Total score is the sum of all item scores and gives a range of 0-33. The MCSDS was administered before cold pressor exposure.

Positive and Negative Affect Schedule: The Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988) was used to provide an assessment of emotional state in the context of cold pressor pain. The PANAS is a 20-item self-report measure of positive and negative emotion, comprising 10 positive affect (PA) items and 10 negative affect (NA) items. Item scoring on both subscales is on a 5-point scale from 1 (*very slightly or not at all*) to 5 (*extremely*). Total PA and NA scores therefore range from 10-50. Both subscales have shown satisfactory reliability and validity (Watson et al., 1988). The state form of the PANAS was administered twice in each coping condition: before cold pressor exposure (but after coping instructions were given) and after cold pressor exposure (when pain had ceased).

Anticipatory Responses questionnaire: A simple questionnaire was devised to assess anticipatory reactions to cold pressor pain, in males and females. Three aspects of

participants' expectations of their own responses to cold pressor exposure were measured; these are referred to collectively as 'Anticipatory Responses'. The first of these was an estimate of expected tolerance for cold pressor exposure, in minutes and/or seconds ('Tolerance Estimate'). The remaining Anticipatory Response measures were 100mm VAS rating scales of fearfulness of cold pressor exposure ('Fear of CPT') and of expected self-control of sensations in the exposed hand during cold pressor ('Sensation Control') respectively. Anticipatory Responses measures were obtained at 3 time points: i) before any discussion of coping strategy (baseline), ii) after sensory focusing coping instructions were given, just before entry to cold pressor (pre-CPT) and iii) after imaginal distraction coping instructions were given, just before entry to cold pressor (pre-CPT).

Coping manipulation check questionnaire: To assess whether participants had adhered to the coping instructions, and how effective they had found the strategies for coping with the cold pressor pain, a simple coping manipulation check questionnaire was devised and administered after each cold pressor exposure. Simple categorical ratings were used to indicate adherence or lack of adherence to coping instructions. The response format for perceived effectiveness of strategy was a 5-point Likert-type scale, where 0 = 'not at all effective', 1 = 'slightly effective', 2 = 'moderately effective', 3 = very effective', 4 = 'extremely effective'.

5.2.6 Coping manipulation

All participants were instructed to use avoidant coping during one cold pressor task and non-avoidant coping in the other. Avoidant coping involved participants distracting themselves by visualising their own living room in detail whereas non-avoidant coping entailed focusing on the detail of the physical sensations elicited by the cold pressor (adapted from Cioffi & Holloway, 1993). Both sets of instructions contained five sentences and had similar grammatical structure, with the aim that they should differ solely in meaning, rather than in additional ways unrelated to the purpose of the study. The exact instructions given appear below. Coping condition was counterbalanced (ABBA) to control for order effects.

Avoidant coping instructions: *'When you put your hand in the cold water, we would like you to picture in your mind's eye your own living room at home. Imagine it in as much detail as possible; move around the room looking at all the familiar things there, such as ornaments, pictures and pieces of furniture. All the time your hand is in the cold water keep picturing your living room as vividly and strongly as you can; make sure you notice everything about your room, the colours in it, and the characteristic smell of the room, the feel of all the different textures in it. Imagine yourself there in your living room, walking across it, perhaps putting on some music you like, or your favourite TV programme, and sitting down on a comfortable chair. Keep vividly imagining your room the whole time your hand is in the cold water.'*

Non-avoidant coping instructions: *'While your hand is in the cold water, we would like you to pay very close attention to the sensations you feel in that hand. Focus in on the feelings you have in your hand in as much detail as possible, noticing the exact location, quality and intensity of those feelings. All the time your hand is in the cold water, keep monitoring as closely as you can all the different sensations you experience; make sure you notice everything about how your hand feels. Pay close attention to all aspects of the feelings in your hand, noticing if those feelings change, if they increase or decrease or if they move at all. Keep your attention fully focused on the sensations in your hand the whole time it is in the cold water.'*

5.2.7 Procedure

Participants were given a standardised explanation of the experimental procedure, including assurances regarding the safety of the cold pressor task, notification of their right to withdraw, and assurances of confidentiality and anonymity in data analysis. After completing the preliminary questionnaires (Anticipatory Responses, PCS, STAI-T, DASS21) participants were taken into the laboratory and shown the cold pressor equipment. Immediately after this, and before pain induction, participant Thought Records were obtained. Coping instructions were then given to participants to read and a verbal check for understanding was made. The Anticipatory Responses questionnaire and the PANAS were administered immediately before each cold pressor task. After withdrawal from the cold tank participants placed their hand on a towel and passively awaited the cessation of pain, without flexing or rubbing the hand. All participants underwent two cold pressor exposures and completed the SF-MPQ, the PANAS and the

coping manipulation check questionnaire immediately after each one. A 15-minute intertrial interval was timed from the point at which participants signalled pain recovery after the first cold pressor exposure was terminated, during which the MCSDS was completed. Participants gave informed consent in writing before the pain induction procedure and were debriefed at the end of the experiment. All participants were tested individually by the same experimenter.

5.2.8 Statistical analysis

Independent-samples t-tests were conducted to examine potential differences in negative affect, pain catastrophizing, trait anxiety, social desirability, Thought Records and age between males and females. Analysis of variance was used to examine the effects of gender and coping instructions on emotional states, and also to evaluate the effects of gender and time of measurement on Anticipatory Responses. Analysis of variance was also used to examine the effects of gender and coping instructions on pain indices. Analysis of coping condition order was also incorporated. Correlational analyses were applied to examine relationships between pain indices and all other variables.

5.3 Results

5.3.1 Data screening

All raw data were screened and examined for normality of distribution. Appropriate transformations were applied where necessary, as recommended by Tabachnick and Fidell (1996). Accordingly, the following variables were logarithmically transformed: pain threshold, pain tolerance, pain recovery, Tolerance Estimate and age of participants. Square root transformation was applied to negative affectivity scores (stress, anxiety, depression), Fear of CPT, Sensation Control and Thought Record scores. As before, statistical analysis was conducted on transformed data where appropriate but all means and standard deviations presented are raw scores.

5.3.2 Coping adherence

Only data from participants who reported adherence to the coping instructions given was included in statistical analysis. According to the coping manipulation check, in the non-avoidant coping condition 60.4% of participants reported they had used only the strategy as instructed, and the remaining 39.6% followed the instructions but also used

an additional (self-selected) strategy. Similarly, in the avoidant coping condition 62.3% of participants reported they had used the coping instructions only, and 37.7% had used distraction as instructed plus an additional strategy.

5.3.3 Thought Records, negative affectivity, pain catastrophizing, trait anxiety, social desirability and age

Means and standard deviations of Thought Records, negative affectivity (depression, anxiety, stress), pain catastrophizing, trait anxiety and social desirability scores and age are presented in Table 5.1

Table 5.1: Mean Thought Record scores, questionnaire scores and ages of males and females, Experiment 2 (standard deviations in parentheses).

	Males	Females
Thought Record	3.33 (4.60)[1]	5.09 (4.04)[2]
DASS		
Stress	11.58 (6.03)	14.96 (8.88)
Anxiety	5.79 (4.92)	6.04 (7.15)
Depression	8.46 (9.10)	6.29 (7.12)
PCS		
Total*	16.42 (7.96)	21.41 (9.46)
Rumination*	6.08 (2.90)	8.72 (3.71)
Magnification	3.12 (1.92)	3.86 (2.86)
Helplessness	7.21 (4.12)	8.83 (4.99)
STAI-T	44.04 (10.44)	40.45 (7.73)
MCSDS	11.58 (5.81)	9.41 (4.83)
Age	30.13 (6.34)	27.55 (5.77)
n	24	29

Key: DASS = Depression Anxiety and Stress Scales, PCS = Pain Catastrophizing Scale, STAI-T = State-Trait Anxiety Inventory - Trait, MCSDS = Marlowe-Crowne Social Desirability Scale, * = $p<.05$, ** = $p<.01$, [1] n = 18, [2] n = 22

Independent samples t-tests were conducted on the above measures between males and females. Women scored significantly higher than men on the total catastrophizing scale (t (51) = -2.09, p<.05) and on the rumination subscale of the PCS (t (51) = - 2.90, p<.01). No other gender differences were found among these variables.

5.3.4 Anticipatory Responses

Means and standard deviations of Anticipatory Responses prior to cold pressor exposure (Tolerance Estimate, Fear of CPT, Sensation Control) at baseline, after reading avoidant coping instructions and after reading non-avoidant coping instructions are presented in Table 5.2

Table 5.2: Mean Anticipatory Response scores of males and females, Experiment 2 (standard deviations in parentheses).

Anticipatory Response	Males	Females
Tolerance Estimate*		
Baseline	164.2 (154.75)	79.23 (75.30)
Avoid	157.37 (188.90)	66.87 (58.36)
Non-avoid	156.37 (163.00)	83.37 (95.08)
Fear of CPT***		
Baseline	9.6 (10.41)	27.80 (26.53)
Non-avoid	16.63 (18.71)	36.70 (26.00)
Avoid	17.02 (19.25)	43.72 (25.21)
Sensation Control**		
Baseline	56.70 (27.26)	39.30 (26.18)
Non-avoid	45.47 (28.89)	31.58 (18.41)
Avoidant	48.32 (21.70)	34.62 (23.54)
n	24	29

*= p<.05, ** = p<.01, *** = p<.001

Large standard deviations were apparent in the Anticipatory Responses data. This indicates high variability in the raw scores, which is probably due to the nature of the data (e.g., self-rated estimates of pain tolerance) rather than the existence of outliers. Transformations, which were applied to all Anticipatory Response variables, improved the normality of the distributions prior to statistical analysis. A series of repeated measures (2 x 3) analyses of variance were conducted to evaluate the effects of gender (male vs. female) and condition (baseline vs. avoidant coping vs. non-avoidant coping) on Tolerance Estimate, Fear of CPT and Sensation Control.

5.3.4.1 Tolerance Estimate

There was a significant main effect of gender (F (1,51) = 5.16, p<05), with males rating their anticipated tolerance of CPT higher (mean = 167.61, SD = 169.69) than females did (mean 78.09, SD = 67.35). No main effect of coping condition or significant interaction between gender and coping condition was found.

5.3.4.2 Fear of CPT

There was a highly significant main effect of gender on Fear of CPT (F (1,51) = 16.86, p<001), with females reporting greater fearfulness (mean = 36.40, SD = 22.97) of the cold pressor task than males (mean = 11.62, SD = 10.97). A significant main effect of condition on Fear of CPT was also found (F (2,51) = 8.98, p<.001). Simple effects analysis revealed that fear of CPT reported at baseline was lower (mean = 19.50, SD = 23.01) than in both the avoidant condition (mean = 30.10, SD = 26.37; F (1,51) = 14.15, p<.001) and the non-avoidant condition (mean = 25.90, SD = 25.02; F (1,51) = 7.72, p<.01). However, Fear of CPT in the avoidant condition did not differ significantly from fear of CPT in the non-avoidant condition. No significant interaction between gender and condition was found.

5.3.4.3 Sensation Control

There was a significant main effect of gender (F (1,51) = 9.40, p<01), with males reporting greater expected Sensation Control (mean = 52.40, SD = 21.67) than females (mean = 35.11, SD = 19.18). A significant main effect of condition was also found (F (2,51) = 4.38, p<.05). Simple effects analysis showed that expected Sensation Control was significantly higher at baseline (mean = 48.0, SD = 28.09) than in the non-avoidant coping condition (mean = 38.0, SD = 25.03; F (1,51) = 8.92, p<.01). Expectancy of

Sensation Control did not differ significantly between baseline and the avoidant coping condition, nor between non-avoidant and avoidant coping conditions. No significant interaction between gender and condition was found.

5.3.5 Pain indices

Means and standard deviations of behavioural and self-report pain index scores for males and females in avoidant and non-avoidant coping conditions are presented in Table 5.3

Table 5.3: Mean pain index scores of males and females in avoidant and non-avoidant coping conditions, Experiment 2 (standard deviations in parentheses).

Pain Index	Males		Females	
	Non-avoidant	Avoidant	Non-avoidant	Avoidant
Threshold	23.57 (17.61)	26.91 (26.38)	23.55 (24.17)	20.48 (16.08)
Tolerance	136.23 (118.65)	141.43 (118.38)	90.02 (102.09)	106.52 (107.70)
Recovery*	92.65 (121.61)	126.22 (113.79)	115.14 (134.79)	103.23 (91.85)
Intensity	51.46 (22.61)	48.35 (26.06)	58.07 (21.43)	57.12 (22.42)
Sensory*	10.67 (5.96)	10.83 (5.61)	14.31 (5.15)	13.65 (6.18)
Affective*	2.50 (2.70)	1.87 (1.72)	3.79 (2.96)	3.34 (3.05)
n	24	24	29	29

* = p<.05, ** = p<.01

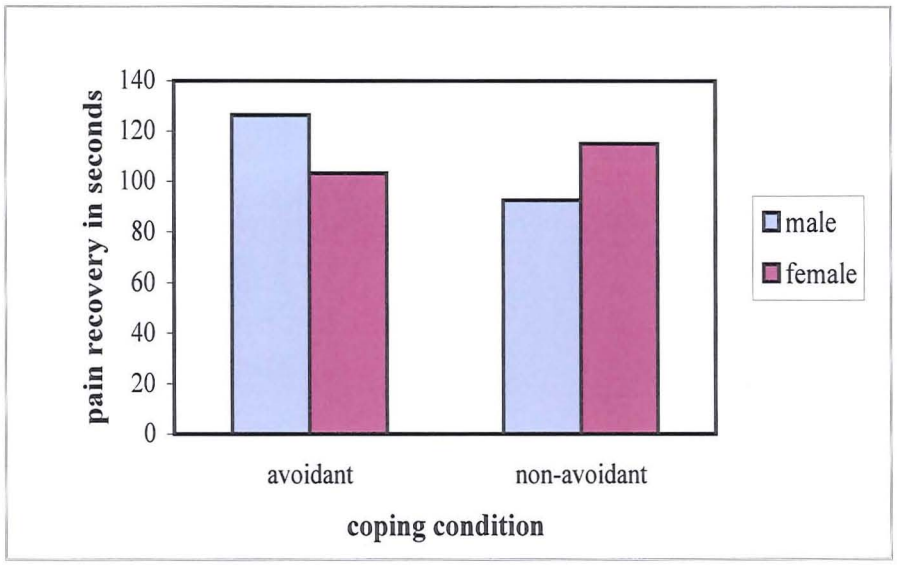
5.3.5.1 Behavioural pain indices

A series of mixed groups ANOVAs were conducted to evaluate the effect of gender and coping strategy on behavioural pain responses. In each analysis the between-groups factor was gender (male vs. female) and the within-subjects factor was coping condition

(avoidant vs. non-avoidant). The dependent variables were variously pain threshold, pain tolerance and pain recovery, measured in seconds.

No significant main or interaction effects of gender or coping were found for pain threshold or pain tolerance. A significant interaction between gender and coping was found for pain recovery (F (1,49) = 5.30, p<.05; see Figure 5.1). Post-hoc paired-samples t-tests showed that pain recovery was significantly faster for males when they used non-avoidant coping than when they used avoidant coping (t (23) = -2.17, p<.05). However, this difference did not remain significant when Bonferroni –type corrections were applied. For women, pain recovery did not differ significantly across coping conditions. No effects of coping condition order were found.

Figure 5.1: Mean pain recovery times in seconds for males and females in avoidant and non-avoidant coping conditions, Experiment 2.



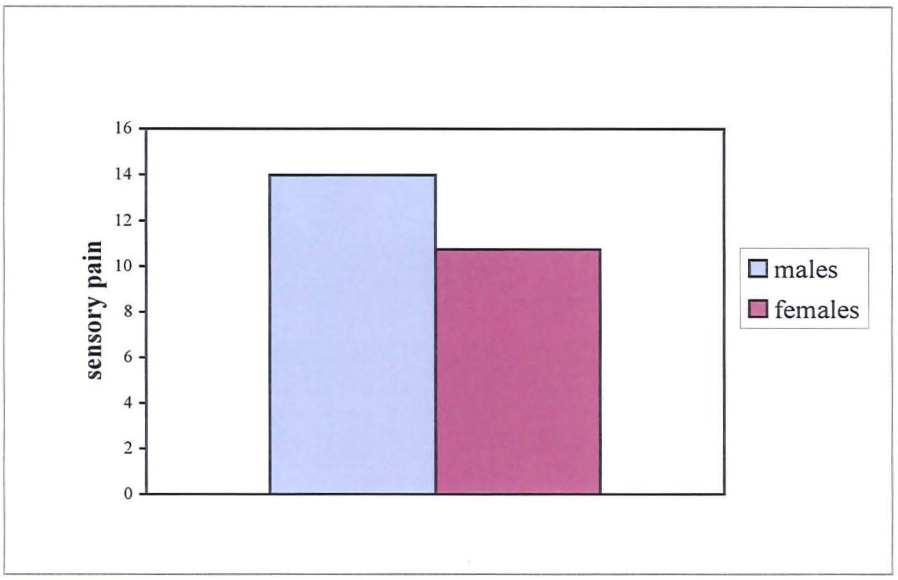
5.3.5.2 Self-report pain indices

Mixed-group ANOVAs were also used to evaluate the effects of gender and coping strategy on self-report pain indices. As before, in each analysis the between-groups factor was gender (male vs. female) and the within-groups factor was coping condition (avoidant vs. non-avoidant). The dependent variables were sensory pain, affective pain and pain intensity.

A significant main effect of gender was found for sensory pain (F (1,49) = 4.60, p<.05; see Fig 5.2). This effect was in the predicted direction, with females reporting higher

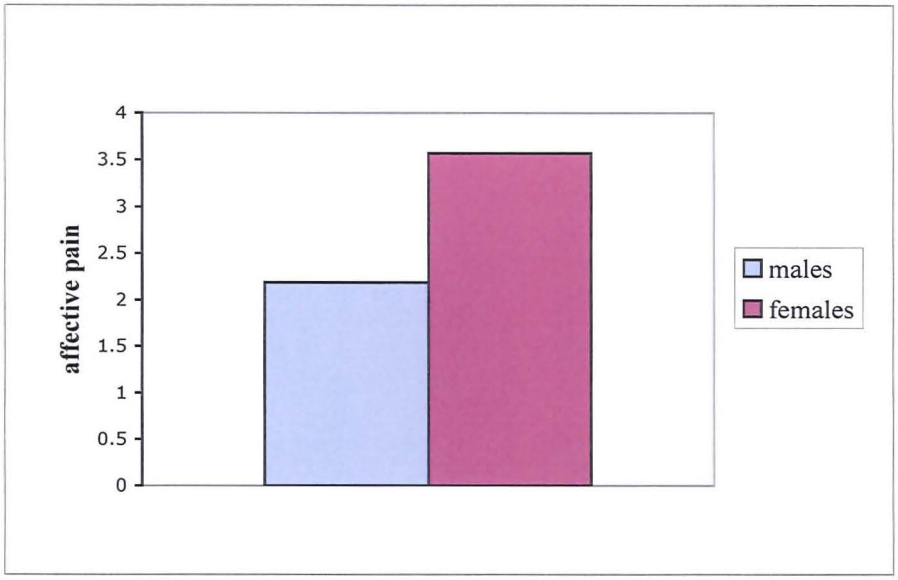
sensory pain (mean = 13.98, SD = 5.36) than males (mean = 10.75, SD = 5.45). No significant main effect of coping or interaction effects were found for sensory pain.

Figure 5.2: Mean sensory pain ratings for males and females, Experiment 2



A significant main effect of gender was also found for affective pain (F (1,49) = 4.14, p<.05; see Fig 5.3), with females reporting higher affective pain (mean = 3.57, SD = 2.76) than males (mean = 2.19, SD = 1.93). There was a near-significant main effect of coping on affective pain (F (1, 49) = 3.91, p<.056), with higher affective pain reported in the non-avoidant coping condition (mean = 3.21, SD = 2.89) than in the avoidant coping condition (mean = 2.68, SD = 2.62). No significant interaction was found between gender and coping on affective pain. As with the behavioural pain indices, no significant effects of coping condition order were found for self-report pain indices.

Figure 5.3: Mean affective pain ratings of males and females, Experiment 2



5.3.6 Emotion

Means and standard deviations of positive and negative emotion (PANAS) scores measured by gender, coping strategy and time of testing are presented in Table 5.4.

Table 5.4: Mean emotion scores measured by gender, coping condition and time of testing, Experiment 2 (standard deviations in parentheses)

		Males		Females	
Emotion	**Time**	**Non-avoid**	**Avoid**	**Non-avoid**	**Avoid**
Positive	**Pre-CPT**	25.50 (7.74)	26.12 (7.71)	26.00 (8.48)	25.45 (7.33)
	Post-CPT	23.20 (9.26)	24.83 (8.09)	25.59 (9.50)	25.07 (10.16)
Negative*	**Pre-CPT**	12.46 (3.23)	12.62 (2.75)	15.21 (4.06)	14.76 (3.42)
	Post-CPT	10.62 (2.96)	11.79 (2.60)	13.03 (3.99)	12.28 (2.90)
n		24	24	29	29

* = p<.05, ** = p<.01

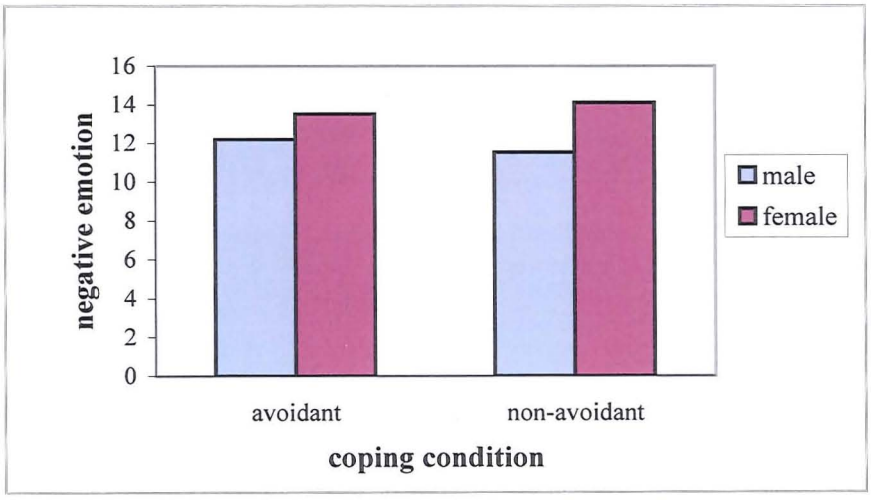
5.3.6.1 Positive emotion

Repeated measures analysis of variance was performed to examine the effects of gender and coping condition on positive emotion. The between-groups factor was gender (male vs. female), whereas the within-groups factors were coping condition (avoidant vs. non-avoidant) and time of testing (pre-CPT vs. post-CPT). The dependent variable was positive emotional state. No significant main or interaction effects were found.

5.3.6.2 Negative emotion

Similar analysis was conducted on negative emotion. A main effect of gender was found for negative emotion (F (1,51) = 7.26, p<.05), with women reporting greater negative emotion (mean = 13.82, SD = 2.98) than men (mean = 11.87, SD = 2.08). There was also a significant main effect of time (F (1,51) = 36.64, p<.001) with negative emotion scores significantly lower after cold pressor exposure (mean = 12.00, SD = 2.86) than before it (mean = 13.88, SD = 3.10). There was also a two-way interaction effect between gender and coping which approached significance (F (1,51) = 3.77, p<.059; see Figure 5.4).

Figure 5.4: Mean negative emotion scores measured by gender and coping condition, Experiment 2.



5.3.7 Perceived coping effectiveness

Mixed-groups ANOVA of subjective effectiveness ratings for the two types of coping instructions were conducted with coping strategy (avoidant vs non-avoidant) as the within-groups factor and gender (male vs female) as the between-groups factor. No main or interaction effects were found, which indicates no perceived benefit of one type

of coping over the other, either for the sample as a whole or for men and women individually.

5.3.8 Correlational analysis

Pearson's correlations were conducted to examine the relationships between all questionnaire measures and pain indices in both coping conditions. Since both gender effects and coping effects were found for some pain indices, correlations were conducted for all participants in both coping conditions, and separately for males and females in both coping conditions (See Tables 5.5 – 5.10)

5.3.8.1 Correlations between Anticipatory Responses and pain indices

For all participants, a number of significant correlations were found between Anticipatory Responses and pain indices. Both baseline and pre-CPT Tolerance Estimate were positively associated with pain threshold and pain tolerance in both coping conditions. Positive associations were also found between Fear of CPT and self-report pain indices in both coping conditions. In the avoidant coping condition only, positive correlations were also found between baseline anticipated Sensation Control and pain threshold and between pre-CPT Sensation Control and pain tolerance.

However, correlations run separately for males and females revealed quite different patterns of association. For males, pre-CPT Tolerance Estimate was positively associated with pain tolerance in the non-avoidant coping condition but not in the avoidant condition. No associations were found between Fear of CPT and pain indices in either coping condition for males. Surprisingly, baseline Sensation Control was positively associated with sensory pain, but only in the non-avoidant coping condition.

For females, both baseline and pre-CPT Tolerance Estimate were positively associated with pain threshold in both coping conditions. Pre-CPT Tolerance Estimate was positively associated with pain tolerance in both coping conditions. As can be seen from Tables 5.9 and 5.10, a number of significant associations between Fear of CPT and pain indices were found for females in both coping conditions. Baseline Sensation Control was positively associated with pain threshold in both coping conditions for females.

Table 5.5: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for all participants (n = 53), Experiment 2.

	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
baseline	**.300***	**.374****	-.035	-.080	-.173	-.146
pre-CPT	**.336***	**.528****	.195	-.255	-.215	**-.329***
Fear of CPT						
baseline	**-.293***	-.256	-.044	.167	.089	**.274***
pre-CPT	-.162	-.257	-.099	**.382****	.252	**.372****
Sensation Control						
baseline	.239	.192	-.198	-.020	-.234	-.120
pre-CPT	.143	.180	-.038	-.133	-.191	-.210
PANAS POSI						
pre-CPT	.150	**.272***	**.341***	.113	.109	.004
post -CPT	.173	.269	**.275***	-.023	.045	-.173
PANAS NEGA						
pre-CPT	-.054	-.036	-.036	**.444****	.000	.171
post-CPT	-.061	**-.276***	.026	.136	.168	.107
DASS						
Stress	-.023	**-.443****	**-.322***	.083	-.101	.175
Anxiety	-.060	-.236	-.081	.160	.037	.090
Depression	-.182	-.246	-.199	.038	-.142	.065
PCS						
Total	-.237	-.165	-.032	**.439***	**.384****	**.321***
Rumination	-.257	-.142	.066	.424	**.438****	.216
Magnification	-.216	-.108	.012	.188	.175	.191
Helplessness	-.149	-.154	-.120	**.429****	**.319***	**.359****
MCSDS	.215	.086	-.124	.145	.085	.083
STAI-T	-.229	-.176	-.137	-.106	-.149	.024
Age	-.019	.206	.035	**-.362****	-.244	-.209

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** = p<.01

Table 5.6: Correlations between questionnaire scores, age and pain indices with avoidant coping for all participants (n = 53), Experiment 2.

	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	**.276***	**359****	.178	-.044	-.261	-.110
Pre-CPT	**.287***	**.509****	.256	-.108	-.166	-.211
Fear of CPT						
Baseline	**-.395***	-.131	-.059	.207	.206	**.284***
Pre-CPT	**-.282***	-.258	-.066	**.302***	**.284***	**.392****
Sensation Control						
Baseline	**.278***	.263	.087	.083	-.236	-.164
Pre-CPT	.209	**.382****	.171	-.052	-.180	-.225
PANAS POSI						
Pre-CPT	.186	.202	.166	.148	.101	.077
Post -CPT	.198	**.300***	.156	.087	-.073	.024
PANAS NEGA						
Pre-CPT	-.133	-.019	-.042	.084	.254	.231
Post-CPT	-.189	-.188	-.003	.268	**.424***	.080
DASS						
Stress	-.111	**-.361****	-.176	.001	.084	.051
Anxiety	-.033	-.163	.008	.044	-.043	-.075
Depression	-.173	**-.317***	-.054	.005	.059	-.005
PCS						
Total	**-.352****	-.252	-.200	**.397****	**.354***	**.317***
Rumination	**-.325***	-.230	-.144	**.364***	**.344***	**.286***
Magnification	-.122	-.186	-.163	.184	.257	.096
Helplessness	**-.371****	-.216	-.193	**.397****	**.289***	**.348***
MCSDS	.152	.053	-.107	.139	-.085	.084
STAI-T	-.199	-.238	-.024	-.079	.012	-.018
Age	-.111	.258	.256	-.205	-.139	-.090

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** = p<.01

Table 5.7: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for males only (n = 24), Experiment 2.

MALES	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.050	.392	.032	.207	-.002	-.049
Pre-CPT	.213	**.578****	.327	.006	-.002	-.241
Fear of CPT						
Baseline	-.005	.204	.133	-.058	.036	.076
Pre-CPT	-.099	.139	.137	.114	.273	.224
Sensation Control						
Baseline	-.297	-.064	-.053	**.418***	.108	.324
Pre-CPT	-.068	.177	.153	-.008	.014	-.048
PANAS POSI						
Pre-CPT	.331	.033	**.479***	.056	.253	.015
Post -CPT	.391	-.006	.332	-.343	.104	-.248
PANAS NEGA						
Pre-CPT	-.300	.274	.153	.195	-.002	-.027
Post-CPT	-.146	-.332	.125	-.044	.046	-.068
DASS						
Stress	-.183	-.396	-.317	-.195	-.113	.100
Anxiety	-.045	-.323	-.153	.083	-.021	.124
Depression	-.164	.019	-.082	-.134	-.138	-.106
PCS						
Total	-.223	-.035	.052	.188	.240	.251
Rumination	-.213	.018	.083	.381	.385	.186
Magnification	.035	.087	.172	-.224	-.017	.051
Helplessness	-.297	-.121	-.039	.199	.200	.330
MCSDS	.182	-.267	-.023	.111	.158	.275
STAI-T	.029	.091	.055	-.102	-.206	-.191
Age	.057	.266	-.128	**-.453***	-.337	**-.532****

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** = p<.01

Table 5.8 : Correlations between questionnaire scores, age and pain indices with avoidant coping for males only (n = 24), Experiment 2.

MALES	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.120	.344	.253	.038	-.210	-.058
Pre-CPT	.096	.403	.368	-.048	-.302	-.007
Fear of CPT						
Baseline	-.372	.155	.143	.219	.318	.175
Pre-CPT	-.376	.165	.140	.195	.391	.256
Sensation Control						
Baseline	.008	.149	.139	.269	-.080	-.049
Pre-CPT	.097	.367	.309	-.169	-.146	-.240
PANAS POSI						
Pre-CPT	.253	.101	.241	-.145	.133	.006
Post -CPT	.273	.163	.151	-.227	-.016	-.048
PANAS NEGA						
Pre-CPT	-.122	**.504***	.304	-.032	.226	-.005
Post-CPT	-.193	.099	.219	.128	.338	-.108
DASS						
Stress	-.165	-.124	-.095	-.292	-.055	-.160
Anxiety	-.141	-.133	.039	-.099	-.236	-.161
Depression	-.139	-.052	.134	.004	.154	-.084
PCS						
Total	-.362	-.236	-.131	.369	.335	.369
Rumination	-.279	-.249	-.174	**.478***	.360	.383
Magnification	-.011	-.023	.020	.002	.257	-.027
Helplessness	**.498***	-.270	-.140	.376	.274	**.456***
MCSDS	.035	-.172	-.076	.067	-.046	.182
STAI-T	-.081	-.010	.251	.064	.049	-.132
Age	.028	.125	-.073	-.293	-.047	-.215

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** = p<.01

Table 5.9: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for females only (n = 29), Experiment 2.

FEMALES	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
baseline	**.418***	.268	.013	-.189	-.206	-.165
pre-CPT	**.436***	**.426***	.048	**-.465***	**-.368***	**-.392***
Fear of CPT						
baseline	-.345	**-.381***	-.281	.127	-.033	.337
pre-CPT	-.117	**-.374***	**-.480****	**.424***	.076	**.435***
Sensation Control						
baseline	**.413***	.233	-.210	-.136	-.348	-.348
pre-CPT	.225	.067	-.129	-.097	-.276	-.308
PANAS POSI						
pre-CPT	.075	**.475****	.231	.156	-.017	-.014
post -CPT	.100	**.560****	.192	.187	-.065	-.153
PANAS NEGA						
pre-CPT	.120	-.083	-.306	**.521****	.147	.235
post-CPT	.036	-.147	.017	.095	.132	.147
DASS						
Stress	.075	**-.433***	**-.419***	.193	-.191	**.189**
Anxiety	-.071	-.185	-.022	.249	.089	.066
Depression	-.236	**-.561****	-.268	.344	-.069	.271
PCS						
Total	-.203	-.150	-.199	**.551****	**.413***	.329
Rumination	.227	-.104	-.069	.335	**.388****	.169
Magnification	-.281	-.162	-.131	**.397***	.242	.249
Helplessness	-.055	-.115	-.251	**.568****	.355	.355
MCSDS	.214	.331	-.160	.356	.133	-.048
STAI-T	**-.504****	**-.594****	-.298	.022	.017	.344
Age	-.112	.078	.272	-.186	-.078	.131

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p<.05, ** = p<.01

Table 5.10 : Correlations between questionnaire measures, age and pain indices with avoidant coping, females only (n = 29), Experiment 2.

FEMALES	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	**.399***	.310	.085	.017	-.195	-.064
pre-CPT	**.450***	**.560****	.126	-.056	.043	-.348
Fear of CPT						
Baseline	**-.416***	-.178	-.186	.095	.036	.292
pre-CPT	-.184	**-.419***	-.234	.226	.050	**.443***
Sensation Control						
Baseline	**.451***	.258	.035	.128	-.206	-.160
pre-CPT	.243	.339	.095	.136	-.063	-.139
PANAS POSI						
pre-CPT	.112	.276	.080	**.407***	.105	.168
post –CPT	.153	**.398***	.170	.273	-.115	.076
PANAS NEGA						
pre-CPT	-.091	-.239	-.325	.031	.162	.335
post-CPT	-.173	-.365	-.212	.340	**.466***	.215
DASS						
Stress	-.040	**-.471***	-.249	.102	.089	.153
Anxiety	.062	-.191	-.023	.148	.085	-.001
Depression	-.243	**-.622****	-.297	.078	.074	.129
PCS						
Total	-.322	-.199	-.269	.347	.286	.219
Rumination	-.339	-.134	-.122	.208	.230	.141
Magnification	-.175	-.238	-.305	.234	.218	.138
Helplessness	-.259	-.140	-.244	**.370***	.246	.232
MCSDS	.245	.191	-.169	.316	-.024	.061
STAI-T	**-.417***	**-.581***	**-.441***	-.135	.088	.211
Age	-.303	.310	**.624***	-.065	-.119	.105

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait , * = p<.05, ** = p<.01

5.3.8.2 Correlations between emotion and pain indices

For all participants, more associations were found between emotion and pain indices in the non-avoidant than the avoidant coping condition. Correlations conducted separately by gender revealed more associations between emotion and pain indices for females than for males, in both coping conditions.

5.3.8.3 Correlations between negative affectivity and pain indices

For all participants, stress was negatively correlated with pain tolerance and pain recovery in the non-avoidant coping condition, while stress and depression were both negatively correlated with pain tolerance in the avoidant condition.

For males, no significant associations were found between negative affectivity and pain indices in either coping condition. However, as can be seen from Tables 5.9 and 5.10, significant relationships between negative affectivity and pain indices were found in both coping conditions for females.

5.3.8.4 Correlations between catastrophizing and pain indices

For the full sample, numerous significant associations were found between pain catastrophizing scales and pain indices in both coping conditions. For males, no such relationships were found in the non-avoidant coping condition but some associations were apparent in the avoidant condition. For females, although positive associations between catastrophizing scales and self-report pain indices were found in both coping conditions, these were far more numerous in the non-avoidant coping condition.

Overall, there were more relationships between cognition, emotion and pain responses for female participants than for their male counterparts.

5.4 Discussion

The main aims of Experiment 2 were to establish the replicability of gender differences in cold pressor pain, to test the relative effects of avoidant versus non-avoidant coping on cold pressor pain responses and emotional states, and to examine the potential interaction of gender and coping on such responses. A secondary aim was to assess potential differences in male and female pain-related cognition and emotion.

Gender differences in the predicted direction were evident for all pain indices, i.e., females showed lower mean threshold and tolerance to cold pressor pain, and higher self-report pain responses than males overall. However, the gender differences in behavioural pain responses (pain threshold, pain tolerance, pain recovery) did not reach significance. As expected, females reported significantly greater sensory pain and affective pain than males, but self-reported pain intensity did not differ significantly between genders.

No significant differences in cold pressor pain threshold have been found between males and females in Experiment 1 or 2. This is consistent with some previous research, and partially replicates the recent findings of Keogh et al. (2000), who found gender differences in pain tolerance and in sensory pain but not in pain threshold. However, the absence of a significant gender difference in pain tolerance in Experiment 2 was unexpected because such differences tend to be more replicable and have a larger effect size than gender differences in pain threshold (Riley et al., 1998).

There was some evidence for differential effects of avoidant and non-avoidant coping on pain responses in Experiment 2, but such effects were limited to pain recovery and affective pain. Greater affective pain was reported with non-avoidant than avoidant coping. As predicted, coping condition selectively affected pain recovery rate; this being faster with non-avoidant coping than with avoidant coping, although this effect was limited to males. This lends some support to the hypothesis that cognitive avoidance of pain while experiencing it can have undesirable after-effects (Cioffi & Holloway, 1993). A likely explanation for the slower pain recovery found with avoidant coping for males is that if attention is successfully diverted away from pain using distraction, monitoring of changes in pain sensation (including awareness of the dissipation of pain) would decrease. A gender-specific benefit of focusing attention on pain was found by Keogh et al. (2000) for sensory pain, with males reporting lower sensory pain when they focused attention upon cold pressor pain (non-avoidant coping). That the present experiment found gender-differentiated effects of coping on a different pain index from Keogh et al. (2000) is surprising since there were strong environmental and methodological similarities between the two experiments. Samples were drawn from the same university population and both experiments were conducted under similar laboratory conditions, using the same pain induction procedure. In both experiments all testing was carried out by one female researcher. One possible reason

for the disparity in findings may be related to the type of avoidance strategy employed. In Experiment 2, instructions were given to use a specified avoidant strategy (imagery of a familiar room) whereas Keogh et al. (2000) gave open-ended instructions for avoidance; participants were instructed to distract themselves from the pain in any way they chose. The Keogh et al. (2000) study does not facilitate conclusions about the usefulness of any one kind of distraction technique relative to another. However, allowing free choice of strategy may have had several effects, such as sanctioning the use of previously-used or favourite methods of distraction, and also enhancing participants' feelings of control. Previous research has shown increased tolerance for cold pressor pain in participants given a choice of coping strategies compared to those given no choice (Rokke & Lall, 1992). Differences in experimental design are also a potential reason for inconsistency between the experiments. In theory, the within-groups design used in Experiment 2 should have enhanced testing power, yet the only interaction of gender and coping found here was on pain recovery. It is possible that the greater impact of individual differences in the between-groups design used by Keogh and colleagues may have contributed to the differences they found. However, a subsequent study with a mixed design (Keogh & Herdenfeldt, 2002) also found that sensory focusing (non-avoidant coping) was selectively beneficial for males compared to females, although this has limited relevance as the comparison made was with another type of non-avoidant coping strategy (emotional focusing).

Over 60% of all participants in Experiment 2 reported that they had strictly adhered to the coping instructions in both the avoidant and the non-avoidant coping conditions, but neither strategy was perceived as more or less effective than the other. The fact that more than 30% of participants reported the use of additional coping strategies in both coping conditions shows that it is difficult to achieve a 'pure' experimental manipulation of coping. However, all participants who reported they had not adhered to coping instruction at all were dropped from the analysis, and the proportion of the sample who reported using additional strategies was extremely similar in both coping conditions. If non-compliant participants had been included, a true comparison of the two types of coping instruction would have been compromised. It is worth noting that if the level of non-adherence to instructions found here is typical, this potentially weakens any tests of differences between coping strategies in similar experiments lacking a coping manipulation check. As there is no obvious way in which low compliance to

coping instructions could be improved, removal of non-compliant participant data via post-experimental manipulation checks is important.

Regarding the secondary research aim of Experiment 2, some systematic differences in cognition about pain were apparent between men and women. Women reported more overall catastrophizing about pain than men, and specifically a greater tendency to increase attentional focus on pain-related thoughts (rumination). However, women showed no greater tendency than men to exaggerate the threat value of pain stimuli (magnification) or to adopt a helpless orientation to coping with pain (helplessness). Taken together, the findings of Experiments 1 and 2 add to previous research which suggests differences in male and female cognition about pain (Sullivan et al., 1995) and that such differences may be an influential factor in their pain perception (Sullivan & Neish, 1998).

As expected, women in Experiment 2 reported more negative emotion (as well as higher affective pain) than men, which indicates that cold pressor pain may evoke more negative emotions in females than males. There was also some indication that coping condition affected negative emotion in a gender-specific manner. Negative emotion was lower for men than for women when non-avoidant coping was used, but no such difference was evident with avoidant coping. This suggests that for men, focusing on pain benefitted their emotional state more than distracting attention away from it, while women did not perceive the same benefit.

Women reported more negative emotion than men overall in the context of this experiment. However, it is possible that this reflects gender differences in the tendency to express negative emotion, rather than - or perhaps in addition to - differences in the occurrence of such feelings. Gender-role normative influence may have meant that, even if men and women experienced similarly negative emotions due to pain, women more readily communicated this to others (Bem, 1981). The absence of gender differences in positive emotion is not necessarily inconsistent with such gender-role mechanisms, as men do seem more likely to underplay negative emotion than women (Robinson et al., 2000) but the perceived acceptability of expressing positive emotion might be less likely to be gender-differentiated.

There were also some notable differences between males and females in their self-expectations regarding imminent pain (Anticipatory Responses). Men expected greater tolerance and greater ability to control sensations during the cold pressor task than women. Women reported greater anticipatory fear of cold pressor pain than their male counterparts at every point of measurement. These differences may also be consistent with gender-role expectations; perhaps reflecting a greater perceived acceptability for women to express fear of forthcoming pain, and a greater perceived obligation for men to express stoicism or bravery.

5.5 Summary

Experiment 2 confirmed that there are some differences between male and female responses to experimental pain, but suggests that the exact nature of these differences may not be consistent between studies, even when the pain induction methodology and laboratory environment remain the same. In contrast to previous research, limited empirical support for the differential effects of avoidant and non-avoidant coping on pain responses overall was found here, although some evidence was found for the particular utility of non-avoidant coping for men. Nevertheless, subjective ratings of strategy effectiveness indicated that neither men nor women in this experiment perceived a benefit of one coping strategy over the other. However, the disparity between these results and those of similar experiments might be partly attributable to characteristics of the specific coping strategies tested here, in particular, the fully specified avoidant strategy (imaginal distraction by visualisation of a familiar room). Consequently, a replication of the present experiment was planned substituting open-ended avoidant coping (as used by Keogh et al., 2000) for the fixed avoidant coping strategy used here (see Chapter 7). However, for several reasons which are outlined below, a new questionnaire to measure cognitive and emotional focus on pain was devised prior to conducting this replication.

Gender differences in pain-related cognition and emotion were evident in Experiment 2. In particular, men and women differed considerably in the ways they appraised the *prospect* of a forthcoming pain induction and in their expectations of how they would respond to pain. These gender differences in cognition about pain included fearful apprehension, anticipated control, and the tendency for pain to dominate thoughts. These findings emerged through the use of several different questionnaires including the

Coping Strategies Questionnaire (CSQ,: Rosenstiel & Keefe, 1983), the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995), the Positive and Negative Affect Schedule (PANAS; Lovibond & Lovibond, 1995) and the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983). However, an alternative method of assessing cognition and emotion in the context of experimental pain seems to be needed. On a practical level, in repeated measures experiments such as the present one, the demands on participants are considerable and it would be preferable to minimise the number of additional questionnaires they are required to complete. In addition, some of the established measures administered in Experiment 2 are often utilised in pain research but are not specific to pain (e.g., the State-Trait Anxiety Inventory) while others are pain-referent (e.g., the Pain Catastrophizing Scale) but are not specific to experimental pain. In light of these issues the next step taken was to develop a questionnaire which would provide a unitary measure of pain-related cognition and emotion in experimental groups. The new measure was intended for use in the planned replication of Experiment 2 and in all subsequent experiments in this series.

Chapter 6

Development of new scale to measure cognitive and emotional focus on pain

6.1 Introduction

This chapter describes the development of a new questionnaire to assess pain-related cognition and emotion in the context of experimental pain induction. Development of this measure was undertaken following Experiment 2 and prior to the planned replication of that experiment.

Since pain is modulated by the interaction of physiological and psychological mechanisms, differences in the ways that men and women think and feel about pain may directly contribute to gender-differentiated pain responses. In Experiment 2, various aspects of cognition and emotion in the context of cold pressor pain were assessed using a battery of questionnaires, in the absence of an instrument which could integrate their measurement. While psychometric instruments to assess pain-related cognition are available (e.g., the Pain Beliefs and Perceptions Inventory (PBAPI); Williams & Thorn, 1989) they have been developed with chronic pain patients (see DeGood & Shutty, 1992) and are not necessarily ideal for non-clinical or pain-free populations (Skevington, 1995). It therefore seemed that a new scale to measure pain-related cognition and emotion in non-clinical groups, including those aspects on which males and females may differ, was required.

The design of the new questionnaire in this chapter was informed by the findings of the first two experiments. Experiments 1 and 2 indicated that there may be systematic differences in the ways in which men and women think about pain, and in their emotional responses to painful experiences. The gender differences in pain-related cognition found in these two experiments have involved both attention and emotion. Firstly, women reported a greater general tendency for pain to dominate their thoughts whenever they experienced it than men did. Before experimental pain induction women reported more fear of the painful stimulation, and expected to be less able to tolerate and control pain than their male counterparts. Women also reported much higher levels of negative affectivity than men in the context of experimental pain. It seems that pain may be a more daunting prospect for women than for men, perhaps partly due to their greater propensity for cognitive focus on pain, their lower self-expectancy of resilience to pain and their higher levels of negative emotion in response to it. Pain perception can be powerfully influenced by cognitive processes and emotional states (Melzack & Casey, 1968), and while paying close attention to pain can be a useful coping strategy in some

circumstances (Suls & Fletcher, 1985) a high level of cognitive focus on pain combined with negative emotion and poor self-expectations is likely to be disadvantageous.

Relevant items were adapted from existent measures of pain-related cognition and emotion, and included the tendency for pain to dominate thinking, fear or worry about pain, and negative anticipatory thoughts about pain.

6.2 Part A: Questionnaire development

6.2.1 Method

6.2.1.1 Questionnaire design

A 30-item self-report questionnaire was generated to assess fear and worry about pain, the tendency for pain to grab attention and dominate thoughts, and perceived ability to control and/or cope with pain. The format of the questionnaire is a series of item-statements reflecting various cognitions about pain, which are self-rated for applicability on a 5-point Likert-type rating scale where 0 = 'not at all applicable', 2 = 'moderately applicable' and 4 = 'extremely applicable'. 1 and 3 are also marked on the scale, located equidistantly between 0 and 2 and between 2 and 4 respectively, but are not assigned value descriptor labels. The new scale incorporated items adapted from existing measures of pain cognition including the Pain Catastrophizing Scale (PCS; Sullivan, Bishop & Pivik, 1995), the Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983), the Survey of Pain Attitudes (SOPA; Jensen, Turner, Romano & Lawler, 1994), the Pain Anxiety Symptoms Scale (PASS; McCracken, Zayfert & Gross, 1992) and the Pain Vigilance and Awareness Questionnaire (PVAQ; McCracken, 1997). However, the majority of items were derived from participant comments noted during Experiments 1 and 2. Piloting was also used to inform the construction of new scale items. Participant comments featured several recurrent themes, such as low expectation of tolerance, difficulty in shifting attention away from pain, concern that ill-health is the cause of pain and fearfulness/worry about pain. It was reasoned that items derived from spontaneous reactions to pain should be salient to participants. The 30 items in the Focus on Pain Scale (FOPS) are presented in Table 6.1.

Table 6.1 Items in the Focus on Pain Scale (FOPS) and their sources.

FOPS Item no.	Item	Source
1	I have a low threshold for pain	PC
2	I am very sensitive to pain	PVAQ
3	When I feel pain I become afraid that it will get worse	PCS
4	I do not have a high tolerance for pain	PC
5	When I am in pain I keep thinking about how much it hurts	PCS
6	When I feel pain I cannot seem to keep my mind off it	PC
7	When I am in pain I can't concentrate on anything else	PASS
8	When I feel pain it distracts me so much that I can't really do anything else	PC
9	When I am in pain it seems to dominate my thoughts	PASS
10	I dread being in pain	PASS
11	There is a strong connection between my emotions and my pain level	SOPA
12	I believe that I can control how much pain I feel by changing my thoughts	SOPA
13	Just by concentrating I can 'take the edge off' my pain	SOPA
14	I can influence the amount of pain I feel	SOPA
15	I feel frightened when I am in pain	PC
16	It scares me when I feel pain	PC
17	I worry that pain may be a sign of serious illness	PC
18	When I feel pain I worry about what the cause of it might be	PC
19	I fear pain	PC
20	I am very aware of my bodily sensations	PC
21	When I feel pain, I can't stop thinking about it even if I try	PASS
22	I am very aware of my thoughts about pain	PC
23	The way I think about pain does not help me cope with it	PC
24	I cannot control my thoughts about pain	PC
25	I think that pain is essentially uncontrollable	PC
26	When I am in pain I worry about my health more than usual	PC
27	I feel helpless when I am in pain	PC
28	When I am in pain I don't know what to do with myself	PC
29	I do not cope well with pain	PC
30	Just by relaxing I can 'take the edge off' my pain	SOPA

Key: PVAQ = Pain Vigilance and Awareness Questionnaire (McCracken, 1997); PCS = Pain Catastrophizing Scale (Sullivan , Bishop & Pivik, 1995); SOPA = Survey of Pain Attitudes (Jensen, Turner, Romano & Lawler, 1994), PASS = Pain Anxiety Symptoms Scale (McCracken, Zayfert & Gross, 1992), PC = participant comments.

6.2.1.2 Participants

Participants were 646 individuals recruited from among staff, students, prospective students and parents of prospective students at a London university. Missing data and illegible item responses reduced the total sample size to 602 participants. The demographic composition of the whole sample was 446 females (74.1 %) and 156 males (25.9 %) between 16 and 69 years of age (mean = 25.65 years, SD = 8.87). Age

within the two gender groups was very similar: males; mean = 26.37 years, SD = 9.27, range 17-68 years; females; mean = 25.40 years, SD = 8.74, range 16–69 years.

Adequacy of sample size is an important determinant of the reliability of correlation coefficients in factor analysis: Tabachnick and Fidell (1996) have stated that, as a general rule of thumb, at least 300 cases are needed. Similarly, Comrey and Lee (1992) have concluded that 500 cases is a very good sample size for factor analysis and 1000 is excellent. On this basis, 602 cases was considered a suitable sample size for the purposes of the present study.

6.2.1.3 Procedure

All participants completed the Focus on Pain Scale as part of a questionnaire battery during introductory sessions on the undergraduate psychology programme, as part of laboratory classes illustrating the use of questionnaires, as part of Open Day at Goldsmiths College or were approached on campus.

6.2.1.4 Statistical analysis

Exploratory factor analysis was used to identify the factor structure of the FOPS, as the scale is in the initial stages of development (Byrne, 1998). Independent-samples t-tests were applied to male and female scores on the factor-analytically derived subscales of the scale, and on total scale scores, to ascertain whether gender differences in focus on pain were apparent in this sample.

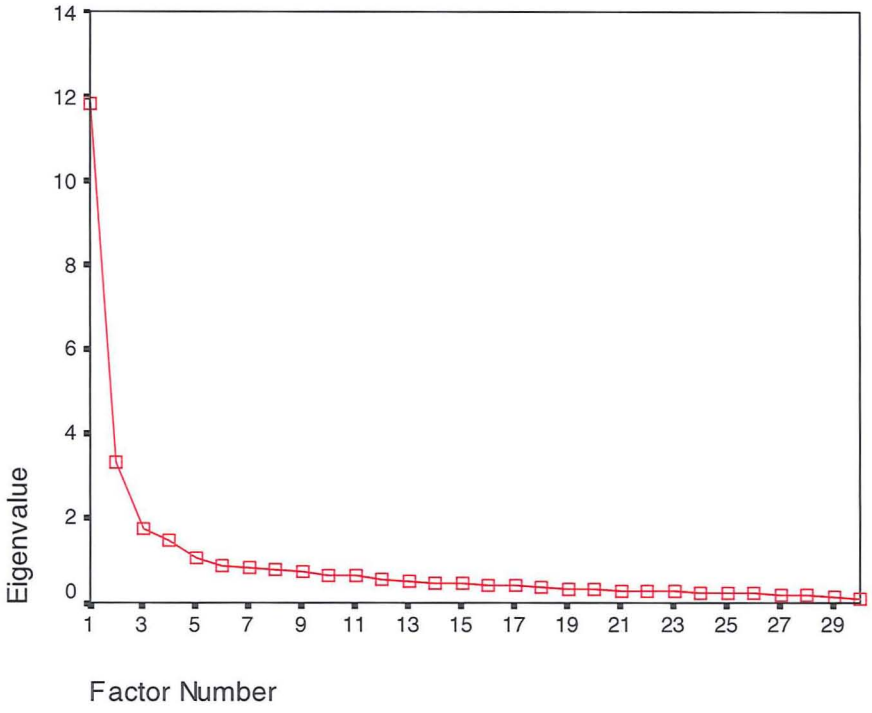
6.2.2 Results

A preliminary Principal Components Analysis (PCA) with Varimax rotation (as recommended by Tabachnick and Fidell, 1996) indicated that the correlation matrix was factorable (numerous correlations above .3 were observed), and yielded no evidence of multicollinearity or singularity as neither the determinant of R nor any of the eigenvalues approached zero. Inspection of eigenvalues and the scree plot from PCA suggested there were three main components within the data.

Principal factor analysis was then performed, and a decision was made regarding the number of factors to extract based on several pre-established criteria applied to the observed data (Gorsuch, 1983). There were five eigenvalues greater than 1.0 but the

first three factors alone accounted for 56.34% of the total variance. All factors after the third each accounted for less than 5% of the variance and so added little to the solution. A factor scree plot of eigenvalues against factors (after Cattell, 1966) indicated that the scree occurred after the third or fourth factor (see Figure 6.1) which also suggested the presence of three or four distinct factors within the scale.

Figure 6.1: Factor scree plot of eigenvalues against factors in Focus on Pain Scale



In order to determine whether a three-factor or four-factor solution best represented the data, inspection of the number of item markers for each factor was conducted. According to Watson, Clark, Weber, Assenheimer, Strauss and McCormick (1995) markers are items that load equal to or in excess of .3 on a factor and load highest on that factor. An additional criterion was also used, that the main loading of a marker should be more than .2 higher than any crossloading (Bedford, 1997). Table 6.2 displays the number of markers per factor for one, two, three, four, five and six-factor solutions. Inspection of the key markers indicated that either a three or four-factor solution would be appropriate according to these two criteria. The three-factor solution showed a lower number of key markers which met Bedford's criterion (19) than the four-factor solution (21). However, the three-factor solution produces a subscale structure within the questionnaire in which all subscales seem meaningful and contain at least four items.

On balance, the three-factor solution was considered to provide the most parsimonious interpretation of the data.

Table 6.2: Number of markers per factor in the Focus on Pain Scale

	Number of Markers for Factor No.				
No. of Factors in Solution	*1*	*2*	*3*	*4*	*5*
1	30(N/A)				
2	26(25)	4(4)			
3	13(7)	13(8)	4(4)		
4	13(8)	9(6)	4(4)	3(3)	
5	12(8)	8(5)	4(4)	3(3)	3(2)
6	12(8)	5(3)	4(4)	3(3)	2(2)

Note. The numerical values without parentheses are the number of markers with factor loadings exceeding .3 (after Watson, Clark et al., 1995). Numerical values within parentheses are the number of markers with factor loadings exceeding .3 and with major loadings at least .2 greater than any cross-loading (after Bedford, 1997).

As recommended by Tabachnick and Fidell (1996), Varimax rotation was used throughout this study to aid interpretation of the factor analysis, and because the scale was intended for use as an IV or DV (measured variable) in subsequent studies. In order to ensure that this rationale had not obscured a preferable solution obtainable with oblique rotation, factor analysis was repeated using Direct Oblimin rotation. There were minimal differences in the pattern of loadings obtained with oblique rotation, indicating a similar factor structure regardless of rotation technique and confirming that the Varimax rotated solution retained and reported here (see Table 6.3) was meaningful.

The items with their highest loading on Factor 1 reflected the interference of pain with usual cognitive function and perceived inability to prevent this, i.e. the attention-grabbing effects of pain. Accordingly, this factor was named 'Pain Dominance'. As the items with their major loading on Factor 2 concerned worry and fear about pain, this factor was named 'Pain Fear & Worry'. Factor 3 contained positively-valenced items reflecting beliefs in own ability to control pain and was consequently labelled 'Pain Control'.

Table 6.3: Varimax-rotated factor loadings of Focus on Pain Scale items

		Factor		
Item No.	Item description	F1	F2	F3
7	Can't concentrate on anything else…	**.831***	.199	-8.165
9	Pain dominates thoughts…	**.825***	.232	-6.212
8	Pain distracts so much…	**.821***	.193	-8.517
21	Can't stop thinking about pain…	**.748***	.395	-.123
6	Cannot keep mind off pain…	**.747***	.341	-.109
28	Don't know what to do with myself…	**.668***	.367	-.192
5	Keep thinking how much it hurts…	**.634***	.430	-.146
27	Feel helpless when in pain…	.572*	.456	-.170
29	Do not cope well with pain…	.556*	.484	-.334
24	Cannot control thought about pain…	.532*	.380	-.235
23	Way I think about pain doesn't help…	.503*	.404	-.299
22	Very aware of my thoughts about pain…	.417*	.353	.163
25	Pain is essentially uncontrollable…	.404*	.284	-.324
15	Feel frightened when in pain…	.237	**.788***	4.880
16	Scares me when I feel pain…	.285	**.784***	2.443
17	Worry pain may be sign of illness…	.139	**.716***	2.230
18	Worry about cause of pain…	.144	**.674***	.122
19	Fear pain…	.354	**.653***	-6.266
26	Worry about health more than usual…	.252	**.631***	3.466
3	Become afraid pain will get worse…	.347	**.572***	-2.991
10	Dread being in pain…	.404	.513*	-2.926
2	Very sensitive to pain…	.316	.495*	-.191
4	Do not have high tolerance…	.270	.438*	-.204
1	Have low threshold for pain…	.202	**.424***	-.201
11	Strong connection between emotions…	.262	.415*	.197
20	Very aware of bodily sensations…	.217	.345*	.186
12	Can control pain by changing thoughts…	-8.556	2.432	**.812***
13	By concentrating, can 'take edge off'…	-.122	6.308	**.809***
14	Can influence amount of pain…	-9.110	1.659	**.774***
30	By relaxing, can 'take edge off'…	-.102	2.687	**.489***

Key: Emboldened values are markers with factor loadings in excess of .3 and with greater than .2 cross-loading. The highest loading for an item (above .3) is indicated by an asterisk.

Some level of crossloading between factors in the scale was expected as subcomponents of this measure of cognitive focus on pain may be related. Eleven items (2, 4, 10, 11, 20, 22, 23, 24, 25, 27 and 29) had less than .2 difference between their major factor loading and crossloading on at least one other factor. Six of these items were within Pain Dominance (22, 23, 24, 25, 27 and 29), and the remaining five were in Pain Fear & Worry (2, 4, 10, 11 and 20). Among these, items 10, 23, 24, 27 and 29 all had fairly high major loadings on one of the factors (above .5) and were conceptually related to

the overall intended purpose of the scale, thus they were retained. Items 2, 4, 11, 20, 22 and 25 which had weaker loadings onto any factor were considered to have provided insufficiently distinct measurement of any unitary construct within the scale and were consequently dropped from the questionnaire. The 24 items retained in the Focus on Pain Scale (FOPS 24) are shown in Table 6.4.

6.2.2.1 FOPS 24 subscale composition and scoring range

The FOPS 24 contains 3 factor-analytically derived subscales. The first of these, Pain Dominance, comprises items 3, 4, 5, 6, 7, 17, 18, 19, 21, 22, and 23, and has a range of possible scores from 0-44. Pain Fear & Worry comprises items 1, 2, 8, 12, 13, 14, 15, 16 and 20 and has a range of scores from 0-36. The final subscale, Pain Control, is composed of items 9, 10, 11 and 24 and has a range of scores from 0-16. The range of score for the total FOPS 24 is 0-96.

6.2.2.2 Gender differences in FOPS 24 scores

Independent-samples t-tests revealed a gender difference ($t(598) = -.2.91$, $p<.01$) in FOPS 24 total scores, with females reporting higher general focus on pain (mean = 40.69, SD = 15.94) than males (mean = 36.47, SD = 14.41). A gender difference was also found for the FOPS 24 Pain Dominance subscale ($t(598) = -2.83$, $p<.01$) with females reporting higher tendency for pain to dominate their thoughts (mean = 17.90, SD = 9.97) than males (mean = 15.36, SD = 9.20) and for the FOPS 24 Fear & Worry subscale ($t(598) = -3.37$, $p<.01$) with females scoring higher (mean = 14.90, SD = 7.58) than males (mean = 12.59, SD = 6.68). No gender difference was found for the FOPS 24 Pain Control subscale. These results suggest that the new questionnaire does measure aspects of pain-related cognition and emotion in which gender differences occur.

Table 6.4: Items retained in the Focus on Pain Scale (FOPS 24)

Original FOPS Item no.	New FOPS Item no.	Item
1	1	I have a low threshold for pain
3	2	When I feel pain I become afraid it will become worse
5	3	When I am in pain I keep thinking about how much it hurts
6	4	When I feel pain I cannot seem to keep my mind off it
7	5	When I am in pain I can't concentrate on anything else
8	6	When I feel pain it distracts me so much that I can't really do anything else
9	7	When I am in pain it seems to dominate my thoughts
10	8	I dread being in pain
12	9	I believe that I can control how much pain I feel by changing my thoughts
13	10	Just by concentrating I can 'take the edge off' my pain
14	11	I can influence the amount of pain I feel
15	12	I feel frightened when I am in pain
16	13	It scares me when I feel pain
17	14	I worry that pain may be a sign of serious illness
18	15	When I feel pain I worry about what the cause of it might be
19	16	I fear pain
21	17	When I feel pain, I can't stop thinking about it even if I try
23	18	The way I think about pain does not help me cope with it
24	19	I cannot control my thoughts about pain
26	20	When I am in pain I worry about my health more than usual
27	21	I feel helpless when I am in pain
28	22	When I am in pain I don't know what to do with myself
29	23	I do not cope well with pain
30	24	Just by relaxing I can 'take the edge off' my pain

6.3 Part B: Reliability and validity of the 24-item Focus on Pain Scale (FOPS 24)

6.3.1 Method

Preliminary psychometric assessments of the reliability and validity of the FOPS 24 were conducted prior to implementing the new scale in the next experiment in this series.

6.3.1.1 Validity of FOPS 24

To evaluate the concurrent validity of the new scale, correlational analysis was conducted on FOPS 24 scores with scores on an established measure of pain cognition obtained from the same individuals. The pain cognition instrument used for this purpose here was The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995). The PCS is a 13-item self-report measure designed to provide an index of catastrophizing about pain in both non-clinical and clinical populations. With this aim, a subgroup of the original sample in this study (n = 511) also completed the PCS. The demographic characteristics

of the subgroup sample were very close to those of the total sample: 382 women (74.8 %) and 129 men (25.2 %) between 16 and 69 years of age (mean = 25.48 years, SD = 8.91). Since some items on the FOPS 24 were derived from the PCS, and the two scales both aim to measure types of maladaptive pain-related cognition, some degree of positive correlation between them was expected.

Table 6.5: Correlations between mean FOPS 24 scores and PCS scores of the subgroup sample (n=511).

	FOPS 24			
	Total	**Pain Dominance**	**Fear & Worry**	**Pain Control**
PCS				
Total	**.642****	**.635****	**.551****	-.059
Rumination	**.574****	**.600****	**.469****	-.096
Magnification	**.503****	**.420****	**.488****	.050
Helplessness	**.573****	**.581****	**.482****	-.064

* = p<.05, ** = p<.01

As can be seen from Table 6.5, the FOPS 24 total score was positively correlated with the PCS total score. Positive correlations were also found between the FOPS 24 Pain Dominance subscale and all PCS subscales, and between FOPS 24 Fear & Worry subscale and all PCS subscales. On the basis of this preliminary assessment the concurrent validity of these FOPS 24 scales appears to be satisfactory. Unsurprisingly, similar associations were not found between the FOPS 24 Pain Control subscale and PCS scores. Since the Pain Control subscale of the FOPS 24 comprises positively-valenced items which reflect confident cognition about pain, it should not be positively correlated with catastrophizing about pain.

Item analyses were conducted on all 24 items to assess the convergent and discriminant validity of the three constructs within the FOPS 24. Pearson's correlations were conducted on each item with its own scale (with the item removed) and with the other two subscales. Highly significant positive correlations ($p<.001$) were found between all items and their own scales, indicating convergent validity. In addition, all items were more highly correlated with their own scale than with other scales, indicating discriminant validity within the measure. Coefficient alphas were computed to obtain

internal consistency estimates of reliability for the three FOPS 24 subscales. The alphas for Pain Dominance, Pain Fear & Worry, and Pain Control were .94, .89 and .81 respectively.

Face validity of the FOPS 24 was expected to be good, since the items in the scale were derived from established measures of pain cognition and the comments of participants in previous cold pressor pain experiments. This was confirmed inasmuch as no participant expressed any difficulty in understanding or responding to the scale items.

6.3.1.2 Reliability of FOPS 24

Two internal consistency estimates of reliability were computed for the FOPS 24; coefficient alpha and a split-half coefficient expressed as a Spearman-Brown corrected correlation. To maximise the equivalence of the two halves of the scale, splitting the items was done taking account of the subconstructs of the scale that the item loadings had indicated, i.e., with equal numbers of items from each factor in each half. Consequently, the first half contained items 1,5, 7, 9, 10, 12, 14, 16, 18, 23, 27, 29 and the second half comprised items 3, 6, 8, 13, 15, 17, 19, 21, 24, 26, 28, 30. The value of coefficient alpha was for the whole measure was .92, and the split-half coefficient was .95, both indicating very good reliability. Inter-item reliability also appears to be good, with very little change in alpha caused by the removal of any item.

6.4 Summary

This study has produced a 24-item self-report scale for the assessment of the tendency to focus on pain, and provided some preliminary evidence that the scale is valid and reliable. Further validation will be necessary to confirm the psychometric properties of the FOPS 24, to ascertain whether the scale possesses temporal stability, and whether it is generalizable to other populations.

This study has indicated that the subscales of the FOPS 24 do provide a realistic measure of the subconstructs within the general tendency to focus on pain. Since there are unequal numbers of items in the factor-analytically derived subscales, further development of the questionnaire will be needed to create more equal subscale size and to ensure that all subconstructs are fully and equally assessed. It is intended that this scale refinement will be undertaken at a future date.

Evaluation of gender differences in the sample used for development of the FOPS 24 questionnaire indicated higher cognitive and emotional focus on pain in females than males. Both the FOPS 24 total scale and the subscales were used to assess cognitive and emotional focus on pain in all subsequent experiments in this thesis, including the planned replication of Experiment 2 which is reported in the following chapter.

Chapter 7

Experiment 3
Gender, Coping Strategies and Cold Pressor Pain II

7.1 Introduction

Experiment 2 prompted two distinct but connected subsequent stages in the overall research programme. The first of these was the development of a unitary measure of pain-related cognition specifically for use in this series of experiments (see Chapter 6). This chapter reports the second stage, an investigation of the effects of gender and coping on cold pressor pain responses using a different coping manipulation than that in Experiment 2.

In contrast to previous research, Experiment 2 provided minimal evidence that directing attention away from cold pressor pain and attending towards pain are differentially effective as coping strategies (cf. Suls & Fletcher, 1985). Nor were the same gender-related differences found in the relative usefulness of avoidant coping versus non-avoidant coping as those reported by Keogh et al. (2000). Despite the empirical basis for selecting imaginal distraction as a highly effective avoidant coping strategy, it is nevertheless possible that this technique was counter-intuitive for some participants. To investigate this possibility a semi-replication of Experiment 2 was conducted, replacing the mandatory imaginal distraction strategy with the open-ended avoidant coping instructions used in the Keogh study, i.e., with participants asked to distract themselves from pain in any way they chose.

The primary aim of Experiment 3 was to ascertain whether the inconsistency between the findings of Experiment 2 and those of Keogh et al. (2000) was related to differences in the specific coping strategies used rather than the direction of attention involved. To that end, a comparison of open-ended avoidant coping and non-avoidant coping for cold pressor pain was conducted within a replication of the experimental methodology of Experiment 2. The secondary research aim of Experiment 3 was to assess potential gender differences in pain-related cognition and emotion.

Gender differences in pain sensitivity and pain-related cognition consistent with those found in Experiment 2 were expected, specifically that females would show greater pain sensitivity and higher pain-related cognition. Gender differences in Anticipatory Responses were also expected to echo those found in Experiment 2, specifically that men would anticipate greater tolerance and sensation control than women, and that women would report more anticipatory fear of cold pressor exposure than men. In view

of the implementation of open-ended avoidant coping instructions, interaction effects between gender and coping on pain responses were anticipated, but no specific hypotheses were generated concerning either the direction of such effects or the pain indices involved. Coping condition was expected to affect pain responses, but no specific predictions were made regarding such effects.

7.2 Method

7.2.1 Design

A mixed design was used, with gender as the between-groups factor (male vs. female) and coping strategy as the within-groups factor (avoidant vs. non-avoidant). The dependent variables were pain threshold, pain tolerance, pain recovery, sensory pain, affective pain and pain intensity. As in previous experiments, measures of pain threshold, pain tolerance and pain recovery were obtained directly from the cold pressor, and measures of sensory pain, affective pain and pain intensity from the SF-MPQ.

Consistent with Experiment 2, emotional state, catastrophizing, negative affect, trait anxiety, social desirability, and expectations about cold pressor (Anticipatory Responses) were assessed. In Experiment 3, cognitive and emotional focus on pain was also measured using the new questionnaire (FOPS 24) developed in the previous chapter.

7.2.2 Participants

The same exclusion criteria were employed as in previous experiments. 42 participants (20 males, 22 females) were recruited from the staff and student population of Goldsmiths College. 4 participants (2 males, 2 females) were dropped from the analysis due to non-compliance with coping strategy instructions during one or both cold pressor trials. The resultant total number of participants was 38 (18 male, 20 female). Age range was 18-37 (mean = 23.17, SD = 6.00). All participants were in good general health, not in pain and had consumed no alcohol or analgesics on the day of testing. A nominal payment of £3.00 was made for participation.

7.2.3 Pain induction

7.2.3.1 Cold pressor apparatus and task

The cold pressor apparatus and task were the same as in Experiment 2.

7.2.3.2 Pain ratings

Measures of pain threshold, pain tolerance and pain recovery were obtained with the same pain rating system and instructions as in Experiment 2. Self-report pain was again assessed using the Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987).

7.2.4 Additional questionnaires

The same questionnaires were administered as in Experiment 2: the Pain Catastrophizing Scale (PCS; Sullivan et al., 1995), the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988), the short version of Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995), the State-Trait Anxiety Inventory, trait form (STAI-T; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983), the Marlowe-Crowne Social Desirability Scale (MCSDS; Crowne & Marlowe, 1960), and the Anticipatory Responses questionnaire. As in Experiment 2, a coping manipulation check questionnaire was administered after each cold pressor exposure.

An extra questionnaire was administered in Experiment 3. The Focus on Pain Scale (FOPS 24) is a measure of pain-related cognition and emotion in non-clinical populations, which was developed for use in this series of experiments. The FOPS 24 comprises 24 item-statements which describe cognitive focus on pain and are rated on a 5-point scale, ranging from 0 (*not at all applicable*) to 4 (*extremely applicable*). A total score (overall focus on pain) and three subscale scores (Pain Dominance, Fear & Worry and Pain Control) are obtained from the FOPS 24. Details of the development and preliminary psychometric properties of the FOPS 24 appear in Chapter 6. As reliability and validity of the scale are not established (although initial assessments are encouraging) use of the FOPS 24 in this experiment is supplementary to the measures of pain-related cognition used in Experiment 2.

7.2.5 Coping manipulation

As in Experiment 2, all participants underwent two cold pressor exposures, using avoidant coping in one and non-avoidant coping in the other. The non-avoidant coping instructions (sensory focusing on the cold pressor) were the same as in Experiment 2. The avoidant coping instructions were open-ended, allowing participants to choose their own method of diverting attention away from pain (after Keogh et al., 2000). The exact instructions given in the avoidant condition appear below. Both sets of instructions

contained five sentences and similar grammatical structure. As in Experiment 2, coping condition was counterbalanced (ABBA) and there was a 15 minute interval between cold pressor exposures.

Open-ended avoidant coping instructions: *' While your hand is in the cold water we would like you to try to avoid all the sensations that the cold water produces in that hand. Try to block all thoughts and feelings about these sensations from your mind. Distract yourself from the sensations produced by the cold water in whichever way seems best for you. For example, some people find that concentrating hard on something else during painful episodes is helpful. All the time your hand is in the cold water, keep distracting yourself and avoiding the sensations in any way you can.'*

7.2.6 Procedure

The experimental procedure was the same as that used in Experiment 2, except that no audiotapes of participants' verbalised thoughts prior to cold pressor exposure (Thought Records) were made in the present experiment.

7.2.7 Statistical analysis

Analysis of variance was used to examine the effects of gender and coping instructions on pain indices. As in Experiment 2, analysis of coping condition order was also incorporated into statistical tests on pain indices. Independent-samples t-tests were conducted to examine potential differences in negative affectivity, pain catastrophizing, focus on pain, trait anxiety, social desirability and age between males and females. Analysis of variance was used to examine the effects of gender and coping instructions on emotional states, and also to evaluate the effects of gender and time of measurement on Anticipatory Responses. Correlational analyses were applied to examine relationships between pain indices and all other variables.

7.3 Results

7.3.1 Data screening

As in previous experiments, transformations were applied to normalise skewed data distributions, as recommended by Tabachnick and Fidell (1996). The following variables were logarithmically transformed: pain threshold, pain tolerance, pain recovery, Tolerance Estimate and age of participants. Negative affectivity scores (stress,

anxiety, depression), Fear of CPT, and Sensation Control were square root transformed. As before, although statistical analysis was conducted on transformed data where applicable, all means and standard deviations presented are raw scores.

7.3.2 Coping adherence

As in Experiment 2, only data from participants who reported adherence to the coping instructions given was included in statistical analysis. In the non-avoidant coping condition, 84.2% of participants reported they had used only the strategy as instructed, and the remaining 15.8% followed the coping instructions but also used an additional (self-selected) strategy. In the avoidant coping condition 94.6% reported strict adherence to the coping instructions while 5.4% reported they had used distraction as instructed but not exclusively.

7.3.3 Negative affectivity, pain catastrophizing, trait anxiety, focus on pain, social desirability and age

Means and standard deviations of negative affectivity (depression, anxiety, stress), trait anxiety, focus on pain, social desirability scores and age of males and females are presented in Table 7.1.

Independent-samples t-tests showed that males and females did not differ significantly in negative affectivity, pain catastrophizing, trait anxiety, focus on pain or social desirability. The male and female groups were found to be significantly different in age (t (36) = 2.60, p<.05), probably due to an unintended sampling bias. Although there has been some evidence for age-related changes in pain tolerance, these are often associated with old age when chronic pain conditions become more prevalent (Skevington, 1995). Some contrasts in pain sensitivity have also been found between younger and older children (e.g., LeBaron, Zeltzer & Fanurik, 1989). As the mean ages of the (all-adult) gender groups in Experiment 4 differed by less than 5 years and their age ranges were fairly similar (males 18-37 years, females 18-33 years), this is unlikely to have had any impact on their respective pain sensitivity.

Table 7.1: Mean questionnaire scores and ages of males and females, Experiment 3 (standard deviations in parentheses).

	Males	Females
DASS		
Stress	14.39 (10.06)	12.35 (8.41)
Anxiety	6.83 (7.43)	5.50 (5.58)
Depression	10.67 (13.24)	5.75 (6.95)
PCS		
Total	17.50 (12.82)	17.65 (7.60)
Rumination	7.11 (5.19)	7.30 (3.36)
Magnification	3.39 (2.72)	3.40 (2.04)
Helplessness	7.05 (5.67)	6.95 (3.33)
FOPS 24		
Total	41.19 (18.45)	38.05 (12.75)
Pain Dominance	15.83 (10.62)	18.15 (8.92)
Fear & Worry	13.50 (7.98)	15.60 (8.06)
Pain Control	9.44 (3.26)	7.65 (3.25)
STAI-T	40.72 (10.65)	40.15 (9.79)
MCSDS	18.72 (3.59)	18.50 (3.14)
Age*	25.50 (6.86)	20.90 (4.18)
n	18	20

Key: DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, FOPS 24 = Focus on Pain Scale, STAI-T = State-Trait Anxiety Inventory-Trait, MCSDS = Marlowe-Crowne Social Desirability Scale, * = $p<.05$, ** = $p<.01$

7.3.4 Anticipatory Responses

Means and standard deviations of Anticipatory Responses scores (Tolerance Estimate, Fear of CPT, Sensation Control) of males and females at baseline, after avoidant coping instructions and after non-avoidant coping instructions are presented in Table 7.2.

Table 7.2: Mean Anticipatory Response scores of males and females, Experiment 3 (standard deviations in parentheses).

Anticipatory Response	Males	Females
Tolerance Estimate**		
Baseline	226.06 (188.28)	92.25 (64.25)
Non-avoidant	234.17 (277.166)	65.72 (53.97)
Avoidant	197.50 (173.46)	99.10 (74.23)
Fear of CPT*		
Baseline	11.78 (9.99)	25.55 (17.90)
Non-avoidant	22.17 (22.58)	23.37 (21.52)
Avoidant	18.00 (19.01)	36.37 (28.28)
Sensation Control		
Baseline	50.67 (29.35)	38.12 (20.22)
Non-avoidant	47.36 (31.34)	40.37 (22.05)
Avoidant	44.08 (29.20)	30.72 (21.89)
n	18	20

* = p<.05, ** = p<.01

Similar to Experiment 2, large standard deviations in the Anticipatory Responses data indicated high variability but transformations improved the normality of the distributions prior to statistical analysis. To compare the Anticipatory Responses of males and females, a series of repeated measures ANOVAs were conducted to evaluate the effects of gender (male vs. female) and time of testing (baseline vs. after avoidant coping instructions vs. after non-avoidant coping instructions) on Tolerance Estimate, Fear of CPT and Sensation Control respectively.

7.3.4.1 Tolerance Estimate

There was a significant main effect of gender (F (1,36) = 10.18, p<01), with males rating their anticipated tolerance of CPT higher (mean = 219.24, SD = 198.06) than females did (mean = 85.69, SD = 57.44). No other significant effects were found.

7.3.4.2 Fear of CPT

There was a significant main effect of gender on Fear of CPT (F (1,36) = 4.77, p<.05), with females reporting greater fearfulness of the cold pressor task (mean = 28.43, SD = 19.87) than males (mean = 17.31, SD = 11.94). No other significant effects were found.

7.3.4.3 Sensation Control

A near-significant main effect of coping condition was found for Sensation Control (F (2,36) = 3.11, p<.057). Post-hoc simple effects analyses revealed that expected Sensation Control was rated significantly higher at baseline than after avoidant coping instructions (F (1,36) = 7.38, p<.05). The ratings of expected Sensation Control given at baseline did not differ significantly from the ratings given after non-avoidant coping instructions were received, nor did the ratings given after avoidant coping instructions differ significantly from those given after non-avoidant coping instructions. No other significant effects were found.

7.3.5 Pain Indices

Means and standard deviations of behavioural and self-report pain indices for males and females in avoidant and non-avoidant coping conditions are presented in Table 7.3.

Table 7.3: Mean pain index scores of males and females in avoidant and non-avoidant coping conditions, Experiment 3 (standard deviations in parentheses).

	Males		Females	
Pain Index	**Non-avoidant**	**Avoidant**	**Non-avoidant**	**Avoidant**
Threshold	17.56 (14.62)	22.10 (17.68)	13.72 (9.07)	15.81 (13.71)
Tolerance**	178.33 (116.13)	222.62 (115.06)	82.54 (83.74)	119.86 (104.89)
Recovery*	100.12 (79.25)	114.78 (79.88)	76.98 (91.32)	90.80 (113.24)
Intensity	5.77 (2.61)	4.84 (2.71)	5.54 (2.35)	5.46 (2.30)
Sensory**	16.39 (6.60)	13.50 (6.52)	13.68 (4.67)	11.95 (4.96)
Affective*	2.50 (2.97)	1.61 (1.88)	2.47 (2.32)	1.84 (2.52)
n	18		20	

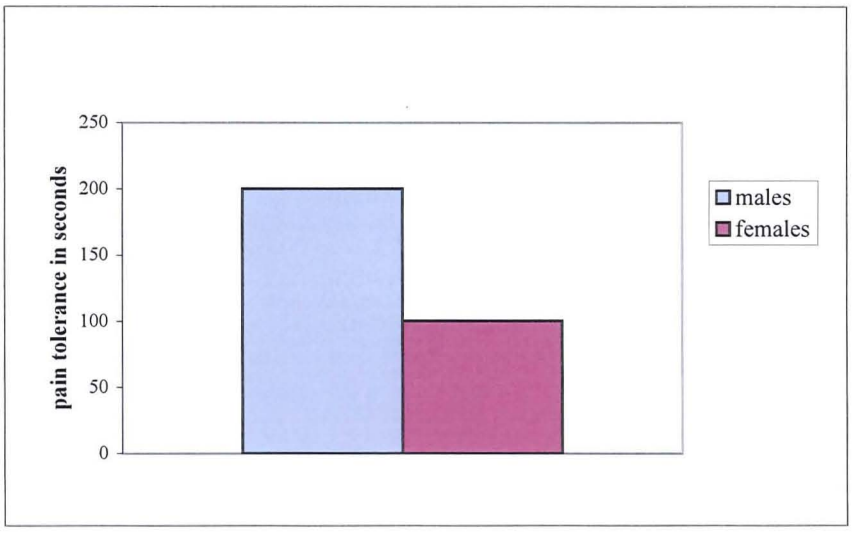
* = p<.05, ** = p<.01

7.3.5.1 Behavioural pain indices

A series of mixed-groups ANOVAs were conducted to evaluate the effect of gender and coping strategy on behavioural pain responses. In each analysis the between-subjects factor was gender (male vs. female) and the within-subjects factor was coping condition (avoidance vs. non-avoidance). The dependent variables were variously pain threshold, pain tolerance and pain recovery.

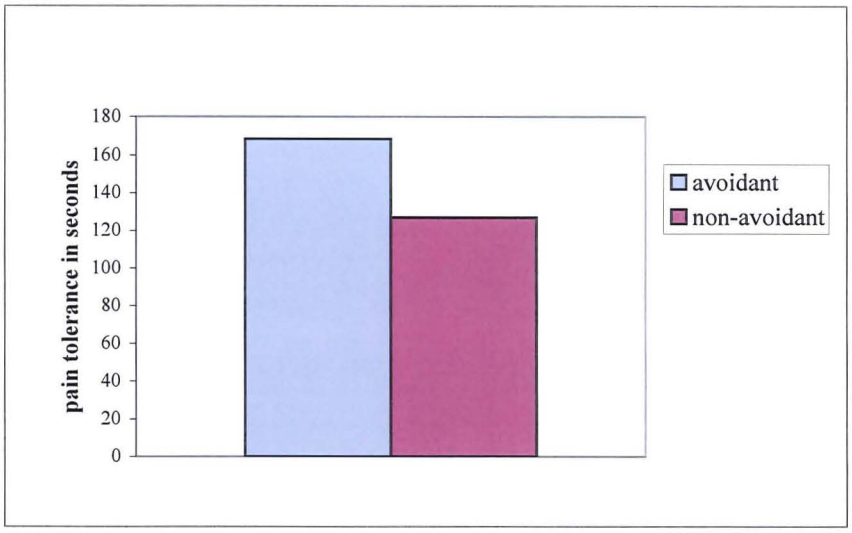
A significant main effect of gender was found for pain tolerance (F (1,34) = 12.81, p<.01; see Fig 7.1), with males showing higher tolerance for pain (mean = 200.47, SD = 99.10) than females (mean = 100.39, SD = 78.70).

Figure 7.1: Mean pain tolerance in seconds for males and females, Experiment 3



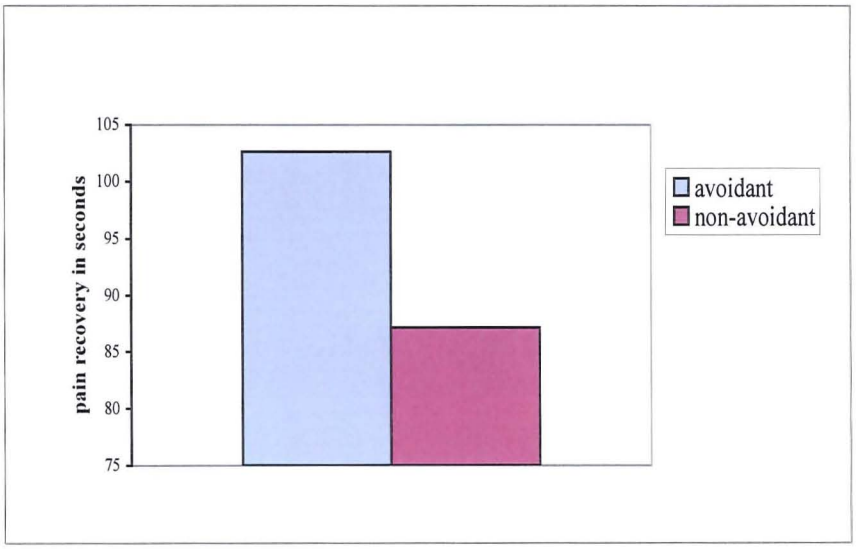
There was a main effect of coping condition on pain tolerance (F (1,34) = 4.98, p<.05; see Fig 7.2) which was higher with avoidant coping (mean = 168.54, SD = 120.15) than with non-avoidant coping (mean = 127.06, SD = 109.71). A main effect of coping condition order on pain tolerance (F (1,34) = 5.97, p<.05) was also found, with higher tolerance in those who used avoidance first and non-avoidance second (mean = 174.44, SD = 95.24) than those who had used the reverse order (mean = 121.15, SD = 102.64).

Figure 7.2: Mean pain tolerance in seconds for avoidant and non-avoidant coping conditions, Experiment 3



There was also a main effect of coping condition on pain recovery (F (1,34) = 8.14, p< .05; see Fig 7.3). Pain recovery was slower with avoidant coping (mean = 102.64, SD = 96.51) than with non-avoidant coping (mean = 87.17, SD = 84.38). No other significant effects were found.

Figure 7.3: Mean pain recovery in seconds for avoidant and non-avoidant coping conditions, Experiment 3

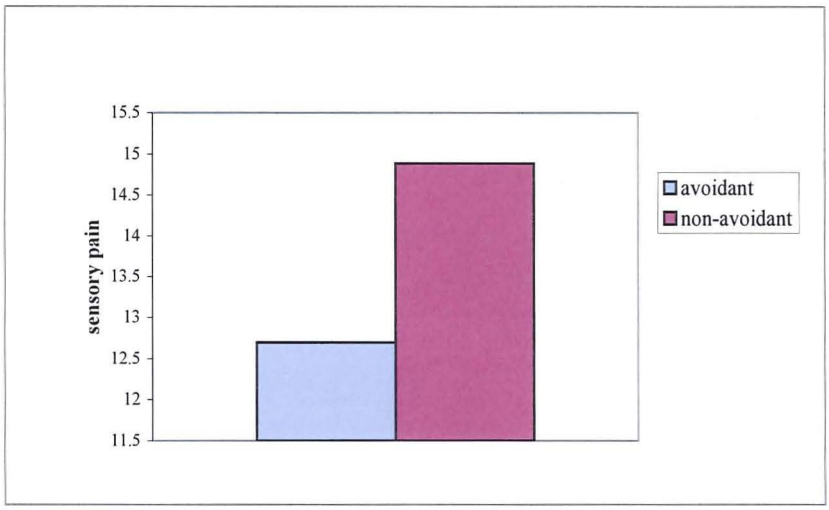


7.3.5.2 Self-report pain indices

Mixed-group ANOVAs were also used to evaluate the effects of gender and coping strategy on sensory pain, affective pain and pain intensity.

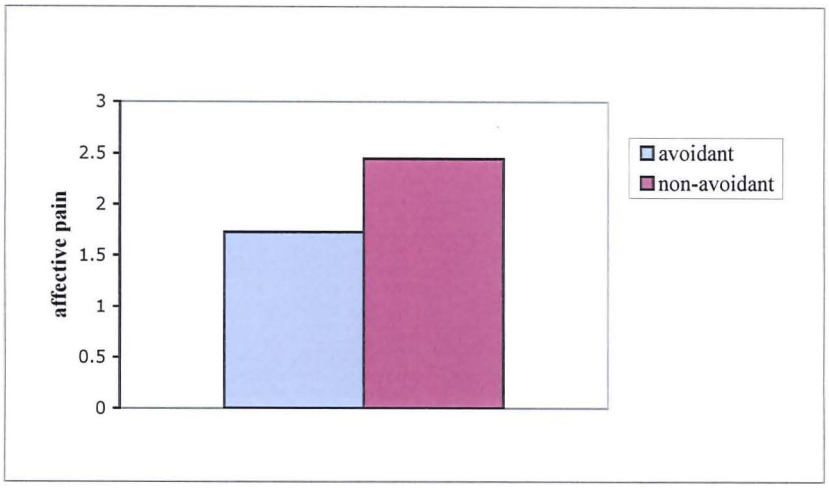
A significant main effect of coping condition was found for sensory pain (F (1,33) = 9.46, p<.01; see Fig 7.4), with higher pain ratings given with non-avoidant coping (mean = 14.89, SD = 5.73) than with avoidant coping (mean = 12.70, SD = 5.74).

Figure 7.4: Mean sensory pain ratings in avoidant and non-avoidant coping conditions, Experiment 3



A significant main effect of coping condition was also found for affective pain (F (1, 33) = 6.17, p<.05; see Fig 7.5), with higher pain ratings given with non-avoidant coping (mean = 2.45, SD = 2.60) than with avoidant coping (mean = 1.73, SD = 2.21). No other significant effects were found.

Figure 7.5: Mean affective pain ratings in avoidant and non-avoidant coping conditions, Experiment 3



7.3.6 Emotion

Means and standard deviations of positive and negative emotion (PANAS) scores for males and females, measured pre and post cold pressor exposures using avoidant and non-avoidant coping strategies are presented in Table 7.4

Table 7.4: Mean emotion scores measured by gender, time of testing and coping condition, Experiment 3 (standard deviations in parentheses).

		Males		Females	
Emotion	**Time**	**Non-avoidant**	**Avoidant**	**Non-avoidant**	**Avoidant**
Positive*					
	Pre	24.67 (6.26)	25.05 (6.76)	25.10 (5.71)	23.50 (5.97)
	Post	24.22 (5.84)	24.56 (7.26)	21.70 (6.57)	22.10 (6.66)
Negative					
	Pre	12.61 (3.01)	11.78 (3.25)	12.50 (3.59)	14.10 (6.61)
	Post	12.00 (3.43)	10.94 (2.04)	12.35 (2.85)	11.65 (1.84)
n		18	18	20	20

* = p<.05, ** = p<.01

7.3.6.1 Positive emotion

Repeated measures analysis of variance was performed to examine the effects of gender and coping condition on positive emotion. The between-groups factor was gender (male vs. female), and the within-groups factors were coping condition (avoidant vs. non-avoidant) and time of testing (pre vs. post). The dependent variable was positive emotional state. A significant main effect of time was found (F (1,36) = 5.85, p<.05) with positive emotion higher overall before (mean = 24.57, SD = 5.47) than after (mean = 23.08, SD = 6.28) cold pressor exposures. No other significant effects were found.

7.3.6.2 Negative emotion

A similar analysis was conducted on negative emotion. No main effects of gender, coping or time were found for negative emotional state. However, similar to Experiment 2, there was a two-way interaction effect between gender and coping which approached significance (F (1,36) = 3.93, p<.056). However, exploratory post-hoc simple effects analyses of negative emotion for men and women in avoidant and non-avoidant coping conditions revealed no significant effects.

7.3.7 Perceived coping effectiveness

Mixed-groups ANOVA of the subjective effectiveness of the two types of coping instructions revealed a significant main effect of coping strategy (F (1,36) =15.28, p<.001) with avoidant coping rated as more effective than non-avoidant coping overall (avoidant mean = 2.61, SD = 1.13; non-avoidant mean = 1.55, SD = 1.50). No other significant effects were found. This indicates that open-ended avoidant coping was generally perceived as more effective than non-avoidant coping.

7.3.8 Correlational analysis

Pearson's correlations were conducted to examine relationships between all questionnaire measures and pain indices in both coping conditions. Since both gender effects and coping effects were found for some pain indices, correlations were conducted for all participants in both coping conditions, and separately for males and females in both coping conditions (See Tables 7.5 – 7.10)

Table 7.5: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for all participants (n =38), Experiment 3.

	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.315	**.461****	.224	-.085	-.184	-.186
pre-CPT	.164	**.483****	**.430***	-.083	**-.324***	**-.344***
Fear of CPT						
Baseline	-.288	-.302	-.239	.281	.234	.231
pre-CPT	-.023	**-.363***	-.085	**.350***	**.540****	**.558****
Sensation Control						
Baseline	.204	.085	.010	.007	.031	-.063
pre-CPT	-.049	.106	.090	.046	-.105	-.241
PANAS POSI						
pre-CPT	.096	-.248	-.034	-.080	.058	.314
post –CPT	.105	-.165	-.005	-.046	.238	**.360***
PANAS NEGA						
pre-CPT	**-.348***	-.134	.064	**.375***	**.414****	.220
post-CPT	-.037	-.204	-.178	**.468***	**.574****	.253
DASS						
Stress	-.143	-.209	.012	.278	**.339***	**.406***
Anxiety	-.024	-.141	-.113	.170	.180	.288
Depression	-.145	-.021	-.024	.264	**.367***	.214
PCS						
Total	-.083	-.292	-.075	.237	**.471****	.314
Rumination	.001	-.288	.011	.253	**.465****	.286
Magnification	-.058	-.257	-.125	.185	**.532****	**.369***
Helplessness	-.146	-.259	-.110	.217	**.385***	.259
FOPS 24						
Total	.029	-.208	-.124	.261	**.487***	.230
Pain Dominance	-.227	-.206	.200	-.039	-.113	-.009
Fear & Worry	-.103	-.269	.039	-.061	.150	**.323***
Pain Control	.195	.079	.023	.153	.061	.077
MCSDS	-.149	**-.327***	-.275	.274	**.473***	.282
STAI-T	-.127	.019	.015	.153	.272	.169
Age	.103	**.324***	**.419****	.007	-.039	.009

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = State-Trait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

Table 7.6: Correlations between questionnaire scores, age and pain indices with avoidant coping for all participants (n = 38), Experiment 3.

	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.303	**.370***	**.348***	-.284	**-.352***	-.172
Pre-CPT	.249	**.465****	**.377***	-.225	**-.331***	-.225
Fear of CPT						
Baseline	-.167	-.249	-.221	.198	**.526****	.236
Pre-CPT	-.018	-.166	**-.400***	.085	**.447***	.314
Sensation Control						
Baseline	.053	-.013	-.033	-.122	-.083	.100
Pre-CPT	.204	.217	.112	-.128	-.057	-.085
PANAS POSI						
Pre-CPT	.316	.129	-.063	.157	.081	.166
Post –CPT	.226	.135	-.163	.073	.056	-.020
PANAS NEGA						
Pre-CPT	-.231	-.248	-.188	.088	**.386***	.207
Post-CPT	-.131	-.248	-.215	**.340***	**.576****	**.410***
DASS						
Stress	-.107	-.039	.056	.173	**.424****	.220
Anxiety	-.121	-.080	-.015	.214	**.366***	**.349***
Depression	-.220	-.044	.070	.281	**.417***	.291
PCS						
Total	.066	-.220	-.121	.177	**.448****	.003
Rumination	.093	-.129	-.120	.232	**.349***	-.030
Magnification	.078	-.247	-.110	.073	**.473***	.152
Helplessness	.030	-.258	-.098	.165	**.444***	-.023
FOPS 24						
Total	.154	-.150	-.138	.321	**.455****	.103
Pain Dominance	**-.330***	.033	.155	.088	-.025	.023
Fear & Worry	-.215	-.143	-.051	.042	.162	.281
Pain Control	.105	-.036	.003	.052	-.049	-.027
MCSDS	-.172	-.172	-.274	.200	.314	.263
STAI-T	-.058	.000	.207	.024	.290	.097
Age	.014	.177	-.131	-.169	-.169	-.079

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

Table 7.7: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for males only (n = 18), Experiment 3.

MALES	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.236	.317	.200	-.430	-.207	-.412
Pre-CPT	-.014	.228	.371	-.286	-.289	**-.632***
Fear of CPT						
Baseline	-.358	-.040	-.179	**.545***	.149	.415
Pre-CPT	.374	-.033	-.192	**.637***	**.528***	**.561***
Sensation Control						
Baseline	.246	-.212	.244	-.201	.056	.005
Pre-CPT	-.166	-.048	.215	-.112	-.156	-.274
PANAS POSI						
Pre-CPT	.328	-.317	-.119	.106	**.496***	.451
Post –CPT	**.478***	-.445	-.324	-.015	**.501***	**.566***
PANAS NEGA						
Pre-CPT	-.366	.339	.322	**.555***	.312	.149
Post-CPT	.018	-.187	-.054	**.535***	**.916***	.399
DASS						
Stress	-.144	-.187	-.401	.199	.296	**.481***
Anxiety	-.151	-.189	**-.492***	-.054	.236	.328
Depression	-.303	-.039	-.264	.103	.296	.234
PCS						
Total	.054	-.215	-.156	.258	.468	.329
Rumination	.179	-.293	-.142	.253	.453	.371
Magnification	.086	-.086	-.164	.289	**.584***	.237
Helplessness	-.067	-.191	-.140	.237	.417	.310
FOPS 24						
Total	.157	-.184	-.279	.283	**.505***	.327
Pain Dominance	-.366	-.148	.105	.028	-.209	.132
Fear & Worry	-.172	-.424	-.249	.030	.118	.431
Pain Control	.249	-.056	.036	.052	.249	.033
MCSDS	.088	-.186	-.284	.233	.514	.224
STAI-T	-.255	.006	-.229	.167	.355	.320
Age	.049	.114	.308	-.161	-.005	-.154

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

Table 7.8: Correlations between questionnaire measures, age and pain indices with avoidant coping, males only (n = 18)

MALES	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.265	-.020	.180	**-.587***	-.369	-.193
Pre-CPT	.250	.028	.291	**-.526***	-.320	-.249
Fear of CPT						
Baseline	-.240	.222	-.191	**.509***	.431	.206
Pre-CPT	.002	.315	**-.492***	.350	.456	.266
Sensation Control						
Baseline	.091	-.165	.192	-.259	-.247	.165
Pre-CPT	.324	.152	.398	-.249	-.194	-.185
PANAS POSI						
Pre-CPT	.373	-.246	-.421	**.487***	**.487***	**.540***
Post –CPT	.452	-.182	-.408	.281	.339	.247
PANAS NEGA						
Pre-CPT	-.403	.105	.044	.236	**.572***	.170
Post-CPT	-.306	.047	-.034	.232	**.667***	.362
DASS						
Stress	-.183	.129	-.305	.094	.293	.387
Anxiety	-.276	.030	-.308	.067	.345	.366
Depression	-.444	.011	-.149	.192	.263	.397
PCS						
Total	.172	-.104	-.203	.267	.325	-.007
Rumination	.241	-.055	-.252	.275	.203	.064
Magnification	.185	-.101	-.148	.339	.444	.071
Helplessness	.093	-.163	-.149	.219	.355	-.075
FOPS 24						
Total	.254	.028	-.315	.338	.379	.109
Pain Dominance	**-.554***	-.134	.065	.278	-.148	.347
Fear & Worry	-.299	-.158	-.349	.286	-.029	.465
Pain Control	.188	-.116	.005	-.017	-.047	-.058
MCSDS	-.024	-.004	-.118	.232	.235	.418
STAI-T	-.215	.043	-.069	-.026	.276	.220
Age	-.029	-.165	.250	-.369	-.153	-.112

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

Table 7.9: Correlations between questionnaire scores, age and pain indices with non-avoidant coping for females only (n = 20), Experiment 3.

FEMALES	NON-AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.430	.315	.104	.020	-.227	-.055
pre-CPT	.357	**.521***	.404	-.058	-.428	-.130
Fear of CPT						
Baseline	-.226	-.202	-.165	.325	.393	.151
pre-CPT	-.377	**-.617***	.038	.096	**.547***	**.571****
Sensation Control						
Baseline	.123	.202	-.328	.216	-.014	-.173
pre-CPT	.130	.235	-.079	.279	-.028	-.206
PANAS POSI						
pre-CPT	-.204	-.217	.061	-.328	**-.500***	.167
post –CPT	-.313	-.211	.160	-.209	-.023	.174
PANAS NEGA						
pre-CPT	-.353	**-.498***	-.128	.226	**.533***	.284
post-CPT	-.109	-.220	-.293	**.448***	.099	.080
DASS						
Stress	-.174	-.413	.255	.330	.400	.346
Anxiety	.116	-.254	.169	.418	.114	.241
Depression	.069	-.261	.151	.433	**.495***	.176
PCS						
Total	-.362	**-.522***	.038	**.223**	**.488***	.305
Rumination	-.325	-.372	.231	.297	**.492***	.164
Magnification	-.291	**-.526***	-.089	.026	**.451***	**.554***
Helplessness	-.311	**-.480***	-.090	.189	.331	.185
FOPS 24						
Total	-.199	**-.463***	-.018	.162	**.463***	.089
Pain Dominance	-.014	.216	.369	-.067	.025	-.169
Fear & Worry	-.007	-.087	.344	-.105	.198	.236
Pain Control	.112	-.067	-.102	.144	-.165	.100
MCSDS	**-.485***	**-.588***	-.296	.339	.419	.349
STAI-T	.035	.006	.234	.131	.167	-.002
Age	.152	.272	.344	.005	-.132	.203

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

Table 7.10: Correlations between questionnaire scores, age and pain indices with avoidant coping for females only (n = 20), Experiment 3.

FEMALES	AVOIDANT					
	Pain Thresh	**Pain Tol**	**Pain Recov**	**Senso Pain**	**Affec Pain**	**Pain Intens**
Tolerance Estimate						
Baseline	.345	**.453***	.360	-.168	-.370	-.066
pre-CPT	.292	**.564****	.319	-.157	-.378	-.132
Fear of CPT						
Baseline	-.057	-.358	-.110	.046	**.627****	.205
pre-CPT	-.132	-.296	-.246	-.078	**.513***	.320
Sensation Control						
Baseline	-.037	-.016	-.349	.007	.069	.077
pre-CPT	.062	.155	-.235	-.040	.068	.095
PANAS POSI						
pre-CPT	.228	.403	.198	-.325	-.221	-.255
post –CPT	-.083	.306	-.045	-.255	-.135	-.286
PANAS NEGA						
pre-CPT	-.276	-.344	-.226	.082	.331	.216
post-CPT	-.061	-.429	-.312	**.574***	**.526***	.439
DASS						
Stress	-.065	-.263	.242	.237	**.518***	.105
Anxiety	.037	-.272	.174	.391	.400	.359
Depression	.075	-.285	.209	.385	**.646****	.209
PCS						
Total	-.140	-.443	-.029	.010	**.670****	.014
Rumination	-.167	-.236	.039	.175	**.554***	-.207
Magnification	-.089	**-.459***	-.080	-.355	**.538***	.268
Helplessness	-.094	**-.477***	-.055	.063	**.618***	.070
FOPS 24						
Total	-.024	**-.496***	-.030	.266	**.580***	.135
Pain Dominance	-.005	.305	.311	-.139	.061	-.446
Fear & Worry	-.105	-.054	.239	-.205	.289	.048
Pain Control	-.030	-.192	-.120	.053	-.028	.081
MCSDS	-.377	-.388	**-.453***	.022	.400	.055
STAI-T	.135	-.062	**-.477***	.081	.312	-.037
Age	-.004	.272	**.530***	-.015	-.188	.096

Key: PANAS POSI = Positive emotion, PANAS NEGA = Negative emotion, DASS = Depression Anxiety Stress Scales, PCS = Pain Catastrophizing Scale, MCSDS = Marlowe-Crowne Social Desirability Scale, STAI-T = StateTrait Anxiety Inventory-Trait, * = p< .05, ** = p< .01

7.3.8.1 Correlations between Anticipatory Responses and pain indices

For all participants, a number of significant correlations were found between Anticipatory Responses and pain indices. Tolerance Estimate ratings given at baseline and after coping instructions were positively associated with pain tolerance in both coping conditions. Fear of CPT was negatively associated with pain tolerance in the non-avoidant coping condition, and with pain recovery in the avoidant condition. Positive associations were also found between Fear of CPT and self-report pain indices in both coping conditions. No correlations were found between anticipated Sensation Control and any pain index in either coping condition.

However, similar to Experiment 2, different patterns of association were revealed when correlations were conducted separately for males and females. For males, the Tolerance Estimate ratings they gave after non-avoidant coping instructions were negatively correlated with pain intensity in the non-avoidant coping condition, whereas in the avoidant condition the ratings they gave at baseline and after avoidant coping instructions were negatively associated with sensory pain. For males, Fear of CPT was positively associated with all self-report pain indices in the non-avoidant condition, but only with sensory pain in the avoidant condition.

For females, the Tolerance Estimate ratings given at baseline were positively associated with pain tolerance in the non-avoidant condition only, but ratings given after coping instructions were positively associated with pain tolerance in both coping conditions. Fear of CPT ratings after coping instructions were given were negatively associated with pain tolerance but only the non-avoidant condition. Fear of CPT ratings both at baseline and after coping instructions were positively associated with affective pain for females, but only in the avoidant condition.

7.3.8.2 Correlations between emotion and pain indices

As can be seen from Tables 7.5 and 7.6, for all participants, more associations were found between negative emotion and pain indices than between positive emotion and pain indices in both coping conditions. In contrast to Experiment 2, correlations conducted separately by gender revealed more associations between emotion and pain indices for males than for females overall.

7.3.8.3 Correlations between negative affectivity and pain indices

For all participants, there were several positive correlations between negative affectivity and self-report pain in both coping conditions. For males, a positive association was found between stress and sensory pain, and a negative association between anxiety and pain recovery in the non-avoidant condition only. For females, depression was positively associated with affective pain in the non-avoidant coping condition, whereas stress and depression were both positively associated with affective pain in the avoidant coping condition.

7.3.8.4 Correlations between pain catastrophizing and pain indices

For all participants, positive associations were found between all pain catastrophizing subscales and affective pain in both coping conditions. The PCS magnification subscale was positively correlated with pain intensity in the non-avoidant coping condition only. For males, magnification was positively associated with affective pain in the non-avoidant condition, but no other correlations between catastrophizing and pain indices were found. However, for females there were numerous correlations between catastrophizing subscales and pain indices in both coping conditions (see Tables 7.9 and 7.10).

7.3.8.5 Correlations between focus on pain and pain indices

For all participants, Pain Dominance was negatively associated with pain threshold in the avoidant coping condition only, whereas Fear & Worry was positively associated with pain intensity in the non-avoidant condition only. Total Focus on pain was positively associated with affective pain in both coping conditions. For males, Pain Dominance was negatively associated with pain threshold in the avoidant coping condition only, whereas Total Focus on pain was positively associated with affective pain in the non-avoidant coping condition only. For females, Total Focus on pain was negatively associated with pain tolerance and positively associated with affective pain in both coping conditions.

7.4 Discussion

Experiment 3 was essentially a replication of Experiment 2 except that open-ended distraction instructions were used in the avoidant coping condition instead of a specified imaginal distraction strategy. On the basis that the findings of Experiment 2 were

inconsistent with previous research, the primary aim of the experiment was to compare the effects of self-selected avoidant coping (using the same open-ended instructions as Keogh et al., 2000) with non-avoidant coping (sensory focusing).

In contrast to Experiment 2, and more consistent with Keogh et al. (2000), Experiment 3 did provide some evidence that avoidance and non-avoidance are differentially effective coping strategies for cold pressor pain. These effects were found for four out of the six dependent variables, whereas only affective pain differed significantly between fixed avoidance and non-avoidance in Experiment 2. In the present experiment, pain tolerance was higher and pain recovery slower when participants were allowed to choose a way of distracting attention away from pain than when they were instructed to focus on it. In addition, two self-report indices of pain (sensory pain, affective pain) were lower with open-ended avoidance than with non-avoidance. However, unlike Keogh et al. (2000), who found that sensory pain diminished with non-avoidance in males only, no interaction between gender and coping condition was found for any pain index in Experiment 3. Limited evidence of differences between male and female responses to cold pressor pain was found in Experiment 3. Although examination of means showed gender differences in the predicted direction for all pain indices, only the gender difference in pain tolerance reached statistical significance.

A free choice of distraction strategy seems to have conferred some other advantages. In the coping manipulation check, a very high percentage of participants reported they were able to adhere solely to avoidant coping when they were free to choose their own method of distraction (94.6%). This suggests that it is easier to engage in a coping strategy for pain if it is self-selected. There were also subjective benefits; open-ended avoidant coping was generally perceived as more effective than non-avoidant coping, whereas there was no subjective difference in effectiveness between fixed avoidant and non-avoidant coping in Experiment 2.

The gender differences in pain catastrophizing found in Experiment 2 were not replicated here, nor did men and women in Experiment 3 differ in cognitive and emotional focus on pain as measured on the FOPS 24. No gender differences were found in emotional states but correlational analyses indicated more associations between emotion and pain responses for women than for men overall.

Experiment 3 did indicate that men and women differ in the way they think and feel about a painful experience as they approach it. Gender differences in Anticipatory Responses to cold pressor pain were found and were similar to those observed in Experiment 2. In both experiments, women reported more fear of the imminent painful experience, and lower expectations of tolerance than men.

7.5 Summary

As a partial replication of Experiment 2, substituting open-ended avoidant coping for fixed avoidant coping, the present experiment did indicate that there might be a clearer impact of avoidant coping on pain responses if participants are free to choose their own method of distraction. Open-ended avoidant coping was also subjectively rated more effective than non-avoidance by all participants. Experiment 3 reiterated that males and females differ both behaviourally and psychologically in their responses to cold pressor pain responses, but did not demonstrate that they benefitted differentially from avoidant or non-avoidant coping strategies. These conclusions are necessarily speculative as Experiment 3 did not directly compare open-ended avoidant coping with fixed avoidant coping. However, since the sample population and experimental methodology of Experiments 2 and 3 were the same, the descriptives from the cold pressor pain responses of males and females in fixed avoidant, open-ended avoidant and non-avoidant coping strategies can be inspected together using a combination of data from both experiments. This cross-experiment data (nominally Experiment 3a) is presented in the following chapter.

Chapter 8

Experiment 3a
Cold pressor pain responses with three different coping strategies

8.1 Introduction

This chapter presents cross-experimental descriptive data from the pain responses of healthy males and females employing three different strategies to cope with the cold pressor stimulus, using combined data from Experiments 2 and 3. Taking pain response data from first cold pressor exposures only in both experiments, three coping condition groups were derived with approximately equal numbers of males and females in each group. The total number of participants was 54 (26 males, 28 females), with an age range of 18-43 years (mean = 26.06, SD = 6.58). Participants were all staff or students at Goldsmiths College. The non-avoidant coping instructions used in Experiments 2 and 3 and the fixed-avoidant coping instructions used in Experiment 2 appear in Chapter 5. The open-ended avoidant coping instructions implemented in Experiment 3 can be found in Chapter 7.

Variation in pain research methodologies adversely affects the viability of comparisons between studies (Eccleston, 1995), but because there was methodological consistency and standardisation across all experiments in this series, viewing the mean pain responses from Experiments 2 and 3 together was considered meaningful. However, inferential tests to directly compare of the effects of fixed avoidant, open-ended avoidant and non-avoidant coping instructions on the cold pressor pain responses of males and females could not be conducted because this is effectively a hierarchical design with the avoidant coping types nested within Experiments 2 and 3, and non-avoidant coping data taken from both experiments. Complex nested designs are difficult to analyse and are not recommended where interactions are likely (Clarke-Carter, 1997). In addition the cell sizes produced from the combined data set were quite small.

This chapter therefore contains a table of descriptive data for visual inspection only. On the basis of previous research, gender differences are to be expected in pain responses, specifically greater pain sensitivity in females than males. On the same basis, differences between mean pain responses in the three coping conditions are also likely to be seen.

The remainder of the chapter provides a general discussion of the effects of gender and coping on cold pressor pain responses in Experiments 1, 2 and 3.

8.2 Pain indices

Means, standard deviations, skewness and kurtosis of pain indices for males and females in fixed avoidant, open-ended avoidant and non-avoidant coping conditions are presented in Table 8.1.

8.3 Discussion

As can be seen from Table 8.1, mean pain threshold and pain tolerance values were generally higher for males than females (with the exception of pain threshold in the open-ended avoidant coping condition). Interestingly, all pain recovery times were slower for males than for females. However, it is not known whether any of these apparent gender differences were statistically significant. There were no obvious indications of differential effects of coping strategy type on pain responses. For future work, incorporating all three types of coping strategy within a single experiment would permit a direct comparison of the relative impact of fixed avoidant, open-ended avoidant and non-avoidant coping on pain responses.

Table 8.1: Descriptive pain data from males and females in fixed avoidant, open-ended avoidant and non-avoidant coping conditions, Experiment 3a.

		Males			Females		
		Fixed avoidant	Open-ended avoidant	Non-avoidant	Fixed avoidant	Open-ended avoidant	Non-avoidant
Thresh	Mean	21.14	17.35	26.35	19.28	19.63	11.59
	SD	14.75	13.52	16.08	10.48	16.75	7.54
	Median	14.04	15.94	24.64	20.24	13.36	10.28
	Min	10.32	3.66	5.79	5.21	3.95	3.15
	Max	56.09	41.10	59.49	36.68	49.97	22.65
	Kurtosis	4.14	-.29	1.36	-.59	.19	-1.55
	Skewness	1.93	.80	1.07	.12	1.21	.39
Tol	Mean	203.04	241.37	132.52	115.92	171.90	35.90
	SD	134.83	108.57	115.79	118.15	110.66	23.05
	Median	269.16	234.05	65.98	51.54	14.35	30.50
	Min	14.44	61.82	41.58	7.60	24.06	14.28
	Max	300.00	300.00	300.00	300.00	300.00	86.19
	Kurtosis	-1.70	.009	-1.53	-.920	-1.99	2.09
	Skewness	-.822	-1.442	.87	.89	.123	1.45
Recov	Mean	146.53	132.43	150.07	119.28	90.29	66.35
	SD	129.29	78.41	169.12	86.93	97.67	29.49
	Median	112.63	128.53	82.19	100.56	57.61	63.24
	Min	17.20	31.45	26.80	26.68	14.00	18.49
	Max	402.29	240.146	497.63	262.07	341.78	117.70
	Kurtosis	.42	-1.31	1.16	-.73	5.47	.25
	Skewness	.93	.24	1.50	.78	2.19	.11
Senso	Mean	12.56	13.62	12.00	16.78	11.60	14.11
	SD	5.64	6.50	4.82	4.55	4.72	3.18
	Median	12.00	12.33	13.00	16.33	10.83	14.40
	Min	5.00	4.00	5.00	11.00	3.00	8.00
	Max	23.00	23.00	17.00	26.00	20.00	19.00
	Kurtosis	.084	-.69	-1.64	.99	.49	1.01
	Skewness	.46	.25	-.490	.89	.04	-.58
Affect	Mean	2.33	1.87	1.89	5.44	1.40	3.67
	SD	1.87	1.96	1.96	3.54	2.63	3.00
	Median	2.50	1.50	1.66	4.20	.50	4.00
	Min	.00	.00	.00	2.00	.00	.00
	Max	5.00	5.00	6.00	11.00	8.00	7.00
	Kurtosis	-1.68	-1.29	1.31	-1.45	4.59	-1.95
	Skewness	-.03	.53	1.09	.77	2.19	-.052
Intens	Mean	4.71	5.17	5.87	5.98	4.96	5.71
	SD	2.86	2.39	1.91	2.88	2.30	2.28
	Median	5.55	4.80	6.50	6.75	5.32	6.50
	Min	.50	1.75	2.50	2.70	.00	1.25
	Max	8.10	9.80	7.90	10.00	8.15	8.10
	Kurtosis	-1.74	1.47	-.78	-1.94	1.50	.31
	Skewness	-.24	.77	-.67	.07	-.92	-.98
	N	9	8	9	10	9	9

8.4 General discussion of gender comparison experiments

Experiments 1, 2, and 3 have found some evidence of gender differences in cold pressor pain responses. However, such differences do not directly replicate and the pain indices which show gender differences varies between experiments. In Experiment 1, which employed a between-groups design (N = 106), gender differences were found in both behavioural and self-report measures of pain (pain tolerance and sensory pain respectively). In Experiment 2, gender differences in sensory pain and affective pain were found but no significant differences were found on behavioural pain indices. This was surprising, as the implementation of a within-groups design was expected to reduce the impact of individual differences and so increase the likelihood of detecting gender effects.

It is possible that the gender differences found in Experiment 1 were at least partly an artefact of individual differences. The absence of gender-differentiated pain tolerance in Experiment 2 could be the result of lower power due to the smaller sample size (N = 53) than in Experiment 1 (despite the repeated measures design). However, the fact that pain tolerance did differ significantly between men and women in Experiment 3 with a sample size of N = 38 (18 male, 20 female) detracts from the likelihood of insufficient power at N = 53 in Experiment 2. The reasons for such inconsistencies are not clear, but since the experimental methods used in these studies have been extremely consistent, these conflicting findings are not attributable to methodological variation.

Across Experiments 1, 2 and 3 men and women differed in some cold pressor pain responses, and also demonstrated some differences in pain-related cognition, negative emotion in the context of pain, and coping style. Taken together, the findings of the experiments reported here indicate that women have lower tolerance for pain and report more sensory and affective pain than men. In addition, women seem to experience more negative thoughts, feelings and expectations about pain than their male counterparts. That said, as with the pain responses, these gender differences in pain-related cognition and emotion vary considerably.

The asymmetry of male and female pain responses may be at least partly attributable to social response bias in pain reporting and coping (see Chapter 3). However, there is also compelling evidence for a biological basis to gender differences in pain sensitivity. For

example, recent animal research echoes the pattern of sex-related disparity observed in human pain studies, with male sensitivity frequently found to be lower than female (see Chapter 2). There are also a number of reasons to consider the influence of reproductively-linked hormones on pain perception. For example, there are many more clinical pain syndromes of physiological and hormonal origin which predominantly affect females than males (Berkley, 1997). The fact that sex differences in pain prevalence emerge around adolescence - a transition dominated by extreme hormonal changes in both males and females - also strongly suggests that hormone levels play a significant part in such differences (Von Korff, Dworkin, Le Resche & Kruger, 1988). Furthermore, CNS regions which are involved in pain perception and inhibition (such as the PAG, rostroventral medulla and spinal dorsal horn) are also populated with receptor binding sites for gonadal steroid hormones which suggests a functional connection between them (Sternberg & Wachterman, 2000).

As discussed in Chapter 2, there is some evidence that female pain responses are affected by hormonally-mediated changes in perception across the ovarian cycle. More research is needed to clarify these effects, but it may be that for women of reproductive age, hormonal fluctuation across the menstrual cycle alters pain sensitivity and emotional state in an interconnected manner (Riley et al., 1999). It is feasible that such a factor could confound gender effects on sensory and/or emotional responses to pain. Menstrual phase is also known to produce alterations in female blood pressure (Dunne, Barry, Ferriss, Grealy & Murphy, 1991), which may also contribute to gender differences in pain sensitivity (Fillingim & Maixner, 1996; Nyklicek, Vingerhoets & Van Heck, 1999). Menstrual phase was not assessed in the gender comparison experiments (1, 2 and 3) but the pain responses of female participants may have been affected by hormonal conditions at the time of testing.

8.5 Summary

In summary, it seems that the experience of pain is more likely, more aversive and less tolerable for women than it is for men. Additionally, the experience of pain is arguably more complex for females than for males; women seem to respond to painful experiences with more negative emotions and less positive expectations than men. Furthermore, the female reproductive cycle may exert influence over sensory and emotional perception, which in turn may cause temporal changes in female pain

responses. In light of these possibilities, the next experiment was designed to investigate the impact of hormonal factors on the pain perception of healthy adult females by comparing cold pressor pain responses in different phases of the menstrual cycle.

Chapter 9

Experiment 4
Menstrual Cycle and Cold Pressor Pain

9.1 Introduction

If gonadal hormones are a significant part of the biological mechanisms of sex-related differences in pain sensitivity, this is likely to have important implications for pain management, particularly for women. As discussed in Chapter 2, a variety of methodologies have been used to examine the impact of gonadal hormones on pain responses in humans and rodents. Although pharmacological and surgical manipulation of hormone levels have been used in rodent studies, methods which utilise naturally-occurring alterations in hormone levels are more appropriate and viable in human research. For example, comparing pain responses across different phases of the menstrual cycle permits non-invasive examination of the effects of gonadal hormone fluctuation on female pain. The female reproductive cycle provides a natural test-bed for the effects of changing hormone levels on female pain sensitivity (Berkley, 1992). Exploiting the hormonal fluctuation of the normal ovarian cycle, comparisons can be made between pain responses in different menstrual phases with contrasting levels of gonadal hormones. Alterations in pain sensitivity across menstrual phase have long been documented (see Fillingim & Ness, 2000), but there has been relatively little empirical investigation of menstrual cycle effects on cold pressor pain.

Variation in female pain perception across the menstrual cycle may explain the inconsistency of gender differences in pain responses found across Experiments 1, 2, 3 and 3a. Experiment 4 was therefore designed to evaluate the impact of menstrual phase on the cold pressor pain responses of healthy females, using the menstrual cycle paradigm outlined in Chapter 2 to compare two phases with strongly contrasting hormonal profiles. A detailed exposition of the complex neuroendocrine regulation of the human reproductive system is not necessary here. However, as relevant background to the present experiment, an overview of the major hormonal events of the human menstrual cycle and the rodent estrous cycle is provided below.

9.1.1 Hormonal events and the reproductive cycle

The two main gonadal hormones secreted during the female ovarian cycle are estrogen and progesterone. Estrogen, which is primarily produced by the ovaries, controls morphological changes during female puberty (e.g., gonadal growth, breast development and deposit of body fat), and also causes cyclic uterine changes during the human menstrual (or rodent estrous) cycle. Progesterone, which is secreted by the

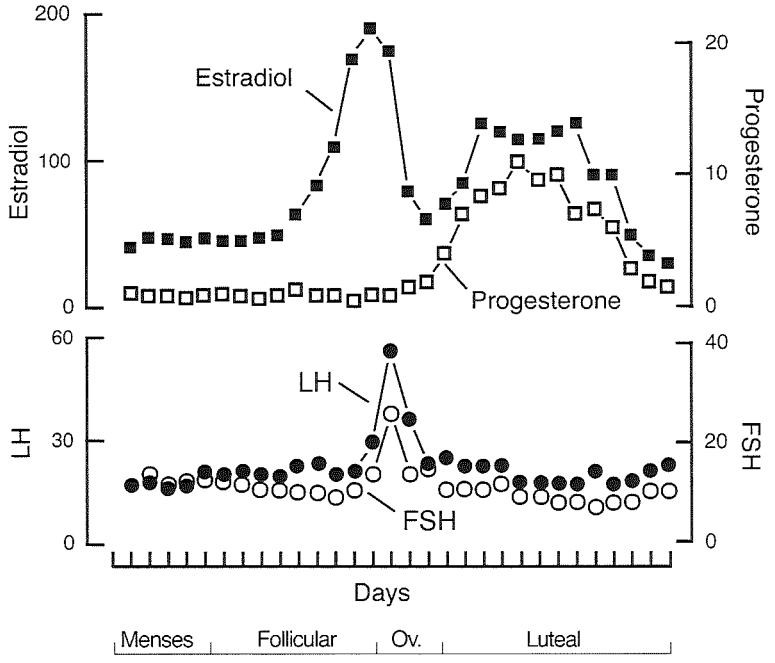
corpus luteum after ovulation (and by the placenta during gestation), stimulates growth of the uterine lining. Radical alterations in the levels of estrogen and progesterone across the cycle are governed by the endocrine system, specifically by the release of two gonadotropic substances, follicle stimulating hormone (FSH) and luteinising hormone (LH), from the pituitary gland.

A sequential pattern of hormonal events characterises the human menstrual cycle. Four stages are commonly distinguished: follicular, ovulatory, luteal and menstrual but operational definition of these phases varies (Yen, 1998). In the follicular (postmenstrual) phase, FSH and LH are secreted at low-moderate levels. Estrogen gradually increases during this phase, peaking just before ovulation. Progesterone remains at a low and stable level throughout the follicular phase, rising slightly before mid-cycle. Approximately one day before ovulation, there is a dramatic surge in LH and FSH. This surge triggers a series of events, starting with release of follicular steroid hormones containing small amounts of progesterone and culminating in the rupture of the follicle and the release of the egg (ovulation). Shortly after ovulation, the corpus luteum forms from the ruptured follicle and secretes a flood of estrogen and progesterone, giving rise and a name to the luteal (postovulatory) phase. Progesterone levels peak in the mid-luteal phase and there is also a rise in estrogen during this stage (although not to the level which occurs immediately prior to ovulation). Rising estrogen and progesterone levels create negative feedback to the pituitary which slows down secretion of LH and FSH across the luteal phase, causing the corpus luteum to degenerate. As the corpus luteum breaks down, secretion of estrogen and progesterone falls, thus negative feedback to the pituitary terminates and LH and FSH levels start to rise again, triggering a new ovulatory cycle. At this point the uterine lining sloughs off and menstruation begins. See Figure 2.1 for a schematic diagram of the main hormonal events of the human ovarian cycle. Research has utilised the changes in hormone levels which occur throughout the menstrual cycle to evaluate the effects of estrogen and progesterone fluctuation on pain perception.

The rodent estrous cycle is broadly analogous to the human menstrual cycle. Female rodents, like humans, are cyclic ovulators whose reproductive system features large shifts in estrogen and progesterone levels. As in the human menstrual cycle, four phases characterised by particular hormonal events are also discernible within the 4-5 day

rodent estrous cycle (Sternberg & Wachterman, 2000). The first of these, known as metestrus, features a high level of progesterone and low levels of estrogen and luteinising hormone (LH). This is followed by diestrus, when LH secretion is relatively high, estrogen level is rising and progesterone is falling. These two phases are considered approximate to the perimenstrual phase in the human cycle (Giamberardino, 2000). During the subsequent proestrus phase, estrogen, progesterone, LH and follicle-stimulating hormone (FSH) all reach peak levels. In the fourth stage, known as estrus, all of these hormones fall to low levels and the rodent becomes sexually receptive. As with the human menstrual cycle, the rodent estrous cycle has been used to explore the effects of hormonal alterations on pain sensitivity.

Figure 9.1: Schematic diagram of hormonal events of the human ovarian cycle



Adapted from Fillingim and Ness (2000). Reproduced with permission from IASP Press.

9.1.2 Methodological issues in menstrual cycle research

Upon examination of previous research into the impact of menstrual cycle on female pain sensitivity it quickly becomes evident that, as with experimental pain research generally, methodological variability is both prevalent and problematic. In particular, two inter-related issues which affect the comparability of different studies are the way in which menstrual phase is delineated, and the accuracy with which ovulation is

pinpointed. Unfortunately, there has been little consistency in the way these have been achieved. Operational definitions of menstrual phases are not standardised and methods of identifying the different phases vary substantially For example, some researchers have defined days 15-21 as the ovulatory phase (e.g., Goolkasian, 1980). Others have given no operational definition of phases within the cycle (e.g., Procacci, Zoppi, Maresca & Romano, 1974). Calendar counting has often been used to estimate phases of the cycle (e.g., Amodei & Nelson-Grey, 1989) and these calculations have commonly been based on a 28-day cycle (see Riley, Robinson, Wise & Price, 1999). However, a number of factors call the accuracy of this practice into question. For example, although the median length of the interval between menses in most women of middle reproductive age (i.e., not proximate to menarche or menopause) is 28 days (Yen, 1998), the range is 25-35 days. Consequently, if ovulation is not conclusively established, phase divisions may be spurious and the validity of experimental comparisons between them becomes questionable. This variability in length of the menstrual cycle across individuals can be primarily accounted for by the extent to which the length of the follicular phase can vary, whereas the luteal phase has been found to be extremely stable (12-15 days). Ovulation, which occurs approximately 14 days before menstruation, has traditionally been estimated by counting backwards from the first day of menses (Ferin, Jewelewicz & Warren, 1993). On this basis, ovulation in a 35-day cycle is likely to occur on day 21. There have been attempts to pinpoint ovulation by more objective methods. For example, Veith, Anderson and Slade (1984) identified menstrual phase by detection of the shift in basal body temperature which accompanies ovulation. However, since this shift is very small (.2-1°F) and must be assessed upon awakening and before any activity (Kass-Annese & Danzer, 1986), this method is dependent on participant competence as well as compliance and cannot be researcher-verified. Some recent studies have improved on calendar counting to identify menstrual cycle phase by use of 'ovulation detection' test kits (e.g., Fillingim, Maixner, Girdler, Light, Sheps & Mason, 1997). Approximately 24-36 hours before ovulation, there is a sudden surge of two types of gonadatropin; luteinising hormone (LH) and follicle stimulating hormone (FSH). These tests detect the LH surge which precedes ovulation, and thus permit accurate identification of menstrual cycle phase. Such tests are a good alternative to hormonal assay of blood, which is a definitive method of ascertaining menstrual phase but an unpleasant invasive procedure and so is not always appropriate or possible in the context of experimental pain induction. Use of ovulation detection test

kits has been recommended as a control against error variance due to fluctuation of cycle lengths (Riley et al., 1999)

An assessment of emotional state was incorporated into Experiment 4 since fluctuations in reproductive hormone levels can substantially affect emotional states in women (Parry & Haynes, 2000), and these in turn may affect pain perception. Negative emotional states such as irritability and depression are commonly reported in the late luteal phase and has been known as pre-menstrual syndrome (PMS). In its extreme form such cyclic emotional lability is clinically defined as pre-menstrual dysphoric disorder (PMDD) in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994).

A factor which may also affect female-specific pain responses, and therefore warrants consideration here is dysmenhorrhea, or pain associated with menstruation. As discussed in Chapter 2, most women experience dysmenhorrhea to some extent (Dawood, 1985; Andersch & Milsom, 1982). There has been some evidence that dysmenhorrhea accentuates the impact of menstrual phase on pain threshold (Giamberarderino et al, 1997). However, other studies have indicated that menstrual phase alterations in pain sensitivity occur only in women who experience no pain accompanying menstruation (e.g. Goolkasian, 1983), and still others have found no such phasic differences between dysmenhorrheics and non-dysmenhorrheics for pain threshold or tolerance (Aberger, Denney & Hutchings, 1983; Amodei & Nelson-Gray, 1989). The impact of menstrual pain on female pain sensitivity overall is unclear, but was considered a potential confound in the present experiment. Since dysmenhorrheic and non-dysmenhorrheic women may respond differently to pain and a total absence of dysmenhorrrhea seems to be unusual, only women who reported pain accompanying menstruation were included in Experiment 4.

Blood pressure was also assessed in Experiment 4, since it has been found to alter across menstrual phase (Dunne, Barry, Ferriss, Grealy & Murphy, 1991; Miller & Sita, 1994) and may affect pain sensitivity. Research has indicated an inverse relationship between arterial blood pressure (BP) and pain sensitivity (e.g., McCubbin & Bruehl, 1994) but BP-related hypoalgesia has not been consistently found in women (Maixner & Humphrey, 1993; Bruehl, McCubbin & Harden, 1999). Much of the research to date

on the relationship between BP and pain sensitivity has been conducted with men only (e.g., Bruehl et al, 1992) and studies including gender comparisons have yielded conflicting findings. For example, Fillingim and Maixner (1996) found an inverse relationship between resting systolic BP and pain sensitivity in men only, whereas Nyclicek, Vingerhoets and Van Heck (1999) found reduced pain sensitivity in hypertensive females relative to normotensive females but no such difference in males. However, Myers, Robinson, Riley, and Sheffield (2001) recently reported that women showed lower pain threshold, pain tolerance and resting BP than men, and also found an inverse relationship between systolic blood pressure and pain sensitivity for both men and women. The pattern of blood pressure variation across the menstrual cycle is inconsistent (Kelleher, Joyce, Kelley & Ferriss, 1986; Greenberg, Imeson, Thompson & Meade, 1985) as are the BP indices which show phasic changes (Freedman, Ramcharan, Hoag & Goldfien, 1974; Hassan, Carter & Tooke, 1990). The association between elevated BP and reduced pain sensitivity suggests that changes in resting BP during the menstrual cycle may contribute to altered pain responses across different phases. Indeed, at least one study has provided some support for this proposition. Testing normotensive women, Pfleeger, Straneva, Fillingim, Maixner and Girdler (1997) found that sensitivity to ischemic pain was greater in the luteal than the follicular phase of the menstrual cycle, and that BP correlated with pain sensitivity in both phases, but did so most strongly in the luteal phase. Following Pfleeger et al. (1997), measurement of resting BP in each menstrual phase was incorporated into Experiment 4.

Experiment 4 was designed to address two primary aims, the first of which was to was to compare the cold pressor pain responses of healthy women in two distinct phases of the menstrual cycle. The second aim was to ascertain whether healthy women would report more pain experiences throughout the follicular or the luteal phase of their menstrual cycle. It was expected that women would show higher pain threshold, higher pain tolerance and report less pain in the follicular than in the luteal phase. This prediction was made on the basis of previous experimental research, although few studies have examined cold pressor pain responses across menstrual phase (see Riley et al., 1999).

The secondary aims of Experiment 4 included investigation of potential alterations in blood pressure and emotional state across menstrual phases. Lower blood pressure was expected during the luteal phase compared to the follicular phase (Dunne et al., 1991),

and it was also expected that blood pressure would correlate with behavioural pain indices (pain threshold, pain tolerance). Emotional alteration across the menstrual cycle was expected, specifically more negative emotion in the luteal phase than the follicular phase. Finally, two aspects of pain-related cognition were evaluated. Firstly, since previous research indicates that women catastrophize more about pain than men (Sullivan et al, 1995 and Chapter 5 of this thesis), Experiment 4 sought to ascertain whether the extent of pain catastrophizing - specifically about cold pressor pain - would be related to pain responses and/or differ between menstrual cycle phases. Secondly, Experiment 4 sought to assess whether women's cognitive and emotional focus on pain would be related to their pain responses. It was expected that greater catastrophizing and a greater tendency to focus on pain would be associated with lower pain tolerance and greater pain report.

9.2 Method

9.2.1 Design

A repeated-measures design was employed with one within-groups factor: menstrual phase (follicular vs. luteal). The main dependent variables were pain threshold, pain tolerance, pain recovery, sensory pain, affective pain and pain intensity. Additional variables assessed were diary pain indices, blood pressure, emotional state, focus on pain and state catastrophizing.

9.2.2 Participants

40 female university students from Goldsmiths College were recruited via posters and flyers around the campus. All women recruited were of reproductive age and reported regular menstrual cycles and experiencing pain accompanying menstruation. A lower age cutoff of 18 years was employed because irregular and anovular menstrual cycles are more common in the early years post-menarche (see Yen, 1998). All participants were in good general health and not in pain on days of testing. 8 participants dropped out of the experiment at various stages, and the data from a further 4 was not included due to unconfirmed ovulation. The resultant sample comprised 28 healthy females with an age range of 18-44 years (mean=29.11, SD=8.42). The same exclusion criteria were employed as in previous cold pressor experiments in this series (see Chapter 4). Women suffering from reproductive disorders were also excluded from this experiment as were those taking hormonal medication of any kind, including oral contraceptives, Hormone Replacement Therapy (HRT) or fertility treatment.

9.2.3 Pain diaries

Pain diaries were kept for a minimum of one full menstrual cycle preceding the experimental test sessions. These served a dual purpose: firstly to facilitate accurate tracking of the menstrual cycle preceding experimental sessions, and secondly to assess self-report pain experienced across the follicular and luteal phases (pain intensity, sensory pain and affective pain). Pain diary entries comprised logging whether each day was menstrual or non-menstrual and completion of the SF-MPQ at approximately the same time each evening to rate any pain experienced during the day. The cause of pain was stated, if known. Participants were also required to record and report menstrual cycle parameters (i.e., start and end dates of menstruation) for a minimum of two full cycles before the experimental test sessions commenced.

9.2.4 Identification of menstrual phase

In order to make a meaningful comparison of pain responses between the follicular and luteal phases, it was important to clearly distinguish these phases. To this end, it was necessary to ascertain the date of ovulation for each participant. Ovulation for each participant was estimated by calendar counting based on the menstrual cycle history obtained for each woman and confirmed using Clearplan hormone detection tests on first morning urine samples. The date for the first ovulation test was calculated on the basis of individual menstrual history. In this manner, the first ovulation test was scheduled for 17 days before the likely date of next menses[1]. All ovulation tests were self-administered by participants and all test results were researcher-verified. Positive tests were verified for all but 4 participants, whose data were subsequently dropped from the analysis. On this basis, follicular phase was operationally defined in Experiment 4 as days 4-9 inclusive (counting the onset of menstruation as Day 1). This block of days is mid-follicular (Yen, 1998), with estrogen rising towards its preovulatory peak level and progesterone levels low and stable. Luteal phase was operationally defined as 5-10 days after ovulation (after Ferin et al., 1993; Pfleeger et al., 1997); hence luteal phase testing took place on days 19-24 inclusive. This block of days is mid-luteal and features peaking levels of progesterone, coupled with some elevation of estrogen. For one participant in this sample the hormone surge was not

[1] This was based on the conventional estimate of ovulation approximately 14 days before menstruation, in conjunction with the likelihood that the LH surge is detectable 24-36 hours prior to ovulation. In addition, the manufacturers of Clearplan recommend that ovulation detection testing commences 17 days prior to expected date of the next menstrual period.

detected until Day 19; the luteal phase experimental session for this individual was therefore scheduled for Day 24 (the latest available day within the luteal test phase used here).

9.2.5 Blood pressure measurement

Resting blood pressure was measured using a MARS MS-700 automatic oscillometric digital blood pressure monitor. Three successive blood pressure readings were taken with a rest of 1 minute between measurements, using the participant's left arm (after Shapiro, Jamner, Lane, Light, Myrtek, Sawada & Steptoe, 1996).

9.2.6 Pain induction

9.2.6.1 Cold pressor apparatus and task

The cold pressor apparatus and task were the same as in previous experiments. Participants were given no instructions for coping with the discomfort of the cold pressor.

9.2.6.2 Pain ratings

As before ratings of pain threshold, pain tolerance and pain recovery, measured in seconds, were obtained directly from the cold pressor task. As before, self-report measures of pain intensity, sensory pain and affective pain were obtained with the Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987). The SF-MPQ was completed immediately after each cold pressor exposure.

9.2.7 Additional questionnaires

Profile of Mood States: Emotional states were measured using the Profile of Mood States scales (POMS; McNair, Lorr & Droppelman, 1992). The POMS was chosen over the PANAS (which was used in Experiments 2 and 3) to provide a more specific measure of emotions. This self-report measure consists of 65 descriptors of feelings, which constitute six factor subscales; Tension-Anxiety, Depression-Dejection, Anger-Hostility, Confusion-Bewilderment, Vigour-Activity and Fatigue-Inertia. The response format is a 5-point Likert-type scale ranging from 0 (*not at all*) to 4 (*extremely*). Respondents circle the number which best reflects the extent of each feeling in the past week, including the present day. Scoring of each POMS subscale involves an individual weighting system as proscribed in the test manual, and the range of scores possible is

not defined. However, higher scores on any subscale reflect greater occurrence of a given emotional state. The POMS was administered before cold pressor exposure in each menstrual phase.

Pain Catastrophizing Scale (state version): To assess catastrophizing cognition during the cold pressor pain induction (rather than pain catastrophizing as a general cognitive trait), an adapted version of the Pain Catastrophizing Scale (PCS, Sullivan, Bisop & Pivik, 1995) was used. The PCS instructions and item-statements were reworded slightly to refer directly to the pain elicited during cold pressor immersion. The original response format and scoring of the PCS were unaltered and are outlined in Chapter 5, but only a total score was used here. The state version of the PCS was administered after cold pressor exposure in each menstrual phase.

Focus on Pain Scale 24: As in Experiment 3, the Focus on Pain Scale 24 (FOPS 24), a self-report measure developed for use in this series of experiments, was used to assess cognitive and emotional focus on pain. Details of the development and preliminary psychometric properties of the FOPS 24 appear in Chapter 6. The FOPS 24 was completed soon after recruitment into the experiment, before the experimental sessions were scheduled.

9.2.8 Procedure

Daily pain diary entries and logging menstrual cycle parameters took place for a minimum of 1 full cycle prior to experimental testing sessions. Two identical experimental sessions were conducted to compare cold pressor pain responses in the follicular and luteal phases of the menstrual cycle. Experimental sessions were counterbalanced across menstrual phase (ABBA) to protect against phase order effects. On this basis, half of the participants were tested either side of ovulation within a single menstrual cycle (follicular-luteal), whereas the other half were tested either side of menstruation across two menstrual cycles (luteal-follicular).

Each experimental session comprised a single cold pressor task, assessment of mood state and measurement of resting blood pressure. Mood state was assessed at the beginning of each experimental session. As in Experiment 3, measurement of blood pressure was made after participants had been seated for approximately 15 minutes and

before any discussion of the cold pressor task. Blood pressure was measured three times at 1-minute intervals, and mean values calculated from these. Participants were debriefed and paid £5.00 upon completion of all parts of the experiment.

9.2.9 Statistical analysis

Paired-samples t-tests were used to evaluate the effects of menstrual phase on experimental pain indices, diary pain indices, mood states, blood pressure and state catastrophizing. Correlational analysis was used to examine relationships between cold pressor pain indices and all other variables assessed. Correlations were also applied to examine potential associations between diary pain indices and focus on pain.

9.3 Results

9.3.1 Data screening

Skewed data distributions were normalised by transformation. The following variables were logarithmically transformed: pain threshold, pain tolerance, pain recovery. Affective pain, emotional states and diary pain indices were square root transformed.

9.3.2 Tests of difference between menstrual phases

9.3.2.1 Diary pain

Self-report sensory pain, affective pain and pain intensity scores from the pain diaries were used to compare the extent of pain experienced during different menstrual cycle phases. Using the same menstrual phase delineation as in the experimental sessions, means were calculated for diary pain indices in follicular (days 4-9) and luteal (days 19-24) phases.[2] See Table 9.1 for means and standard deviations.

[2] The same range of days as used for experimental sessions were used to obtain follicular and luteal measures of diary pain indices. However, it should be noted that since the pain diaries were completed in the menstrual cycle prior to that in which the experimental sessions took place, pain diary menstrual phases were not confirmed by ovulation detection tests.

Table 9.1: Mean diary pain indices in follicular and luteal menstrual phases, Experiment 4 (standard deviations in parentheses).

	Follicular phase	Luteal phase
Pain Diary		
Sensory pain	1.55 (1.93)	.86 (1.13)
Affective pain	.26 (.49)	.21 (.37)
Pain intensity	9.31 (10.26)	6.24 (7.79)

* p<.05 **p<.01

Paired samples t-tests were conducted on diary pain indices (sensory pain, affective pain, pain intensity) in the follicular and luteal phases. No significant differences were found between diary pain indices in the follicular and luteal phases.

9.3.2.2 Emotion

Means and standard deviations of emotional states subscale scores are presented in Table 9.2. Counter to predictions, all mean negative emotion scores were higher in the follicular than the luteal phase. By contrast, mean scores on the single positive emotion subscale (Vigour-Activity) were higher in the luteal phase.

Table 9.2: Mean emotional states scores in follicular and luteal menstrual phases, Experiment 4 (standard deviations in parentheses).

Emotional state	Follicular phase	Luteal phase
POMS		
Anger-Hostility	12.71 (11.23)	9.25 (7.03)
Confusion-Bewilderment*	10.54 (5.09)	7.96 (4.55)
Depression-Dejection	10.21 (9.56)	7.54 (7.51)
Fatigue-Inertia	8.93 (5.56)	8.26 (6.12)
Tension-Anxiety	14.50 (8.05)	11.25 (7.31)
Vigour-Activity	16.04 (6.91)	18.41 (5.69)

* p<.05 **p<.01

However, paired-samples t-tests revealed that the differences in emotional states between the follicular and luteal phases were significant on only one subscale; 'Confusion-Bewilderment', which was higher in the follicular than in the luteal phase (t (27) = 2.94, $p<.$ 01). There was a trend towards significance for 'Tension-Anxiety' (t (27) =-1.93, $p<.07$, 2-tailed) which was also higher in the follicular than the luteal phase.

9.3.2.3 Blood pressure

Mean resting systolic, diastolic and mean arterial blood pressure values in the follicular and luteal phases are presented in Table 9.3. Mean arterial pressure was calculated using the standard formula MAP = Pd + (Ps-Pd)/3, where Pd and Ps represent diastolic and systolic pressure respectively.

Table 9.3: Mean blood pressure values in follicular and luteal menstrual phases, Experiment 4, (standard deviations in parentheses).

Blood pressure index	Follicular phase	Luteal phase
Systolic	115.76 (13.00)	112.55 (14.01)
Diastolic	73.65 (10.89)	73.36 (12.53)
Mean arterial	87.69 (10.64)	86.42 (12.05)

* p<.05 **p<.01

Paired-samples t-tests revealed no significant differences between follicular and luteal phases for any blood pressure index.

9.3.2.4 Experimental pain

Means and standard deviations of behavioural and self-report pain indices obtained during the experimental CPT sessions of Experiment 4 are presented in Table 9.4. As can be seen, pain threshold and pain tolerance were both higher in the follicular than the luteal phase. However, paired-samples t-tests revealed no significant differences in pain threshold, pain tolerance or pain recovery times between the menstrual phases. Large standard deviations show that there was a high level of variation in behavioural pain responses in both phases.

Table 9.4: Mean experimental pain indices in follicular and luteal menstrual phases, Experiment 4 (standard deviations in parentheses).

Pain index	Follicular phase	Luteal phase
Threshold	19.70 (23.24)	17.75 (12.17)
Tolerance	115.72 (121.91)	109.83 (113.56)
Recovery	90.18 (103.65)	96.32 (141.95)
Intensity	59.55 (21.83)	60.96 (21.86)
Sensory	11.04 (6.67)	11.36 (5.98)
Affective	1.86 (2.98)	1.64 (2.67)

* p<.05 **p<.01

Paired samples t-tests were also conducted on pain intensity, sensory pain and affective pain scores (derived from the SF-MPQ) in both menstrual phases. No significant differences were found between self-report pain indices in the follicular and luteal phases.

9.3.2.5 State catastrophizing

Means and standard deviations of state catastrophizing during cold pressor were calculated for follicular and luteal phases (follicular mean = 11.86, SD = 9.59, luteal mean = 12.36, SD = 9.61). A paired-samples t-test showed that state catastrophizing did not differ significantly between phases.

9.3.3 Correlational analysis

Pearson's correlations were conducted to (i) investigate whether habitual cognition about pain (focus on pain) was related to the level of pain experienced throughout menstrual cycle phases (diary pain indices) and (ii) investigate potential relationships between questionnaire data (emotional states, focus on pain, state catastrophizing), blood pressure and experimental pain indices in the follicular and luteal phases. See Tables 9.5-9.8.

9.3.3.1 Correlations between focus on pain scores and diary pain indices

Table 9.5: Correlations between focus on pain scores and diary pain indices in follicular menstrual phase (n=28), Experiment 4.

	Sensory Pain	Affective Pain	Pain Intensity
FOPS 24			
Total	-.109	-.095	.023
Pain Dominance	.105	-.039	.210
Fear & Worry	.216	-.084	.274
Pain Control	**.431***	.067	.237

* p< .05, ** p< .01

As can be seen from Table 9.5, the only significant relationship found between focus on pain and diary pain indices in the follicular phase was between sensory pain and the Pain Control subscale, which reflects self-confident cognition about pain. Since there is no obvious explanation for this positive association, it may be spurious.

Table 9.6: Correlations between focus on pain scores and diary pain indices in luteal menstrual phase (n=28), Experiment 4.

	Sensory pain	Affective Pain	Pain Intensity
FOPS 24			
Total	.040	.173	.158
Pain Dominance	.099	-.027	.179
Fear & Worry	.120	-.104	.149
Pain Control	-.096	-.157	-.177

* p< .05, ** p< .01

No significant correlations were found between focus on pain scores and diary pain indices in the luteal phase.

9.3.3.2 Correlations between questionnaire scores, blood pressure and experimental pain indices

As can be seen from Table 9.7, a number of modest correlations were found between questionnaire data and experimental pain indices in the follicular phase. State catastrophizing was negatively correlated with pain threshold and pain tolerance, and positively correlated with affective pain and pain intensity, indicating that greater catastrophizing during cold pressor exposure was associated with greater pain sensitivity and higher self-reported pain. There were also negative correlations between pain intensity and two negative mood state subscales (Confusion-Bewilderment, Depression-Dejection) but since there is no obvious explanation for these apparently inverse but modest relationships, they may be spurious especially in light of the small sample size (N=28). An inverse relationship was found between diastolic blood pressure and affective pain, which concurs with previous research showing lowered self-report pain in conjunction with elevated blood pressure.

As can be seen from Table 9.8, there were fewer correlations between questionnaire data and pain indices in the luteal phase. Similar to the follicular phase, state catastrophizing was negatively correlated with pain threshold and pain tolerance, and positively correlated with affective pain and pain intensity. The associations between catastrophizing and these self-report pain indices were stronger in the luteal than the follicular phase. A positive correlation was found between mean arterial pressure (MAP) and pain tolerance in the luteal phase, indicating that higher blood pressure was associated with greater tolerance for cold pressor pain. No other significant relationships were found in the luteal phase.

Table 9.7: Correlations between experimental pain indices, questionnaire data, and blood pressure in follicular menstrual phase (n=28), Experiment 4.

	Pain Thresh	Pain Tol	Pain Recov	Sens Pain	Affect Pain	Pain Intens
FOPS 24						
Total	-.121	-.032	-.150	.006	.018	.151
Pain Dominance	.046	-.170	.111	.095	-.138	.122
Fear & Worry	.207	-.077	.109	.262	-.031	.120
Pain Control	.063	.001	-.065	.106	.113	-.030
POMS						
Tension-Anxiety	.055	.241	.367	-.324	-.049	-.358
Confusion-Bewilderment	-.247	.200	**.409***	-.108	-.141	**-.419***
Depression-Dejection	-.243	.167	.308	-.105	.036	**-.398***
Anger-Hostility	-.168	.067	.345	-.024	.174	-.246
Vigour-Activity	.069	.077	-.076	.266	**.440***	.185
Fatigue-Inertia	-.150	-.101	.261	-.061	-.100	-.167
PCS S	**-.471***	**-.519****	-.308	-.024	**.474***	**.493****
Blood pressure						
Systolic	.222	.205	.133	-.237	-.142	-.124
Diastolic	.077	.107	.045	-.120	**-.432***	-.348
Mean arterial	-.140	.068	-.041	.301	-.062	-.113

Key: FOPS 24 = Focus on Pain Scale, POMS = Profile of Mood States, PCS S = Pain Catastrophizing Scale – state, * p< .05, ** p< .01

Table 9.8: Correlations between questionnaire scores, blood pressure and experimental pain indices in luteal menstrual phase (n=28), Experiment 4.

	Pain Thresh	Pain Tol	Pain Recov	Sens Pain	Affect Pain	Pain Intens
FOPS 24						
Total	-.271	-.108	-.010	.189	.252	.159
Pain Dominance	-.140	-.229	-.094	-.051	.000	-.007
Fear & Worry	.023	-.213	-.217	.130	.129	-.062
Pain Control	.140	-.121	-.257	.070	.000	-.145
POMS						
Tension-Anxiety	-.020	.032	-.003	-.109	.187	.307
Confusion-Bewilderment	.084	.314	.230	-.308	.038	.085
Depression-Dejection	.138	.220	.087	.016	.070	.123
Anger-Hostility	-.056	-.089	-.107	.078	.288	.229
Vigour-Activity	-.211	-.077	-.070	-.173	.135	-.209
Fatigue-Inertia	-.185	-.268	-.328	-.057	-.343	.001
PCS S	**-.454***	**-.544****	-.221	.324	**.734****	**.586****
Blood pressure						
Systolic	.184	.225	.178	.040	-.019	-.130
Diastolic	.224	.347	.270	-.028	-.012	-.108
Mean arterial	.359	**.541****	.274	-.140	-.150	-.287

Key: FOPS 24 = Focus on Pain Scale, POMS = Profile of Mood States, PCS S = Pain Catastrophizing Scale – state, * p< .05, ** p< .01

9.4 Discussion

The primary objective of Experiment 4 was to examine the effects of menstrual cycle on female pain responses. This was achieved using a combination of pain diaries and cold pressor pain induction across menstrual phase. Although consensus in the research literature regarding the effects of menstrual phase on pain responses and cardiovascular activity has remained elusive, predictions were generated here in light of the agglomerated findings of contemporary reviews and meta-analyses (e.g., Riley et al., 1999; Fillingim & Ness, 2000). On this basis, lower pain report and pain sensitivity were expected in the follicular phase compared to the luteal phase. Secondary aims were to investigate the impact of menstrual cycle on resting blood pressure and emotional states. Blood pressure was expected to vary across menstrual cycle, and to

correlate with pain threshold and/or tolerance. More negative emotion and state catastrophizing in the luteal phase than the follicular phase were also anticipated.

No differences were found between cold pressor pain responses in the follicular and luteal phases. Hence, the menstrual cycle effects on experimental pain perception reported in previous studies were not replicated in Experiment 4, despite methodological improvements on some of these. Furthermore, there was no evidence of alterations in the experience of pain across the menstrual cycle in the pain diaries participants kept throughout a complete ovarian cycle.

Experiment 4 has provided scant evidence of menstrual cycle impact on emotional states; only one negative emotion subscale (Confusion-Bewilderment) differed between phases and the direction of difference was unexpected, i.e., greater in follicular than luteal phase. This is inconsistent with the increase of negative emotion which is a widely-reported feature of the late luteal phase of the menstrual cycle. The extent to which women reported they had catastrophized about pain during cold pressor exposure was related to the level of pain they reported and inversely related to their tolerance for it in both phases. However, there was no significant difference in catastrophizing scores between the follicular and luteal phases, which suggests that despite adaptation of the scale to assess state catastrophizing (specifically about cold pressor exposure) this variable may be essentially traitlike.

Since no differences in pain indices or blood pressure were found between the follicular and luteal phases in Experiment 4, this detracts somewhat from the possibility that blood-pressure related hypoalgesia occurs in women. Associations were found between BP and pain responses in both menstrual phases, but these involved different indices of blood pressure and pain in each phase. An association between mean arterial pressure (MAP) and pain tolerance was found only in the luteal phase of Experiment 4, whereas the only blood pressure-pain index relationship found in the follicular phase was between diastolic blood pressure (DBP) and affective pain. These findings both concur with and differ from those of Pfleeger et al. (1997), who reported correlations between BP and pain tolerance in both follicular and luteal menstrual phases, but no associations between BP and self-report pain.

There are a number of possible explanations for the findings of Experiment 4. They may signify that some of menstrual cycle effects reported in previous research are not robust and replicable, and rather than reflecting hormonal effects may instead be artefacts of individual differences or of inaccurate menstrual phase definition. For example, potential confounding of menstrual phase comparisons may arise when phases are calendar calculated based on the convention of a 28-day average cycle. Substantial variability in cycle length and ovulation timing was found in Experiment 4, which reiterates the importance of ascertaining ovulation by an objective test (assay of urine or blood) to ensure that testing takes place in the desired phases.

Another possibility is that some aspect of the methodology in Experiment 4 masked menstrual phase differences in cold pressor pain responses. For half of the sample both test sessions took place within a single menstrual cycle (i.e., follicular phase followed by luteal phase) whereas for the remaining half test sessions spanned two menstrual cycles (i.e., luteal followed by follicular). While this protocol was chosen on theoretical grounds (counterbalancing test phase reduces the risk of order effects), and has been used by other researchers (e.g., Pfleeger et al., 1997) this could potentially have confounded menstrual phase differences.

In addition to variability in the timing of hormonal shifts across the ovarian cycle, there are ultradian rhythms which exert pulsatile changes in circulating levels of hormones. Together these can create large variations between individual cycles, even successive cycles in the same woman (Ferin et al., 1993). To investigate this possibility, statistical analyses of experimental pain indices, blood pressure and state catastrophizing across menstrual phase were repeated controlling for testing within one or two cycles (test mode). Only one variable (affective pain) was affected by test mode, and was rated higher by those tested within a single cycle than by those tested across two menstrual cycles. However, there was neither a phase difference in affective pain nor an interaction between phase and test mode, so this apparent effect of test mode may be an artefact of variability in the affective pain data.

9.5 Summary

Experiment 4 provided no evidence of menstrual phase effects on pain responses and little indication that emotional states were affected by menstrual phase. Although some

association was apparent between blood pressure and pain responses within menstrual phases, blood pressure was not found to alter across the menstrual cycle. Overall, the predictions made in Experiment 4 were not supported and the predominantly null findings which have emerged concur with some previous studies but are inconsistent with many others. However, many of the menstrual cycle effects on experimental pain sensitivity previously reported were found with other types of noxious stimuli, such as ischemic pain (e.g., Fillingim et al., 1997; Pfleeger et al. 1997) rather than cold pressor pain. It is also notable that while some previous studies have found menstrual phase-related alterations in cold pressor pain (e.g.,Hapidou & Cantanzero, 1988) others have failed to do so (e.g., Amodei & Nelson-Gray, 1989).

Since relatively little research has directly addressed the impact of menstrual phase on cold pressor pain specifically, it was important to ascertain whether the pattern of findings in Experiment 4 was aberrant. Accordingly, a semi-replication was planned incorporating some specific changes, but essentially keeping to the same methodology. In light of the possible confounding effects of testing across menstrual cycles, experimental sessions in Experiment 5 were to be scheduled in the follicular and luteal phases of a single, confirmed ovulatory cycle.

Chapter 10

Experiment 5
Menstrual Cycle, Coping Strategies and Cold Pressor Pain

10.1 Introduction

Experiment 4 indicated that female pain responses, blood pressure and emotional states were not affected by changing hormone levels across the menstrual cycle. Since these findings conflict with previous research (e.g., Pfleeger et al., 1997) it is important to ascertain whether they are replicable. Although the inclusion of testing across as well as within menstrual cycles seemed to have little impact in Experiment 4, this could only be fully assessed in an experimental replication with this potential confound removed. Accordingly, Experiment 5 was a follow-up retaining the same basic procedural methodology as Experiment 4 but testing all participants within one ovarian cycle. The main aim of Experiment 5 was therefore to ascertain whether female cold pressor pain responses would vary between the follicular and luteal menstrual phases of a single, confirmed ovulatory cycle.

In addition, this experiment also sought to investigate the effects of attentional direction on female pain responses during each of these phases. Some previous research has indicated that men and women in pain may be differentially affected by avoidant and non-avoidant coping strategies, and that it is unclear which approach is best for women (Keogh et al., 2000; Experiment 2 in this thesis). It is possible that menstrual phase is a confounding factor in evaluations of the effectiveness of coping strategies for pain in females of reproductive age. Because alterations in emotional state and aspects of cognitive functioning (such as ability to concentrate) across the menstrual cycle have previously been documented (Parry & Haynes, 2000) women could find different types of attentional coping more or less viable and/or effective in different phases of the cycle. In light of this possibility, Experiment 5 was designed to examine the effects of avoidant and non-avoidant coping instructions on cold pressor pain responses in the follicular and luteal phases of the menstrual cycle.

10.2 Method

10.2.1 Design

A repeated-measures design was employed with two within-groups factors: menstrual phase (follicular vs. luteal) and coping condition (avoidant vs. non-avoidant). The primary dependent variables were pain threshold, pain tolerance, pain recovery, pain intensity, sensory pain and affective pain. As in Experiment 4, additional dependent

variables were diary pain indices, blood pressure, emotional state, focus on pain and pain catastrophizing.

10.2.2 Participants

48 adult females, including some students and university staff, were recruited in south-east London via advertisements in local press and on local radio as well as on noticeboards at several higher education institutions. The inclusion and exclusion criteria employed were the same as in Experiment 4. 18 participants were lost from the experiment at various stages. The resultant sample comprised 30 healthy females with an age range of 21-45 years (mean = 31.47, SD = 7.22).

10.2.3 Pain diaries

As in Experiment 4, pain diaries were kept for a minimum of one full menstrual cycle preceding the experimental test sessions to provide self-report indices of pain (pain intensity, sensory pain and affective pain) experienced during the follicular and luteal phases. The structure and modus operandi of pain diaries was as described in Chapter 9. As before, participants recorded and reported menstrual cycle parameters (i.e., start and end dates of menstruation) for a minimum of two full cycles before the experimental test sessions commenced.

10.2.4 Identification of menstrual phase

As before, the date of ovulation for each participant was estimated by calendar counting using individual menstrual cycle history and confirmed using Clearplan hormone detection tests on first morning urine samples. All ovulation tests were self-administered by participants and all test results were researcher-verified. Only data from those participants whose ovulation was confirmed were included in the analysis. As in Experiment 4, the follicular phase was operationally defined as days 4-9 inclusive (counting the onset of menstruation as Day 1) and luteal phase was operationally defined as 5-10 days after ovulation. On this basis luteal phase testing took place on days 19-26 inclusive for all participants.

10.2.5 Blood pressure measurement

As in Experiment 4, resting blood pressure was measured before cold pressor exposure using a MARS MS-700 automatic oscillometric digital blood pressure monitor.

10.2.6 Pain induction

10.2.6.1 Cold pressor apparatus and task

The cold pressor apparatus and task were the same as in previous experiments.

10.2.6.2 Pain ratings

As before, pain threshold, pain tolerance and pain recovery measured in seconds, were obtained directly from the cold pressor task. Self-report measures of pain intensity, sensory pain and affective pain were obtained with the Short-Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) completed immediately after each cold pressor exposure.

10.2.7 Additional questionnaires

As in Experiment 4, the Profile of Mood States scales (POMS; McNair, Lorr & Droppelman, 1992) and the Focus on Pain Scale (FOPS 24) were administered. The Pain Catastrophizing Scale (PCS; Sullivan, Bishop & Pivik, 1995) was administered in its original (trait) form and all subscale scores used. A simple coping manipulation check questionnaire was administered after each cold pressor exposure. Respondents indicated the extent to which they had been able to adhere to the coping instructions on a 5-point Likert-type scale, ranging from 0 (*not at all*) to 4 (*completely*).

10.2.8 Coping manipulation

The non-avoidant coping strategy used was the same as in Experiments 2 and 3; the exact coping instructions given appear in Chapter 5. The same open-ended avoidant coping strategy was used as in Experiment 3; the exact instructions can be found in Chapter 7. Coping conditions were fully counterbalanced (ABBA) across menstrual phase (FL). On this basis, half the sample were randomly allocated to the use of avoidant coping first and non-avoidant coping second in the follicular phase experimental session and to the reverse order in the luteal phase session. The other half of the sample used non-avoidant coping first and avoidant coping second in the follicular phase experimental session and the reverse order in the luteal phase session.

10.2.9 Procedure

As in Experiment 4, two identical experimental sessions were conducted to compare cold pressor pain responses, emotional states and blood pressure in the follicular and

luteal phases of the menstrual cycle. Both sessions took place within a single menstrual cycle for all participants. Emotional states and blood pressure were assessed at the beginning of each experimental session, before any discussion of the cold pressor task or coping instructions. In each experimental session, participants underwent two cold pressor exposures using contrasting attentional coping strategies, with a 15-minute intertrial interval. At the end of the experimental session in the follicular phase ovulation detection tests were issued with full written instructions and an assigned date on which to commence testing. Start dates for ovulation testing were calculated on the basis of individual menstrual histories, with the first ovulation test scheduled for 17 days prior to the likely date of next menses. All participants made a total of four visits to the laboratory, and were reimbursed £15.00 travel expenses at the end of the final experimental session. Test sessions were approximately 1 hour long.

10.2.10 Statistical analysis

As in Experiment 4, a series of paired-samples t-tests were conducted to evaluate the effects of menstrual phase on diary pain indices, emotional states and blood pressure. Repeated measures ANOVAs were used to evaluate the effects of menstrual phase and coping instructions on experimental pain indices. Correlational analysis was used to examine relationships between experimental pain indices and all other variables assessed. Correlations were also applied to examine potential associations between diary pain indices, focus on pain and pain catastrophizing.

10.3 Results

10.3.1 Data screening

Where applicable, skewed data distributions were again normalised by transformation. The following variables were log transformed: pain threshold, pain tolerance and pain recovery. Affective pain, mood states and diary pain indices were square root transformed.

10.3.2 Tests of difference between menstrual phases

10.3.2.1 Diary pain

Sensory pain, affective pain and pain intensity scores from the pain diaries were used to compare the extent of pain experienced during different menstrual cycle phases. The same menstrual phase delineation was used as in the experimental sessions, means were

calculated for pain indices in follicular (days 4-9) and luteal (days 19-24) phases. See Table 10.1 for means and standard deviations.

Table 10.1: Mean diary pain indices in follicular and luteal menstrual phases, Experiment 5 (standard deviations in parentheses).

		Follicular phase	Luteal phase
Diary pain			
	Sensory	.87 (1.35)	1.38 (1.78)
	Affective	.29 (.41)	.47 (1.07)
	Intensity	7.03 (8.26)	4.45 (5.05)

* p<.05 **p<.01

As in Experiment 4, a series of paired samples t-tests were conducted to compare diary pain indices (sensory pain, affective pain, pain intensity) measured in the follicular and luteal menstrual phases. No significant differences were found in self-reported pain (diary pain indices) from the two menstrual phases.

10.3.2.2 Emotion

To ascertain whether emotional states had altered across menstrual phase, paired-samples t-tests were applied to POMS subscale scores in follicular and luteal phases. See Table 10.2 for means and standard deviations.

Table 10.2: Mean emotion scores in follicular and luteal menstrual phases, Experiment 5 (standard deviations in parentheses).

Emotional state	Follicular phase	Luteal phase
POMS		
Anger-Hostility	10.9 (10.38)	9.9 (9.19)
Confusion-Bewilderment	8.7 (5.97)	8.5 (6.76)
Depression-Dejection*	12.9 (13.09)	10.6 (12.78)
Fatigue-Inertia	10.1 (7.13)	9.9 (7.61)
Tension-Anxiety	9.8 (6.73)	9.8 (7.69)
Vigour-Activity	16.1 (6.73)	15.6 (6.70)

* p<.05 **p<.01

Only one emotion subscale differed significantly between menstrual phases: Depression-Dejection was reported as higher in the follicular phase than the luteal phase ($t(29) = 2.22$, $p<.05$). This difference was not in the expected direction.

10.3.2.3 Blood pressure

Mean resting systolic, diastolic and mean arterial blood pressure values were calculated from the average measurements taken in the follicular and luteal phases (See Table 10.3).

Table 10.3: Mean blood pressure values in follicular and luteal menstrual phases, Experiment 5 (standard deviations in parentheses).

Blood pressure index	Follicular Phase	Luteal Phase
Systolic	117.12 (12.16)	117.71 (11.77)
Diastolic	76.01 (9.53)	77.31 (12.68)
Mean arterial	89.71 (9.45)	90.78 (11.90)

* $p<.05$ **$p<.01$

As can be seen from Table 10.3, mean blood pressure values in the two menstrual phases were very similar. Paired-samples t-tests on systolic, diastolic and mean arterial pressure confirmed that there were no significant differences in blood pressure readings.

10.3.2.4 Experimental pain

Means and standard deviations of behavioural and self-report pain indices obtained with avoidant and non-avoidant coping during the experimental sessions of Experiment 5 are presented in Table 10.4.

A series of repeated-measures ANOVAs were conducted on behavioural pain scores (obtained directly from cold pressor). The within-groups factors were phase (follicular vs luteal) and coping condition (avoidant vs non-avoidant). No main effects of menstrual phase or coping condition, or interaction effects between these factors were found for pain threshold, pain tolerance or pain recovery. This indicates that these pain indices did not differ across menstrual phase or coping condition.

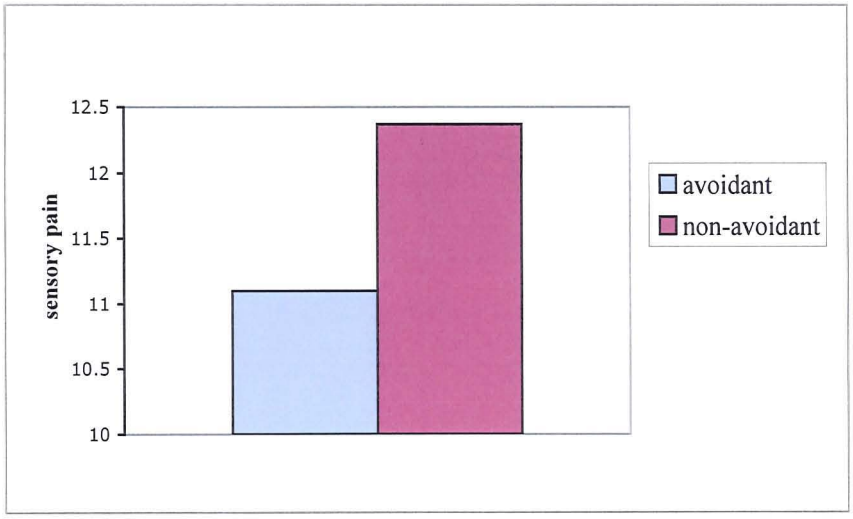
Table 10.4: Mean experimental pain indices with avoidant and non-avoidant coping in follicular and luteal menstrual phases (standard deviations in parentheses)

Pain Index	Follicular phase		Luteal phase	
	Non-avoidant	Avoidant	Non-avoidant	Avoidant
Threshold	26.99(23.71)	32.63 (33.69)	31.85 (34.08)	35.45 (28.86)
Tolerance	101.14 (92.54)	111.93 (100.59)	106.46 (99.25)	113.78 (102.93)
Recovery	63.56 (55.98)	47.22 (43.58)	59.90 (55.98)	57.20 (52.01)
Intensity	66.50 (28.01)	60.72 (26.44)	65.32 (24.41)	64.03 (23.13)
Sensory*	12.70 (6.63)	11.23 (5.47)	12.03 (6.26)	10.97 (5.73)
Affective	2.63 (2.76)	2.03 (2.31)	1.87 (2.29)	1.47 (1.43)

* p<.05 **p<.01

Similar analyses were also conducted on self-report sensory pain, affective pain and pain intensity (derived from the SF-MPQ). A main effect of coping condition was found for sensory pain (F (1,29) = 6.31, p<.05; see Fig 10.1), with higher pain reported in the non-avoidant coping condition (mean = 12.37, SD = 6.03) than in the avoidant condition (mean = 11.10, SD = 5.00). No significant main effect of phase or interaction effect was found for sensory pain.

Figure 10.1: Mean sensory pain ratings in avoidant and non-avoidant coping conditions, Experiment 5



A near-significant main effect of coping condition was found for pain intensity, which was higher in the non-avoidant (mean = 65.91, SD = 25.37) than in the avoidant (mean = 62.37, SD = 22.87) condition (F (1,29) = 3.89, p<.059). There was no main effect of phase or interaction effect on pain intensity. No significant effects were found for affective pain.

10.3.2.5 Coping adherence

On the basis of the coping manipulation check questionnaire, no participant reported that they had been completely unable to adhere to either type of coping instructions. A repeated-measures ANOVA of self-report ratings of ability to adhere to coping instructions was conducted with menstrual phase (follicular vs. luteal) and coping condition (avoidant vs. non-avoidant) as within-groups factors. A significant main effect of menstrual phase was found (F (1,29) = 6.61, p<.05) with higher ability to adhere to coping instructions reported in the luteal phase (mean = 2.80, SD = .65) than in the follicular phase (mean = 2.42 , SD = .75). A main effect of coping condition was also found (F (1,29) = 5.04, p<.05) with higher ability to adhere to instructions reported in the non-avoidant condition (mean = 2.80, SD = .62) than in the avoidant condition (mean = 2.42 , SD = .84). No significant interaction was found.

10.3.3 Correlational analyses

A series of Pearson's correlations were performed to investigate potential relationships between: (i) trait questionnaire measures and diary pain indices (ii) trait questionnaire scores and experimental pain indices in avoidant and non-avoidant coping conditions (iii) emotional states scores, blood pressure values and experimental pain indices in avoidant and non-avoidant coping conditions.

Separate analyses were conducted on follicular and luteal phase data. Tables 10.5 – 10.9 show the significant associations found between these sets of variables. Means and standard deviations of trait variables are presented within Table 10.5. For clarity, correlations of pain indices in avoidant and non-avoidant coping conditions are subdivided within tables.

(i) As can be seen from Table 10.5, the only significant relationships found between trait measures and diary pain indices were with the Magnification subscale of catastrophizing and were restricted to the luteal phase. This suggests that a greater

tendency to magnify painful feelings is associated with more self-report pain during the luteal stage of the menstrual cycle.

(ii) As can be seen from Table 10.6, in the follicular phase, higher scores on Magnification (subscale of catastrophizing) were associated with higher affective pain scores in both coping conditions. The other catastrophizing subscale scores (Total, Rumination and Helplessness) were negatively associated with pain tolerance but only in the avoidant coping condition. Similarly, focus on pain scores (Total score, Pain Fear & Worry subscale score) were negatively associated with pain threshold in the avoidant coping condition only. This might indicate that the greater tendency to attend to pain cognitively and emotionally, which high scores on these trait measures indicates, hampers the effectiveness of avoidant coping somewhat.

Table 10.7 shows that there were fewer significant associations between trait measures and experimental session pain indices in the luteal phase. Magnification was again found to be positively associated with affective pain but only in the non-avoidant coping condition. Catastrophizing Total and Rumination were negatively associated with pain threshold in the non-avoidant coping condition only.

(iii) As is clear from Tables 10.8 and 10.9, there were very few significant associations between emotion scores, blood pressure values and experimental pain indices in either menstrual phase.

10.3.3.1 (i) Correlations between trait questionnaire scores and diary pain indices

Table 10.5: Correlations between trait questionnaire scores and diary pain indices in follicular and luteal menstrual phases (n=30), Experiment 5.

			Sensory Pain	Affective Pain	Pain Intensity
	Trait measure	**Mean (SD)**			
DIARY	**PCS T**	19.50 (10.15)	.129	.003	.248
FOLLICULAR	**PCS R**	7.97 (4.41)	.158	-.064	.324
PHASE	**PCS M**	3.90 (2.49)	.194	.085	.335
	PCS H	7.63 (4.63)	.029	.021	.056
	FOPS-T	45.27 (19.02)	.288	.192	.301
	FOPS-PD	19.53 (11.49)	.310	.192	.299
	FOPS-FW	17.53 (9.75)	.281	.226	.307
	FOPS-PC	8.20 (4.31)	-.189	-.176	-.162
DIARY	**PCS T**		.139	.297	.145
LUTEAL	**PCS R**		.150	.193	.114
PHASE	**PCS M**		**.420***	**.420***	**.574****
	PCS H		-.064	.242	-.099
	FOPS-T		.096	.132	.058
	FOPS-PD		-.034	-.007	-.123
	FOPS-FW		.185	.194	.182
	FOPS-PC		.096	.160	.175

Key: FOPS-T = Focus on pain total, FOPS-PD = Pain Dominance, FOPS-FW = Pain Fear & Worry, FOPS-PC = Pain control, PCS T = Pain catastrophizing total, PCS R = Rumination, PCS M = Magnification, PCS H = Helplessness, * p< .05, ** p< .01

10.3.3.2 (ii) Correlations between trait questionnaire scores and experimental pain indices

Table 10.6: Follicular phase correlations between trait questionnaire scores and experimental pain responses in avoidant and non-avoidant coping conditions (n=30), Experiment 5.

	NON–AVOIDANT COPING					
	Pain threshold	**Pain tolerance**	**Pain Recovery**	**Sensory pain**	**Affective Pain**	**Pain intensity**
PCS T	-.260	-.209	-.073	.017	.182	.051
PCS R	-.331	-.291	-.051	.001	.076	.109
PCS M	-.184	-.023	.130	-.012	**.442***	.101
PCS H	-.155	-.169	-.181	.043	.087	-.046
FOPS-T	-.102	-.105	-.173	.091	.031	-.200
FOPS-PD	-.023	-.177	-.285	.079	-.052	-.118
FOPS-FW	-.130	-.114	-.098	.191	.146	-.181
FOPS-PC	-.097	.267	.219	-.240	-.055	-.157
	AVOIDANT COPING					
	Pain threshold	**Pain tolerance**	**Pain Recovery**	**Sensory pain**	**Affective Pain**	**Pain intensity**
PCS T	-.278	**-.431***	-.267	.051	.284	.055
PCS R	-.325	**-.465****	-.291	.007	.252	.099
PCS M	-.138	-.212	.055	.098	**.367***	.189
PCS H	-.225	**-.387***	-.337	.051	.184	-.075
FOPS-T	**-.412***	-.263	-.152	.038	.168	-.163
FOPS-PD	-.322	-.234	-.335	.025	.183	-.114
FOPS-FW	**-.478****	-.263	.002	.143	.186	-.146
FOPS-PC	.119	.059	.218	-.220	-.168	-.082

Key: FOPS-T = Focus on pain total, FOPS-PD = Pain Dominance, FOPS-FW = Pain Fear & Worry, FOPS-PC = Pain control, PCS T = Pain catastrophizing total, PCS R = Rumination, PCS M = Magnification, PCS H = Helplessness, * p< .05, ** p< .01

Table 10.7: Luteal phase correlations between trait questionnaire scores and experimental pain responses in avoidant and non-avoidant coping conditions (n=30), Experiment 5.

	NON–AVOIDANT COPING					
	Pain threshold	**Pain tolerance**	**Pain Recovery**	**Sensory pain**	**Affective pain**	**Pain intensity**
PCS T	-.287	-.121	-.056	.172	.182	-.096
PCS R	-.306	-.130	.007	.218	.076	-.030
PCS M	-.275	-.077	.022	.108	**.442***	-.074
PCS H	-.190	-.100	-.142	.112	.087	-.141
FOPS-T	-.137	-.003	.235	.006	.031	-.217
FOPS-PD	-.179	-.032	.035	.003	-.052	-.148
FOPS-FW	-.112	-.043	.269	.099	.146	-.149
FOPS-PC	.125	.168	.335	-.203	-.055	-.228
	AVOIDANT COPING					
	Pain threshold	**Pain tolerance**	**Pain Recovery**	**Sensory pain**	**Affective Pain**	**Pain intensity**
PCS T	**-.418***	-.150	-.281	.249	.170	-.022
PCS R	**-.453***	-.172	-.194	.263	.141	.008
PCS M	-.361	-.031	-.155	.217	.364	-.042
PCS H	-.290	-.148	-.348	.179	.041	-.034
FOPS-T	-.256	-.053	.023	.029	-.009	-.120
FOPS-PD	-.212	-.082	-.049	-.009	-.118	-.138
FOPS-FW	-.272	-.083	-.030	.137	.060	-.030
FOPS-PC	.052	.174	.098	-.156	.138	-.094

Key: FOPS-T = Focus on pain total, FOPS-PD = Pain Dominance, FOPS-FW = Pain Fear & Worry, FOPS-PC = Pain control, PCS T = Pain catastrophizing total, PCS R = Rumination, PCS M = Magnification, PCS H = Helplessness, * p< .05, ** p< .01

10.3.3.3 (iii) Correlations between blood pressure, emotional states and experimental pain indices

Table 10.8: Follicular phase correlations between emotion scores, blood pressure values and experimental pain indices in avoidant and non-avoidant coping conditions (n=30), Experiment 5.

	NON-AVOIDANT COPING					
	Pain Threshold	**Pain Tolerance**	**Pain Recovery**	**Sensory Pain**	**Affective Pain**	**Pain Intensity**
POMS T/A	.238	.252	.257	-.118	-.170	-.347
POMS C/B	.228	.317	**.484***	.098	.109	.030
POMS D/D	.045	.228	.187	-.020	-.116	.043
POMS A/H	.045	.151	.250	.078	.092	.218
POMS V/A	.010	-.008	-.070	-.032	.074	.026
POMS F/I	.124	.189	.217	-.147	-.148	-.199
SBP	-.066	-.136	.122	.056	.156	.181
DBP	-.213	-.102	.045	.386	.323	**.414***
MAP	-.171	-.010	.083	.284	.284	.356
	AVOIDANT COPING					
	Pain Threshold	**Pain Tolerance**	**Pain Recovery**	**Sensory Pain**	**Affective Pain**	**Pain Intensity**
POMS T/A	-.120	.042	-.063	-.030	.225	-.346
POMS C/B	.196	.222	.292	.174	**.422***	-.026
POMS D/D	.061	.035	.013	.028	.261	.012
POMS A/H	.199	.102	.176	.081	.302	.194
POMS V/A	.095	.090	.081	-.034	-.318	.000
POMS F/I	-.058	.133	-.001	-.043	.110	-.227
SBP	.008	.266	.136	.148	.062	-.002
DBP	-.151	.057	.187	**.535****	.229	.321
MAP	-.098	.153	.184	**.423***	.181	.215

Key: POMS T/A = Profile of Mood States Tension-Anxiety, POMS C/F = Confusion-Bewilderment, POMS D/D = Depression-Dejection, POMS A/H = Anger-Hostility, POMS V/A = Vigour-Activity, POMS F/I = Fatigue-Inertia, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, MAP = Mean arterial pressure, * p< .05, ** p< .01

Table 10.9: Luteal phase correlations between emotion scores, blood pressure values and experimental pain indices in avoidant and non-avoidant coping conditions (n=30), Experiment 5.

	NON-AVOIDANT COPING					
	Pain Threshold	**Pain Tolerance**	**Pain Recovery**	**Sensory Pain**	**Affective Pain**	**Pain Intensity**
POMS T/A	.207	.139	.203	.031	**.431***	-.157
POMS C/B	.139	.170	.163	-.027	.352	-.140
POMS D/D	.191	.057	.030	.066	.268	-.110
POMS A/H	.027	.021	.075	-.016	.286	-.091
POMS V/A	.074	.272	.061	.142	-.351	.109
POMS F/I	.093	.096	-.006	-.008	.237	-.108
SBP	.032	.082	-.037	.239	.213	.080
DBP	-.135	.036	-.087	.248	.335	.145
MAP	-.086	.052	-.074	.255	.308	.129

	AVOIDANT COPING					
	Pain Threshold	**Pain Tolerance**	**Pain Recovery**	**Sensory Pain**	**Affective Pain**	**Pain Intensity**
POMS T/A	.029	.104	-.031	-.071	.244	-.136
POMS C/B	-.082	.131	-.263	-.130	.282	-.110
POMS D/D	.025	.112	-.185	-.041	.198	-.207
POMS A/H	-.150	-.040	-.234	-.024	.329	-.115
POMS V/A	.276	**.380***	.348	.158	-.219	.065
POMS F/I	-.098	.015	-.208	.088	.289	-.244
SBP	-.022	.123	-.074	.104	.091	.100
DBP	-.194	.111	-.155	.068	.055	.217
MAP	-.145	.119	-.134	.083	.069	.187

Key: POMS T/A = Profile of Mood States Tension-Anxiety, POMS C/F = Confusion-Bewilderment, POMS D/D = Depression-Dejection, POMS A/H = Anger-Hostility, POMS V/A = Vigour-Activity, POMS F/I = Fatigue-Inertia, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, MAP = Mean arterial pressure, * p< .05, ** p< .01

10.4 Discussion

The main research objective of Experiment 5 was to ascertain whether testing women within a single menstrual cycle would result in different effects of menstrual phase on pain responses, blood pressure and emotional states from those found in Experiment 4. Additionally, Experiment 5 sought to examine whether women would find avoidant and non-avoidant coping strategies differentially effective for cold pressor pain, and furthermore whether the effects of these strategies would vary across menstrual phase.

Overall, as in Experiment 4, little impact of menstrual phase was found in Experiment 5. No differences were found in female experimental pain responses between the follicular and luteal phases of the menstrual cycle. Nor did the extent of self-reported pain experiences throughout the menstrual cycle (diary pain indices) differ between the menstrual phases. As in the previous experiment, blood pressure did not differ significantly between the menstrual phases but there was an association between BP and self-report pain which was restricted to the follicular phase. Only one emotion subscale (Depression-Dejection) varied across menstrual phase, and the direction of difference (higher in the follicular than the luteal phase) was counter-intuitive both to previous research findings and to the predictions made in this experiment. As in Experiment 4, some associations were found between pain catastrophizing, focus on pain and pain indices but the patterns of these associations varied among menstrual phases and coping conditions.

Taken together, Experiments 4 and 5 did not indicate that menstrual phase altered female sensitivity to cold pressor pain. It is possible that this lack of impact is in some way related to the nature of the noxious stimulus, although on the basis of previous findings that gender-differences are more consistent with experimentally-induced pain which resembles naturally-occurring pain, such as cold pain (Fillingim & Maixner, 1995), biologically-related factors such as hormonal fluctuation across menstrual phase might also be expected to affect cold pressor pain responses. However, since few previous studies have examined the effects of menstrual phase on cold pressor pain, firm conclusions cannot yet be drawn.

The attentional direction of coping (avoidant vs. non-avoidant) did affect some pain responses in Experiment 5. The self-reported sensory pain and pain intensity

experienced during cold pressor exposure was exacerbated when attention was focused upon it (non-avoidant coping). However, this effect was not phase-specific and so suggests that avoidant and non-avoidant coping strategies do not differentially affect pain responses across the menstrual phases. Women in Experiment 5 did perceive that they were more able to adhere to a non-avoidant than an avoidant coping strategy overall, and also that they were more able to adhere to coping instructions generally in the luteal phase than the follicular phase. However, there was no indication that they found avoidant or non-avoidant coping easier to implement in either menstrual phase. Together with the lack of phase-specific impact of coping instructions on pain responses, this suggests that, in this experiment at least, menstrual phase neither impaired nor enhanced the effectiveness of attentional coping strategies for cold pressor pain.

10.5 Summary

Experiment 5 has broadly replicated the findings of Experiment 4, revealing little impact of menstrual phase on pain responses, emotional states or blood pressure and in this regard has not provided evidence for menstrual cycle as part of the underlying mechanisms of female pain sensitivity. The concurrence of these results with those of Experiment 4 confirms that those findings were not an artefact of testing across menstrual cycles, and indicates that menstrual phase effects on cold pressor pain are inconsistent. Some evidence for differential effects of avoidant and non-avoidant coping on cold pressor pain has emerged from Experiment 5, specifically that non-avoidance exacerbated self-report pain, but also that such effects were unrelated to menstrual phase.

Chapter 11

Overview and Evaluation

11.1 Introduction

To recapitulate, epidemiological and clinical research indicates that males and females differ in their vulnerability and sensitivity to pain. Gender differences in human pain perception may be manifest both psychologically and behaviourally, and can affect emotional responses and coping behaviours as well as pain indices such as threshold and tolerance. Although laboratory-based research has also revealed gender differences in pain responses to various noxious stimuli, such differences seem to be inconsistent.

Gender is a particularly complex factor in human pain perception, since it encompasses both biological and psychosocial elements. At a biological level, considerable anatomical and physiological dimorphism between males and females is likely to affect pain perception (Berkley, 1997) and psychosocial factors associated with gender, such as learnt behaviours and gender-typed social roles, are also potentially powerful determinants of pain responses (Bendelow, 1996). However, Fillingim (2000) has argued that the distinction between psychosocial and biological factors in pain perception is a false one, because "psychosocial factors inevitably produce their effects via neurophysiological mechanisms, and because neurophysiological influences also affect psychosocial processes" (p.4). Fillingim's contention highlights a fundamental problem for pain research; namely that it is very difficult to isolate any single factor from its embedded position within the fabric of pain perception. Furthermore, it is often the juxtaposition of multiple elements which exerts dynamic effects on pain responses, rather than the sole agency of any single variable. In view of this, the rarity of clean, definitive answers to pain research questions becomes more understandable, if no less frustrating. However, if systematic differences do exist between male and female pain perception, there is considerable potential for tailoring pain management protocols accordingly.

11.2 Aims of thesis

At the broadest level, the aim of this thesis is to add to the ongoing research process of identifying and investigating the contributory factors in human pain perception. More specifically, the research conducted here sought to investigate the impact of gender and cognitive coping strategies on pain perception, within an experimental pain induction paradigm.

Although there has been a substantial amount of previous research into the effects of gender and coping on experimental pain responses, great variability in research methodologies limits the extent to which comparisons can readily be made between studies or collective conclusions drawn from among them. The empirical work reported in this thesis was an attempt to investigate the effects of these factors in a series of controlled experiments maintaining a consistent methodology throughout. Continued investigation of gender and coping in the context of experimental pain is worthwhile because of the relevance that both these factors are likely to have to clinical pain, to chronic pain and to the acute pains and injuries which may occur in the course of everyday life. As discussed in Chapter 3, there is already evidence that men and women differ in their approaches to coping with pain and their perceptions of how they should respond to it. The potential interaction between gender and coping was therefore considered important, as prior research has also indicated that men and women may benefit from different coping approaches (e.g., Keogh et al., 2000).

11.3 Overview of experimental findings

The present series of experiments addressed two main issues: the impact of gender on cold pressor pain responses, and the effects of avoidant and non-avoidant coping on experimental pain in males and females. In addition, pain-related cognition and emotion were evaluated, as were the effects of menstrual phase on pain sensitivity in females. A brief recapitulation of the primary aims and principal findings of these experiments follows.

Experiment 1 was an exploratory investigation of male and female pain responses and self-reported coping style, and the relationships between these. Gender differences in behavioural and self-report pain responses were apparent but men and women in Experiment 1 were not found to differ in coping style. Although associations were evident between gender and pain responses and between coping style and pain responses, these relationships were found to be independent of each other. In Experiment 2, a coping manipulation paradigm was used to directly examine the effects of an avoidant coping strategy (imaginal distraction) and a non-avoidant coping strategy (sensory focusing) on the cold pressor responses of males and females. Gender differences were found in self-report pain indices only, and non-avoidant coping was found to exacerbate affective pain. A single interaction between gender and coping

strategy was found in this experiment; pain recovery was slower for males when they used avoidant coping. Since males and females in Experiment 2 differed on various measures of pain-related cognition and emotion, a new questionnaire was then developed to facilitate integrated assessment of cognitive and emotional focus on pain in all subsequent experiments (Study 1). In light of the incongruence of the results of Experiment 2 with previous research, Experiment 3 was designed to investigate whether open-ended avoidant coping (free choice of distraction strategy) would have different impact on the cold pressor responses of males and females than the fixed avoidant technique (imaginal distraction) used in Experiment 2. Higher pain tolerance was again found for males compared to females, but no gender differences were found for self-report pain indices. Coping exerted effects on four pain indices in Experiment 3, specifically open-ended avoidant coping extended both pain tolerance and pain recovery, and non-avoidant coping produced higher sensory pain and affective pain ratings. However, no interaction between gender and coping was found. Experiment 3a presents cross-experiment descriptive data from the cold pressor pain responses of males and females in fixed avoidant, open-ended avoidant and non-avoidant coping conditions using combined data from Experiments 2 and 3, for visual inspection. Experiments 4 and 5 investigated the impact of the menstrual cycle on female experimental pain responses, blood pressure and emotional states. This was done to address the possibility that the normal ovarian cycle may cause fluctuations in female pain sensitivity (see Fillingim & Maixner, 1995; Berkley, 1996) and so could confound the effects of gender on pain responses. Menstrual phase was found to have negligible impact on pain responses, blood pressure or emotional states in either of these experiments. Experiment 5 also examined the effects of avoidant and non-avoidant coping on pain responses across the menstrual cycle. Non-avoidant coping was found to exacerbate self-report pain but this effect was general rather than phase-specific.

11.4 Evaluation of findings

The overall pattern of results from this series of experiments has provided limited evidence for effects of gender or of coping on cold pressor pain responses and is characterised by similar inconsistencies to those found in the existent literature. Several issues are particularly notable. Firstly, although some gender differences in pain responses were apparent and, as predicted, reflected greater pain sensitivity in females than in males, such differences were found to be statistically significant in only one or

two pain indices in any single experiment. Secondly, the pain indices for which such differences were found varied between experiments despite consistency of laboratory setting, experimental methodology, experimenter and sample population. The direction of attention during cold pressor exposure (avoidant and non-avoidant coping) did have limited effects on pain responses, but gender-specific effects of avoidant and non-avoidant coping strategies were not generally found. The single exception to this was in Experiment 2 where pain recovery was slower for males when using open-ended avoidant coping, but a similar effect was not found for females. Some gender-differentiated patterns of association were evident in pain-related cognition and emotion throughout this series of experiments, but no differences were found between male and female scores on the new questionnaire designed to assess cognitive and emotional focus on non-clinical pain. Finally, virtually no impact of menstrual phase on female pain was detected within this research programme.

The key question arising from the present findings is why should gender differences in experimental pain responses be intermittent? In fact, there are precedents in the pain literature not only for the existence of gender effects but also for their absence, indicating that the inconsistencies found here are not unusual. Indeed, although there is a general publication bias in the research literature towards positive findings (Easterbrook, 1987) which ensures that negative findings are always under-represented, conflicting findings are a well-documented feature of the experimental pain research literature. Referring to the failure of psychophysics to find an algorithmic relationship between noxious stimulation and evoked pain, Wall (1979) commented on the 'wild variability' of results. Almost a decade later, Rollman and Harris (1987) reiterated that a high level of individual differences in experimental pain responses remained common. These authors reported that pain threshold and pain tolerance values can vary more than eightfold between participants exposed to the same stimulus. Such variability reduces experimental power and increases the likelihood of negative findings, and so may make gender differences in pain sensitivity harder to detect.

Inconsistencies between the results of methodologically disparate studies of gender differences in pain perception are likely to be attributable to numerous factors both extrinsic and intrinsic to the individuals tested. However, in this thesis such discrepancies have occurred between experiments within a methodologically consistent

series, which points strongly to intra-participant variables such as individual differences as their source. A number of factors which may have contributed to the unexpected pattern of findings in this thesis are considered below.

11.5 Potential explanations for inconsistent findings

There is now some evidence for biologically-based individual differences in pain sensitivity which are under genetic control and which contribute to inconsistencies among the responses within experimental groups. Such effects could at least partly explain the disparity between the results of different experiments in this series. As discussed in Chapter 2, sex-related differences in pain sensitivity have been found in other (non-human) mammalian species. While extrapolation to humans requires extreme caution, this does suggest that response bias or socially driven gender role behaviours may not fully explain gender-differentiated pain responses. Recent research with rodents has demonstrated strain-dependent sex differences in pain sensitivity, which suggests that genotype may be a determinant of pain sensitivity (Mogil, 2000). Sex-related differences in pain sensitivity could also be genetically-mediated in other mammals including humans, but this has yet to be established. A study of pressure pain sensitivity in adult monozygotic (MZ) and dizygotic (DZ) twins found strong correlations between pain thresholds within twin pairs (MacGregor, Griffiths, Spector & Baker, 1997). However, the correlations in MZ twin pairs were not much higher than in DZ pairs which was interpreted as more indicative of environmental than genetic influence on pain sensitivity. Further research is clearly needed, but if there is even partial genetic determination of human pain sensitivity, this could explain the intermittent occurrence of gender differences, especially when sample sizes are modest.

Altered female pain sensitivity across the menstrual cycle is a biological factor which might confound gender-differentiated pain responses, although no evidence of such alterations was found within the present series of experiments. However, a methodological factor related to biology which could have obscured menstrual cycle effects here is the body location exposed to noxious stimulation. There is some evidence that the impact of menstrual cycle on the experimental pain sensitivity of females varies with the segmental site (inside or outside the viscerotomes of the uterus) and depth of the tissue stimulated (cutis, subcutis or muscle) (Giamberardino, Berkley, Iezzi, Bigontina & Vecchiet, 1997). Giamberardino and colleagues found that pain thresholds

were highest in the luteal phase but that menstrual phase had more impact on abdomenal than limb sites overall. Dysmenhorrhea was found to accentuate the effects of menstrual phase on pain thresholds in muscle and subcutis, but importantly not in skin thresholds. In the present series of experiments only dysmenhorrheics were included and noxious stimulation was applied only to the skin of the hand, wrist and lower forearm (limb sites). Although there were theoretical and practical grounds for these methodological restrictions, they may have hindered the detection of menstrual phase effects on female pain responses in this thesis.

It is also possible that the lack of reliable gender effects in the present series of experiments was partly related to the type of noxious stimulus and methods of pain measurement used. In their review of gender differences in experimental pain, Fillingim and Maixner (1995) concluded that females are generally more sensitive than males to all forms of experimental pain, including cold pressor pain. They also reported that such differences occur most consistently with noxious stimuli which produce deep, tonic pain similar to naturally-occurring pain (e.g., cold, mechanical pressure, ischaemia). Extending this review to a meta-analysis of gender effects on experimental pain sensitivity, Riley et al. (1998) reported that where such differences were found females always demonstrated greater pain sensitivity than males. However, as the published studies using cold pressor pain were not statistically adequate for the calculation of effect sizes and consequently were excluded from the meta-analysis, firm conclusions regarding the impact of gender on this type of experimental pain must await further research.

As outlined in the introduction to this thesis, the relative lack of direct investigations of gender differences in cold pressor pain responses to date was the primary reason for investigating this stimulus type in the present series of experiments. Consequently, this pain induction technique was used throughout even though it has certain limitations as a noxious stimulus. As described in Chapter 4, cold pressor is a useful experimental pain induction method; a particular benefit is that the pain evoked has naturalistic qualities (Gracely, 1994) and good face validity. Cold pressor has been attributed with ecological validity as an analogue of clinical pain in terms of quality, duration and urgency (Turk, Meichenbaum & Genest, 1983). Indeed, the affective qualities of tonic pain such as cold pressor has been found to resemble clinical pain more closely than phasic pain stimuli

such as electrical or noxious heat (Chen & Treede, 1985; Rainville et al., 1992). The use of cold pressor is also manageable in practical and methodological terms, and pain induction was rigorously executed and temperature standardised throughout this series of experiments to ensure that all participants were exposed to same stimulus. All these considerations aside, it is nonetheless important to recognise that (in common with all experimental pain induction methods) cold pressor has certain shortcomings and that these might have affected the detection of consistent gender differences in pain responses. For example, cold pressor threshold and tolerance are confounded with time inasmuch as the pain produced increases with duration of exposure. In addition, the relatively slow onset and offset of pain with cold pressor, which precludes fast repetition of exposures. In designs with repeated exposures, as with some of these experiments, the overall duration of testing sessions are necessarily extended to incorporate an intertrial recovery interval. It is possible that prolonged testing sessions might have an impact on participant responses over and above the noxious stimulation involved.

A limitation of pain tolerance as an index of pain which has previously been noted is an endurance component in this pain response (Gracely, 1994; Wolff, 1971). Pain tolerance data from cold pressor stimulation, in common with other noxious stimuli such as mechanical pressure and ischaemia, tends to have a binomial distribution with clustering of very low and very high scores. This may represent relatively pain-sensitive and pain-tolerant groups within the same sample, but also may reflect a proportion of participants who are *electing* to tolerate a little or a lot of pain respectively. Experimental designs which could tease apart such potential factors in pain tolerance data would be a worthwhile goal for future research, especially since endurance may be a factor with particular relevance to male pain responses (see below for further discussion of this issue). However, since the intermittence of gender effects between experiments in this series was apparent in self-report pain ratings obtained with a reliable and widely-used questionnaire (S-F MPQ; Melzack, 1987) as well as in pain threshold and tolerance data, the present findings do not seem likely to have been an artefact of the methods of pain measurement used.

Another factor which should be considered (and which could be termed both methodological and psychosocial) is a possible experimenter effect. Although

standardised experimental procedures often include all participants being tested by one researcher (as was done throughout this series of experiments) in order to eliminate the potential for variations in testing style between researchers to affect participant responses, the use of a single experimenter also represents another potential source of bias. In particular, the gender of the experimenter might affect participant behaviour and/or self-report of pain. At least one study has found evidence that men tested by a female researcher reported less pain in response to cold pressor stimulation than those tested by a male researcher (Levine & DeSimone, 1991). There was also a tendency for females to report higher levels of pain to a male researcher than to a female but not to a statistically significant extent. Interestingly, although women reported more cold pressor pain than men overall, gender differences in pain report were not found by participants tested by same-gender researchers. These findings were interpreted as indicating gender differences in the communication of pain, rather than pain sensitivity, and a tendency for men to conform more strongly to traditional gender-role behaviours in the context of pain. However, other research has found no impact of experimenter gender on the pain responses of men or women (Otto & Dougher, 1985; Feine et al., 1991) and it is notable that the Levine study specified that in order to evoke gender-related motives the researchers were selected for their attractiveness and instructed to dress in an attractive manner. This deliberate manipulation is likely to have exaggerated the impact of experimenter gender on participants in that particular study. Nevertheless, even in the absence of such enhancements, there could have been a weaker effect of experimenter gender on the pain responses of participants in the present series of experiments.

It is unclear why the cognitive coping strategies implemented during this series of experiments had such limited impact on pain responses generally and almost no gender-specific effects. This is unlikely to be attributable to purely methodological factors since established techniques and measures were used. However, cognitive coping strategies implemented during an experimental pain induction have limited salience because participants know the pain is time-limited and harmless. In addition, a maximum pain induction of 300 seconds provides relatively little time in which to implement coping strategies, which may have weakened their effects generally as well as any contrast between them. Although investigation of pain coping strategies with healthy participants in a laboratory setting allows good experimental control, the experiences

evoked are markedly different to those of individuals coping with pain from disease or injury. The relative utility of different coping strategies for men and women may perhaps be more fully addressed in the context of naturally-occurring, rather than artificially-induced pain.

The negligible impact of the coping manipulations used here was particularly unexpected in light of previous research using similar paradigms which found distinct differences between the effects of sensory focusing (non-avoidant coping) and distraction (avoidant coping) on cold pressor pain, notably the work of Howard Leventhal and colleagues. For example, Leventhal, Brown, Shacham and Engquist (1979) compared the effects of attending to hand sensations with the effects of viewing a visual distraction on cold pressor pain responses and found that sensory monitoring produced less distress than distraction. In a similar experiment Ahles, Blanchard and Leventhal (1983) found that an experimental group instructed to express their emotions during cold pressor (by moaning and groaning) reported more distress than controls instructed to talk about irrelevant experiences whereas a group instructed to describe their sensations reported less distress than the control group. Leventhal and Everhart (1980) proposed that the sensory and affective aspects of pain are simultaneously processed but contribute independently to the overall experience of pain. According to Leventhal this parallel-processing occurs preconsciously and attention determines which aspect of pain comes into awareness. Central to the theory are pain schemata, which develop through pain experiences and are then invoked (and may be modified) whenever pain subsequently occurs. Logically, the aversiveness of pain and its association with illness mean that most individuals develop a pain schema which comprises not only the sensory features of pain but also its distressing qualities. The findings of the two experiments described immediately above can be explained within this model if attending to the sensory features of pain overrides the schema and leads to a pain experience perceived mainly in sensory terms, with consequent reduction of the distress which normally accompanies it. This effect has also been shown to persist into subsequent trials in which no sensory monitoring instructions were given (suggesting that the pain schema has been modified) and to be replicable and is therefore considered robust (Ahles et al., 1983; Leventhal et al., 1979; Dar & Leventhal (1993). However, the results of the present series of experiments do not support the parallel-processing model of pain since no reduction of affective pain ratings was found with sensory

monitoring, the non-avoidant coping technique used here. Indeed, in both Experiments 2 and 3 there was some evidence for an opposing effect: higher affective pain rating with sensory monitoring (non-avoidant coping) than with a distraction strategy (avoidant coping). However, there was some indication of a positive after-effect from sensory monitoring in these experiments as pain recovery was more rapid with this non-avoidant coping technique.

The present series of experiments has provided scant evidence for gender-differentiated effects of different coping methods, but there were some indications of differences in the ways that men and women think and feel about pain which could affect the way they cope with it in real life but would not necessarily be apparent in a laboratory setting. For example, similar to previous research (e.g. Sulllivan et al., 1995), female participants here tended to catastrophize more about pain than males, or at least to *report* this tendency more. Males professed to be less fearful of imminent pain and to anticipate greater ability to tolerate it than their female counterparts. These findings are in accord with the wider research literature, which suggests that persistent normative social pressures may exert idiosyncratic influence on each gender with respect to coping with pain. It seems that men are generally expected - by themselves and others - to be stoic and uncomplaining when in pain. There may be a male tendency to try to cope in pragmatic, self-reliant ways, and social support for men in pain seems unlikely to be solicited or proffered. Perceived normative social pressure towards male bravery when in pain also carries a potential negative side-effect of feelings of failure or inadequacy should the mask slip. For women, coping with pain presents a different set of problems. Paradoxically, although they are more likely to experience pain, fear it more and seem to find it more intense than men, women are commonly perceived as biologically predisposed to cope with pain. This basis of this assumption seems to be tautological - simply the likelihood that women will experience pain as a function of reproductive processes. Therefore, although it seems that women in pain *seek* help more readily than men, in both medical and social support terms, such perceptions may compromise how much help is given. Interestingly, there is potential for pain to be underestimated and undertreated in both males and females because of assumptions about how they will cope with it.

As mentioned earlier in this chapter, individual differences are likely in the extent to which experimental participants are motivated to endure pain. Although participants here were instructed to maintain exposure to the noxious stimulus for as long as they could bear it, motivational factors influence tolerance measures in all experimental pain studies, since voluntarism and the right to withdraw are both prerequisite and explicit. However, motivation to tolerate pain might also be gender-differentiated in a manner likely to strengthen gender differences in pain responses rather than dilute them. For example, as a result of gender-role normative influence, males might be more motivated to tolerate pain doggedly than their female counterparts (see Robinson et al., 2001). Certainly, although there were both male and female participants who tolerated the maximum possible duration of exposure to cold pressor within the present series of experiments, more males did so than females in each gender comparison experiment. Of course, this could be a direct reflection of lower pain sensitivity in more males than females, but could also be at least partially the result of a gender-specific response bias.

Interestingly, over the course of this series of experiments simply observing the behaviour, comments and demeanour of male and female participants has led me to form some impressions of differences between the two genders, although it should be noted that such differences were not formally assessed. Generally, females seemed more fearful of impending pain than males – or at least they communicated or revealed these feelings more than males, both verbally and non-verbally. With few exceptions, male participants expressed far less than females about pain, or indeed anything else, in the laboratory setting. These differences were manifest verbally and behaviourally, including facial expressions. In particular, with most male participants there was very little emotional expression, and a sense that stoicism was a deliberately adopted coping strategy with many of them. This of course represents a confound to the pain tolerance results in this, and all other similar gender comparisons in experimental pain responses, but also gives some indication of gender-differentiated attitudes and behaviours which are likely to impact on coping with naturally-occurring acute pain such as injury, and clinical pain. Unfortunately, in the absence of any systematic measurement no conclusions can be drawn regarding the relative contribution of gender-specific response biases to the pain data in this series of experiments. However, such mechanisms could help to account for the occurrence of gender differences in pain responses, and could also explain why such differences are sporadic. Even where the

methodology is unchanged between studies, individual differences in gender role stereotypical attitudes and behaviours are likely, which in turn could affect the extent to which gender differences are manifest in any given sample.

Overall, my observations during the present series of experiments have indicated greater emotional expressivity and more apprehension about cold pressor pain in female participants than males. A short discussion of the likely impact of social and cultural factors in such 'gendered' responses follows. It is noteworthy that, despite the conceptual inclusion of sociocultural factors as determinants of psychological and cognitive variables within the Gate Control model (Melzack & Wall, 1988) there has been relatively little research into such factors within psychology or medicine. However, in other disciplines the impact of social and cultural influences have long been considered of central importance. In particular, sociologists have been extremely critical of what has been termed the 'medicalisation' of pain, in which mind-body dualism has perpetuated a reductionist perspective with research focused on the study of sensation and neurophysiology to the exclusion of experiential data. Hochschild (1983) has claimed that even social psychologists have focused on cognition and been careful to avoid discussion of feelings because they believe that to do so renders their work more scientific. This may relate to wider issues of justifying the status of psychology as a science, by adherence to similar methods to those used in the 'natural' sciences.

With an emphasis on empiricism and hypothetico-deductive reasoning, and a broad reliance on quantitative research methods, psychology has been criticised as 'socially naïve' (Harre & Secord, 1972) because it has not taken account of social context, society and its dynamics (Buss, 1975). Certainly, the need for pain research to broaden out of the constraints of the biomedical paradigm, and particularly to take account of emotional and sociocultural factors, is increasingly clear. Until relatively recently, emotion has largely been ignored in both medicine and social science, and been ascribed key importance only within psychotherapy. The general need to develop a sociology of emotions was first highlighted by feminist social science in the 1970s, and more recently medical sociology continues to challenge the assumptions of biomedicine and call for the integration of human experience and phenomenology in the study of health and illness, including pain. Recently, Oakley (2000) has asserted that we now need to

link scientific knowledge about pain and emotion to the everyday experiences of real people.

Certain perspectives contend that the pain experiences of women in particular should be re-evaluated because they are likely to have been misconstrued in the past. Feminist theory has claimed that women in pain who resort to medical treatment have been in a peculiarly disadvantaged position due to male dominance and bias within the healthcare professions (see Bendelow, 2000). In the view of feminist sociology, traditional medicine has operated as a form of social control and women's secondary social status (which is attributed to their intrinsic connections to family and 'lower' functions associated with the private domain of the home when contrasted with men, who are more strongly linked to 'higher' mental processes in the public world of paid work) is believed to have been reinforced by the predominance of male physicians, especially in the field of obstetrics. Furthermore, feminist sociology has asserted that science has devalued the female body for centuries (Martin, 1987) and that male medics have perpetuated stereotyped and prejudiced views about women's health matters (Ehrenreich & English, 1974). The validity of such claims remain open to debate, but there is anecdotal, historical and empirical evidence to suggest that gender-differentiated diagnosis and treatment occur in health care practice and that women in pain tend to be taken less seriously than men (McCaffery & Ferrell, 1992; Bendelow, 2000).

Interestingly, despite the negative consequences that gender stereotyping by men is claimed to have had for women, when the content of such stereotyping by men and women is examined both genders tend to express broadly similar beliefs with respect to pain. For example, Bendelow (2000) reported a qualitative study in which she explored how men and women perceived, evaluated and behaved in response to their own pain symptoms and to find out whether they believed that social characteristics, especially their gender, were important in this context. Bendelow found that perceptions of pain coping abilities were 'strongly gendered' but were mainly consistent across men and women respondents. Male respondents reported active discouragement of emotional expression in boyhood, and believed that they should be stoic when in pain. Women were believed to be more able to cope with pain because it is more 'natural' to them, due to their reproductive function in life, whereas pain was considered 'abnormal' or

outside the usual experience of men. Both sexes expressed the belief that it is more culturally acceptable for women to express pain than for men to do so.

It has long been argued that there are social and cultural differences in how people perceive and respond to pain both in themselves and in others, and that how and whether people communicate their pain to others is culturally determined (e.g., Helman, 1979). In this view, reactions to pain are socially contextual rather than involuntary and whether pain is kept private (concealed from others) or expressed publicly depends on which of these is socially-approved behaviour. Importantly for this discussion, gender socialisation occurs through social learning influences, affects a wide range of behaviours, and starts early in life. Most individuals develop a stable sense of gender identity in childhood and learn gender-role congruent behaviours (in the same way that they learn everything else) through modelling, imitation, reinforcement and punishment (Bandura, 1986; Kohlberg, 1966; Mischel, 1966). Of particular relevance is evidence that males grow up experiencing more radical consequences than females if they deviate from gender norms. For example, research has shown that boys are rewarded more than girls for gender-congruent behaviour and also punished more than girls for gender-incongruent behaviour, particularly by their fathers (Langlois & Downs, 1980). Boys who do not conform to masculine gender norms are often ridiculed and ostracised from male peer groups whereas peer responses to girls who transgress feminine gender norms is far more variable and may not be negative (Fagot, 1977). Early experiences of particularly rigid gender stereotyping for males is likely to lead to a strong avoidance of gender role transgressions in men, which in the context of pain probably explains motivation to withstand pain and to repress or conceal pain-related emotion. Although gender-role socialisation has rarely been directly examined in pain research, it does seem that the expression of pain and pain-related emotion is likely to be sanctioned in females while males perceive social pressure to conceal such feelings. In a recent review Myers, Riley & Robinson (2003) cite empirical evidence that girls and boys tend to respond to pain in ways which conform to their respective gender roles. For girls this includes greater emotional expressivity and a tendency to seek social support in order to cope. Such tendencies in females may have evolutionary origins; expression and interpretation of emotion is likely to be an integral part of the communication between mothers and offspring, and perhaps between female group members in this highly social

species. Certainly a tendency for women to prefer, seek and use social support as a coping resource has been well-documented (Jensen et al, 1994; Unruh et al., 1999).

Research has indicated that gender-role beliefs may be particularly linked to experimental pain responses for men. For example, in a study of male and female responses to pressure pain using the Bem Sex Role Inventory (BSRI: Bem, 1974) to measure stereotypical masculine and feminine personality traits, Otto & Dougher (1985) found an enhancement of pain threshold in men who scored high on stereotypical masculinity when compared to women or men with lower masculinity scores, whereas gender scores were not related to pain in women. However, using the same measure Myers et al., (2001) found that although BSRI scores were related to pain tolerance they did not explain sex differences in cold pressor pain.

More recently, studies of sex-related stereotypic attributions of pain sensitivity, endurance and willingness to report pain have also revealed findings consistent with gender role stereotypes. For example, Robinson, Riley, Myers, Papas, Wise and Waxenberg (2001) found that both men and women believe women to be more pain-sensitive than men, men to be less willing to report pain than women, and men to have higher endurance for pain than women. In addition, men believed their own endurance for pain was higher than that of a 'typical' man. These findings reveal interesting details of gender-specific socially learned responses to pain, especially a fundamental competitiveness (or gender-stereotyped congruence) in male attributions shown in their belief that their own pain tolerance exceeded not only that of females but also other males. A similar study has recently indicated that gender role expectations as well as sex significantly predict the differences in thermal pain responses between men and women (Wise, Price, Myers, Heft and Robinson, 2002).

It certainly seems likely that both the drive for men to appear impervious to pain, and the tendency for women to express pain-related emotion which have been observed in these experiments and many others demonstrate the pervasive influence of gender role beliefs, attitudes, and persistent conformity to stereotypical femininity and masculinity norms.

11.6 Implications of research

Since the results of this series of experiments are inconclusive, there are no direct implications of this research. However, negative or inconsistent findings are as important as positive ones in some respects. For example, they perform a valuable function in stimulating investigation of factors which may have masked the expected effects. Ultimately, this helps to configure the map by which clear conclusions will eventually be reached. For example, the fact that gender differences in pain responses do not invariably replicate but *often* do, suggests that such differences are not artefactual but are perhaps contingent upon certain configurations of many variables. This reiterates that human pain responses are not governed by singular factors, and also helps to explain why the effects of any given factor may be inconsistent. A clear limitation of these experiments is that all the potential factors which contribute to gender differences in pain responses are not accounted for, although as Lautenbacher (1997) has pointed out, such factors are so numerous that this may not even be a realistic objective. If this work makes an incremental contribution to the overall progress of pain research, it does so by demonstrating the intermittently recurrent nature of gender differences in pain perception and by underlining the need for further research to clarify the relationships involved.

An issue which has come into focus during these experiments is whether a wholly quantitative experimental methodology is an adequate tool to investigate the effects of gender on pain perception. In pursuit of empirical evidence and scientific rigour, experimental psychology - including the present research - has tended to approach gender differences in pain responses as if they are analogous to a signal-to-noise ratio. The objective then is to filter out the noise so that the signal becomes clear and measurable, but the borrowed metaphor effectively highlights the dissonance between human pain responses and the sort of testing rationales and methods more appropriate to physics. The critical issue is that in pain perception, gender differences may be an empirically demonstrable 'signal', but here the 'noise' comes from an array of contributory factors rather than unconnected sources of interference. Furthermore, the interaction of psychological and physiological factors in pain perception means that an individual – whether male or female - may experience different levels of pain from the same noxious stimulus on separate occasions. This level of flexibility in pain responses could easily intensify or diminish gender differences in experimental pain sensitivity even if testing conditions do not change and so might account for the inconsistent

gender differences in pain responses found here. Unfortunately, such inconsistency is incompatible with a broadly accepted scientific criterion for a real and robust effect - replicability under standardised conditions. However, if variability on this basis *characterises* pain perception, such a criterion may simply be inappropriate to pain research. Certainly, the effects of potentially important factors in pain perception, such as gender differences, could be overlooked or dismissed erroneously on the basis of such intermittence as was found in this series of experiments.

There are also other reasons to conclude that some changes of approach are needed in experimental pain research. For example, the frame of assessment in laboratory investigations of pain responses is typically very narrow; predetermined aspects of pain are measured, such as threshold or tolerance for a painful stimulus or unidimensional measurement scales of pain intensity or severity. An inevitable consequence of using standardised measures is that the data obtained is limited by the questions asked. Even a multidimensional pain questionnaire only allows an individual to endorse certain preallocated descriptive terms to convey their experience. Nowhere is the participant able to freely describe what they feel. Quantitative data amenable to statistical analysis is obtained, but important information may be missed. If individuals were given the opportunity to describe their experience of pain *in their own terms* qualitative analysis of their descriptions of pain might reveal more information about the experience. This might be particularly pertinent to the impact of gender in pain responses, since gender differences may operate at a subjective level not easily uncovered by standard questionnaires. For example, in Experiments 2 and 3 simple assessments devised for the studies showed that men and women differed considerably in terms of their trepidation and expected ability to withstand the impending cold pressor pain. Caution is needed in interpretation of the data from these measures because they cannot be assumed to possess the reliability and validity of standardised measures but they did provide interesting indications of cognitive and emotional differences between men and women as they approached a painful experience.

The pervasiveness of the biomedical model has certainly meant that if a biological basis for gender differences in pain behaviour can be found there is an implicit sense of greater justification in reporting their existence, as is clear from the continued research emphasis on the identification of underlying physiological mechanisms. However, on

reflection, it seems that some of the distinctions made in the study of human pain simply refer to differences in the level of analysis and explanation being used at any given moment. At a physiological level, changes in neurochemical levels and electrical activity are measurable, and the correlated alterations in cognitions and behaviours can be explained in these terms. At an experiential level, explanations of pain-related emotions and cognitions need to centre on less instantly tangible factors such as social influence and learning processes, and the analysis of such factors may need to be qualitative rather than quantitative. The key point here is that many levels of explanation are needed to fully account for pain phenomena, and these should not be regarded as mutually exclusive. For example, without knowledge of the anatomical and physiological bases of perceptual systems the mechanisms by which the experiential qualities of pain are produced would remain mysterious. Conversely, without self-report from the person experiencing pain, even the most precisely-measured patterns of cortical activation or neurochemical change could not improve our understanding of pain.

The incorporation of methodologies more usual in other social sciences would allow psychological research to take account of certain factors in pain perception which have hitherto been largely overlooked. According to Greenhalgh (1998), sociology and phenomenology are needed to enhance understanding of pain, and narrative accounts are as important in this context as evidence based medicine. Skevington (1995) has argued persuasively for a social psychological model for pain study, emphasising particularly that although psychopathology has been investigated in the context of chronic pain, emotionality in the normal range should not be ignored. She makes the important point that, since there are well-documented gender differences in the extent to which emotions are expressed, especially those pertaining to health and illness, the social and cultural aspects of pain-related emotions for men and women certainly require further investigation. Skevington also recommends the use of qualitative methodologies (which are still under-represented in the mainstream published literature) to evaluate the social and cultural aspects of sex-specific types of pain, such as those arising from reproductive biology.

On the basis of this series of experiments, I wish to add my voice to many others within psychology and other disciplines who have already commented on the need for a shift

towards a more integrated approach in pain research. For example, Rollman (1992) attested that behavioural responses are a vital part of the assessment of pain but are only meaningful when allied to an expressed human experience. Similarly, Skevington (1995) has argued that in addition to laboratory protocols, more naturalistic methods could be used to encompass the psychosocial and cultural context of pain and provide qualitative as well as quantitative data. More recently, Price (1999) has argued for shift towards the integration of first-person experiential data, third-person observation and assessment, psychophysical methodologies and neural imaging techniques to facilitate more meaningful study of pain.

11.7 Recommendations for future research

There are several ways in which future studies could address some of the limitations of the experiments reported in this thesis. Firstly, useful extensions to this research could be designed on a larger scale than was possible here. For example, comparison of male and female responses to several different pain stimuli within each experiment would clarify whether these findings were specific to cold pressor pain. Use of repeated-measures designs in such multi-stimulus comparisons could be used to reduce the impact of individual differences.

Although cold pressor is widely used as an experimental pain induction technique, gender differences in cold pressor pain responses remain inconsistent, as is also the case with other other noxious stimuli (e.g., thermal heat pain). Full evaluation of the impact of gender on all types of experimental pain, including cold pressor, will clearly require further research. In particular, investigations using larger group sizes than have been typical in cold pressor studies to date would allow inclusion of this noxious stimulus in meta-analyses of gender differences in experimental pain. As far as I know, no meta-analysis of gender differences in cold pressor pain has yet been conducted.

As mentioned earlier in this chapter, an important objective which remains for future research is to try to tease apart gender differences in pain sensitivity from differences in pain responses. This is likely to be a difficult task, but one approach would be to implement a cognitive manipulation protocol designed to sidestep the effects of gender-specific normative influences. For example, one possibility would be to compare the effects of an experimental rationale and instructions which convey that sensory acuity to pain - rather than ability to tolerate pain - is the focus of research interest (implying high

sensitivity as a positive attribute) with the effects of a rationale and instructions which convey the reverse. While such a short-term experimental manipulation would not be expected to alter stable, learned attitudes and behaviours, it might temporarily reduce their impact on pain responses.

As discussed earlier in this chapter, possible experimenter effects related to the administration of all testing by one female researcher could have affected the cold pressor pain responses found in the present research. For the future, testing by at least two experimenters (one male and one female) would permit control and evaluation of such effects. Another alternative might be some form of remote test administration, for example via computerised instructions, to remove the impact of any individual experimenter, although this could lead to a loss of data through reduced participant compliance.

Finally, further investigation of the effects of menstrual phase on female pain responses should incorporate measurement of gonadal hormone levels. Although ovulation detection confirmed that testing was carried out in the follicular and luteal phases of participants' ovarian cycle, the actual levels of estrogen and progesterone secretion on the days of testing were not ascertained and might have been atypical. Full evaluation of the impact of gonadal hormone levels on female pain sensitivity requires laboratory assay of blood or plasma samples obtained at the time of testing. Future experiments could also incorporate contrast groups of females on oral contraceptives (whose gonadal hormone levels are artificially moderated). The impact of gonadal hormone levels on pain sensitivity could also be investigated in human males using blood assay, since the little research in this area to date has focused on the exogenous manipulation of hormones in male rodents by surgical or pharmacological means rather than evaluation of naturally-occurring hormonal levels.

11.8 Conclusion

As the diverse strands of pain research progress, evidence accumulates for a biopsychosocial model which can successfully account for the extreme variability of pain experiences where older, dualistic theories of pain have failed. Numerous determinants of pain perception have now been identified and continued investigation of their dynamic interplay is needed to reach a full understanding of the complexity of pain

experiences. It is noteworthy that, despite significant advances in knowledge since the inception of Gate Control theory, neither the physiological nor the psychological mechanisms of human pain perception are yet fully elucidated. However, research increasingly reveals these underlying mechanisms as systems in constant flux. Human experimental pain responses are therefore affected by a plethora of interconnected and fluctuating physiological and psychological states internal to the individual as well as stable extrinsic factors such as the test paradigm and stimulus used. Furthermore, laboratory pain is elicited in an unusual social milieu created and exclusively inhabited by participant and researcher, and differs greatly in meaning from clinical pain.

The appropriateness of a biopsychosocial model of pain becomes increasingly compelling, and certainly offers the only comprehensive explanation for gender differences in pain perception. While the contributions of biological and psychological factors to such differences are becoming clearer, the impact of social and cultural influences requires more research attention. For example, impressions gained from this series of experiments and findings from some recently published experimental pain studies have indicated that gender role influences may be important determinants of sex-related differences in human pain responses. We may need to turn back to social learning theories to understand how gender role expectations are generated across a lifetime, via the impact of childhood modelling of behaviour, appraisal and reinforcing influences from family and peers, and in turn how such expectations may affect pain responses.

In conclusion, this series of experiments has reiterated the need for an integrated and comprehensive approach to the investigation of human pain responses. In particular, it seems that typical experimental research methods may need to be adapted in order to evaluate gender differences in pain responses more fully and that a combination of qualitative and quantitative paradigms could provide a more progressive approach for psychological research into pain. Finally, it is worth noting that the somewhat elusive qualities of gender differences in pain responses observed here do not signify that such differences are unimportant. If gender does have an impact on pain experiences, even if it is not ubiquitous the implications for treatment of males and females in pain should be comprehensively explored.

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APPENDICES

Appendix 1

Cold pressor instructions to participants, Experiment 1

'When you receive a signal from the experimenter, please place your hand into the tank of cold water. Please make sure your fingertips are in contact with the bottom of the tank all the time your hand is in the water. Using your other hand, please press the first button marked 'Just Noticeable Pain' as soon as the sensations in your hand are at all painful. Please press the remaining buttons ('Mild Pain', 'Moderate Pain', 'Severe Pain') in sequence from left to right to rate the level of discomfort you feel as it changes. Press as many of the buttons in sequence as is appropriate to the changes in sensations you feel. Remember, you should keep your hand in the water for as long as you possibly can.'

Appendix 2

Mean pain change rating times in seconds for males and females, Experiment 1 (standard deviations in parentheses)

Pain Index	Males		Females	
Mild Pain	31.99 (22.63)	n=51	23.21 (25.02)	n=55
Moderate Pain	66.97 (60.56)	n=47	39.00 (41.38)	n=54
Severe Pain	77.12 (49.64)	n=30	49.34 (38.77)	n=43

Note. As can be seen from the table above, fewer men than women rated the pain level as ever having reached 'Severe' (30 of the 51 male participants compared to 43 of the 55 females). This pattern (which is likely to be unaffected by the problems encountered with the rating system) is consistent with the well-documented finding that females experience and/or report more severe pain than their male counterparts in response to the same noxious stimulus.

Appendix 3

Instructions for Thought Record, Experiment 2

'Before you put your hand in the cold water tank, we want you to speak your thoughts aloud as they occur to you for a short time, just 5 minutes. I will leave you on your own in here for 5 minutes to do this, and I will knock on the door to let you know when the time is up. It is quite easy to do; just say out loud anything and everything that comes into your head, and keep verbalising your thoughts for the whole 5 minutes. Please say everything that comes into your head however trivial or even silly you think it is. We will make a tape recording while you think aloud, but please note that no-one but the researchers will listen to it and the tape will be erased afterwards. Your name is not required and your responses will, as always, be confidential and anonymous. Are you quite clear about what you should do? Please 'think aloud' for the next 5 minutes after I leave the room. Please say out loud anything and everything that comes into your mind and keep speaking for the whole 5 minutes.'

Appendix 4

Modified cold pressor instructions to participants

'When you receive a signal from the experimenter, please place your hand into the tank of cold water.Please make sure your fingertips are in contact with the bottom of the tank all the time your hand is in the water. Using your other hand, please press the first button marked 'Just Noticeable Pain' as soon as the sensations in your hand are at all painful. Remember, you should keep your hand in the water for as long as you possibly can. When you really feel you cannot bear to keep your hand in the water any longer, please lift it out and lay it on the towel on your lap. Please do not move, rub or flex your hand. After your hand is removed from the cold tank, as soon as the sensations in it are no longer painful, please press the second button, marked 'No More Pain'.