

Copyright is owned by the Author of the thesis. Permission is given for a copy to be downloaded by an individual for the purpose of research and private study only. The thesis may not be reproduced elsewhere without the permission of the Author.

**THE EFFECTS OF ANXIETY SENSITIVITY ON PAIN
TOLERANCE FOLLOWING EXPOSURE AND
AVOIDANCE:
AN EXPERIMENTAL ANALYSIS**

A thesis presented in partial fulfilment
of the requirements for the degree of
Master of Science in Psychology
at Massey University

Kim Alexandra Coldham-Fussell

1999

ABSTRACT

Avoidance behaviour is a prominent and pervasive component of chronic pain behaviour, and is thought to play an active part in the development and maintenance of chronic pain problems. It has been suggested that avoidance behaviour results in increased expectations and fear of pain, which in turn leads to increased avoidance (Lethem, 1983; H. Philips, 1987). However, this concept has not been examined directly to date.

Fear of pain is thought to be a powerful contributor to these avoidance behaviours, in the same way that fear contributes to the avoidance behaviour associated with phobias. The efficacy of exposure therapy in the treatment of phobic avoidance has led to recent suggestions regarding the utility of exposure in the modification of pain-related avoidance.

The present study compared the effects of exposure, non-exposure, and avoidance of pain on subsequent pain tolerance in 90 participants, using the iontophoretic administration of potassium ions as the pain stimulus. It was predicted that exposure would lead to an increase in pain tolerance when compared with non-exposure, and that avoidance would lead to a decrease in tolerance, when compared with both exposure and non-exposure. The effect of anxiety sensitivity, an individual difference variable thought to exacerbate fear of pain, was also examined. It was predicted that high levels of anxiety sensitivity would amplify the tolerance changing effects of exposure and avoidance.

No significant differences were found in pain confrontation following the three experimental conditions. High levels of anxiety sensitivity were associated with a lower initial pain tolerance, and larger increases in tolerance following all the experimental conditions, when compared with low levels of anxiety sensitivity. This was interpreted in terms of a general exposure effect. A number of methodological issues were raised, and directions for future research were discussed.

ACKNOWLEDGEMENTS

I would like to thank my supervisor, Malcolm Johnson, for his time, support, advice, patience, and knowledge. I am very grateful for your help and encouragement.

Thanks also go to Dr. Steve Humphries, who not only developed and wrote the computer programmes, but also contributed ideas and helpful suggestions. Thank-you for enduring request after request for assistance good-naturedly.

I would also like to thank the participants, who bravely volunteered their time and nervous systems.

To my parents, and to Doris, thank you for all the support and encouragement.

And finally, thanks go to Stevo, who throughout the duration of the study came to realise that I could be a painful stimulus in my own right, and who proved to have remarkable tolerance - and to Melanie, who managed to feign interest most of the time.

TABLE OF CONTENTS

ABSTRACT	ii
ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS	iv
LIST OF FIGURES AND TABLES	vii
CHAPTER ONE: INTRODUCTION	
1.0 Historical Conceptualisations of Pain	1
1.1 The Gate Control Theory	2
1.2 The Components of Pain	3
1.3 Chronic Pain.....	4
CHAPTER TWO: AVOIDANCE AND CHRONIC PAIN	
2.0 The Function of Pain	7
2.1 Avoidance	7
2.2 Consequences of Avoidance	8
2.3 The Operant Model of Avoidance	9
a. Positive Reinforcers	10
b. Negative Reinforcers	11
2.4 Cognitive-Behavioural Models of Avoidance	12
a. The Chronic Pain Avoidance Model	12
b. The Over/Under Prediction Model	14
c. The Fear-Avoidance Model of Exaggerated Pain Perception	15
2.5 Review of the Fear-Avoidance Literature	16
2.6 Summary	19
CHAPTER THREE: FACTORS WHICH IMPACT ON FEAR OF PAIN	
3.0 Catastrophic Cognitions.....	20
3.1 Anxiety Sensitivity.....	21

3.2	Exposure and Avoidance	24
a.	Exposure	24
b.	Avoidance	26
3.3	Summary	27

CHAPTER FOUR: THE PROPOSED RESEARCH

4.0	Rationale of the Current Study	29
4.1	The Hypotheses	31

CHAPTER FIVE: METHOD

5.0	Participants	32
5.1	Apparatus	32
a.	Pain Stimulus	32
b.	Discrimination Task	35
c.	Measures	35
5.2	Procedure	39
a.	Experimental Design	39
b.	Experimental Session	41
c.	Post-Experimental Follow-up	45
5.3	Statistical Analysis	45

CHAPTER SIX: RESULTS

6.0	Primary Analyses	47
a.	Exposure, Non-Exposure, and Avoidance	47
b.	The Effects of Anxiety Sensitivity	49
6.1	Additional Analyses	52
a.	Tolerance Change in Manipulated and Experimental Arms	52
b.	Comparison of Reaction Time Data	52
c.	Gender Differences	54

CHAPTER SEVEN: DISCUSSION

7.0 Review of Hypotheses and Findings	55
a. Exposure, Non-Exposure, and Avoidance	55
b. Anxiety Sensitivity	57
c. Experimental vs Manipulated Arm	59
d. Reaction Times and Errors	60
e. Gender	61
7.1 Methodological Issues	62
7.2 Directions for Future Research	65
7.3 Research Summary	66

REFERENCES	67
-------------------------	----

APPENDIX A: Information Sheet	82
APPENDIX B: Consent Form	84
APPENDIX C: Medical Checklist	85
APPENDIX D: Anxiety Sensitivity Index	86
APPENDIX E: Debrief Statement	87
APPENDIX F: Raw Data	89

LIST OF FIGURES AND TABLES

FIGURES

Figure 1: Model of chronic pain avoidance behaviour	13
Figure 2: Theoretical pain-perception curves for 'confronters' and 'avoiders'	16
Figure 3: Pathways between pain severity, anxiety sensitivity, fear of pain, and pain-related escape and avoidance	24
Figure 4: Electrode placement	34
Figure 5: Mean tolerance change (%) for the high anxiety sensitivity and low anxiety sensitivity groups in the non-exposure and avoidance conditions	51

TABLES

Table 1: Configuration of staircase tolerance test	37
Table 2: Research sequence	40
Table 3: Mean tolerance change (%) following the three experimental conditions	48
Table 4: Distribution of low and high AS among the three conditions	49
Table 5: Percentage tolerance change following the experimental conditions for low and high anxiety sensitivity	50
Table 6: Mean reaction time to the tone task across conditions	52
Table 7: Mean reaction time to the tone task for three subgroups of the avoidance condition	53

CHAPTER ONE

INTRODUCTION

The experience of pain is an immensely complex phenomenon, which is mediated by both physiological and psychological factors. The impact of variables such as attention, cognition, and environment on pain perception has been recognised only recently, and has radically altered our conceptualisation and understanding of pain. The following sections will review historical and recent models of pain, the components of pain, and the chronic pain syndrome.

1.0 HISTORICAL CONCEPTUALISATIONS OF PAIN

The recognition and study of pain as a multidimensional phenomenon is relatively new. Although writers of the early 19th century referred to the psychological aspects of pain (Gamsa, 1994), sensory models prevailed soon after, and organic causes were presumed to underlie all pain.

Perhaps the most widely accepted sensory model is the specificity theory, derived from the work of Descartes in 1694 (cited in Melzack & Wall, 1982). This theory asserts that following tissue injury or pathology, impulses are transmitted from specific pain receptors in the skin directly to a pain centre in the brain. Although the theory has appeal on an intuitive level, there are a number of problems with it. Firstly, the assertion is made that there is a fixed, direct line between peripheral receptors and the brain. However, neurosurgical techniques to sever the so-called pain pathways have proved largely unsuccessful in reducing pain, with some cases reporting more pain following the operation (Livingston, 1943).

Secondly, the theory implies that the quality and intensity of the pain sensation is determined by, and is directly proportional to, the intensity of the noxious or damaging stimulus. Although there does tend to be a correlation between pain perception and severity of tissue damage, a wealth of evidence demonstrates that this is not an

invariable linear relationship, as the model suggests. For example, pain is sometimes reported in the absence of apparent pathology, as in phantom limb pain (Livingston, 1943), and in chronic pain (Philips, 1977). Conversely, in other circumstances, tissue damage is evident, but does not seem to cause pain, or causes less pain than would be expected. Examples include injured soldiers who deny feeling pain and do not require analgesic medications (Beecher, 1956), ritualistic tissue damage that provokes no sign of pain (Melzack & Wall, 1965), and relief from pain with placebo (Sternbach, 1968).

Thirdly, the specificity model does not account for emotional and motivational factors implicit in pain experience. As argued by Melzack and Casey (1968), pain has an unpleasant affective quality that demands attention, disrupts on-going behaviour, and motivates action, such as withdrawal or escape, and as such, it is more than a simple sensory phenomenon. This motivational-affective dimension of pain is illustrated by clinical studies on frontal lobotomy. Lesions of the frontal lobe do not disrupt sensory pathways, yet patients following lobotomy rarely complain about pain or request analgesia (Freeman & Watts, 1948; cited in Melzack & Casey, 1968). Similarly, patients with a condition known as pain asymbolia are able to perceive noxious stimuli - they recognise pin pricks as sharp - but do not withdraw from the source, or complain (Osunkotin, Odeku, & Luzzato, 1968). Melzack and Casey (1968) interpreted such phenomena in terms of a disruption of the motivational properties of the pain, while the sensory properties remain intact.

In response to the failure of sensory models to account for the complex and variable nature of pain, Melzack and Wall (1965) developed the gate control theory of pain. This was the first attempt to integrate physiological, psychological and clinical findings into a coherent account of pain perception. Although the model has been revised over the years (Melzack, 1986), the general principle remains valid, and underpins much of our understanding of pain today.

1.1 THE GATE CONTROL THEORY

When peripheral nerve fibres are stimulated, the nerve impulses are transmitted to the brain via the spinal cord. The gate control theory, as expounded by Melzack & Wall (1965), states that, rather than being relayed directly, the information is influenced, or

modulated, at the spinal cord. This modulation is achieved by the mechanism of a 'gate' located within the dorsal horns of the spinal cord. The degree of pain perceived by the brain is dependent on how far the gate is open, as well as the extent of peripheral stimulation. Transmission of noxious impulses from the peripheral nerve fibres to the brain is dependent upon the activation of transmission (T) cells in the dorsal horn. Both myelinated and unmyelinated fibres influence these cells; the larger myelinated fibres inhibit T cell transmission, while the smaller unmyelinated C fibres facilitate it.

This spinal gating mechanism is influenced by other peripheral receptors, as well as by nerve impulses descending from the brain. Sensory input is also modulated throughout its projection from the spinal cord to the regions of the brain responsible for the perception of pain. Pain is experienced when the number of nerve impulses reaching these regions exceeds a critical level.

This theory is able to explain phenomena not accounted for by sensory models. Furthermore, allowance is made for cognitive and emotional factors, as they may exert their influence from the brain to the gate control system. It is well established in the literature that cognitive variables, such as attention (Miron, Duncan, & Bushnell, 1989), sense of control (Rosenbaum, 1980), and expectation (Anderson & Pennebaker, 1980) systematically influence the perception of pain, as do emotional factors, such as anxiety (Cornwall & Donderi, 1988) and fear (Gil, Abrams, Phillips, & Keefe, 1989).

1.2 THE COMPONENTS OF PAIN.

Arising from the gate control theory was the recognition that the concept of pain incorporates three distinguishable components, which have been variously labelled the sensory, evaluative, and affective components (Melzack & Casey, 1968), or the physiological, subjective, and behavioural components (Lethem, Slade, Troup, & Bentley, 1983; Philips, 1977). The physiological component refers to the degree of nociceptive stimulation caused by damage or pathology of the tissue. The subjective component of pain refers to how intense or aversive the individual *experiences* the pain to be. Fordyce (1988) further divided this component into dimensions of pain perception, and suffering. Pain perception involves the subjective sensation of pain,

with or without the presence of nociception. Suffering was defined as an affective or emotional response to pain. The behavioural component refers to the behaviours, known as pain behaviours, exhibited by the individual that indicate distress and subjective pain experience. According to the specificity model, all three of these components should be in synchrony; that is, nociception, experience of pain and suffering, and pain behaviour should be congruent. However, as discussed, a number of instances exist where a marked desynchrony between the components exists. To illustrate, physiological pain may be present, as has occurred in wounded soldiers (Beecher, 1956), in the absence of the subjective and behavioural components. The physiological and subjective components of pain may occur following injury in the absence of behavioural demonstrations, as in a social display of 'toughness'. Subjective experience of pain may also occur in the absence of painful stimulation. In an intriguing study, Anderson and Pennebaker (1980) found that an ambiguous stimulus was perceived as pleasurable or painful, depending on experimental suggestion. Finally, the behavioural component may exist in isolation, as occurs in malingering.

Recognition of the importance of these three components is reflected in the currently accepted definition of pain: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or *described in terms of such damage*" [italics added] (International Association for the Study of Pain [IASP], 1994, p. 210).

1.3 CHRONIC PAIN

Chronic pain is the subjective experience of persistent pain, in the absence of biomedical indicators such as tissue damage, or beyond the point of predicted healing (IASP, 1994). Chronic pain may thus be thought of as a desynchrony between the three components of pain, as exemplified by the following definition:

" Pain experience and/or pain behaviour (and/or physiological responses to pain stimulation) which are (is) out of all proportion to demonstrable organic pathology or current levels of nociceptive stimulation" (Lethem et al., 1983, p. 402).

Consistent with the concept of desynchrony in chronic pain are numerous studies which attest to only a moderate correlation between physical dysfunction and subjectively experienced pain (Kaplow et al., 1993; Philips, 1977; Waddell, Main, Morris, Di Paola, & Gray, 1984). For example, although muscle tension has traditionally been assumed to be responsible for pain level and subsequent pain behaviour in chronic tension headache, Philips (1977) found that muscle tension levels were not significantly correlated with pain intensity and frequency, or with pain behaviour. Waddell, Newton, Henderson, Somerville, and Main (1993) found in patients with chronic musculoskeletal pain, pain severity accounted for only 14% of the variance of disability in the activities of daily living. It is clear that psychological variables contribute to the continuing pain and disability in chronic pain.

Chronic pain syndromes may be grouped into at least two broad categories: neuropathic syndromes, which include post-herpetic neuralgia, causalgia, and phantom limb pain, and musculoskeletal syndromes, which include fibromyalgia, repetitive strain injury, and low back pain. Chronic low back pain is a common presentation in pain programmes, and dominates the research in chronic pain syndromes. For this reason, many of the studies discussed have low back pain as their focus. However, it is recognised that the various syndromes share underlying causal similarities (Asmundson, Norton, & Norton, 1999; Philips & Jahanshahi, 1986; Rose, Klenerman, Atchison, & Slade, 1992).

In the majority of cases, chronic pain problems are preceded by physiological injury or dysfunction (Turk & Rudy, 1992). As the injury heals, physiological, psychosocial, cognitive, and behavioural variables interact to maintain and exacerbate the pain problem. It has been estimated that approximately 90% of adults will have an episode of acute low back pain at some time (McNaughton, 1996). In 90% of cases, subjective pain and pain behaviour reduces gradually over the first 4 to 12 weeks (Philips & Grant, 1991). For the remaining 10%, the disability persists, and becomes a chronic pain problem. The diagnostic cut-off point is generally 3 months post-injury (IASP, 1994), a criterion that is supported by studies that show that individuals who have not recovered from an acute pain episode within 3 months are those who go on to become chronic pain

sufferers (Philips & Grant, 1991). One study found they could predict this outcome at 2 months (Klenerman et al., 1995).

Chronic pain is debilitating and distressing, as it has the potential to disrupt virtually every aspect of life. Persistent pain interferes with daily activities, concentration, and ability to work (Roy, 1992), and is associated with anxiety (Krishnan, et al., 1985) and depression (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997). Fishbain and colleagues (1997) reviewed 87 studies of chronic pain and depression, and concluded that depression tended to be a consequence rather than an antecedent of chronic pain problems. Additional stresses facing chronic pain sufferers include work disability, financial difficulties, loss of roles, family stress, disruption of activities, loss of social support, and sleep disturbance. As pointed out by Turk and Rudy (1992), having a painful condition that eludes diagnosis, and being in so-called 'medical limbo', is in itself significantly distressing.

Unfortunately, the problem of chronic pain is very common. In Britain, almost 5% of the population seek medical advice about a back problem (Tyrer, 1992). This is an underestimation of the true prevalence of the problem, as not everyone with back pain seeks help. In New Zealand, back pain accounts for 30% of all ACC claims, with an estimated cost of over \$360 million per year (McNaughton, 1996). A large proportion of this is spent on people who develop chronic pain problems.

These problems are associated with huge costs, both for the individuals and families affected, and for society, through ACC payouts, unemployment, and overuse of the medical system. It is of obvious benefit to further understanding of the causes and contributing factors of this syndrome.

CHAPTER TWO

AVOIDANCE AND CHRONIC PAIN

2.0 THE FUNCTION OF PAIN

The function of pain is to alert the organism to injury, and to promote recuperative behaviours, such as rest, inhibition of unnecessary activity, and the seeking of medical attention (Bolles & Fanselow, 1980; Wall, 1979). To illustrate, after burning their hand on a hot stove element, an individual might visit their G.P., bandage the hand, and avoid unnecessary use of that hand. In addition, pain acts as a powerful conditioning stimulus, in that it promotes avoidance of the activity or situation that caused the injury. Using the same example, the individual is likely to exercise caution in future when around stove elements. In this way, pain is adaptive; its aversive characteristics enable us to learn which situations are potentially harmful, so that we may survive. This is reflected in the phrase "to learn the hard way".

The value of pain is demonstrated by people who are born with a total lack of sensitivity to pain. In this unusual condition, known as congenital analgesia, individuals may be unaware that they have an injury, and must learn to avoid inflicting damage to themselves (Fields, 1987). It is potentially life threatening, as complications can develop from wounds they have unknowingly sustained.

As a response to acute injury, avoidance of physical activity and situations associated with the injury is an adaptive response to pain that promotes healing and recovery, and reduces the risk of further damage. When avoidance continues after tissue healing is complete, however, it becomes a maladaptive response that may serve to maintain disability. This commonly occurs in chronic pain.

2.1 AVOIDANCE

Avoidance behaviour has long been associated with chronic pain problems. Whereas it was once thought of as an indicator of pain severity, it is now thought of as a habitual behavioural response that plays an active role in the maintenance of disability (H. Philips, 1987). It has been found that increasing chronicity of the pain problem is

associated not with increased severity of pain, but rather with increasing avoidance behaviour (Philips & Jahanshahi, 1986). Thus, the avoidance behaviour becomes desynchronous with both the physiological and subjective components of pain.

The avoidance seen in chronic pain sufferers is not restricted to physical activity. It encompasses a wide range of activities, including work, social interactions, and leisure pursuits (Anciano, 1986; Philips & Hunter, 1981). In addition, the overuse of analgesic medications is seen as a component of avoidance behaviour, in that anticipated pain may be avoided before its onset, through the use of analgesics (Fordyce, 1977).

Avoidance behaviour is the most common type of pain behaviour exhibited by chronic pain sufferers. Philips and Jahanshahi (1986) conducted a factor analysis of the components of pain behaviour, utilising 267 chronic headache sufferers. They used a checklist of 49 items, and found 13 factors of pain behaviour, which could be grouped into complaint, self-help strategies, use of medication, crying and distraction, and avoidance. They found six independent avoidance factors, of which the strongest was 'social avoidance', accounting for 21.9% of the variance. Other factors they found included avoidance of housework, daily mobility, daily exercise, and stimulation. Overall, they found that avoidance behaviour was the most prominent component of pain behaviour, accounting for 42.6% of the total variance.

In short, chronic pain sufferers display extensive avoidance and withdrawal from a wide range of activities. Such widespread behavioural avoidance in turn leads to a number of dysfunctional consequences.

2.2 CONSEQUENCES OF AVOIDANCE

Extensive avoidance of activities leads to a number of maladaptive physical and psychological consequences, which are thought to lead to a self-perpetuating cycle of pain and avoidance. Physical sequelae of inactivity may include loss of muscular strength and weight gain (Bortz, 1984), and for those with back pain, loss of spinal mobility and the development of adhesions (Lethem et al., 1983). Bed rest is associated

with substantial losses in bone calcium and a shortening of muscle tendons (Bortz, 1984), which may induce further pathology of the musculoskeletal system and worsen back pain. Deyo, Diehl, and Rosenthal (1986) found that, for back injury, bed rest was associated with increasing disability. In this study, two days of rest resulted in a better outcome than seven days. Avoidance may thus increase pain upon movement, rather than reducing pain levels.

Psychological effects of avoidance include social isolation, disability in daily living, and depression (Bortz, 1984; H. Philips, 1987). Lethem, et al. (1983) have suggested that stabilisation of the sick role (acceptance of the 'invalid status') associated with withdrawal from everyday activities may lead to increased responsivity to positive and negative reinforcers of pain behaviours. Avoidance also appears to have a negative impact on self-control of pain. A study utilising a heterogeneous group of chronic pain sufferers found that avoidance behaviour was significantly correlated with self-efficacy beliefs ($r = -0.62$; $p = 0.001$; H. Philips, 1987). In addition, it has been found that avoidance is related to increased somatic preoccupation (Crombez, Vervaeke, Lysens, Baeyens, & Eelen, 1998).

A further consequence of avoidance is the increasing desynchrony between pain experience and behaviour, and pain intensity (Crombez, Vervaeke et al., 1998; Lethem et al., 1983). The limitation of exposure to painful stimulation results in fewer opportunities to calibrate pain sensation against pain experience. In this way, expected pain can outweigh actual pain, and promote fear of pain.

It is clear that the consequences of avoidance influence actual and perceived pain, and are associated with a number of unadaptive consequences. But why does avoidance behaviour persist after healing is complete? The following section will review current theories of avoidance behaviour, and associated literature.

2.3 THE OPERANT MODEL OF AVOIDANCE

Fordyce (1976, 1977) applied learning theory to the avoidance and other pain behaviours observed in chronic pain. His operant conditioning model proposed that the

unconditioned and immediate response to a stimulus that causes injury or tissue damage is withdrawal, and avoidance of activity that causes pain. This is called respondent behaviour, as it involves an automatic response to an unconditioned stimulus. Fordyce (1977) proposed that as healing progresses, these behaviours may become subject to the principles of operant conditioning. Operant conditioning occurs when behaviour is reinforced, or becomes more likely to occur, due to environmental contingencies. According to this view, persistence of the avoidance behaviours associated with chronic pain is maintained by the positively or negatively reinforcing consequences of this behaviour.

a. Positive Reinforcers

Initially, Fordyce (1976) emphasised the importance of positive reinforcement of pain behaviours. Positive reinforcers include social and financial rewards, such as attention from family members and health professionals, and compensation payments. If these rewards occur only when the individual is in pain, pain behaviour is reinforced.

A number of studies have evaluated the effect of positive reinforcement on avoidance behaviours. Investigations into the role of marital reinforcement have generally demonstrated a positive relationship between spouse solicitousness and avoidance behaviour. Utilising an experimental design, Block, Kremer, & Galer (1980) found that patients with relatively solicitous spouses reported significantly higher pain levels when the spouse rather than a neutral person was observing. Patients with relatively non-solicitous spouses showed the opposite trend, reporting lower levels of pain in the presence of the spouse. A partial replication of this study found similar results (Lousberg, Schmidt, & Groenman, 1992). They found that patients exercised for a shorter time, exerted themselves less physically, and reported more pain when in the presence of a solicitous spouse.

Other studies have revealed that spouses who are satisfied with their marital relationship appear to respond emotionally to the pain sufferer's display of pain (Block, 1981), and the degree of their marital satisfaction determines the degree of reinforcement and attention given to the pain sufferer (Flor, Turk, & Rudy, 1989). A related finding is that

pain and avoidance behaviour are correlated with perceived social support (Gil, Keefe, Crisson, & Van Dalfsen, 1987). However, not all studies have found a relationship between environmental support and avoidance behaviour (Petroni, 1969).

Studies looking at the role of another positive reinforcer, financial compensation, have yielded results that are more equivocal. Some studies have found that compensation status is relevant in the prognosis of the chronic pain patient, and others have not. After reviewing the literature, Fishbain and colleagues concluded that while compensation does appear to be associated with poorer prognosis in chronic pain sufferers, the literature does not provide evidence that the poor prognosis is directly related to financial gain (Fishbain, Rosomoff, Cutler, & Rosomoff, 1995). Another review concluded that settlement of compensation claims was not associated with significant improvement in the pain problem (Mendelson, 1992), which suggests that monetary gain does not account for the maintenance of disability in chronic pain sufferers.

Although it appears that positive reinforcement may contribute to the maintenance of avoidance behaviour in chronic pain, it has been argued that this alone is not a sufficient explanation for the persistence of avoidance. For many sufferers, the 'secondary gains' associated with inactivity and avoidance are far outweighed by 'secondary losses', such as loss of roles, isolation and depression (H. Philips, 1987; Roy, 1992).

b. Negative Reinforcers.

Fordyce also considered the importance of avoidance learning in the maintenance of avoidance behaviours (Fordyce, 1977; Fordyce, Shelton, & Dundore, 1982). According to this view, pain behaviours may persist if they are negatively reinforced by the avoidance of feared or aversive responsibilities. Consistent with this, Rowat and Knarfl (1985) found that a reduction in familial tension and conflict was related to pain behaviour. Meyers and Lyon (1979) found that the frequency and severity of stressful life events diminished as avoidance behaviour increased. Reduction in social anxiety due to reduced social responsibility (Asmundson, Norton, & Jacobson, 1996) has also been noted as a consequence of avoidance behaviour.

More importantly, the avoidance of anticipated pain has been posited as a powerful negative reinforcer of avoidance behaviours (Fordyce, 1977). Avoidance behaviour is

reinforced because it prevents anticipated or expected increases in pain and suffering. In this context, limping and distorted posture are seen as avoidance behaviours, as they may serve to prevent expected or anticipated increments in pain. As noted by Fordyce, this avoidance may become very resistant to change because it is based on anticipated consequences of activity, rather than actual consequences. In this way, the behaviour may persist long after the pain itself has diminished.

The role of expectations of pain and the anticipated negative consequences of activity have remained important in current conceptualisations of avoidance behaviour in chronic pain. However, recent theories have extended their focus to include cognitive variables, in addition to the overt behaviour emphasised by the operant model. The following section will discuss these theories.

2.4 COGNITIVE-BEHAVIOURAL MODELS OF AVOIDANCE

a. The Chronic Pain Avoidance Model

H. Philips (1987) formulated a cognitive-behavioural model of how avoidance behaviour develops and becomes a sustaining factor in chronic pain. While recognising the influence of both current pain levels and positive reinforcement, she argued that neither was sufficient to explain the persistence of avoidance behaviour. Drawing on a cognitive theory of avoidance (Seligman and Johnston, 1973), she stressed the relationship between expectations and behaviour. Specifically, this model states that the chronic pain sufferer begins to expect that exposure to certain situations or activities will result in increased pain and suffering, that overall pain levels are reduced by avoidance, and that control over pain is best achieved by avoiding such activities. These beliefs are exacerbated by memories of past aversive episodes of pain, and feelings about one's inability to cope with pain. A self-defeating cycle ensues, where avoidance leads to increased expectations of pain on exposure, which, in turn, leads to increased avoidance (see Figure 1).

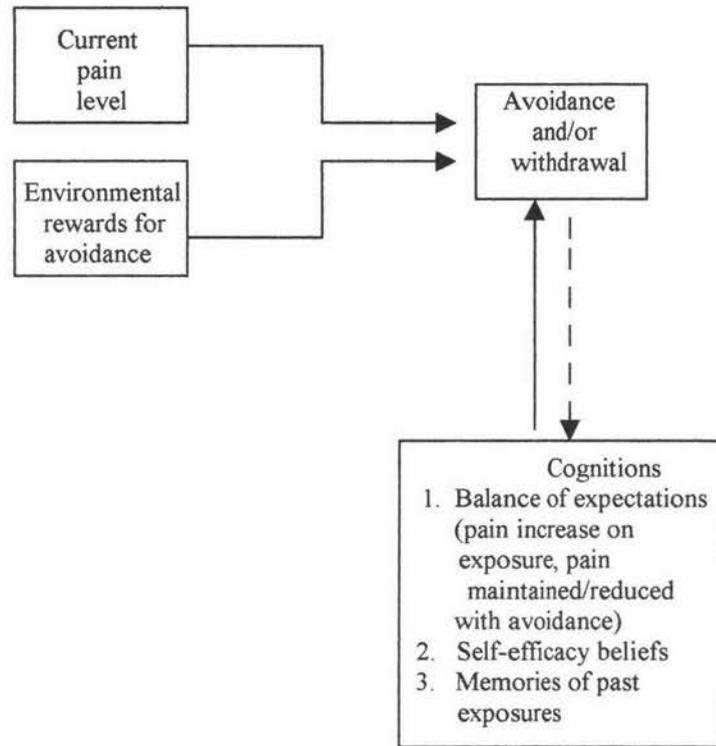


Figure 1. Model of chronic pain avoidance behaviour.

Note. From "Avoidance behaviour and its role in sustaining chronic pain", by H.C. Philips, *Behaviour Research and Therapy*, 25, 273 - 279. Copyright 1987 by Pergamon Press Ltd. Reprinted with permission.

Support for the role of expectations in pain avoidance exists. Studies have found that expectations of pain in clinical pain populations often results in avoidance behaviour which cannot be accounted for by pain severity alone (Crombez, Vervae, Lysens, Eelen & Baeyens, 1996; Linton, 1985; Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995). Similarly, Philips and Jahanshahi (1985) found that avoidance of a noise stimulus in chronic headache sufferers was related to expectations of pain. Providing further support for H. Philip's model was the finding in this study that these expectations were related to the memory of an aversive episode of the noise stimulus. Evidence suggests that chronic pain sufferers may exaggerate memories of past pain episodes (Linton & Melin, 1982), particularly when anxious or depressed (Bower, 1981; Kent, 1985), or following an episode of intense pain (Eich, Reeves, Jaeger, & Redford, 1985). Finally, rather than increasing control over pain, avoidance behaviour is associated with

reductions in self-efficacy (Dolce, Crocker, Moletteire, & Doleys, 1986; C. Philips, 1987).

In the conclusion to her theory, H. Philips (1987) pointed to the similarities between chronic pain and chronic fear, in that they are both aversive experiences that result in avoidance behaviour. With reference to fear reduction methods, she made the suggestion that exposure to avoided activities may impact on negative expectations, and reduce avoidance behaviour.

b. The Over/Under Prediction Model

Closely allied to the idea of expectation is the concept of prediction. Recent research indicates that inaccurate predictions of pain may contribute to the avoidance behaviour in chronic pain (Arntz, van Eck, & de Jong, 1991; Rachman & Arntz, 1991). Rachman and Lopatka (1988) described three types of pain prediction: i) underprediction, in which the intensity of the impending pain is underestimated; (ii) overprediction, in which the intensity of the impending pain is overestimated; and (iii) accurate prediction. Numerous experimental studies have documented how inaccurate predictions of pain affect subsequent expectations of pain intensity; predictions of pain intensity increase following an underprediction, and decrease following an overprediction (Arntz & van den Hout, 1988; Rachman & Arntz, 1991). With repeated exposures, predictions become increasingly accurate. A marked asymmetry in this effect has been reported (Arntz, 1996), whereby underprediction appears to produce an immediate and prolonged increase in expected pain, and overprediction produces a relatively weak and short-lived effect on future expectancies. The same asymmetry has been observed in subjective reports of fear (Rachman, 1994).

Studies of chronic pain patients have failed to demonstrate a consistent pattern of inaccurate prediction. Some studies have found a tendency in this group to overpredict pain (Linton & Melin, 1982; Rachman & Eyrl, 1989), whereas others have found the opposite trend, a tendency to underpredict pain (Arntz and Peters, 1995; McCracken, Gross, Sorg, & Edmands, 1993; Murphy, Lindsay, & de C. Williams, 1997). Consistent with the postulated link between expectations of pain and avoidance (H. Philips, 1987), overprediction of pain is associated with increased avoidance (Rachman & Lopatka,

1988). McCracken et al. (1993) found that higher predictions of pain were related to less range of movement among chronic pain patients.

The finding that underpredictions have prevailed in some studies in chronic pain is puzzling. Asmundson et al. (1999) offered an explanation for this apparent inconsistency. They suggested that the errors in prediction may be mediated by fear of pain. Specifically, they proposed that overprediction may be associated with elevated fear of pain and avoidance, while underprediction may be related with a low fear of pain. This has been supported by the finding that high fear of pain, as measured by the Pain Anxiety Symptoms Scale, is related to overprediction, while low fear of pain is related to underprediction (McCracken et al., 1993).

c. The Fear-Avoidance Model of Exaggerated Pain Perception

The notion that fear of pain may impact on avoidance behaviour was further elaborated by Lethem et al. (1983) in their Fear-Avoidance Model of Exaggerated Pain Perception. They proposed that the desynchrony between the physiological, subjective, and behavioural aspects of chronic pain is similar to the desynchrony seen in other emotional responses, such as phobic responses (Rachman & Hodgson, 1974).

The theory states that desynchrony occurs in some individuals, and not in others, due to avoidance behaviour. The central tenet of the model is 'fear of pain', which they see as an important determinant of pain avoidance behaviour. As with other fears, there are two extremes of coping response available to the individual: confrontation or avoidance. Where an individual falls on this continuum is determined by fear of pain. Individuals with a low fear of pain are able to confront their pain and gradually increase their exposure to painful activities and to the experience of pain itself. Confrontation is seen as an adaptive response, as the expectation of pain is calibrated with the actual experience of pain, and the components of pain remain in synchrony. In contrast, individuals with a strong fear of pain tend to avoid painful activities and the experience of pain. In line with the previous models discussed, avoidance is seen as a maladaptive response. By avoiding exposure to pain, there are few opportunities to calibrate (exaggerated) pain perception with pain sensation, and desynchrony of these two

components occurs. Figure 2 illustrates the desynchrony between pain perception and pain sensation thought to occur as a result of avoidant behaviour.

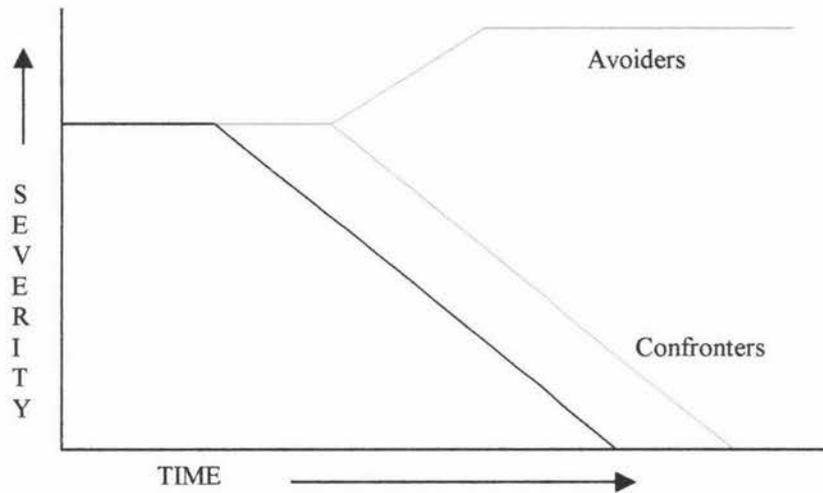


Figure 2. Theoretical pain-perception curves for 'confronters' and 'avoiders'.

Pain sensation — Pain perception - - -

Note. From "Outline of a Fear-Avoidance Model of exaggerated pain perception - I", by J. Lethem, P.D. Slade, J.D.G. Troup, & G. Bentley, 1983, *Behaviour Research and Therapy*, 21, 401 - 408. Copyright 1983 by Pergamon Press Ltd. Reprinted with permission.

In addition, it was proposed that avoidance results in the exacerbation and maintenance of fear, which in turn, increases the likelihood of an avoidance response. Finally, it was suggested that increased responsiveness to environmental reinforcers was likely to occur as avoidance behaviour developed.

2.5 A REVIEW OF THE FEAR-AVOIDANCE LITERATURE

The theory that fear of pain may instigate avoidance behaviour posited by Lethem et al. (1983) has been the subject of several recent investigations. The following review will highlight recent studies that have examined three fears, fear of pain, fear of work-related activities, and fear of movement/(re)injury, and their relationship with avoidance behaviour.

Fear of Pain

The first attempt to measure pain-related fear in chronic pain patients was the development of a 6-item Fear Self-Statements subscale (Gil et al., 1989), which was included in an expanded version of the Coping Strategies Questionnaire (Rosenstiel and Keefe, 1983). When utilised in a study of chronic pain due to sickle cell disease, elevated scores on the subscale contributed to predictions of greater severity of painful episodes, and greater avoidance, as measured by level of activity (Gil et al., 1989).

Subsequently, McCracken, Zayfert, and Gross (1992) developed and validated a more comprehensive measure of pain-related fear and anxiety, the Pain Anxiety Symptoms Scale (PASS). This rationally derived self-report inventory consists of four subscales: fearful pain-related thoughts and appraisals, cognitive symptoms of anxiety, physiological symptoms of anxiety, and escape/avoidance. They administered the PASS, the Multidimensional Pain Inventory (MPI), which includes a measure of the impact of pain on daily life, and the Pain Disability Index (PDI), a measure of perceived disability, to 104 chronic pain patients. They found that PASS scores related significantly to both interference with activities of daily living due to pain, and to levels of self-reported disability. Consistent with concept of the role of fear in maintaining avoidance, McCracken et al. (1993) found that high scores on the PASS were related to greater restriction of movement, or avoidance, on a passive straight leg-raising test. This was further supported by findings that PASS scores and avoidance pain behaviours, as measured by the Pain Behaviour Checklist (PBC), are positively correlated (McCracken, Gross, Aikens, and Carnrike, 1996).

A prospective study by Klenerman et al. (1995) found that pain-related fear is a precursor rather than a consequence of chronic pain problems. In a comparison of the predictive value of psychosocial, demographic, psychological, and physical variables on the development of chronic pain, they found that fear-avoidance beliefs were the most powerful.

Fear of pain thus appears to impact on levels of disability and avoidance in chronic pain. More recently, studies have focussed on more specific fear variables implicated in avoidance: fear of work-related activities and fear of movement/(re)injury.

Fear of Work-Related Activities

It appears that the fear experienced by chronic pain sufferers is not restricted to pain, but generalises to activities and situations that are expected to cause pain. In line with this, Waddell et al. (1993) developed the Fear-Avoidance Beliefs Questionnaire (FABQ), a 16-item measure which assesses beliefs about how work and physical activity affect pain levels, and whether they should be avoided. The instrument consists of two scales: fear-avoidance beliefs about work-related activity (e.g., "My work aggravated my pain"), and physical activity (e.g., "I should not do physical activities which (might) make my pain worse"). Using a sample of 184 chronic back pain patients, they found fear-avoidance beliefs about work and physical activity to be strongly related with disability of daily living and loss of work, even after allowing for severity of pain. Crombez, Vlaeyen, Heuts, & Lysens (1999) found both scales of the FABQ to be positively correlated with self-reported disability, over and above pain intensity, pain duration, and negative affect. In addition, they found that scores on the FABQ-Physical scale were significant predictors of poor behavioural performance on a physical task (a trunk-extension-and-flexion test).

Fear of Movement/(Re)Injury

A further line of investigation has looked at a more specific fear-avoidance belief, that movement and physical activity will cause (re)injury. Evidence suggests that this fear, also known as "kinesiophobia", is related to avoidance behaviour. The Tampa Scale for Kinesiophobia (Kori, Miller, & Todd, 1990) is a questionnaire that is aimed at the assessment of fear of (re)injury due to movement. Using a Dutch version of this scale, Vlaeyen et al. (1995) found that chronic pain patients with a high degree of fear of movement/(re)injury showed more fear and avoidance when performing the physical task of lifting a 5.5kg bag, and also showed more escape behaviour, by dropping the bag significantly sooner than less fearful individuals. Crombez et al. (1999) found that high scorers on the TSK in a chronic pain sample showed greater fear of pain and (re)injury, and demonstrated poorer behavioural performance on an exercise task than did confronters. They also found the TSK to be superior in predicting avoidance of the task than pain intensity and duration. Similarly, Crombez, Vervaeke et al. (1998) found that avoiders were more somatically preoccupied, and reported more fear of (re)injury, than confronters during a behavioural test.

2.6 SUMMARY

The operant and cognitive-behavioural models discussed differ in their focus, but share a number of similarities. Firstly, each of the models emphasises the dysfunctional and self-perpetuating nature of avoidance behaviour. Secondly, the cognitive-behavioural models have all drawn comparisons between pain-related avoidance and fear-related avoidance. Thirdly, the concepts of negative expectations, erroneous predictions of pain, and fear of pain are closely related, and have all been posited as being causal factors in the development and maintenance of avoidance behaviour. The pivotal role of fear of pain was argued by Lethem et al. (1983), who posited that fear of pain instigated avoidance behaviour. Recent research has provided support for the role of fear in avoidance behaviour, finding that pain-related fear is related to poor behavioural performance, and is more disabling than pain itself. In addition, fear of work-related activities and fear of (re)injury have been shown to impact on avoidance behaviour in chronic pain.

CHAPTER THREE

FACTORS WHICH IMPACT ON FEAR OF PAIN

Recently, the focus of research has extended to include potential dispositional traits that may directly impact on propensity of individuals to engage in fear and avoidance behaviours following acute injury. Two such traits that have received recent attention are the tendency to interpret pain-related information in a catastrophic manner, and anxiety sensitivity. Following a discussion of these two variables, the effects of avoidance and confrontation of pain-related fear will be discussed.

3.0 CATASTROPHIC COGNITIONS

Recent research has focussed on the role of catastrophic cognitions in mediating pain-related fear and avoidance. Sullivan, Bishop, and Pivik (1995) conceptualised catastrophising as "an exaggerated negative orientation toward noxious stimuli" (p.524), and proposed that through exposure to painful situations or to others' catastrophic reactions to pain, individuals may develop catastrophising beliefs or schema about the high threat value of pain. Sullivan et al. (1995) subsequently developed and validated the Pain Catastrophizing Scale (PCS), a self-report index for use with both clinical and non-clinical populations. Items were based on dimensions of catastrophising that have been emphasised by different researchers (Chaves & Brown, 1987; Rosenstiel & Keefe, 1983; Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979), which included the tendency to increase attentional focus on pain-related thoughts, to assume a helpless orientation to coping with pain, and to exaggerate the threat value of pain. These three components, rumination, helplessness and magnification, have been supported by factor analyses (Osman et al. 1997; Sullivan et al., 1995).

Research suggests that catastrophic styles of thinking about pain are associated with increased pain sensitivity and distress. Prior to the development of the PCS, Spanos et al. (1979) found that a catastrophising style was related to increased sensitivity and reduced tolerance to pain in a population of chronic pain sufferers. Utilising the PCS,

Sullivan et al. (1995) found, in a non-clinical sample, that high pain catastrophisers reported greater pain intensity during induction of cold-pressor pain than low catastrophisers, and reported more negative pain-related thoughts and emotional distress. These results were replicated with a group who had undergone an aversive electrodiagnostic medical procedure (Sullivan et al., 1995). Crombez, Eccleston, Baeyens, & Eelen (1998) offered a possible explanation for the greater pain intensity apparently experienced by catastrophisers. They argued that because these individuals show an inability to suppress or divert attention away from pain-related thoughts, pain-related information is perceived as more intense and unpleasant.

Catastrophising is thought to increase fearful responses to pain (Crombez, Eccleston et al., 1998). This reflects the excessive appraisals of the threat value of pain central to the pain catastrophising construct. McCracken and Gross (1993) found a strong association between pain catastrophising and fear of pain in a chronic pain population. Similarly, catastrophic thinking about pain has been found to be significantly related to fear of movement and (re)injury (Crombez et al., 1999; Vlaeyen, et al., 1995). These authors raised the possibility that pain catastrophisers may focus more on the negative aspects of the experience and are more likely to interpret physical arousal as pain cues.

Research on coping with chronic pain has shown that women are more likely to catastrophise than men (Jensen, Nygren, Gamberale, Goldie, & Westerholm, 1994). This is consistent with research that suggests that females are more likely than males to adopt a ruminative and emotionally expressive style when coping with stressful situations (Nolen-Hoeksema, 1987).

In summary, pain catastrophising seems to increase negative expectations about pain, about its intensity, and about the individual's ability to cope with the pain.

3.1 ANXIETY SENSITIVITY

Anxiety sensitivity (AS) is the fear of the sensations associated with anxiety, such as palpitations, breathlessness, and dizziness. This fear arises from the belief that these

sensations have harmful somatic, psychological, or social consequences (Reiss, 1991; Reiss & McNally, 1985). For example, palpitations are feared if they are believed to be a sign of impending heart attack; concentration difficulties are feared if they are believed to be an indication of insanity; and shakiness is feared if it is believed that it will elicit ridicule from others.

According to the expectancy model of fear (Reiss, 1987, 1991; Reiss & McNally, 1985), AS is one of three fundamental or trait sensitivities: (1) injury/illness sensitivity; (2) fear of negative evaluation; and (3) AS. The Anxiety Sensitivity Index (ASI) was developed as a measure of this construct, for use with both clinical and normal populations (Reiss, Peterson, Gursky, & McNally, 1986). Debate has centred on whether the ASI is uni-factorial (Reiss et al., 1986) or multi-factorial (Peterson & Heilbronner, 1987; Telch, Shermis, & Lucas, 1989) in nature. However, it is now generally agreed that a hierarchical structure, with lower-order factors loading on a single, higher-order factor, provides the best description (Lilienfeld, Turner, & Jacob, 1993; Taylor & Cox, 1998; Zinbarg, Barlow, & Brown, 1997).

Reiss and colleagues have argued that AS is an individual difference variable that acts to amplify fear reactions (Reiss, 1991; Reiss & McNally, 1985). For example, when exposed to an anxiety provoking event, a person with high AS will become anxious, and then become anxious about being anxious, thus amplifying the anxiety. Following this reasoning, it follows that people with elevated AS scores will fear situations where they *expect* to feel anxious:

"People who are afraid of anxiety should develop a fear of any situation in which there is even a small chance/expectation of becoming anxious; because there are many such situations, people who are extremely sensitive to anxiety should develop fears of many situations" (Reiss, 1991, p. 147).

Consistent with this, evidence shows that compared with low AS individuals, those with high AS have a greater propensity to fear and avoid a variety of stimuli, such as animals (McNally & Steketee, 1985) and insects (Taylor, Koch, & Crockett, 1991), and agoraphobic situations (McNally & Louro, 1992; Taylor, 1993). Other studies have

found high correlations between ASI scores and scores on a variety of fear survey schedules (Reiss et al., 1986; Reiss, Peterson, & Gursky, 1988).

Until recently, most studies of AS have focussed on anxiety disorders, with a particular focus on the relationship between AS and the occurrence of panic. Studies have shown that elevated AS is associated with an increased incidence of unexpected and cued panic attacks in both clinical (Rapee, Ancis, & Barlow, 1988; Reiss et al., 1986) and non-clinical (Asmundson & Norton, 1993; Cox, Endler, Norton, & Swinson, 1991) samples. Due to recent attention given to the role of fear in chronic pain, interest has now been generated in the possible role of AS in amplifying fear of pain reactions. In the context of chronic pain, this could mean that an individual with high AS may fear, and avoid, activities and situations associated with pain due to a fear of the sensations of anxiety this may provoke.

Several studies indicate that AS is related to fear of pain in chronic sufferers. Asmundson & Norton (1995) found in a group of 70 chronic pain patients that those with high AS exhibited greater cognitive disruption and anxiety in response to pain, and greater pain-related fear ($r = 0.48$). They also showed more escape and avoidance behaviours, independent of pain severity. Finally, using the PASS, a moderate correlation was found between AS and pain-related avoidance ($r = 0.32$). Asmundson and Taylor (1996) further investigated the role of AS in pain-related fear and escape/avoidance. Using structural equation modelling, they tested and validated the prediction that AS directly exacerbates fear of pain, and indirectly promotes pain-related avoidance, via its influence on fear of pain. This indirect effect was significant even when controlling for pain severity. Figure 3 depicts these pathways.

To summarise, these results suggest that high AS individuals may be more avoidant of pain and pain-related activities than those with medium and low AS.

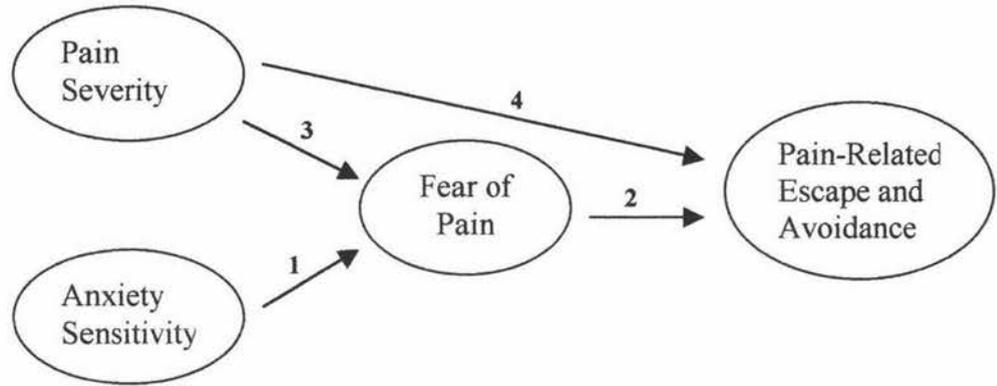


Figure 3. Pathways between pain severity, anxiety sensitivity, fear of pain, and escape/avoidance.

Note. From "Role of anxiety sensitivity in pain-related fear and avoidance", by G.J.G. Asmundson and S. Taylor, 1996, *Journal of Behavioral Medicine*, 19, 577 - 586. Copyright 1996 by Plenum Publishing Company. Reprinted with permission.

3.2 EXPOSURE AND AVOIDANCE

The previous sections have outlined the role of fear of pain in the avoidance behaviours observed in chronic pain. It has been suggested that the fear and avoidance in chronic pain may share similarities with other fears, such as phobic avoidance. As other fears have shown to be mediated by exposure to, and avoidance of, the feared stimulus, it follows that pain-related fear may also be mediated this way. The following section will discuss evidence that shows how fear, in particular, pain-related fears, are affected by exposure and avoidance.

a. Exposure

Repeated exposure to a feared stimulus is a widely used and accepted method of fear reduction. The efficacy of imaginal and *in vivo* (real life) exposure is well-documented in the treatment of disorders in which fear is implicated, such as simple phobias (Vodde & Gilner, 1971), agoraphobia (Munby & Johnston, 1980), panic disorder (Craske, Rowe, Lewin, & Noriega-Dimitri, 1997), social phobia (Scholing & Emmelkamp, 1993), obsessive-compulsive disorder (Lindsay, Crino, & Andrews, 1997), and post-

traumatic stress disorder (Foa & Goldstein, 1978). The mechanism or mechanisms by which exposure reduces fear is not well explicated at present, although it has been argued that it is due to the process of extinction (Marks, 1978; Mathews, 1978; Vodde & Gilner, 1971). This hypothesis assumes that fear is reduced as a function of repeated presentations of the feared stimulus in the absence of any real aversive consequences.

Following her suggestion that the avoidance seen in chronic pain may be similar to the avoidance in common fears, H. Philips (1987) suggested that exposure may be of use in the reduction of these behaviours. A similar suggestion was made by Lethem et al. (1983), who thought that exposure to the feared stimulus, pain, would lead to a reduction in fear, and a concomitant reduction in avoidance. Lethem suggested that exposure treatment should be undertaken in order to produce disconfirmation between expectations of pain and actual experience. Following the extinction hypothesis, an individual who fears pain may expect that they will not be able to cope with the pain, that they may (re)injure themselves, that they will be unable to cope with the anxiety the pain produces, and so on. When repeated exposure to the pain itself does not result in these outcomes, these fearful expectations are calibrated with the actual experience, and fear diminishes.

The reduction of fear is commonly gauged by the observable reduction of avoidance behaviours, or increases in confrontation behaviour. In the treatment of phobias, for example, the efficacy of the treatment is often evaluated via the use of a behavioural approach test, which measures how close and for how long the fearful person is able to be near their feared stimulus. In agoraphobia, the length of time the fearful person is able to remain in their feared situation may be the measure of fear reduction. With regard to the fear of pain, reduction in fear could be measured by the increase in tolerance of the painful activity or situation. In a clinical setting, this is often measured in terms of the intensity and duration of the performance of a specific exercise. In the laboratory, fear reduction may be measured by increases in tolerance to a painful stimulus.

To date, pain studies have provided support for the notion that exposure leads to reduced fear, and increased tolerance for pain. This is illustrated by the success of

exercise quotas in reducing pain-related fear and avoidance. In this procedure, patients must persist at a set 'quota' of a feared activity or exercise, despite pain, until the quota is achieved. A number of studies have shown reductions in fear and avoidance as a result of this technique. For example, Dolce et al. (1986) found in a heterogeneous sample of chronic pain patients, exposure via an exercise quota system led to a reduction in fear-related thoughts, and an average increase in exercise tolerance of 42%. Other studies have found similar results (Doleys, Crocker, & Patton, 1982; Fordyce, 1977).

One experimental analysis on the effects of exposure to a painful stimulus has been conducted to date. Philips and Jahanshahi (1985) gave one group of chronic migraine and tension headache sufferers exposure to an aversive noise stimulus, while another group were not exposed. Participants in the exposure condition were required to listen to the noise stimulus for 60 seconds longer than they had opted for. Results showed that tolerance of the stimulus increased significantly when compared to the no-exposure group. Exposure under conditions of relaxation was found to significantly increase subsequent tolerance, by 14.1 seconds. Interestingly, this result was found in the migraine group, but not in the tension headache group. This was explained in terms of the possible susceptibility of migraine sufferers to the particular auditory stimulus used.

b. Avoidance

Avoidance behaviour in chronic pain leads to a number of maladaptive physiological and psychological consequences. It has been proposed that among these consequences is an increase in pain-related fear that, in turn, leads to increased avoidance. H. Philips (1987) stated that "it seems possible that avoidance of stimulation may in fact fortify and strengthen expectations of pain increment upon exposure, thus strengthening pain avoidance behaviour itself" (p. 276). Indirect support for this comes from studies of chronic headache, where avoidance behaviour increases over time, even when the severity of pain remains constant (Philips & Hunter, 1981). One study from the anxiety literature found that when phobics and obsessive ritualisers were instructed to avoid all contact with stimuli which evoked fear or rituals, an increase in self-reported fear and increased avoidance of the stimuli followed (Greist, Marks, Berlin, Gournay, & Noshirvani, 1980). From an operant perspective (Fordyce, 1977), avoidance behaviours

may be reinforced by reducing the anticipation of pain. Lethem et al. (1983) also theorised that avoidance led to exacerbation of fear, which she reasoned was in part due to the lack of opportunity to calibrate expectations of aversive outcomes with the actual consequences.

Although the idea that avoidance increases fear of pain and subsequent avoidance is appealing, there is little direct evidence to support it. Philips and Jahanshahi (1985) included an avoidance condition in their study of the effects of exposure on tolerance of a noise stimulus. They did find that avoidance was associated with a reduction in tolerance of the stimulus, which would appear to provide support for the above-mentioned ideas. Unfortunately, participants in the avoidance condition were simply not exposed to the stimulus; active avoidance did not occur. Whether this truly constitutes avoidance, with the implicit sense of preventing an aversive outcome, is questionable. Thus, no studies have attempted to manipulate avoidance experimentally to date.

3.3 SUMMARY.

Lethem et al. (1983) developed a model of the avoidance behaviours seen in chronic pain, in which fear of pain influences the probability that an individual will confront or avoid painful activities and situations following acute injury. The concept that fear of pain is an important variable in the development of avoidance behaviours has gained support from clinical and experimental research.

Recent studies have examined the impact of various individual difference variables on the propensity of an individual to have a fearful response to pain. Pain catastrophising, the tendency to interpret pain in a catastrophic manner, has been implicated in fear of pain. Studies have found that chronic pain sufferers with a tendency to catastrophise pain not only perceive pain as being more intense than low catastrophisers, but also tend to fear movement and (re)injury. The role of anxiety sensitivity, the fear of sensations associated with anxiety, has also been investigated. Chronic pain sufferers with high anxiety sensitivity not only tend to fear pain more, but, as predicted by Lethem's model, show more pain-related avoidance behaviours.

Given that fear of pain is associated with avoidance behaviours in chronic pain, it has been suggested that the treatments developed for the attenuation of other fears, including phobias, may be useful in the reduction of pain-related fears (Lethem et al., 1983; Philips, 1987). Exposure therapy, where an individual is repeatedly exposed to a feared stimulus, has been highly successful in the reduction of a variety of fears, including phobias, OCD, and PTSD. Research to date has confirmed the utility of exposure-based therapies in reducing pain-related fears in the chronic pain population to date. However, experimental research in this area is presently lacking.

Research indicates that the avoidance behaviours seen in chronic pain are, at least in part, due to fear of pain. In addition, the possibility that these avoidance behaviours themselves may act to further increase fear, and subsequent avoidance, has been raised (Lethem et al., 1983; Philips, 1987). However, this notion has not been tested experimentally to date.

CHAPTER FOUR

THE PROPOSED RESEARCH

4.0 RATIONALE OF THE CURRENT STUDY

Fear of pain is thought to play a prominent role in the development of the avoidance behaviours commonly seen in chronic pain (Lethem et al., 1983). Research in fear (Munby & Johnston, 1980) and pain (Dolce et al., 1986) has shown that exposure to a feared stimulus results in reduced fear of the stimulus. This is thought to be due to the process of extinction, whereby fear is reduced as a function of repeated presentations of the feared stimulus in the absence of any real aversive consequences (Vodde & Gilner, 1971). Fear reduction due to exposure is typically measured by the increase in tolerance for the feared stimulus. In evaluating the effects of exposure to pain on subsequent fear of pain, increase in pain tolerance would serve as the measure of fear reduction.

A corresponding notion is that avoidance of a feared stimulus actually exacerbates fear, and promotes further avoidance (Lethem et al., 1983). In the context of pain-related fear, this fear increase would be observable as a reduction in tolerance for the pain stimulus. However, support for the fear-increasing effects of avoidance are meagre at present. Given that avoidance behaviour is important in the development and maintenance of chronic pain, the possible negative effects of early avoidance are of relevance. The extent to which active avoidance of pain influences subsequent fear of pain needs to be determined in more detail.

The main objectives of the present study are two-fold. The first aim is replicate the findings of Philips and Jahanshahi (1985) that exposure to a painful stimulus results in increased tolerance for that stimulus, when compared with non-exposure to the stimulus. The study of Philips and Jahanshahi was based on a clinical sample of chronic headache sufferers. The current study, however, will utilise a non-clinical sample. A common problem with pain studies involving clinical samples is that current pain levels are difficult to control for. It is expected that the general principle of the fear-reducing effects of exposure will be evident in the current sample, given the intrinsically aversive nature of pain (Melzack & Casey, 1968).

To test this prediction, two groups, one receiving pain stimulation, and the other not, will be compared on subsequent tolerance for the stimulus. An increase in tolerance will be thought of as a reduction in fear, while a decrease in tolerance will be thought of as an increase in fear.

Non-exposure to a painful stimulus would, under ideal conditions, be expected not to affect pain tolerance level. However, in the present study, the tolerance tests themselves constitute a degree of exposure. Hence, it is not predicted that the non-exposure group will show no change in tolerance, but rather that tolerance will increase to a greater degree in the exposure group.

The second aim is to investigate the effect of active avoidance on subsequent pain tolerance. It has been suggested that avoidance may act to increase fear of pain, and subsequent pain avoidance (Lethem et al., 1983; H. Philips, 1987), but this has not been experimentally tested as yet. It is therefore predicted that avoidance of the pain stimulus would result in a decrease in tolerance when compared to the exposure condition. Because non-exposure is not thought to impact on fear of pain, it is also predicted that avoidance of the pain stimulus would result in a decrease in pain tolerance when compared with the non-exposure condition. Thus, the three groups to be compared are exposure, non-exposure, and avoidance.

In addition to these main predictions, the effect of anxiety sensitivity will be examined. Because anxiety sensitivity is associated with fear of pain (Asmundson & Taylor, 1996), it is firstly predicted that participants with high AS will have a lower tolerance for pain than those with low AS. It is also predicted that the tolerance increasing effects of exposure will be amplified in those with high AS when compared with those with low AS. This is based on the assumption that a high level of fear will be more amenable to change than a low level. Similarly, it is predicted that the tolerance decreasing effects of avoidance will be amplified in those with high AS when compared with those with low AS. If avoidance of pain increases fear of pain, it is hypothesised that those with a fearful predisposition will be more affected.

4.1 THE HYPOTHESES

The specific hypotheses to be tested are:

1. Exposure to the pain stimulus will lead to increased tolerance for the pain stimulus when compared to non-exposure to the stimulus.
2. Avoidance of the pain stimulus will lead to a decrease in tolerance for the pain stimulus when compared to exposure and non-exposure to the stimulus.
3. High scorers on the Anxiety Sensitivity Index will have a lower initial pain tolerance than low scorers on the Anxiety Sensitivity Index.
4. High anxiety sensitivity will lead to increased tolerance for the pain stimulus following exposure when compared to low anxiety sensitivity.
5. High anxiety sensitivity will lead to reduced tolerance of the pain stimulus following avoidance when compared to low anxiety sensitivity.

CHAPTER FIVE

METHOD

5.0 PARTICIPANTS

100 students were recruited on a voluntary basis from Massey University. Compensation for time and other costs of \$10 was paid. 10 sets of data were excluded due to tolerance scores exceeding the upper limit (24.5mA) of the pain stimulus. 47 males and 43 females comprised the remaining pool of 90 participants. The mean age was 22 years ($SD = 5.2$), ranging from 18 to 41 years. Prior to participation, volunteers read an information sheet describing the requirements of the study and signed a consent form (see Appendices A and B). In order to control for demand characteristics, participants were not aware of the true nature of the experiment. They were led to believe that the main interest of the study was the effect of task performance on pain perception. Volunteers were not included for the study if they had participated in a pain study previously, or if they had any contraindicating conditions that would put them at risk or distort results, as determined by a medical checklist (see Appendix C). Participants were informed that they were free to withdraw from the study at any time.

5.1 APPARATUS.

a. The pain stimulus

Potassium iontophoresis, the application of potassium ions (K^+) through the epidermal barrier of the skin, was used as the experimental pain stimulus (Benjamin & Helvey, 1963). Iontophoresis (ion transfer) is the movement of ions under the influence of an applied DC electric current (Tyle & Kari, 1988). By making the body part of the DC circuit, the potassium ions at the anode are able to move through the epidermal layer. The release of intracellular potassium ions occurs naturally during tissue trauma (Benjamin 1959; Uchida & Murao, 1974), and is associated with pain. By introducing potassium ions iontophoretically, nerve fibres are stimulated in a non-invasive, non-damaging manner (Benjamin & Helvey, 1963; Humphries, Long, & Johnson, 1994).

Potassium iontophoresis was selected for use in the current study as it does not have many of the problems inherent in other experimental pain stimuli, such as cold pressor pain and thermal pain (Benjamin & Helvey, 1963; Edens & Gil, 1995), and possesses many of the characteristics required of an experimental pain stimulus.

Firstly, the sensation produced by the stimulus is clearly identifiable as pain (Humphries et al., 1994). Subjective reports range from a pricking, stinging sensation at low intensities to an intense burning at higher levels (Benjamin & Helvey, 1963; Humphries et al., 1994).

Secondly, the pain intensity can be precisely controlled and measured, and the intensity can be rapidly changed. There is a linear relationship between the number of ions administered and the degree of pain intensity (Benjamin & Helvey, 1963; Humphries et al., 1994). The number of ions transferred depends upon the duration and level of the current, and is independent of skin resistance (Benjamin & Helvey, 1963).

Thirdly, the application of potassium ions is not damaging, even at the highest tolerable level of pain (Benjamin & Helvey, 1963). Carry-over effects, such as lingering residual pain and inflammation are relatively small, as the stimulus can be ramped off quickly (Humphries, Johnson, & Long, 1996). This is advantageous in experimental designs which involve repeated presentations of the stimulus.

Finally, experimental data indicate that while between-subject variability in stimulus tolerance is large, within-subject variability is relatively small (Benjamin & Helvey, 1963; Voudouris 1981, cited by Voudouris, Peck, & Coleman, 1985). This intra-subject reliability is particularly important in the context of the present study, as the experimental manipulations are predicted to change tolerance levels.

The pain stimulus was delivered by an iontophoretic pain generator designed and developed by the Massey University School of Psychology. This appliance consisted of a computer controlled constant-current power source, and was designed to deliver a selected current ranging between 0 and 24.5mA. The amount of potassium ions delivered was directly proportional to the applied current.

The placement of the electrodes used in the study was similar to that described by Benjamin and Helvey (1963) and Voudouris et al. (1985), and have been used successfully in previous studies at Massey University (Humphries et al., 1994; Johnson, Breakwell, & Douglas, 1998). Two sets of apparatus were used, as both forearms were used in the study.

The anode consisted of a circular silver plate (4.5 cm diameter) fitted inside a plastic ring, which was attached to the volar surface of the forearm by means of elastic bands (see Figure 4). An electrical wire ran from the plate through a hole in the ring to the DC power source. The bowl formed by the ring and the underlying skin was filled with sufficient potassium chloride solution (KCl) to cover the plate (w/v 3% KCl in distilled water). To reduce seepage of the solution between the bowl and the arm, the KCl solution was turned into a gel by adding 1.75g of biological grade agar to every 200mls of heated KCl.

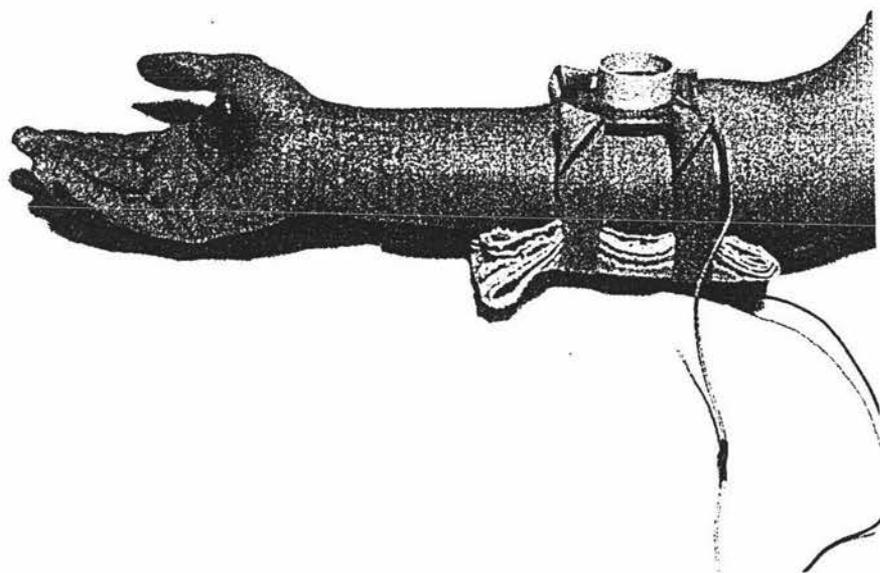


Figure 4. Electrode placement. The anodal bowl containing the potassium chloride gel is on the volar surface of the arm, with the opposing cathode plate covered with saline-saturated gauze on the dorsal surface of the arm.

The cathode was a 4cm x 13cm silver plate, with an electrical wire to the power source, covered with saline saturated medical gauze (4% w/v sodium chloride in distilled water). The medical gauze prevented direct skin contact with the cathode, thereby precluding the possibility of electrical skin burns. The saline solution was used to maintain conductivity between the arm and the plate. This cathode was attached to the dorsal surface of the arm, directly opposite the anode, with the same elastic bands.

b. Discrimination Task

A simple auditory discrimination task was included in the study, primarily to provide a method by which the avoidance group could avoid pain. By performing accurately on this task, they could prevent the occurrence of the pain stimulus. The other two groups also performed the task, to maintain equivalence between groups.

A series of 300 tones, with equal numbers of high (750 Hz) and low (250 Hz) pitched tones, were emitted by the internal speaker of the computer. These tones were easily distinguishable, and were presented in a quasi-random sequence: no more than four tones of the same pitch were presented consecutively. Tones were of 200ms in duration, with an inter-tone interval that varied randomly between three settings of 1500, 2000, and 2500ms, in order to reduce anticipatory responding. Participants responded by pressing the appropriate button on a two-button console with the thumb. Because the anodal bowl restricted movement of the arm, the console was strapped to the middle and index fingers of the arm not receiving pain stimulation, buttons facing palm-up. Two sets of 150 tones were used, with a short interval between, in an effort to reduce fatigue or boredom. The sets were comprised of the same sequence of tones, each arranged in reverse order of the other. The order of presentation of these two sets was counterbalanced. Reaction times and errors made were recorded by computer.

c. Measures

i. Pain tolerance measurement

Tolerance tests were used to gain measures of initial and subsequent pain tolerance. For these tests, a computer controlled¹ staircase configuration of pain increment was used, known as the Method of Limits (Engan, 1971). In this method, each response

Footnote 1: All computer software for the running of the pain stimulus, including the staircase, was written by Dr. S.A. Humphries, School of Psychology, Massey University.

determines the intensity of the next stimulus. This is a sensitive procedure that is able to obtain a highly accurate measure of pain tolerance, as it is able to calculate tolerance to within 0.05 mA. To establish tolerance to this degree of specificity using a procedure which uses pain increases of equal intervals would require the presentation of a prohibitive number of pain trials. To illustrate, following the first presentation of the pain stimulus, a response that the next pain trial should be higher in intensity results in an intensity increment of 1 mA. Following two consecutive responses in the same direction (e.g. that the pain should be higher), the increment increases by 2 mA. Each 'step' then increases incrementally by 2mA until a response is made in the opposite direction (that the pain should be lower). The next trial pain decreases in intensity by 1 mA. Alternating responses (more pain, less pain, more pain etc.) result in progressively smaller changes to the pain intensity. Table 1 shows response patterns over 16 trials for a hypothetical pain tolerance of 7.85mA.

Each tolerance test consisted of 16 presentations (trials) of the pain stimulus. Preliminary trials indicated that this was the minimum number of presentations needed to obtain a stable and reliable tolerance score. The measure of tolerance was obtained by calculating the average pain intensity of the last four trials, which was recorded by the computer. Participants responded to each presentation, indicating whether the next pain stimulus should be higher or lower in intensity. Each presentation ramped up over 500ms, remained at a constant for 3000ms, and ramped down over 500ms, a total duration of 4000ms. The current was ramped to prevent the sensation of electric shock which has been associated with sudden current changes (Balogun, 1986).

Table 1: Configuration of staircase tolerance test. Hypothetical response pattern of an individual with a tolerance of 7.85 mA.

Trial	Pain Level	Response	Change to next pain level
1	1 mA	More pain	+ 1 mA
2	2 mA	More pain	+ 1 mA
3	3 mA	More pain	+ 2 mA
4	5 mA	More pain	+ 2 mA
5	7 mA	More pain	+ 2 mA
6	9 mA	Less pain	- 1 mA
7	8 mA	Less pain	- 1 mA
8	7 mA	More pain	+ 0.5 mA
9	7.5 mA	More pain	+ 0.5 mA
10	8.0 mA	Less pain	- 0.25 mA
11	7.75 mA	More pain	+ 0.15 mA
12	7.90 mA	Less pain	- 0.10 mA
13	7.80 mA	More pain	+ 0.10 mA
14	7.90 mA	Less pain	- 0.10 mA
15	7.80 mA	More pain	+ 0.10 mA
16	7.90 mA	Less pain	

ii. The Anxiety Sensitivity Index (ASI)

The Anxiety Sensitivity Index (ASI; Reiss et al., 1986) was used as the measure of anxiety sensitivity (see Appendix D). It is a widely used 16-item self-report inventory that assesses fears of anxiety and anxiety sensations, and is intended for use with both clinical and non-clinical populations. Examples of items include 'When I notice my heart is beating rapidly, I worry that I might have a heart attack' and 'it scares me when I feel faint'. Each item is rated on a 5-point scale, ranging from *very little* (scored as zero points) to *very much* (scored as four points). The AS score is the sum of the scores on the 16 items. The ASI has demonstrated good psychometric properties. Internal consistency, as measured by Cronbach's alpha coefficient, has been estimated at .80 (Telch et al., 1989) and at .88 (Peterson & Heilbronner, 1987). Adequate test-retest reliability has also been reported, ranging between 0.71 and 0.75 (Reiss et al., 1986). Maller (1988; cited in Reiss, 1991) compared ASI scores taken in 1984 and in 1987 and found a correlation of 0.71. This suggests that the ASI measures a stable personality factor, rather than a transitory state. In addition, the ASI has been shown to measure a unique construct. The hypothesis that AS is simply trait anxiety (Lilienfeld, Jacob, & Turner, 1989) has been refuted by evidence that has demonstrated independence of these two constructs (Peterson & Heilbronner, 1987) and studies in which AS explains clinical phenomena not explained by trait anxiety (McNally, 1989; Reiss et al., 1986). Other studies have provided support for the validity of the ASI by showing that anxiety disorders, and a variety of fears, including fear of pain, are associated with elevated AS (Asmundson & Norton, 1995; McNally & Louro, 1992; Rapee et al., 1988).

The finding that females tend to yield higher scores on the ASI than males has been noted repeatedly in the literature (Reiss et al., 1986; Stewart, Taylor, & Baker, 1997). For example, Peterson and Heilbronner (1987) found in a sample of 122 college students, the mean score for males was 19.7, whereas the mean score for females was 23.6. This difference was explained in terms of a gender difference in the valence of anxiety symptoms, or a gender difference in the willingness to report sensitivity to anxiety symptoms.

iii. Self-report measure

A numerical Graphic Rating Scale (GRS) was used to assess changes in pain-related fearfulness (Jensen & Karoly, 1992). Scales of this type have been used successfully in

the measurement of pain-related variables in previous studies (Crombez, Eccleston, Baeyens, & Eelen, 1996; Crombez et al., 1998). The scale asked participants to rate their level of fearfulness during the second set of tolerance tests as compared to the level experienced during the first set of tolerance tests. The GRS was anchored by -5 (less fearful) and +5 (more fearful).

5.2 PROCEDURE

a. Experimental Design

The study had a mixed-experimental design, incorporating both between- and within-subject components. Tolerance change was compared between groups, as a function of the experimental manipulation. However, the measure of tolerance change itself was obtained by taking repeated measures of tolerance from each participant. This use of repeated measures is appropriate given the large inter-subject variability in tolerance associated with potassium iontophoresis (Benjamin & Helvey, 1963).

The purpose of the study is to determine the effect of exposure to painful stimulation on fear of pain, and subsequent tolerance of that stimulus. However, it is likely that repeated painful stimulation of the same site may lead to increased sensitivity to pain at that site. This would represent a confound in the present study. If tolerance to the stimulus decreased, it would be unclear whether this decrease represented a response to the experimental manipulation, or simply a reflection of sensitisation of the site. For this reason, it was decided that the tolerance tests would be conducted on one arm (which will be referred to as the experimental arm), and the manipulation (exposure, non-exposure or avoidance) would be carried out on the other (referred to as the manipulated arm). The site of administration of potassium ions and the nature of the stimulus was identical for both arms. Changes in fear of pain due to exposure or avoidance was expected to generalise between arms, as it is a cognitive process not thought to be associated with a particular site.

Pain intensity during the exposure and avoidance conditions was delivered at 80% of the initial tolerance level. Because laterality effects have been found for pain responding (Wolff & Jarvik, 1964), it could not be assumed that the tolerance of one

arm would be the same as that of the other. For this reason, it was necessary to take tolerance tests of both arms. For the experimental arm, the purpose of the initial tolerance test was to establish a baseline measure of tolerance. For the manipulated arm, the initial tolerance test was given so that an appropriate level of pain could be calculated and administered during the exposure condition. Following the manipulation, a second tolerance test was given to each arm. For the experimental arm, this was in order to gain a post-experimental measure of tolerance, which would then be compared with the first tolerance test of that arm. This provided the dependent variable 'tolerance change'. For the manipulated arm, the second tolerance test was given so that sensitisation effects, if present, could be evaluated. This would be indicated if, within the same condition, tolerance changes were markedly different between arms. To reduce the effects of sensitisation, the initial tolerance tests, the manipulation, and the subsequent tolerance tests were presented in an alternating sequence. Table 2 shows the sequence of the research.

Table 2. Research Sequence.

Arm 1 (manipulated arm)	Tolerance test 1
Arm 2 (experimental arm)	Tolerance test 1
Arm 1 (manipulated arm)	Condition 1, 2, or 3.
Arm 2 (experimental arm)	Tolerance test 2
Arm 1 (manipulated arm)	Tolerance test 2

The arm that was used as the experimental arm was counterbalanced across groups: for half the participants the dominant arm was the experimental arm, and for the other half, the non-dominant arm was used. This was to counteract possible laterality effects.

Anxiety sensitivity was also counterbalanced between groups, so that each of the three conditions had equal numbers of high and low scorers on the Anxiety Sensitivity Index. Previous studies have established AS groups on the basis of normative data (Asmundson, Norton, Wilson, & Sandler, 1994; Asmundson, Norton, & Veloso, 1998). These studies have defined high AS as ASI scores of greater or equal to 30, medium AS

as scores of between 30 and 12, and low AS as scores of less than or equal to 12. However, in the present study, ASI scores were generally lower than the normative data. Only two participants fulfilled the normative criterion for high AS. In an effort to find some measure of low and high AS within the sample, the ASI scores were divided into three equal groups, with participants scoring in the top third classified as high AS, and those scoring in the bottom third classified as having low AS. Although studies have suggested possible gender differences in AS (Reiss et al., 1986; Stewart, et al., 1997), the distribution of ASI scores for both males and females was very similar, with the same median. For this reason, it was not necessary to divide the ASI scores according to gender. In addition, gender was counterbalanced between groups.

b. Experimental Session

Participants were assigned to one of three conditions; exposure, non-exposure, or avoidance. This was done randomly, within the constraints of the counterbalancing procedures mentioned previously. During the performance of the discrimination (tone) task, the exposure group received a pain stimulus every seventh tone, a total of 42 pain stimulations. The non-exposure received no pain, and the avoidance group received a variable number of pain stimulations, depending upon their accuracy on the task. If an incorrect response was made, or a response was made outside the response window (1500ms), a pain stimulus was delivered. By responding with 100% accuracy, the delivery of the pain stimulus could be avoided completely. The intensity of the pain stimulations for the exposure and avoidance groups during the tone task was 80% of each participant's tolerance for that arm (the manipulated arm). The duration (4000ms) and ramp-times of the pain stimulus were identical to the pain stimuli used in the tolerance tests.

Prior to arrival at the experimental laboratory, each participant had read the information sheet and completed the Consent Form, Medical Checklist, and the ASI. On arrival, further information was elicited, such as age and hand dominance. Before attaching the electrodes, both forearms were lightly scrubbed with a soapy wash, and rinsed. The volar surfaces were then further cleaned with a sterile alcohol swap. The purpose of this procedure was reduce and stabilise skin resistance, in order to optimise consistency of pain levels. The participant was seated at a table with both forearms resting palm-up on

the table. The electrodes were attached to both arms, and the anodal bowls filled with potassium-chloride gel.

Prior to the first tolerance test of the manipulated arm, two initial pain 'ramps' were administered, in order to further reduce skin resistance, and to familiarise the participant with the pain stimulus. The pain ramp consisted of a continuous and rapid increase in pain intensity from 0 mA to a maximum of 24.5 mA. The participant was able to terminate the ramp by pressing a button on a console. The intensity of the stimulus would then rapidly decrease back to 0 mA. Because both hands were positioned palm-up on the table, the console was placed upright directly next to the fingertips of the opposite hand. By moving the hand forward slightly, the participant was able to engage the button without disturbing the anodal bowl. The following instructions were given:

" I will now give you the first pain stimulus. It feels like a pricking or stinging at low levels, and a burning sensation at higher levels. The pain cannot damage your skin or cause you any harm. The sensation will start at a very low level, and increase quite quickly. When you want it to stop, press this button, and the pain will immediately drop away. The purpose of this ramp is to reduce the resistance of your skin, and give you a chance to see what it feels like. It is not the tolerance test, and the level you reach will not be recorded. You will feel this on your left/right (whichever appropriate) arm. Are you ready?"

The level reached on the second ramp was divided by 3 to provide an entry-level intensity for the staircase tolerance test. The participant was then given the first tolerance test on the same arm. The following instructions were given:

" Now I am going to give you a tolerance test on the same arm. What is meant by tolerance is the highest level of pain that you are willing to take, your limit. This test is different from the ramp that you have just had. You will feel a pain at a very low level. It will last 4 seconds, and then you will hear a beep. Then I want you to respond using this console. If the pain is below your limit, I want you to push this button marked 'more pain'. The next 4 seconds of pain

will then increase slightly in intensity. Keep on pressing the 'more pain' button after each pain until you reach your limit. Then press the button marked 'too much pain'. The next 4 seconds of pain will be at a slightly reduced intensity. Then decide with each pain whether it is above or below your limit, and press the appropriate key. There are 16 pain trials altogether. Eventually you should be pushing the buttons alternately, as we narrow in on your tolerance level. The jumps are relatively large at first, and get smaller and smaller as you zero in on your tolerance level. The 'STOP' button is for your peace of mind; it shuts down the pain apparatus. Do you understand? Are you ready?"

The average of the last four pain levels was recorded as the tolerance score. The electrical wires were then detached from the generator, and the wires from the other arm connected. The console was also moved to the opposite hand. The procedure was then repeated for the opposite arm, with the following instructions:

"I am now going to test your other arm. First, I'll give you the two ramps to start. Remember to push this button."

"Now I will give you the staircase tolerance test. I want you to remain as consistent as possible with regards to the sensation you decided was your tolerance. That is, I want you to use the same criteria for what you're willing to tolerate. Do you understand? Are you ready?"

The participant was then introduced to the auditory discrimination task. The 2-buttoned tone response console was attached to the fingers of the experimental arm with a velcro strap. The electrode wires of the manipulated arm were connected to the generator. The following instructions were given to all participants:

"You will now be given a task to perform. You will hear a series of tones. There are two tones; a high pitched one and a low pitched one. After you hear each tone, I want you to respond by pushing one of these two buttons. There are 300 tones altogether, and they are presented in two blocks of 150 tones, so

there will be a short break in between. The tones are presented quickly, but there is enough time for you to respond. Respond as quickly and as accurately as you can. I'll give you a practice of 20 tones before we start. Because this is a practice, you will get feedback about your accuracy. If you press the wrong button, or do not press any button, you will hear a shrill beep. You will also hear a beep if you respond too slowly. You have 1.5 seconds to respond. You will not feel any pain during this practice, and your performance will not be recorded. Are you ready? The first tone you hear will be high pitched. "

If the participant did not achieve an accuracy level of at least 60% (12 of 20 tones), a second practice session was given. This is because the task was not intended to be difficult, or to represent a distracter from the pain stimuli. The following instructions were then given:

"Very good. I will now give you the first set of 150 tones. You will not hear the shrill beep if you make a mistake."

Depending upon which condition the participant was assigned to, the following instructions were given:

1. **Exposure:** "You will feel a pain on your right/left (manipulated) arm every seventh tone".
2. **Non-exposure:** "You will not feel any pain during this task".
3. **Avoidance:** "If you push the wrong button you will immediately feel a pain on your left/right arm. If you do not respond at all, you will feel a pain. If you respond too slowly, you will feel a pain. These pain presentations will each last for 4 seconds, and will be at 80% of the intensity you indicated was your tolerance for that arm. Are you OK with that? Respond as quickly and as accurately as you can. Are you ready?"

The second set of tones was presented following a 60-second interval, with the following instructions:

"I will now give you the second set of 150 tones. Are you ready?"

Following the tone task, the tone response console was removed from the fingers. The electrical wires of the manipulated arm were disconnected, and wires of the experimental arm were attached to the generator. The pain response console was placed in position, by the fingertips of the manipulated arm. The participant was then told:

"I am now going to test your tolerance on your left/right (experimental) arm. It will be the same as the first test of this arm, except there won't be the continual ramps; we're going straight to the staircase. Do you remember? Are you ready?"

After this test, the second tolerance test was given to the manipulated arm. The appropriate wires were attached to the generator, and the console was moved to the hand of the experimental arm. The instructions were the same as for the previous test.

The electrodes were removed from the participant's arms, and they were given the opportunity to wash and dry their arms. The participant then completed the GRS scale. Finally, the participant was thanked for their time, paid \$10, and told that a statement of the results of the study would be sent to them.

c. Post-Experimental Follow-up.

A debrief statement was sent to each of the participants on completion of the analysis of results. This statement outlined the rationale of the study, addressed the deception entailed in the presentation of the study, and briefly reviewed the results (see Appendix E).

5.3 STATISTICAL ANALYSIS

All data were analysed using SPSS.PC forWindows, version 8.0.0. The between-subjects data were analysed with one-way analyses of variances (ANOVAs) and independent-samples t-tests. Two-way ANOVAs were used to test for interactions

between conditions and anxiety sensitivity level. For the t-tests, when Levene's Test for Equality of Variances indicated that equal variances of the groups could not be assumed, degrees of freedom and level of significance for unequal variances were reported.

CHAPTER SIX

RESULTS

Tolerance changes for all analyses were calculated by subtracting the first tolerance score from the second tolerance score. A change in tolerance from 2 mA to 3 mA is more substantial proportionally than a tolerance change from 22 mA to 23 mA, even though the change in both cases is 1 mA. For this reason, the tolerance change in all analyses was computed in terms of percentage change:

$$\text{Tolerance Change (\%)} = \frac{\text{Tolerance 2} - \text{Tolerance 1}}{\text{Tolerance 1}} \times 100$$

The mean tolerance change for experimental arm for the sample overall was 13.94 ($N = 90$, $SD = 25.70$). A one-sample t-test showed that this change was significant ($t(89) = 5.15$, $p < .001$).

6.0 PRIMARY ANALYSES

All primary analyses, those concerned directly with the hypotheses, were conducted on data yielded from the experimental arm (the arm which did not receive pain stimuli during the conditions; see section 5.2a).

a. Exposure, Non-Exposure and Avoidance

Independent-samples t-tests were conducted to test for differences in tolerance change between the three experimental conditions (exposure, non-exposure, and avoidance). Table 3 shows the means and standard deviations of tolerance change following each condition. First, tolerance change was compared following exposure and non-exposure. The difference between these means was not significant ($t(51.75) = 0.59$, $p = .56$). Thus, the first hypothesis, that exposure to the pain stimulus would increase pain tolerance when compared to non-exposure to the stimulus, was not supported.

Secondly, the effects of exposure and avoidance were compared with respect to tolerance change. Participants in the avoidance condition received a mean of 5.2 pain

stimuli ($SD = 9.8$), ranging from 0 to 44. 82.2% of these stimuli were delivered due to an incorrect response (error type a), and 17.8% were delivered due to a response that was too slow ($>1500ms$) (error type b). Over 80% of the participants in this group received five or less pain stimuli. The remaining participants received between 10 and 44 stimulations. To confirm that this group was not affected by the amount of pain they received, a preliminary one-way analysis of variance (ANOVA) was undertaken to ascertain whether those in the avoidance condition that received no pain ($n = 9$) differed with respect to subsequent tolerance change from those that received a moderate number of pain stimuli (between 1 and 5 inclusive; $n = 17$) and those that received a considerable number of pain stimuli (between 10 and 44 inclusive; $n = 5$). This test showed no significant differences between these sub-groups ($F(2, 28) = 0.45, p = .64$), and so the avoidance group is considered to be homogenous for the purpose of analysis.

No significant effect of tolerance change was found when the avoidance condition was compared with the exposure condition ($t(53.91) = 0.88, p = .38$), or with the non-exposure condition ($t(58) = 0.26, p = .80$). Thus, the second hypothesis, that avoidance would lead to a decrease in tolerance when compared to exposure and non-exposure to the stimulus, was not supported by the data.

Table 3. Mean tolerance change (%) following the three experimental conditions.

CONDITION	TOLERANCE CHANGE (%)		
	<i>n</i>	Mean	Standard Deviation
Exposure	30	10.80	20.4
Non-exposure	29	14.54	27.3
Avoidance	31	16.42	29.0

Results of a one-way ANOVA showed that changes in self-reported fear as measured by the GRS also did not significantly differ between conditions ($F(2, 87) = 1.20, p = .31$). Fearfulness decreased by a mean of 2.05 points ($SD = 1.85$) for the exposure condition, by 1.45 points ($SD = 1.72$) for the non-exposure condition, and by 1.42 points ($SD = 1.78$) following the avoidance condition.

b. The Effects of Anxiety Sensitivity.

ASI scores of the sample had a mean of 13.80 ($SD = 6.16$), a median of 12.0, and ranged between 3 and 32. Scores of 11 or less were defined as low AS, and scores of 16 or more were defined as high AS. Because the more extreme scores were of interest, the middle scores were excluded from the analyses. The high and low AS groups were approximately evenly distributed among the conditions (see Table 4).

Table 4. Distribution of low and high AS among the three conditions.

CONDITION	ANXIETY SENSITIVITY GROUP (<i>n</i>)		
	Low	High	Total
Exposure	9	11	20
Non-exposure	10	10	20
Avoidance	11	9	20
Total	30	30	60

The hypothesis that high scorers on the ASI would have lower initial tolerance than low scorers was supported by the data. High scorers tolerated a mean of 8.71 mA ($SD = 5.60$) during the initial tolerance test, whereas low scorers tolerated a mean of 11.76 mA ($SD = 5.55$). An independent-samples t-test revealed that this difference was significant ($t(58) = 2.12, p = .04$).

The hypothesis that high AS would lead to increased tolerance of the pain stimulus following exposure when compared to low AS was also supported. Following exposure, tolerance increased in the high AS group, and decreased in the low AS group. An independent-samples t-test showed that this difference was significant ($t(18) = 2.06, p = .05$). Table 5 shows mean tolerance change for high and low AS following the three conditions.

However, the hypothesis that, following the avoidance condition, high AS would lead to decreased tolerance of the pain stimulus when compared with low AS, was not

supported. Tolerance decreased in the low avoidance group, but increased by nearly 35% in the high AS avoidance group. An independent-samples t-test showed that this tolerance difference was significant ($t(11.27) = 3.41, p = .008$). Rather than showing a decrease in tolerance following avoidance, the high AS group showed a substantial and significant increase in tolerance.

An additional independent-samples t-test showed that no significant differences in tolerance change for high and low AS existed following the non-exposure condition ($t(18) = 0.69, p = .50$).

Table 5. Percentage tolerance change following the experimental conditions for low and high anxiety sensitivity.

CONDITION	TOLERANCE CHANGE (%)			
	Low AS		High AS	
	Mean	SD	Mean	SD
Exposure	- 4.12*	12.3	12.84*	21.9
Non-Exposure	17.26	33.3	8.41	23.2
Avoidance	- 3.01**	16.2	34.78**	32.2

* $p \leq .05$ ** $p < .005$

Overall, the combined data for all three conditions showed that the high AS group had a mean tolerance increase of 17.94% ($SD = 27.35$) and the low AS group showed a mean tolerance increase of 3.41% ($SD = 23.98$). Whereas the low AS group showed only a small increase from the first to the second tolerance test, regardless of condition, the high AS group showed a substantial increase in tolerance from the first to the second tolerance test. An independent-samples t-test revealed that the difference between these overall tolerance changes for high and low AS was significant ($t(58) = 2.19, p = .03$).

Additional analyses were then conducted to test for interactions between the AS groups and the three experimental conditions. A two-way analysis of variance, with experimental condition and AS group (high and low) as between-subject factors, and tolerance change as the dependent variable, revealed a significant interaction ($F(2, 54) = 4.62, p = .01$). Additional two-way ANOVAs were undertaken in order to identify which specific conditions were causing the interaction. It was revealed that the interaction occurred between the non-exposure and avoidance groups ($F(1, 36) = 7.57, p = .009$) (see Figure 5). In the low AS group, tolerance increased following non-exposure, and decreased following avoidance. In contrast, the high AS group showed only a slight increase following non-exposure, and a relatively large increase following avoidance.

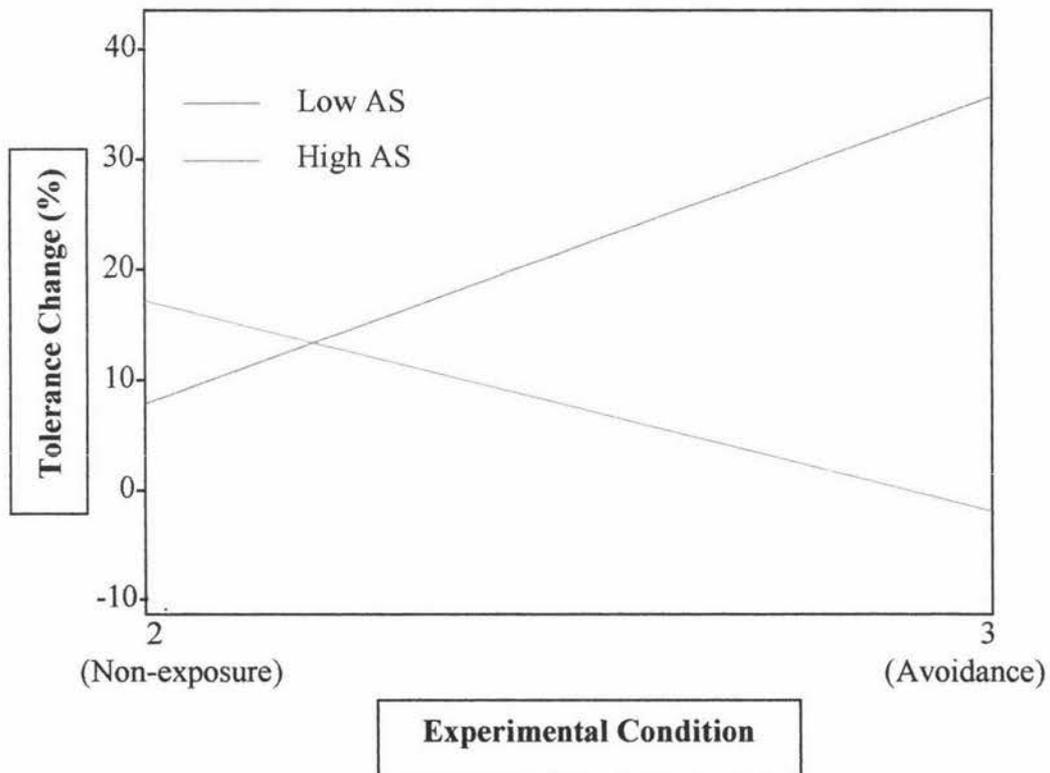


Figure 5. Mean tolerance change (%) for the high anxiety sensitivity and low anxiety sensitivity groups in the non-exposure and avoidance conditions.

Finally, results of a two-way ANOVA revealed no significant differences in self-reported change in fear level, as measured by the GRS, between high and low AS ($F(1, 54) = 0.24, p = .63$), or between AS group and condition ($F(2, 54) = 0.51, p = .60$).

The means and standard deviations for this analysis are presented in Table 1 of Appendix F.

6.1 ADDITIONAL ANALYSES

A number of additional analyses were undertaken in order to further explore the data.

a. Comparison of tolerance change between manipulated arm and experimental arm.

Tolerance change following the experimental conditions was compared between the experimental arm, and the manipulated arm. This was to ascertain whether a sensitisation effect had occurred on the manipulated arm due to repeated pain stimulations. Tolerance changes for the manipulated arm were 10.59% ($SD = 29.09$) for the exposure condition, 13.64% ($SD = 31.29$) for the non-exposure condition, and 10.77% ($SD = 38.36$) for the avoidance condition. A one-way ANOVA showed no significant differences between these changes ($F(2, 87) = 0.08, p = .93$). Furthermore, a mixed-design two-way ANOVA showed no significant differences in tolerance change between the manipulated and experimental arms following the conditions ($F(2, 87) = 0.25, p = .78$).

b. Comparison of Reaction Time Data.

Independent-samples t-tests were conducted to investigate whether reaction times (RT) to the tone task were affected by the experimental conditions. Table 6 shows the mean RT for each condition.

Table 6: Mean reaction time to the tone task across conditions.

CONDITION	REACTION TIME (ms)	
	Mean	Standard Deviation
Exposure	522.03	78.9
Non-exposure	483.27	69.2
Avoidance	530.73	102.7

A significant difference was found in mean RT between the exposure and non-exposure conditions ($t(56) = 1.98, p = .05$), and between the non-exposure and avoidance conditions ($t(57) = 2.06, p < .05$). Reaction time was increased in both the exposure and avoidance conditions, when compared with the non-exposure condition.

To check whether accuracy on the task was influenced by reaction time, an ANOVA was conducted to test for an interaction between condition and number of errors made on the task. Two scores (41 and 42 errors) were removed from the data for this analysis, as they fell beyond the third standard deviation of error scores, and were thus considered to be extreme, non-typical outliers. The remaining 88 scores ranged between 0 and 26 errors. A significant interaction was not found ($F(2, 85) = 0.14, p = .87$). Additional ANOVAs confirmed that the incidence of type a (inaccurate response) and type b (slow response) errors did not vary systematically across conditions: ($F(2, 85) = 0.08, p = .93$) and ($F(2, 85) = 0.84, p = .44$), respectively. The error data is presented in Table 2 of Appendix F.

To test whether the increased RT in the avoidance condition was associated with painful stimulation, the mean reaction times for three subgroups within the avoidance condition, those that received no pain ($n = 9$), those that received a moderate number of pain stimuli (between 1 and 5 inclusive; $n = 17$), and those that received a considerable number of pain stimuli (between 10 and 44 inclusive; $n = 5$), were compared. Table 7 shows the mean RT for each subgroup of the avoidance condition. A significant difference in RT between these groups was found ($F(2, 28) = 6.03, p = .007$).

Table 7: Mean reaction time to the tone task for three subgroups of the avoidance condition.

SUBGROUP	REACTION TIME (ms)	
	Mean	Standard Deviation
No pain	504.86	77.56
Moderate pain	507.28	72.55
Considerable pain	657.09	148.97

Independent-samples t-tests showed that the group that received a considerable number of pain stimuli during the avoidance condition had a significantly longer mean RT than the group that received no pain ($t(12) = 2.56, p = .025$), and the group that received a moderate number of pain stimuli ($t(20) = 3.17, p = .005$). Closer examination of the group that received a considerable number of pain stimuli showed that the stimuli were delivered predominately due to an incorrect response ($X = 21.00, SD = 14.31$), rather than due to a response that was too slow ($X = 3.79, SD = 1.89$).

The RTs of the avoidance condition subgroup that received a considerable number of pain stimuli were then compared with the RTs of the participants in the exposure condition. This was to test whether the administration of painful stimuli alone accounted for the extended RTs in these groups. An independent-samples t-test showed that the RT of the avoidance subgroup were significantly longer than the RTs of those in the exposure condition ($t(4.38) = 1.98, p = .004$).

c. Gender Differences.

Males and females differed in their initial (pre-manipulation) pain tolerance. The mean initial tolerance was 11.60 mA ($SD = 6.21$) in males and 9.14 mA ($SD = 4.52$) in females. Results of an independent-samples t-test showed that this difference was significant ($t(83.83) = 2.12, p = .03$).

A two-way ANOVA showed that no significant differences existed between males and females with regard to tolerance change following the conditions ($F(2, 87) = 1.62, p = .20$). This raw data for this analysis is presented in Table 3 of Appendix F.

CHAPTER SEVEN

DISCUSSION

7.0 REVIEW AND INTERPRETATION OF HYPOTHESES AND FINDINGS.

a. Exposure, Non-Exposure, and Avoidance.

The fear literature has provided ample evidence for the use of exposure therapy in attenuating fear, and in increasing confrontation of the feared stimulus (Craske et al., 1997; Vodde & Gilner, 1971). Treatment studies in chronic pain have also provided support for the efficacy of exposure therapy in decreasing fear and avoidance of painful activities and situations (Dolce et al., 1986; Doleys et al., 1982). The one study to date that analysed the effects of exposure to pain in an experimental setting similarly found that exposure to pain increased subsequent pain tolerance in chronic headache sufferers (Philips & Jahanshahi, 1985). However, this had not been tested in a non-clinical population. The first hypothesis predicted that exposure to the pain stimulus would result in increased confrontation (operationally defined as tolerance level) of that stimulus, when compared with non-exposure.

The notion that avoidance of feared pain stimuli leads to increased fear, and subsequently increased avoidance, has been oft-repeated in the chronic pain literature, although not directly tested in the area of pain. The second hypothesis therefore predicted that, compared with exposure and non-exposure conditions, participants who actively avoided the pain would show a decrease in pain tolerance.

However, these first two hypotheses were not supported. All three conditions resulted in comparable increases in tolerance level. Self-reported changes in fear also did not vary between the conditions, fear decreasing to a similar degree following all three conditions.

The failure of the exposure condition to increase pain tolerance when compared with non-exposure is perturbing, given the weight of evidence in support of the hypothesis. There are a number of possible explanations for this finding. Firstly, the tone

discrimination task that participants performed while receiving painful stimuli during the exposure condition may have acted as a distracter from the pain. Several participants in the exposure condition made the comment that the pain seemed less noticeable because they were concentrating on the tone task. Evidence from the fear literature suggests that distraction may reduce the efficacy of exposure procedures in reducing fear (Rodriguez & Craske, 1995). Related studies have shown that purposeful attending to the feared stimulus during an exposure-based treatment results in the greatest reductions in fear (Grayson, Foa, & Steketee, 1982). As suggested by Borkovec and Grayson (1980):

"objective presentation of stimuli does not guarantee functional exposure to those stimuli...events which interfere with or facilitate the subjects' awareness and/or processing of that information (the feared stimuli) will critically influence the effect of those procedures on a targeted emotional behavior" (p. 531).

It is thus possible that the tone task reduced the effectiveness of the exposure condition in reducing fear, and increasing tolerance, in the present study.

Another possible explanation for the lack of findings is that overall levels of fear of pain were too low to be further reduced, by any procedure. As participants were recruited on a voluntary basis, it is logical to assume that for this particular sample, initial levels of fear were initially low. However, it was expected that as pain is a naturally aversive stimulus (Melzack & Casey, 1968), a reasonable level of fear would exist prior to the manipulation. In support of this, levels of self-reported fear did decrease in both groups. Therefore, it appears that fear was reduced, but similarly following both the exposure and non-exposure conditions.

Results therefore indicate that tolerance was increased following the exposure condition, and fear was reduced, but not significantly more than the non-exposure condition. This suggests that the problem may have been with the non-exposure condition. One possible issue is that the tolerance tests may have constituted a

considerable degree of exposure in themselves. By the time the second tolerance test commenced on the arm of interest, the experimental arm, all participants had experienced a minimum of 32 pain stimuli, regardless of condition. This may have been sufficient exposure to confound the exposure condition. This would explain why both the exposure and non-exposure conditions displayed comparable increases in tolerance, and comparable decreases in fear of pain.

The interpretation of the lack of difference in tolerance change following the avoidance condition when compared with the exposure and non-exposure conditions is more complex. Although a number of researchers have theorised that avoidance exacerbates fear of pain, and increases subsequent avoidance (Fordyce, 1977; Lethem et al., 1983; H. Philips et al., 1987), this effect has not yet been demonstrated. Therefore, one possible interpretation is that no such effect of avoidance exists - that avoidance of pain does not lead to increased fear and avoidance. However, a lack of demonstrated effect in a study does not necessarily indicate that the effect does not exist (Ott & Mendenhall, 1985). In the context of this study, it would be precipitate to conclude that avoidance does not decrease tolerance, as other possibilities exist.

One such possibility is that avoidance does lead to a reduction in tolerance, but that this experiment failed to demonstrate this. As with the non-exposure condition, it is possible that the pain stimuli associated with the tolerance tests constituted enough exposure to undermine the avoidance manipulation. Further research is needed to clarify this issue.

b. Anxiety Sensitivity

Research has found that high levels of AS are implicated in fear of pain and pain-related avoidance, by amplifying sensations of anxiety that pain may produce, and promoting cognitive disruption (Asmundson & Norton, 1995; Asmundson & Taylor, 1996). It follows that avoidance of pain should be evident in terms of a lowered tolerance for pain. For this reason, it was predicted that participants with high levels of AS would show a lower initial tolerance for the pain stimulus than participants with low AS. This hypothesis was supported, even though the general level of AS among the participants was low.

High levels of pain-related fear were expected to be more modifiable, or amenable to change, than low levels of fear. Given that high AS is associated with pain-related fear, it was predicted that high AS participants would show increased tolerance for pain following exposure when compared to participants with low AS.

The effects of AS level on tolerance change following avoidance of the pain stimulus were also examined. It was thought that if the avoidance of pain promotes increased fear and avoidance of pain, that it would be especially marked in the high AS group, as this group has higher levels of initial fear. Thus, it was predicted that participants with high AS would show decreased tolerance of the pain stimulus following the avoidance condition when compared with participants with low AS. This hypothesis was not supported by the data. In fact, the high AS group showed a significant increase in pain tolerance, of nearly 35% of their initial tolerance. Self-reported fear levels decreased to a similar degree in the low and high AS groups.

At first glance, the results appeared to support the prediction that high AS leads to greater confrontation of pain following exposure to the pain stimulus than low AS. Following exposure, the high AS group showed a significantly larger increase in tolerance than the low AS group. However, contrary to prediction, tolerance also increased markedly for the high AS group following avoidance of the pain stimulus. Because tolerance increased for the high AS group across both the exposure and the avoidance conditions, the tolerance increase following exposure cannot be assumed to be the direct result of the exposure manipulation. Therefore, although the data support the prediction that high AS leads to increased tolerance following exposure when compared with low AS, the hypothesis cannot be accepted with confidence.

A possible interpretation may be found upon closer examination of the data. Tolerance increased in the high AS group following both the exposure and avoidance conditions. In contrast, the low AS group showed a decrease in tolerance following the exposure and avoidance conditions. When the tolerance change data was collapsed across all three conditions, an interesting result emerged. Overall, regardless of condition, the high AS group showed a large tolerance increase from the first to the second tolerance

test. The low AS group, however, showed only a small tolerance increase from the first to the second tolerance test. This could be interpreted in terms of the pain exposure associated with the tolerance tests, discussed earlier. The tolerance tests may have, in themselves, constituted a considerable degree of exposure. Consistent with the assumption that exposure would exert a larger effect on tolerance in those with high initial levels of fear than those with low initial levels, it appears that this exposure exerted more of an effect for those with high AS than those with low AS. This may indicate that chronic pain sufferers with high levels of anxiety sensitivity may particularly benefit from exposure therapy.

This is a possible explanation for the overall tolerance changes for the high and low AS groups, but does not address the specific pattern of changes within each group in response to the experimental conditions. An interaction was found between low and high AS and the non-exposure and avoidance conditions. The low AS group showed an increase in tolerance following the non-exposure condition, and tolerance decrease following the avoidance condition. In contrast, the high AS group showed the opposite effect, with a moderate tolerance increase following non-exposure, and a dramatic increase following avoidance. It is possible that for those with high AS, successful avoidance of the pain stimulus gave a sense of control over the pain, which in turn, reduced fear of the pain. This is consistent with the notion that avoidance of pain reduces fear and anxiety (Asmundson & Norton, 1995). However, it is generally argued that successful avoidance, and concomitant reductions in fear and anxiety, reinforce avoidance behaviour (Fordyce, 1977; Lethem et al., 1983). This was not apparent in the subsequent tolerance test; confrontation, rather than avoidance of pain, as indicated by tolerance increase, occurred in the high AS group following avoidance.

c. Experimental vs. Manipulated Arm.

Initial and subsequent tolerance tests were conducted on both arms. The arm that received pain stimuli during the exposure and avoidance conditions was referred to as the 'manipulated arm'. Changes in tolerance on this arm as a result of the experimental conditions would be problematic to interpret, as a change in tolerance could be due either to an effect of the experimental manipulation, or due to physical desensitisation due to repeated stimulation of the same site. For this reason, it was decided to analyse

tolerance changes of the arm that did not receive pain stimuli during the conditions, referred to as the 'experimental arm'. It was argued that, as reduction in fear was thought within the context of the present study to be a cognitive process, it would not be associated with a particular site of the body, and would generalise between arms.

Although not hypothesised, it was of interest to ascertain whether a sensitisation effect had occurred on the manipulated arm. If sensitisation had occurred, a reduced tolerance would be found following the exposure condition, and to a lesser extent, following the avoidance condition, as both these conditions received pain stimuli. However, when compared with the tolerance changes following these conditions of the experimental arm, no differences were found. Thus, no sensitisation effect occurred on the manipulated arm as a result of the experimental conditions.

d. Reaction Times and Errors.

Consistent with Melzack and Casey's (1968) motivational-affective analysis of pain, recent research has shown that pain demands attention, and disrupts on-going behaviour (Crombez, et al., 1996; Eccleston, 1994; Eccleston & Crombez, submitted). Although not of central importance to the current study, the effects of the pain stimuli on reaction times to the tone task were examined. Results showed that reaction times were significantly increased during exposure condition, when compared with the non-exposure condition. This suggests that the pain interrupted attention, and interfered with performance of the task, a finding congruent with previous research (Crombez et al, 1994; Eccleston, 1994). In addition, it was found that reaction times to the tone task were significantly increased during the avoidance condition, when compared with the non-exposure condition.

One possible explanation for this could be that, as the delivery of pain in this condition was contingent upon an incorrect response, reaction time was slower in this group in an effort to enhance accuracy. However, analysis of the frequency of errors in each condition revealed that a similar number of errors were made in all three conditions.

The possibility that reaction times were increased during the avoidance condition as a distracting effect of the pain stimuli administered was then examined. It was found that those in the avoidance condition who had received a considerable number of pain stimuli, due to errors made, showed a marked increase in reaction time when compared with those in the avoidance condition who had received no painful stimuli, or who had received a moderate number of pain stimuli. Further support was thus found for the notion that pain disrupts attention.

In addition, a further interesting finding was noted. The RTs of those in the exposure condition, who had received a total of 42 pain stimuli throughout the tone task, were compared with the RTs of those in the avoidance condition who had received a considerable number of pain stimuli, an average of 24 stimulations. The RTs of this subset of the avoidance group were significantly longer than the RTs of those in the exposure condition, even though the exposure condition had received more pain stimuli. The difference between the delivery of pain in these two groups is that in the exposure condition, the onset of pain was predictable (every seventh tone), whereas in the avoidance condition, the onset of pain was unpredictable - pain occurred only when an error had been made. Studies of the effect of temporal predictability of pain have found that unpredictable pain is more intrusive, and more demanding on attentional resources (Crombez, Baeyens, & Eelen, 1994; Matthews, Scheier, Brunson, & Carducci, 1980). Thus, during performance of the tone task, attention was disrupted by the painful stimulus, particularly when the onset of this stimulus was unpredictable.

e. Gender.

Research has yielded conflicting results on the impact of gender on pain tolerance. Weisenberg (1977), in a review of the literature, cited four studies of experimental pain that found no differences in pain tolerance between the genders, and three studies which found that males had a higher tolerance to pain than females. Results of the current study found that males tolerated significantly more pain than females during the initial tolerance tests. However, Levene and de Simone (1991) found that the gender of the experimenter had a significant impact on tolerance reports in male participants. Specifically, males reported a higher tolerance in the presence of a female experimenter,

than in the presence of a male experimenter. As the experimenter in the current study (myself) was female, the finding that males tolerated more pain than females may be due to this reason, rather than due to an inherent difference in pain tolerance between the genders.

7.1 METHODOLOGICAL ISSUES

A critical issue in this study was obtaining accurate estimates of tolerance. As already discussed, the staircase procedure of pain increment used in the tolerance tests was selected for its sensitivity and accuracy in generating tolerance (Engan, 1971). A stimulus duration of 4000ms was used in these tolerance tests for two reasons. Firstly, preliminary trials showed that shorter durations of the pain stimulus increased the likelihood that the limit of pain intensity, 24.5mA, would be reached before tolerance was indicated. This 'ceiling effect' occurred in approximately 10% of the participants in the current study, and this data was discarded. However, a longer stimulus duration, while eliminating this problem, was considered undesirable, as more pain overall would be administered during the tolerance tests..

The second reason that the duration of pain stimulus was set at 4000ms was so that a reasonable amount of pain could be administered to the individuals in the exposure group. Studies on exposure therapy for reducing phobic fears have shown repeatedly that short exposure durations are inferior to longer durations (Miller & Levis, 1971; Sue, 1975). As discussed, changing the duration of the stimulus markedly changed tolerance to the stimulus. As the members of the exposure group were administered pain stimuli of 80% of their tolerance, it was important that the duration of the exposure stimuli were of the same duration as the tolerance test stimuli, so that the appropriate exposure intensity could be calculated.

Thus, a stimulus duration of 4000ms during the tolerance tests seemed to provide the best balance between providing a sensitive test of tolerance, while keeping pain duration to a minimum, and providing an adequate duration of pain stimulation for use in the exposure condition.

However, this may have been problematic in the context of this study. Although those in the exposure condition were given 42 more pain stimuli than those in the non-exposure condition, every condition actually received 32 pain stimuli during the initial tolerance tests. This may have constituted a considerable degree of exposure, for every condition. It is possible that this contributed to the lack of difference in tolerance change found between the conditions. This is a problem inherent in research interested in the tolerance changing effects of exposure to pain. In order to expose participants to a level of pain subjectively considered as painful, tolerance tests must be taken. A possible solution, while retaining the iontophoretic administration of potassium ions as the pain stimulus, would be to develop tolerance tests that provide an analogue of the intensity to be used in the exposure procedure. For example, it may be found that tolerance to pain at a short duration (e.g. 500ms) has a linear relationship with tolerance at a higher duration (e.g. 5000ms). In this way, total exposure to pain during the tolerance tests could be kept to a minimum, and the appropriate level of intensity could be calculated for pain stimuli of longer durations needed in an exposure condition. As pain tolerance increases as the stimulus duration decreases, a maximum pain intensity of more than 24.5 mA would then be needed for the tolerance tests. Further research is needed to indicate whether a linear relationship exists between tolerance of stimuli of different durations.

A second methodological issue concerns the use of the tone discrimination task during the experimental conditions. The purpose of this task was to provide a means by which participants in the avoidance condition could avoid pain. By performing accurately on this task, they could prevent the occurrence of the pain stimulus. The exposure and non-exposure groups also performed the task, so that equivalence between groups would be maintained. In order to minimise the distracting effect of the task, an attempt was made to select tone pitches (250 and 750 Hz) that would be easily discriminable, while still providing a task for the participants in the avoidance condition to perform. This attempt to minimise the difficulty level of the task was successful, insofar as the mean number of errors made by the participants over all conditions was only 4.81 ($SD = 7.32$). This is a low error rate, considering the task required the discrimination of 300 tones. However, it is possible that the tone task served as a distracter from the pain

stimulus. Evidence from the fear literature suggests that distraction may reduce the efficacy of exposure procedures in reducing fear (Rodriguez & Craske, 1995).

It is thus possible that the tone task reduced the effectiveness of the exposure condition in reducing fear, and increasing tolerance, in the present study. In order to clarify this issue, two exposure groups, one attending to the pain, the other distracted from the pain, could be compared with regard to subsequent fear and tolerance change.

A third issue concerns the measurement of changes in fear of pain due to the experimental conditions. A numerical graphic rating scale was used (Jensen & Karoly, 1992), which has been used successfully in measurement of change in variables such as tension (Crombez et al., 1998) and attentional disruption (Crombez, Eccleston, Baeyens, & Eelen, 1996) in previous pain studies. The results from the GRF showed no significant differences in fear change following any of the analyses. However, tolerance changes were significantly different between the high and low AS groups, following the exposure and avoidance conditions. The lack of concomitant changes in fear, as measured by the GRS, indicates that either tolerance increased or decreased because of a process other than change in fear, or that the GRS was not a sensitive measure in the context of this study. It is possible that asking participants to rate their fear level during the second set of tolerance tests as compared to the level experienced during the first set was not adequately valid, as it required retrospective reporting on fear levels. A more appropriate method would be to take separate measures of fear of pain before, and after, the experimental conditions.

Finally, issues regarding the sample used need to be discussed. The vast majority of the research regarding chronic pain has been conducted using samples taken from a clinical population. Caution must be exercised in the interpretation of the results yielded from the present study, as it has been conducted with a non-clinical sample. As pointed out by Dworkin and Chen (1982), findings from laboratory studies are limited in their generality to a clinical population. For example, it is likely that issues regarding fear of pain and fear of (re)injury are more salient in the chronic pain population than in a non-clinical population. Exposure and avoidance may have more success in changing fear of pain when real and prominent fears exist. In addition, the mean ASI score for the

sample was 13.80 ($SD = 6.16$), which is probably lower than that found in the general population. Although normative data is not available in NZ, the mean ASI score reported for the the Reiss et al. (1986) normative sample was 15.4 ($SD = 8.1$). This difference is not surprising given that the present sample were comprised of individuals who volunteered for participation in a pain study. However, the effects of AS may have been lessened in the current study, given this overall low level of AS.

7.2 DIRECTIONS FOR FUTURE RESEARCH

In addition to the suggestions for future research already made, a number of other possibilities arise from the present study. The use of potassium iontophoresis as a pain stimulus is very useful in a laboratory setting, as it is safe and does not cause any physical damage (Benjamin & Helvey, 1963). However, this very factor reduces the generality between such non-threatening pain, and the potentially threatening and distressing low-back pain experienced by chronic pain sufferers. Future studies examining relative impact of exposure, non-exposure, and avoidance of pain on subsequent tolerance for pain in a sample of chronic pain sufferers may yield results that are more promising.

Secondly, it would be of interest to compare the relative efficacy of exposure to pain in increasing tolerance, with and without relaxation training. Research from the fear literature indicates that exposure based therapies are most effective under conditions of low arousal (Philips & Jahanshahi, 1985; Mathews, 1978).

Finally, the use of a longitudinal study in the study of factors contributing to chronic pain may yield useful results. Chronic pain problems are highly suited to this method, as the time span of the development of the syndrome is relatively short. Research has shown that those who have not recovered from an acute pain episode within three months are those who go on to become chronic pain sufferers (Philips & Grant, 1991). Klenerman et al. (1995) conducted a longitudinal study investigating the relative importance of psychosocial, demographic, and physical factors in the development of chronic low back pain following acute injury. A future longitudinal study could test whether variables such as anxiety sensitivity and pain catastrophising can reliably differentiate between acute pain sufferers who recover, and those who go on to become

chronic pain sufferers. In addition, the value of pain catastrophising in predicting outcome following acute injury could be evaluated. Pain catastrophising is difficult to include in a pain study utilising a non-clinical sample. Not only is the threat value of experimental pain likely to be perceived as minimal, even by participants high on measures of catastrophising, but only those with low levels of catastrophising are likely to volunteer.

7.3 RESEARCH SUMMARY

This study set out to investigate the effects of exposure, non-exposure, and avoidance of pain on subsequent confrontation, or tolerance, of the pain stimulus. Contrary to predictions, findings showed no differences between these conditions with regard to tolerance change. This was discussed in terms of a possible confounding factor, the exposure inherent the tolerance tests. It was also thought that the exposure condition itself may have been compromised by the distracting effects of the discrimination task performed during this manipulation.

The impact of anxiety sensitivity, an individual difference variable thought to be implicated in fear of pain and pain-related avoidance, was also investigated. It was found that, overall, participants with high AS showed a lower initial pain tolerance, which tended to increase significantly following the manipulation, regardless of condition, when compared to low AS participants. This was interpreted in terms of a general exposure effect; individuals with a high degree of fear showed a greater increase in tolerance following general exposure to the pain stimulus than individuals with a low degree of fear.

Findings additional to those hypothesised showed that attention to the discrimination task was interrupted during the presentation of the painful stimuli, particularly when the onset of the pain was unpredictable.

Finally, some possibilities for future research were discussed. A longitudinal design was considered to be of potential use in the study of etiological factors in chronic pain, and the exploration of the relative effects of exposure, non-exposure, and avoidance on pain tolerance within a clinical setting was recommended.

REFERENCES

- Anciano, D. (1986). The pain behaviour checklist: Factor analysis and validation. *British Journal of Clinical Psychology, 25*, 301 - 302.
- Anderson, D.B. & Pennebaker, J.W. (1980). Pain and pleasure: Alternative interpretations for identical stimulation. *European Journal of Social Psychology, 10*, 207 - 212.
- Arntz, A. (1996). Why do people tend to overpredict pain? On the asymmetries between underpredictions and overpredictions of pain. *Behaviour Research and Therapy, 34*, 545 - 554.
- Arntz, A., & Peters, M. (1995). Chronic low back pain and inaccurate predictions of pain: Is being too tough a risk factor for the development and maintenance of chronic pain? *Behaviour Research and Therapy, 33*, 49 - 53.
- Arntz, A. & van den Hout, M.A. (1988). Generalizability of the match/mismatch model of fear. *Behaviour Research and Therapy, 26*, 207 - 223.
- Arntz, A., van Eck, M., & de Jong, P. (1991). Avoidance of pain of unpredictable intensity. *Behaviour Research and Therapy, 29*, 197 - 201.
- Asmundson, G.J.G. & Norton, G.R. (1993). Anxiety sensitivity and its relationship to spontaneous and cued panic attacks in college students. *Behaviour Research and Therapy, 31*, 199 - 201.
- Asmundson, G.J.G. & Norton, G.R. (1995). Anxiety sensitivity in patients with physically unexplained chronic back pain: A preliminary report. *Behaviour Research and Therapy, 33*, 771 - 777.
- Asmundson, G.J.G., Norton, P.J., & Norton, G.R. (1999). Beyond pain: The role of fear and avoidance in chronicity. *Clinical Psychology Review, 19*, 97 - 119.

- Asmundson, G.J.G., Norton, G.R., Wilson, K.G., & Sandler, L.S. (1994). Subjective symptoms and cardiac reactivity to hyperventilation in individuals with high anxiety sensitivity. *Behaviour Research and Therapy*, 32, 237 - 241.
- Asmundson, G.J.G., Norton, G.R., & Veloso, F. (1998). *Anxiety sensitivity and fear of pain in patients with recurring headaches*. Manuscript submitted for publication.
- Asmundson, G.J.G. & Taylor, S. (1996). Role of anxiety sensitivity in pain-related fear and avoidance. *Journal of Behavioral Medicine*, 19, 577 - 586.
- Balogun, J.A. (1986). Pain complaint and muscle soreness associated with high voltage stimulation: Effect of ramp rate. *Perceptual and Motor Skills*, 62, 799 - 810.
- Beecher, H.K. (1956). Relationship of significance of wound to the pain experienced. *Journal of the American Medical Association*, 161, 1609 - 1613.
- Benjamin, F.B. (1959). Release of intracellular potassium as the physiological stimulus for pain. *Journal of Applied Physiology*, 14, 643 - 646.
- Benjamin, F.B., & Helvey, W.M. (1963). Iontophoresis of potassium for experimental determination of pain endurance in man. *Proceedings of the Society for Experimental Biology and Medicine*, 113, 566 - 568.
- Block, A.R. (1981). Investigation of the response of the spouse to chronic pain behavior. *Psychosomatic Medicine*, 43, 415 - 422.
- Block, A.R., Kremer, E.F., & Gaylor, M. (1980). Behavioral treatment of chronic pain: The spouse as a discriminative stimulus. *Pain*, 9, 243 - 252.
- Bolles, R.C. & Fanselow, M.S. (1980). A perceptual-defensive-recuperative model of fear and pain. *The Behavioral and Brain Sciences*, 3, 291 - 323.

- Borkovec, T.D. & Grayson, J.B. (1980). Consequence of increasing the functional impact of internal emotional stimuli. In K. Blankstein, P. Pliner, and J. Policy, (Eds.), *Advances in the study of communication and affect: Assessment and modification of emotional behavior* (Vol. 6, pp. 117 - 137). New York: Plenum Press.
- Bortz, W.M. (1984). The disuse syndrome. *Western Journal of Medicine*, 141, 691 - 694.
- Bower, G.H. (1981). Mood and memory. *American Psychologist*, 36, 129 - 148.
- Chaves, J.F. & Brown, J.M. (1987). Spontaneous coping strategies for the control of clinical pain and distress. *Journal of Behavioral Medicine*, 10, 263 - 276.
- Cornwall, A. & Donderi, D.C. (1988). The effect of experimentally induced anxiety on the experience of pressure pain. *Pain*, 35, 105 - 113.
- Cox, B.J., Endler, N.S., Norton, G.R., & Swinson, R.P. (1991). Anxiety sensitivity and nonclinical panic attacks. *Behaviour Research and Therapy*, 29, 367 - 369.
- Craske, M.G., Rowe, M., Lewin, M., & Noriega-Dimitri, R. (1997). Interoceptive exposure versus breathing retraining within cognitive-behavioural therapy for panic disorder with agoraphobia. *British Journal of Clinical Psychology*, 36, 85 - 99.
- Crombez, C., Baeyens, F., & Eelen, P. (1994). Sensory and temporal information about impending pain: The influence of predictability. *Behaviour Research and Therapy*, 32, 611 - 622.
- Crombez, G., Eccleston, C., Baeyens, F., & Eelen, P. (1996). The disruptive nature of pain: An experimental investigation. *Behaviour Research and Therapy*, 73, 135 - 143.

- Crombez, G., Eccleston, C., Baeyens, F., & Eelen, P. (1998). When somatic information threatens, catastrophic thinking enhances attentional interference. *Pain, 75*, 187 - 198.
- Crombez, G., Vervaeet, L., Lysens, R., Baeyens, F., & Eelen, P. (1998). Avoidance and confrontation of painful, back-straining movements in chronic back pain patients. *Behavior Modification, 22*, 62 - 77.
- Crombez, G., Vlaeyen, J.W.S., Heuts, P.H.T.G., & Lysens, R. (1999). Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. *Pain, 80*, 329 - 339.
- Deyo, R., Diehl, A. & Rosenthal, M. (1986). How many days of bed rest for acute low back pain? *New England Journal of Medicine, 315*, 1064 - 1070.
- Dolce, J.J., Crocker, M.F., Moletteire, C., & Doleys, D.M. (1986). Exercise quotas, anticipatory concern and self-efficacy expectancies in chronic pain: A preliminary report. *Pain, 24*, 365 - 372.
- Doleys, D.M., Crocker, M.F., & Patton, D. (1982). Response of patients with chronic pain to exercise quotas. *Physical Therapy, 62*, 1111 - 1114.
- Dworkin, S.F. & Chen, A.C.N. (1981). Pain in clinical and laboratory contexts. *Journal of Dental Research, 61*, 772 - 774.
- Eccleston, C. (1994). Chronic pain and attention; A cognitive approach. *British Journal of Clinical Psychology, 33*, 535 - 547.
- Eccleston, C. & Crombez, G. *Pain demands attention: A cognitive-affective model of the interruptive function of pain.* Manuscript submitted for publication.
- Edens, J.L. & Gil, K.M. (1995). Experimental induction of pain: Utility in the study of clinical pain. *Behavior Therapy, 26*, 197 - 216.

- Eich, E., Reeves, J.L., Jaeger, B., & Redford, S.B. (1985). Memory for pain: Relation between past and present pain intensity. *Pain, 23*, 375 - 379.
- Engan, T. (1971). Psychophysics II. Scaling methods. In J.W. King and L.A. Riggs (Eds.), *Experimental Psychology* (3rd Ed., pp. 11 - 86). New York: Raven Press.
- Fields, H.L. (1987). *Pain*. McGraw-Hill: New York.
- Fishbain, D.A., Cutler, R., Rosomoff, H.L., & Rosomoff, R.S. (1997). Chronic pain-associated depression: Antecedent or consequence of chronic pain? *Clinical Journal of Pain, 13*, 116 - 137.
- Fishbain, D.A., Rosomoff, H.L., Cutler, R.B., & Rosomoff, R.S. (1995). Secondary gain concept: A review of the scientific evidence. *The Clinical Journal of Pain, 11*, 6 - 21.
- Flor, H., Behle, D.J., & Birbaumer, N. (1993). Assessment of pain-related cognitions in chronic pain patients. *Behaviour Research and Therapy, 31*, 63 - 73.
- Flor, H., Turk, D.C., & Rudy, T.E. (1989). Relationship of pain impact and significant other reinforcement of pain behaviors: The mediating role of gender, marital status and marital satisfaction. *Pain, 38*, 45 - 50.
- Foa, E.G. & Goldstein, A. (1978). Continuous exposure and complete response prevention in the treatment of obsessive-compulsive neurosis. *Behavior Therapy, 9*, 821 - 829.
- Fordyce, W.E. (1976). *Behavioral methods for chronic pain and illness*. St. Louis: Mosby.
- Fordyce, W.E. (1977). Operant conditioning: An approach to chronic pain. In A.K. Jacox (Ed.), *Pain* (pp. 275 - 284). Boston: Little, Brown & Co.

- Fordyce, W.E. (1988). Pain and suffering. *American Psychologist*, 43, 276 - 283.
- Fordyce, W.E., Shelton, J.L., & Dundore, D.E. (1982). The modification of avoidance learning in pain behaviors. *Journal of Behavioral Medicine*, 5, 405 - 414.
- Gamsa, A. (1994). The role of psychological factors in chronic pain. I. A half century of study. *Pain*, 57, 5 - 15.
- Gil, K.M., Abrams, M.R., Phillips, G., & Keefe, F.J., (1989). Sickle cell disease pain: Relation of coping strategies to adjustment. *Journal of Consulting and Clinical Psychology*, 57, 725 - 731.
- Gil, K.M., Keefe, F.J., Crisson, J.E., & Van Dalfsen, P.J. (1987). Social support and pain behavior. *Pain*, 29, 209 - 217.
- Grayson, J.B., Foa, E.B., & Steketee, G. (1982). Habituation during exposure treatment: Distraction vs attention-focusing. *Behaviour Research and Therapy*, 20, 323 - 328.
- Greist, J.H., Marks, I.M., Berlin, F., Gournay, K., & Noushirvani, H. (1980). Avoidance versus confrontation of fear. *Behavior Therapy*, 11, 1 - 14.
- Humphries, S.A., Johnson, M.H., & Long, N.R. (1996). An investigation of the gate control theory of pain using the experimental pain stimulus of potassium iontophoresis. *Perception and Psychophysics*, 58, 693 - 703.
- Humphries, S.A., Long, N.R., & Johnson, M.H. (1994). Iontophoretically applied potassium ions as an experimental pain stimulus for investigating pain mechanisms. *Perception and Psychophysics*, 56, 637 - 648.
- International Association for the Study of Pain (1994). Descriptions of chronic pain syndromes and definitions of pain terms. In H. Merskey and N. Bogduk (Eds.), *Classification of chronic pain* (2nd Ed.). Seattle: IASP Press.

- Jensen, M.P. & Karoly, P. (1992). Self-report scales and procedures for assessing pain in adults. In D.C. Turk and R. Melzack (Eds.), *Handbook of pain assessment* (pp. 135 - 151). New York: Guilford Press.
- Jensen, I., Nygren, A., Gamberale, F., Goldie, I., & Westerholm, P. (1994). Coping with long-term musculoskeletal pain and its consequences: Is gender a factor? *Pain, 57*, 167 - 172.
- Johnson, M.H., Breakwell, G., & Douglas, W. (1998). The effects of imagery and sensory detection distractors on different measures of pain: How does distraction work? *British Journal of Clinical Psychology, 37*, 141 - 154.
- Kaplow, J.C., Slater, M.A., Patterson, T.L., Doctor, J.N., Atkinson, J.H., & Garfin, S.R. (1993). An empirical evaluation of multidimensional clinical outcome in chronic low back pain patients. *Pain, 55*, 107 - 118.
- Kent, G. (1985). Memory for dental pain. *Pain, 21*, 187 - 194.
- Klenerman, L., Slade, P.D., Stanley, I.M., Pennie, B., Reilly, J.P., Atchison, L.E., Troup, J.D.G., & Rose, M.J. (1995). The prediction of chronicity in patients with an acute attack of low back pain in a general practice setting. *Spine, 20*, 478 - 484.
- Kori, S.H., Miller, R.P., & Todd, D.D. (1990). Kinisophobia: A new view of chronic pain behavior. *Pain Management, Jan/Feb*, 35 - 43.
- Krishnan, K.R.R., France, R.D., Pelton, S., McCann, U.D., Davidson, J., & Urban, B.J. (1985). Chronic pain and depression. II. Symptoms of anxiety in chronic low back pain patients and their relationship to subtypes of depression. *Pain, 22*, 289 - 294.
- Lethem, J., Slade, P.D., Troup, J.D.G., & Bentley, G. (1983). Outline of a fear-avoidance model of exaggerated pain perception - I. *Behaviour Research and Therapy, 21*, 401 - 408.

- Levene, F.M. & de Simone, L.L. (1991). The effects of experimenter gender on pain report in male and female subjects. *Pain, 44*, 69 - 72.
- Lilienfeld, S.O., Jacob, R.G., & Turner, S.M. (1989). Comment on Holloway and McNally's (1987) "Effects of Anxiety Sensitivity on the Response to Hyperventilation." *Journal of Abnormal Psychology, 98*, 100-102.
- Lilienfeld, S.O., Turner, S.M., & Jacob, R.G. (1993). Anxiety sensitivity: An examination of the theoretical and methodological issues. *Advances in Behaviour Research and Therapy, 15*, 147 - 183.
- Lindsay, M., Crino, R., & Andrews, G. (1997). Controlled trial of exposure and response prevention in obsessive-compulsive disorder. *British Journal of Psychiatry, 171*, 135 - 139.
- Linton, S.J. (1985). The relationship between activity and chronic back pain. *Pain, 21*, 289 - 294.
- Linton, S.J. & Melin, C. (1982). The accuracy of remembering chronic pain. *Pain, 13*, 281 - 285.
- Livingston, W.K. (1943). *Pain mechanisms*. New York: Macmillan.
- Lousberg, R., Schmidt, A.J.M., & Groenman, N.H. (1992). The relationship between spouse solicitousness and pain behavior: Searching for more experimental evidence. *Pain, 51*, 75 - 79.
- Marks, I.M. (1978). *Living with fear*. New York: McGraw-Hill:
- Mathews, A. (1978). Fear-reduction research and clinical phobias. *Psychological Bulletin, 85*, 390 - 404.

- Matthews, K.A., Scheier, M.F., Brunson, B.I., & Carducci, B. (1980). Attention, unpredictability, and reports of physical symptoms: Eliminating the benefits of predictability. *Journal of Personality and Social Psychology*, 38, 525 - 537.
- McCracken, L.M. & Gross, R.T. (1993). Does anxiety help coping with chronic pain? *The Clinical Journal of Pain*, 9, 253 - 259.
- McCracken, L.M., Gross, R.T., Aikens, J., & Carnrike Jr., C.L.M. (1996). The assessment of anxiety and fear in persons with chronic pain: A comparison of instruments. *Behaviour Research and Therapy*, 34, 927 - 933.
- McCracken, L.M., Gross, R.T., Sorg, P.J., & Edmands, T.A. (1993). Prediction of pain in patients with chronic low back pain: Effects of inaccurate prediction and pain-related anxiety. *Behaviour Research and Therapy*, 31, 647 - 652.
- McCracken, L.M., Zayfert, C., & Gross, R.T. (1992). The Pain Anxiety Symptoms Scale: development and validation of a scale to measure fear of pain. *Pain*, 50, 67 -73.
- McNally, R.J. (1989). Is anxiety sensitivity distinguishable from trait anxiety? Reply to Lilienfeld, Jacob, and Turner (1989). *Journal of Abnormal Psychology*, 98, 193 - 194.
- McNally, R.J. & Louro, C.E. (1992). Fear of flying in agoraphobia and simple phobia: Distinguishing features. *Journal of Anxiety Disorders*, 6, 319 - 324.
- McNally, R.J. & Steketee, G.S. (1985). The etiology and maintenance of severe animal phobias. *Behaviour Research and Therapy*, 23, 431 - 435.
- McNaughton, H. (1996). Managing acute low back pain. *The New Zealand Medical Journal*, 109, 175 - 177.
- Melzack, R. (1986). Neurophysiological foundations of pain. In R.A. Sternbach (Ed.), *The psychology of pain* (pp. 1 - 24). New York: Raven Press.

- Melzack, R. & Casey, K.L. (1968). Sensory, motivational, and central control determinants of pain. In D. Kenshalo (Ed.), *The skin senses*. Springfield: Thomas Publishing.
- Melzack, R. & Wall, P.D. (1965). Pain mechanisms: A new theory. *Science*, *150*, 971 - 979.
- Melzack, R. & Wall, P.D. (1982). *The challenge of pain*. Great Britain: Penguin.
- Mendelson, G. (1992). Compensation and chronic pain. *Pain*, *48*, 122 -123.
- Miller, B.V. & Lewis, D.J. (1971). The effects of varying short visual exposure times to a phobic test stimulus on subsequent avoidance behaviour. *Behaviour Research and Therapy*, *9*, 17 - 21.
- Miron, D., Duncan, G.H., & Bushnell, M.C. (1989). Effects of attention on the intensity and unpleasantness of thermal pain. *Pain*, *39*, 345 - 352.
- Munby, M. & Johnston, D.W. (1980). Agoraphobia: The long-term follow-up of behavioural treatment. *British Journal of Psychiatry*, *137*, 418 - 427.
- Murphy, D., Lindsay, S., & de C Williams, A. (1997). Chronic low back pain: Predictions of pain and relationship to anxiety and avoidance. *Behaviour Research and Therapy*, *35*, 231 - 238.
- Meyers, J.W. & Lyon, H.P. (1979). Stress and distress. *Proceedings of the American Occupational Medicine Association*, *24*, 112 - 139.
- Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin*, *101*, 259 - 282.
- Osman, A., Barrios, F.X., Kopper, B.A., Hauptmann, W., Jones, J., & O'Neill, E. (1997). Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *Journal of Behavioral Medicine*, *20*, 589 - 605.

- Osuntokun, B.O., Odeku, E.L., & Luzzatao, L. (1968). Congenital pain asymbolia and auditory imperception. *Journal of Neurology, Neurosurgery & Psychiatry*, 31, 291 - 296.
- Peterson, R.A. & Heilbronner, R. (1987). The Anxiety Sensitivity Index: Construct validity and factor analytic structure. *Journal of Anxiety Disorders*, 1, 117 - 121.
- Petroni, F. (1969). Significant others and illness behavior: A much neglected sick role contingency. *Sociology Quarterly*, 10, 32 - 41.
- Philips, C. (1977). A psychological analysis of tension headache. In S. Rachman (Ed.), *Contributions to medical psychology* (pp. 91 - 113). Oxford: Pergamon Press.
- Philips, C. (1987). The effects of behavioural treatment of chronic pain. *Behaviour Research and Therapy*, 25, 365 - 377.
- Philips, H.C. (1987). Avoidance behaviour and its role in sustaining chronic pain. *Behaviour Research and Therapy*, 25, 273 - 279.
- Philips, H.C. & Grant, L. (1991). Acute back pain: A psychological analysis. *Behaviour Research and Therapy*, 5, 429 - 434.
- Philips, C. & Hunter, M. (1981). Pain behaviour in headache sufferers. *Behaviour Analysis and Modification*, 4, 256 - 266.
- Philips, H.C. & Jahanshahi, M. (1985). Chronic pain: An experimental analysis of the effects of exposure. *Behaviour Research and Therapy*, 23, 281 - 290.
- Philips, H.C. & Jahanshahi, M. (1986). The components of pain behaviour report. *Behaviour Research and Therapy*, 24, 117 - 125.
- Rachman (1994). The overprediction of fear: A review. *Behaviour Research and Therapy*, 32, 683 - 690.

- Rachman, S. & Arntz, A. (1991). The overprediction and underprediction of pain. *Clinical Psychology Review, 11*, 339 - 355.
- Rachman, S. & Eyril, K. (1989). Predicting and remembering recurrent pain. *Behaviour Research and Therapy, 27*, 621 - 635.
- Rachman, S. & Hodgson, R. (1974). Synchrony and desynchrony in fear and avoidance. *Behaviour Research and Therapy, 12*, 311 - 318.
- Rachman, S. & Lopatka, C. (1988). Accurate and inaccurate predictions of pain. *Behaviour Research and Therapy, 26*, 291 - 296.
- Rapee, R.M., Ancis, J.R., & Barlow, D.H. (1988). Emotional reactions to physiological sensations: Panic disorder patients and non-clinical Ss. *Behaviour Research and Therapy, 26*, 265 - 269.
- Reiss, S. (1987). Theoretical perspectives on the fear of anxiety. *Clinical Psychology Review, 7*, 141 - 153.
- Reiss, S. (1991). Expectancy theory of fear, anxiety, and panic. *Clinical Psychology Review, 11*, 141 - 153.
- Reiss, S. & McNally, R.J. (1985). The expectancy model of fear. In S. Reiss and R.R. Bootzin (Eds.), *Theoretical issues in behavior therapy* (pp. 107 - 121). New York: Academic Press.
- Reiss, S., Peterson, R.A., & Gursky, D.M. (1988). Anxiety sensitivity, injury sensitivity, and individual differences in fearfulness. *Behaviour Research and Therapy, 26*, 341 - 345.
- Reiss, S., Peterson, R.A., Gursky, D.M., & McNally, R.J. (1986). Anxiety sensitivity, anxiety frequency, and the prediction of fearfulness. *Behaviour Research and Therapy, 24*, 1 - 8.

- Rodriguez, B.I. & Craske, M.G. (1995). Does distraction interfere with fear reduction during exposure? A test among animal-fearful subjects. *Behavior Therapy*, 26, 337 - 349.
- Rose, M.J., Klenerman, L., Atchison, L., & Slade, P.D. (1992). An application of the Fear-Avoidance Model to three chronic pain problems. *Behaviour Research and Therapy*, 30, 359 -365.
- Rosenbaum, M. (1980). Individual differences in self-control behaviours and tolerance of painful stimulation. *Journal of Abnormal Psychology*, 89, 581 - 590.
- Rosenstiel, A.K. & Keefe, F.J. (1983). The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. *Pain*, 17, 33 - 44.
- Rowat, K.M. & Knarfl, K.A. (1985). Living with chronic pain: The spouse's perspective. *Pain*, 23, 259 - 271.
- Roy, R. (1992). *The social context of the chronic pain sufferer*. Toronto: University of Toronto Press.
- Scholing, A. & Emmelkamp, P.M. (1993). Exposure with and without cognitive therapy for generalized social phobia: Effects of individual and group treatment. *Behaviour Research and Therapy*, 31, 667 - 681.
- Seligman, M.E.P. & Johnston, J.C. (1973). A cognitive theory of avoidance learning. In F.J. McGuigan and D.B. Lumsden (Eds.), *Contemporary approaches to conditioning and learning* (pp. 69 - 106). Washington D.C.: Winston & Sons.
- Spanos, N.P., Radte-Bodorik, L., Ferguson, J.D., & Jones, B. (1979). The effects of hypnotic susceptibility, suggestions for analgesia, and utilization of cognitive strategies on the reduction of pain. *Journal of Abnormal Psychology*, 88, 282 - 292.

- Sternbach, R.A. (1968). *Pain. A psychophysiological approach*. New York: Academic Press.
- Stewart, S.H., Taylor, S., & Baker, J.M. (1997). Gender differences in dimensions of anxiety sensitivity. *Journal of Anxiety Disorders, 11*, 179 - 200.
- Sue, D. (1975). The effect of duration of exposure on systematic desensitization and extinction. *Behaviour Research and Therapy, 13*, 55 - 60.
- Sullivan, M.J.L., Bishop, S.R., & Pivik, J. (1995). The Pain Catastrophizing Scale: Development and validation. *Psychological Assessment, 7*, 524 - 532.
- Taylor, S. (1993). The structure of fundamental fears. *Journal of Behavior Therapy and Experimental Psychiatry, 24*, 289 - 299.
- Taylor, S. & Cox, B.J. (1998). Anxiety sensitivity: Multiple dimensions and hierarchic structure. *Behaviour Research and Therapy, 36*, 37 - 51.
- Taylor, S., Koch, W.J., & Crockett, D.J. (1991). Anxiety sensitivity, trait anxiety, and the anxiety disorders. *Journal of Anxiety Disorders, 6*, 293 - 311.
- Telch, M.J., Shermis, M.D., & Lucas, J.A. (1989). Anxiety sensitivity: Unitary personality trait of domain-specific appraisals? *Journal of Anxiety Disorders, 3*, 25 - 32.
- Turk, D.C. & Rudy, T.E. (1992). Cognitive factors and persistent pain: A glimpse into Pandora's box. *Cognitive Therapy and Research, 16*, 99 - 122.
- Tyle, P. & Kari, B. (1988). Iontophoretic devices. In P. Tyle (Ed.), *Drug delivery devices: Fundamentals and applications* (pp. 421 - 454). New York: Dekker.
- Tyrer, S.P. (1992). Basic concepts of pain. In S.P. Tyrer (Ed.), *Psychology, psychiatry, and chronic pain* (pp. 3 - 15). Oxford: Butterworth-Heinemann.

- Uchida, Y. & Murao, S. (1974). Potassium-induced excitation of afferent cardiac sympathetic nerve fibres. *American Journal of Physiology*, 226, 603 - 607.
- Vlaeyen, J.W.S., Kole-Snijders, A.M.J., Boeren, R.G.B., & van Eek, H. (1995). Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain*, 62, 363 - 372.
- Vodde, T.W. & Gilner, F.H. (1971). The effects of exposure to fear stimuli on fear reduction. *Behaviour Research and Therapy*, 9, 169 - 175.
- Voudouris, N.J., Peck, C., & Coleman, G. (1985). Conditioned placebo responses. *Journal of Personality and Social Psychology*, 48, 47 - 53.
- Waddell, G., Main, C.J., Di Paola, M.P., & Gray, I.C.M. (1984). Chronic low back pain, psychologic distress, and illness behaviour. *Spine*, 9, 209 - 213.
- Waddell, G., Newton, M., Henderson, I., Somerville, D., & Main, C.J. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*, 52, 157 - 168.
- Wall, P. (1979). On the relation of injury to pain. *Pain*, 6, 253 - 261.
- Weisenburg, M. (1977). Pain and pain control. *Psychological Bulletin*, 84, 1008 - 1044.
- Wolff, B.B. & Jarvik, M.E. (1964). Relationship between superficial and deep somatic thresholds of pain with a note on handedness. *American Journal of Psychology*, 77, 580 - 599.
- Zinbarg, R.E., Barlow, D.H., & Brown, T.A. (1997). Hierarchical structure and general factor saturation of the Anxiety Sensitivity Index: Evidence and implications. *Psychological Assessment*, 9, 277 - 284.

APPENDIX A

PARTICIPANT INFORMATION SHEET

THE INTERACTION BETWEEN PAIN AND TASK PERFORMANCE

My name is Kim Coldham-Fussell. I am a psychology Masters student interested in how pain works and the factors that affect how pain is perceived. My supervisor for this research is Malcolm Johnson, a lecturer and researcher in the Department of Psychology.

I am seeking volunteers for a study that measures the effects of a simple task on the way pain is perceived. If you have participated in a pain study before, please do not volunteer for this study.

If you decide to participate, you will be asked to fill out two short pencil and paper questionnaires. You will then be required to attend a single session by yourself, lasting approximately 45 minutes. In the session, you will be given an experimental pain stimulus, known as potassium iontophoresis. The nature of the pain stimulus is the application of potassium ions to the skin on your wrist. This type of pain stimulus does not produce tissue damage. The pain felt is typically described as a pricking or burning sensation. You will be administered pain of increasing intensity until you indicate your tolerance. During the main study, the pain will remain below your tolerance level.

You will be asked to perform a simple audio task, and your performance on the task will be recorded. You will be given the opportunity to practise this task before the experiment begins.

I am interested in discovering how your performance on the task is influenced by the pain stimulus.

If you are willing to be in this study, you will first fill in a Medical Checklist, which asks personal health questions. This information will be kept confidential until one year after the study, and then destroyed. You will also be required to sign a Consent Form. The Consent Form is not a contract that in any way obliges you to complete the experiment. At any stage you are free to withdraw from the study. Participation is independent from any papers in which you are enrolled, and will not affect any assessment procedure associated with your course of study.

If you agree to take part in this study, you have the following rights:

1. to refuse to answer any particular question we might ask
2. to be given time to consider and discuss participation with others if desired
3. to withdraw from the study at any time, without repercussion
4. to ask questions as they occur to you during participation

5. to provide information on the understanding that it will remain confidential to the researchers. All the information will be stored anonymously, in coded form on a database that only the researchers have access to. It will not be possible to identify you in any reports prepared from the study
6. to be given access to your own personal data
7. to have the opportunity to discuss the experiment immediately after participation.

At the conclusion of the study, you will be sent a full explanation of the experiment along with a summary of the findings.

We hope you will be willing to participate. If you wish to talk to someone about any aspect of this study, you can contact:

Malcolm Johnson (supervisor): School of Psychology, Massey University.
Ph. 350 4130

Kim Coldham-Fussell (researcher): School of Psychology, Massey University.

APPENDIX B
CONSENT FORM

I have read the Information Sheet and have had the details of the study explained to me. My questions have been answered to my satisfaction and I understand that I may ask further questions at any time.

I understand that I have the right to withdraw from the study at any time and to decline to answer any particular questions.

I understand that the information I provide will be used only for this project and publications arising from this research project.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signed:

Name:

Date:

Contact phone number:

Contact address (December 1998):

.....

.....

APPENDIX C
MEDICAL CHECKLIST

Participant name

Please answer the following questions:

- 1) Have you ever had any form of epilepsy? yes/ no
- 2) Do you have any known heart or circulatory condition? yes/ no
- 3) In the past 6 months have you suffered from any painful injury or condition that you think may influence your ability to feel pain? yes/ no
- 4) Is it possible you are pregnant? yes/ no
- 5) Do you have diabetes? yes/ no

If you have answered yes to any of the above questions you are not suitable for this study.

- 6) Are you currently on medication of any type? yes/ no
- 7) Have you ever had an allergic reaction to any medication? yes/ no
- 8) Are you in good health? yes/ no
- 9) Do you suffer from any skin disorders? yes/ no
- 10) Is your hearing impaired? yes/ no

If you have any doubts about your health you should not participate in this study.

Researcher's signature: Date:

Supervisor's signature: Date:

APPENDIX D
THE ANXIETY SENSIVITY INDEX

Below is a list of statements. Read each one carefully and select the code from the box on the right that best describes how true each statement is for you. Enter the number you have selected in the box beside each statement.

0 = very little
1 = a little
2 = some
3 = much
4 = very much

- | | |
|---|--------------------------|
| 1. It is important for me not to appear nervous | <input type="checkbox"/> |
| 2. When I cannot keep my mind on a task, I worry that I might be going crazy | <input type="checkbox"/> |
| 3. It scares me when I feel 'shaky' (trembling) | <input type="checkbox"/> |
| 4. It scares me when I feel faint | <input type="checkbox"/> |
| 5. It is important for me to stay in control of my emotions | <input type="checkbox"/> |
| 6. It scares me when my heart beats rapidly | <input type="checkbox"/> |
| 7. It embarrasses me when my stomach growls | <input type="checkbox"/> |
| 8. It scares me when I am nauseous | <input type="checkbox"/> |
| 9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack | <input type="checkbox"/> |
| 10. It scares me when I become short of breath | <input type="checkbox"/> |
| 11. When my stomach is upset, I worry that I might be seriously ill | <input type="checkbox"/> |
| 12. It scares me I am unable to keep my mind on a task | <input type="checkbox"/> |
| 13. Other people notice when I feel shaky | <input type="checkbox"/> |
| 14. Unusual body sensations scare me | <input type="checkbox"/> |
| 15. When I am nervous, I worry that I might be mentally ill | <input type="checkbox"/> |
| 15. It scares me when I am nervous | <input type="checkbox"/> |

APPENDIX E

DEBRIEF STATEMENT

Dear

I am writing with regards to the pain experiment you participated in during October, 1998. I would first like to thank you very much for your time and participation in the study. This letter will outline the rationale of the study, and summarise the results.

Rationale of the study.

The chronic pain syndrome is associated with avoidance of movement and activities that are expected to cause pain and suffering. Recent studies have suggested that sufferers may begin to fear the pain, and avoid movement and activities even when pain is at a minimal level. The avoidance in chronic pain has been compared with the avoidance seen in phobias. In phobic avoidance, a person avoids a feared object, such as heights, because they expect some aversive outcome, such as losing control, panicking, or falling. In the avoidance of pain, the person may similarly expect to lose control, to sustain injury, or to be unable to cope with the pain. A common treatment for phobic avoidance is exposure treatment, whereby exposure to the feared object or situation teaches the individual that the aversive outcomes they expect do not actually occur. In this way, fear and avoidance is reduced over time.

This study investigated the effects of exposure and avoidance of pain on subsequent pain tolerance. It was expected that repeated exposure to the pain stimulus would result in an increase in pain tolerance, by a process of fear reduction. It was also expected that not having repeated exposure to the pain would have little effect on tolerance. This study also attempted to examine the effects of active avoidance on subsequent tolerance. It was predicted, following the phobia literature, that avoidance of the pain would actually decrease subsequent tolerance, by maintaining the fearful and aversive characteristics of the pain. You were included in the group that was exposed/ not exposed/ avoided the pain.

The experiment was presented to appear as if the interest was in how performance of the audio task affected the way pain was perceived. It was important that you were not aware of the true nature of the study, because expectations can powerfully influence performance in an experiment, in a way that can distort results. Deception is never used lightly in psychological experiments today, and it was agreed by the Human Ethics Committee that it was justified in this study.

Results.

The hypotheses failed to be supported by the study. In fact, no significant differences were found in tolerance change between the 3 conditions, exposure, non-exposure and avoidance. There are a number of possible explanations for this. For example, it was possible that the tolerance tests themselves constituted a considerable amount of exposure in themselves, thus diluting the effects. It was also possible that individuals

who volunteer to be given pain in an experiment have a very low fear of pain to begin with. Finally, the audio task that you performed may have acted to distract you from the pain, which would also limit the effectiveness of the manipulation.

However, the experimental investigation of avoidance on pain tolerance is just beginning. To date, only one other study has attempted to manipulate avoidance in a laboratory. For this reason, this study was useful in examining the possible pitfalls in this area. It thus contributes to pain research, which has relevance for thousands of New Zealanders suffering from chronic pain problems. Your participation has been greatly appreciated. If you are worried about any aspect of the study, or would like to discuss it further, please do not hesitate in contacting me.

Yours Sincerely,

Kim Coldham-Fussell
54 Haultain St., Hamilton.

APPENDIX F

RAW DATA

Table 1. Self-reported change in fear following the experimental conditions for low and high anxiety sensitivity.

CONDITION	GRS CHANGE IN FEAR			
	Low AS		High AS	
	Mean	SD	Mean	SD
Exposure	- 2.17	0.56	-1.77	0.54
Non-Exposure	-2.15	0.57	-1.45	0.57
Avoidance	-0.86	0.54	-1.28	0.60

Table 2. Mean number of errors made on the tone task during the three experimental conditions.

CONDITION	ERRORS					
	Type a error		Type b error		Total error (a + b)	
	Mean	SD	Mean	SD	Mean	SD
Exposure	3.03	3.46	0.48	1.02	3.52	3.98
Non-Exposure	3.33	3.98	0.89	1.65	4.22	5.09
Avoidance	2.92	4.50	1.00	1.94	3.92	5.87

Table 3. Mean tolerance change (%) in males and females across the three experimental conditions.

CONDITION	TOLERANCE CHANGE (%)			
	Males		Females	
	Mean	SD	Mean	SD
Exposure	8.29	18.72	13.31	22.31
Non-Exposure	16.52	25.86	12.10	29.96
Avoidance	7.11	25.80	26.35	29.80