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NGAU MAMAE

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Ngau Mamae
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Ngau Mamae aims to keep clinicians up-to-date in regard to pain diagnosis and management. It will inform on NZ Pain Society (a Chapter of IASP – International Association for the Study of Pain) initiatives and activities. The Editor and Sub-editors seek contributions that will further these aims. Articles, reviews and letters should be submitted by email or supplied on disk to:

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Pennebaker JW. The psychology of physical symptoms. New York: Springer, 1982.

Philips H. Avoidance behaviour and its role in sustaining chronic pain. Behaviour Research Therapy 1987a; 4: 273-279.

Philips H. The effects of behavioural treatment on chronic pain. Behaviour Research Therapy 1987b; 5: 365-377.

Scharloo M, & Kaptein A. Measurement of illness perceptions in patients with chronic somatic illness: a review. In: Petrie K, Weinman J, editors. Perception of health and illness. The Netherlands: Harwood Academic Publishers, 1997. pp. 103-154.

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Contents

Ngau Mamae (Real Pain)

Ngau means to bite or engage in a very real way. Mamae means pain. In combination, the words describe a very real and deeply ingrained, gripping, biting pain.

— Merimeri Penfold

Of Ngati Kuri descent from the Far North. She was born in Te Hapua, educated at Queen Victoria College and qualified as a teacher working in education for many years. Moved to the Maori Studies Department at Auckland University. Was employed by Maori Studies Department at the University of Auckland to provide interpretations for University documents. Passed away in April, 2014.

Does pain always lead to poorer cognitive performance?

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Does pain experience affect the way we think? The general view is that experiencing pain is associated with cognitive deficits. Experimentally, this is seen as poorer scores on memory tasks, poorer attentional control, and general slowing in performance when in pain compared to when not in pain (Moriarty, McGuire & Finn, 2011; Moriarty & Finn, 2014).

These lab findings correspond to the day-to-day experience of people with chronic pain, who report, for example, that they forget things, have trouble paying attention and feel confused, slow, and disoriented, more so than they did before they developed chronic pain (McCracken & Iverson, 2001). Furthermore, chronic pain is associated with functional (e.g. frontal activation; Mao, Zhang, Bao, Liao, Yang, & Zhang, 2014) and structural changes in the brain (Bushnell, Čeko & Low, 2013). Changes in white matter connectivity are observed, and some areas show grey matter loss and others grey matter gain. These changes suggest functional reorganisation of normal processing.

I propose that one contributing factor to the cognitive deficits observed may be that cognitive resources are taken up with looking for further pain-related information in the environment; information that signals further threat or relief from pain. Simultaneously, cognitive resources are directed away from information not directly relevant to pain, information such as that used in the tasks to measure cognitive performance. So rather than being purely depleted, cognition is *motivated* in pain experience. This "motivated cognition" may thus contribute to the deficits we observe with pain-irrelevant information. If so, it requires us to rethink previously observed deficits. Additionally, if motivated cognition is descriptive of processing in pain, further down the track it will be important to determine if it is a general principle, or only present in some types of pain experience or some types of chronic pain. In this article, I summarise the background to the motivated cognition proposal and outline my PhD research programme, which aims to investigate the role that motivated cognition may have in pain experience.

Pain is an experience generated when our protection system detects threat to bodily tissues. The protection system receives input from the periphery which interacts with our knowledge, expectations, and previous experience. If threat, or potential threat, to bodily tissues is perceived, pain is experienced (Moseley, 2007; Moseley & Jones, 2009). Key to this description is two components; the conscious experiential nature of pain and the reduction of threat. These two components make pain similar to other somatic experiences such as hunger and thirst. Craig (2003) proposes that these experiences are homeostatic emotions, or emotions that serve to motivate us to return to our ideal state. Hunger, thirst, and perhaps itch and pain are all conscious sensory experiences that reflect a need for a behavioural response to return the individual to their ideal state (i.e. homeostasis). Furthermore, they are powerful motivators. When we are hungry, for example, it is difficult to focus on much except the need for food.

Hunger, thirst, and perhaps itch and pain are all conscious sensory experiences that reflect a need for a behavioural response to return the individual to their ideal state

Motivated cognition extends the idea of homeostatic emotions to changes in cognition. In fact, others are thinking along similar lines. For example, a very recent paper by Frankenhuis and de Weerth (2014) suggests a similar idea in the context of extreme stress (such as abuse) experienced by children. Frankenhuis and de Weerth propose that attention to information that signals self–relevant threat, at the expense of other information, contributes to the cognitive deficits observed in children who have experienced such stressors.

The premise of motivated cognition and ideas such

as Frankenhuis and de Weerth's (2014) is not that experiencing pain or stress makes it hard to think, rather it is that these experiences shift our priorities towards information relevant to our experiences and away from irrelevant information. This is a paradigm shift in understanding of cognitive deficits. In fact, Frankenhuis and de Weerth suggest reframing this shift in priorities as an "adapted mind" rather than focussing on impairments.

Clinically, such a paradigm shift has implications for treatment of children who have experienced extreme stress, in terms of both the selection of the most suitable treatment to change behaviour and in consideration of any detrimental effect treatment may have (Frankenhuis & de Weerth, 2014). Further elucidation of how pain and cognition modulate each other may have similar clinical implications for patients with chronic pain. First, in order to develop this new perspective on cognition in pain, I am exploring whether motivated cognition is a valid description of specifically *attentional* processing in pain experience by recruiting three participant groups; a group experiencing acute pain, a group experiencing chronic pain, and a group experiencing lifestyle pain (or pain that is seen as being "worth it" in some way, such as pain associated with tattooing). To assess how pain motivates attentional priorities, I will compare patterns of attention to pain relevant and irrelevant information between these three participant groups and to control participants not experiencing pain.

In everyday life we encounter a lot of rapidly changing information. That is, we encounter many stimuli we need to respond to in time. For example, when walking we need to move around other pedestrians and obstacles, watch for traffic, and say hello to friends. We need to identify relevant information in this sequential stream in order to activate the appropriate approach or withdrawal behaviours (Lang & Bradley, 2010). One method for testing serial information processing (also called *temporal attention*) is the classic rapid-serial-visual-presentation task (RSVP task; Raymond, Shapiro & Arnell, 1992). Imagine seeing a series of words flashed at a rate of 10 per second and you will have an idea of what it is like to be a participant in an RSVP task. I will use three variants of the RSVP task to explore how pain affects attention to neutral information and to aversive pain-related information. In the most basic version of the RSVP task, participants view a rapid stream of briefly presented numbers with two letters embedded within the stream. Participants are asked to identify the two letters. The key variable is the time between the first letter (Target 1, T1) and second letter (Target 2, T2). People are generally quite accurate at reporting T1, but reporting of T2 depends on the time between T1 and T2 (target onset asynchrony, TOA). If T2 appears less than 500 ms after presentation of T1, accuracy drops for reporting T2. For the participant, it is often as if there was no T2 in the stream; they just do not see T2. The resource capacity limit explanation of this "attentional blink" (Asplund, Fougnie, Zughni, Martin, Marois, 2014; Chun & Potter, 1995; Dux & Marois, 2009; Marois & Ivanoff, 2005, McHugo, Olatunji, & Zald, 2013) is that when attention is consumed with processing of T1, a bottleneck is created that prevents processing of T2. That is, there are not enough resources left over to process T2. The more attention we pay to the first word, the greater the magnitude of the "blink" is (and the lower the accuracy is for reporting T2). The blink can be used as a measure of how much attention was allocated to the first target, as a larger blink reflects less efficient processing of T2.

In basic RSVP tasks the targets are neutral stimuli like letters and numbers, and so attention to neutral items is assessed. However, in an emotional variant of the task, the letters and numbers can be replaced with words or pictures that have emotional value. Interestingly, the emotional nature of the stimuli used can influence the size of the blink (Arnell, Killman, & Fijavz, 2007; McHugo et al., 2013). The emotional RSVP task allows us to assess two different types of attention to emotion, depending on whether we manipulate the emotional nature of T1 or T2. In one version of the task, T2 is always a neutral stimulus, but T1 can be either emotional or neutral. This allows us to compare how processing resources are consumed by emotional versus neutral stimuli. A larger blink following an emotional compared to a neutral T1 indicates prolonged processing of the emotional stimulus. In an alternate version of the task, T1 is always a neutral stimulus, but T2 can be either emotional or neutral. In this version of the task, the magnitude of the blink is a measure of attentional capture of T2. If emotional stimuli attract attention, they will "overcome" the blink more easily, resulting in a shorter blink than for neutral stimuli.

For each different pain experience (acute, chronic, lifestyle), I will explore how the size of the blink changes depending on the nature of the stimuli

(letters/numbers, neutral words, aversive pain-related words) and order of their presentation (manipulating T1 or T2). If pain generally reduces attentional capacity (as predicted by the deficit view), then I expect to observe a larger blink, that is not modulated by the type of information, across all pain conditions compared to the non-pain condition. However, if pain acts to motivate attention to threatening and pain-relevant information (as predicted by the motivated cognition view), then I expect pain to be associated with a longer blink following an aversive pain-related T1 (induction of the blink) and/or a shorter blink for an aversive pain-related T2 (overcoming of the blink) than in non-pain control conditions. Essentially, people in pain should be better able to process pain-relevant information, but at the expense of their ability to process less relevant information.

The RSVP paradigm has been used in other clinical research examining emotional processing, for example in alexithymia (Grynberg, Vermeulen, & Luminet, 2013), PTSD (Schönenberg & Abdelrahman, 2013), schizophrenia (Strauss, Catalano, Llerena, & Gold 2013), and in spider phobia (Trippe, Hewig, Heydel, Hecht, & Miltner, 2007). Thus far, there is limited, mostly indirect, evidence that could inform understanding of how *pain experience* modulates attention to sequential information.

First, high intensity electric shock stimulation, assessed as painful (note this is acute pain), presented at the same time as T1 led to lower T2 accuracy than no stimulation or lower levels of stimulation (Sippel, 2011). Second, experiencing pain associated with fibromyalgia was associated with decreased T2 detection compared to healthy controls but only when target detection was difficult (Harker, Klein, Dick, Verrier, & Rashiq, 2011). These two studies used neutral letter and number stimuli and demonstrate that T2 accuracy is reduced when experiencing something adverse at T1, suggesting that more attentional resources are engaged with processing a T1 which occurs in a potentially threatening context. That is, there is a cognitive deficit associated with pain experience. However, by using neutral stimuli alone it is not possible to determine whether pain consumes attentional resources generally, or alternatively, if pain experience redirects resources to pain-relevant information and away from pain-irrelevant information.

Schwabe and Wolf (2010) explored motivated cognition, although without direct reference to pain experience. They used a cold pressor test (which is known to be painful) conducted under conditions of social monitoring to induce stress. Participants then completed a RSVP task with both negative (not specifically pain relevant) and neutral T1 and T2 words. Negative T1 (versus neutral T1) resulted in lower accuracy (a longer blink) for neutral T2, and negative T2 resulted in higher accuracy (shorter blink) than neutral T2, but only with a neutral T1. Thus, negative stimuli, compared to neutral information, both induced a longer blink and more effectively overrode the blink, perhaps due to increased attention directed to processing the potential threat and attention capture by the potential threat, respectively. Cold pressor also slightly increased T2 detection generally, which is contrary to the deficit-only view. Note, however, that the pain due to cold pressor was experienced before the attentional task, not during the task as in Sippel (2011) and Harker et al., (2011).

No research has been conducted directly comparing how different pain experiences motivate attentional processing. Induction of an attentional blink with RSVP tasks that tap different aspects of attention is a useful way to do this.

Methods

I have planned four phases of experiments. Phase 0 is the pilot stage where I will develop the tasks I will use. In Phases 1-3, I will examine the effect of different types of pain (acute, chronic, or lifestyle pain) on attentional processing of aversive pain-related stimuli. To do so, I will use three RSVP task variants: 1) a neutral letter/number RSVP task to assess the effects of pain experience on general attentional resources; 2) an emotional word RSVP task with aversive and neutral words presented at T1, to determine how pain information affects induction of the blink compared to neutral information (Manipulating T1); and 3) an emotional word RSVP task with aversive and neutral words presented at T2, to assess how pain information overcomes the blink compared to neutral information (Manipulating T2). See Table 1 for a summary of the planned studies and phases.

Table 1. Summary of planned experiments.

Phase 0 Piloting	Phase 1 Acute physical discomfort	Phase 2 Chronic pain	Phase 3 Lifestyle pain
Pilot neutral RSVP	+ neutral RSVP	+ neutral RSVP	+ neutral RSVP
Pilot manipulating T1 RSVP	+ manipulating T1 RSVP	+ manipulating T1 RSVP	+ manipulating T1 RSVP
Pilot manipulating T2 RSVP	+ manipulating T2 RSVP	+ manipulating T2 RSVP	+ manipulating T2 RSVP
Pilot physical discomfort induction			
Ethical approval granted	Ethical approval granted	Ethical approval granted	

Phase 0

Phase 0 involved the development of three RSVP tasks that demonstrate a blink in healthy, pain free participants, and the development of a way to induce tonic, short lasting physical discomfort to approximate acute pain experience. First, a basic RSVP task with number distractors and two target letters in each stream was run (Experiment 1). See Figure 1 for the trial outline. Participants completed 288 trials.

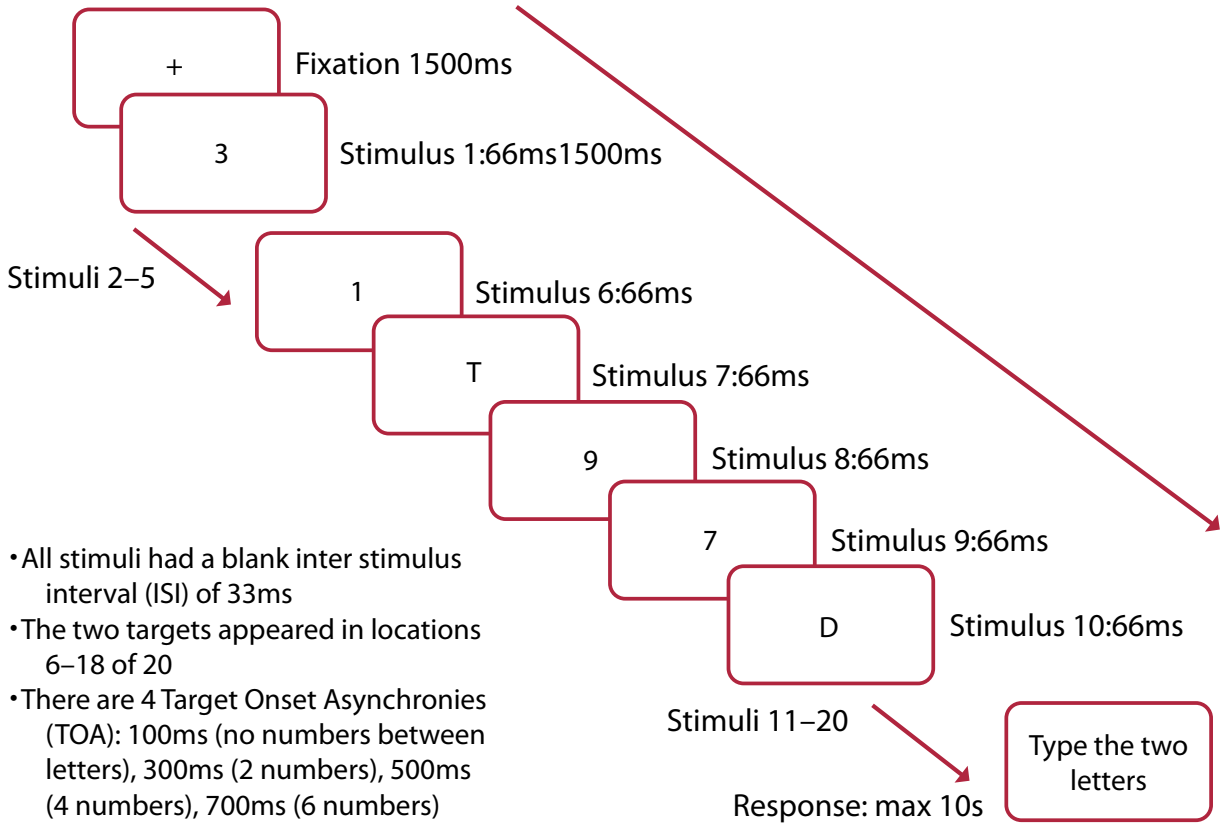

Figure 1. Outline of an example trial in Experiment 1.

Second, RSVP tasks with neutral and aversive words were developed and run. See Figure 2 for the trial outline. The filler words were presented in black ink, and the target words in green. Pairs of aversive and neutral words were gathered from previous attentional bias experiments (using dot-probe and Stroop tasks; Asmundson, Wright, & Hadjistavropoulos, 2005; Dehghani, Sharpe, & Nicholas, 2003; Kaur, Butow & Sharpe, 2013; Keogh, Ellery, Hunt & Hannent, 2001; Sharpe, Ianiello, Dear, Perry, Refshauge & Nicholas, 2012). The aversive words tap different aspects of pain including sensory pain (e.g. burning), affective pain (e.g. despair), health threat (e.g. collapse), disability (e.g. helpless), and physical threat (e.g. harmful) themes. Neutral T1 - neutral T2 pairs and filler neutral words were generated from a psycholinguistic database to match the aversive-neutral pairs on frequency and length. Valence and arousal ratings for the target words were gathered from independent raters.

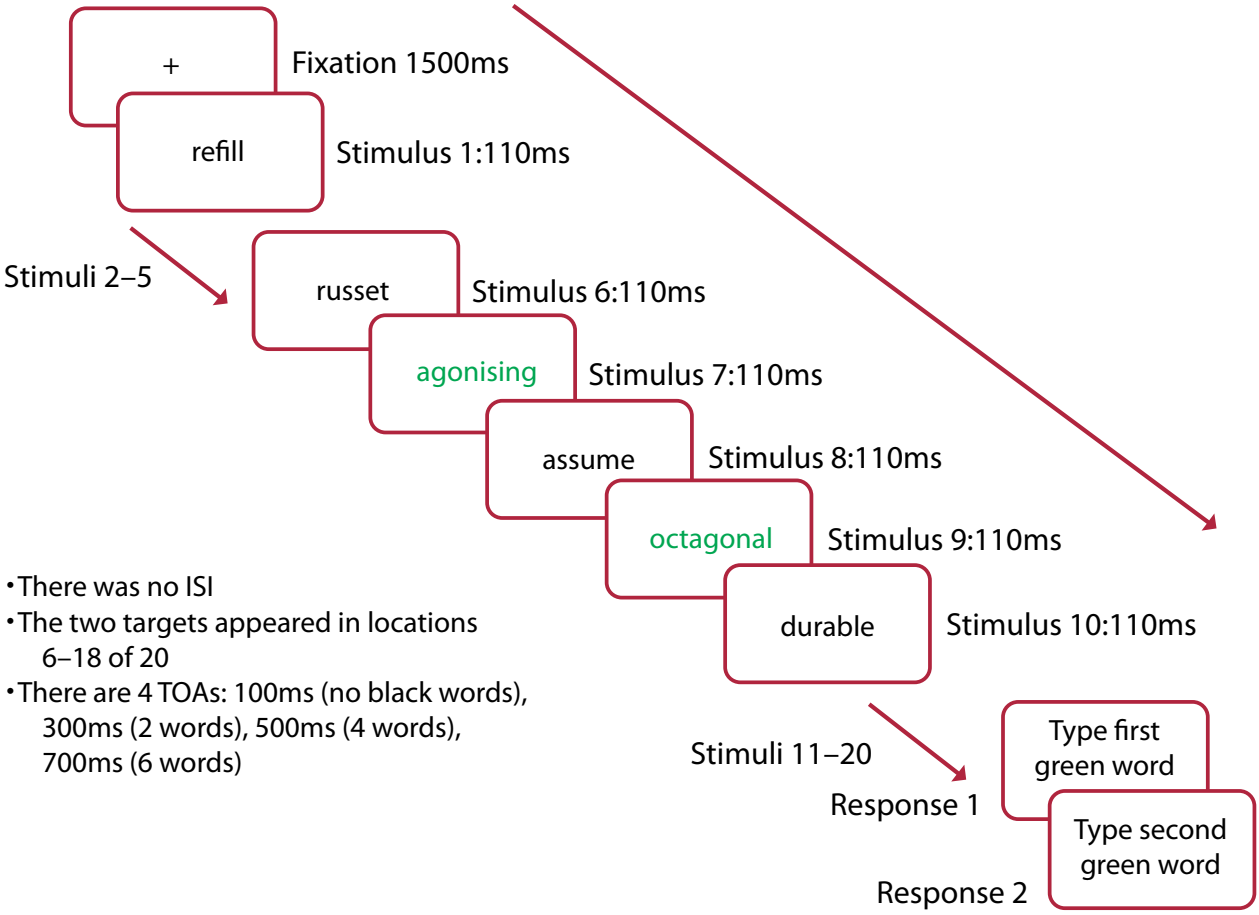

Figure 2. Outline of an example trial in Experiment 2.

In Experiment 2 (manipulating T1), T2 was always neutral but T1 was neutral or aversive (96 trials of each, 192 total). If attention is allocated to potentially threatening information, then an aversive T1 will induce a longer blink (assessed by accuracy of neutral T2 identification) than a neutral T1. In Experiment 3 (manipulating T2), T1 was always neutral but T2 was neutral or aversive (96 trials of each, 192 total). If attention is captured by potentially threatening information, then an aversive T2 will overcome the blink more effectively, resulting in a shorter blink than for a neutral T2 (i.e., increased T2 reporting for aversive words).

Third, a method of physical discomfort induction was assessed that was designed to be somewhat similar to the tonic nature of chronic pain experience. This method involves pressure on the skin on either the left or right lower shin, which produces a fairly consistent aching experience between people (Forgione & Barber, 1971). I have modified a tendon support to deliver pressure via glass beads (Figure 3).

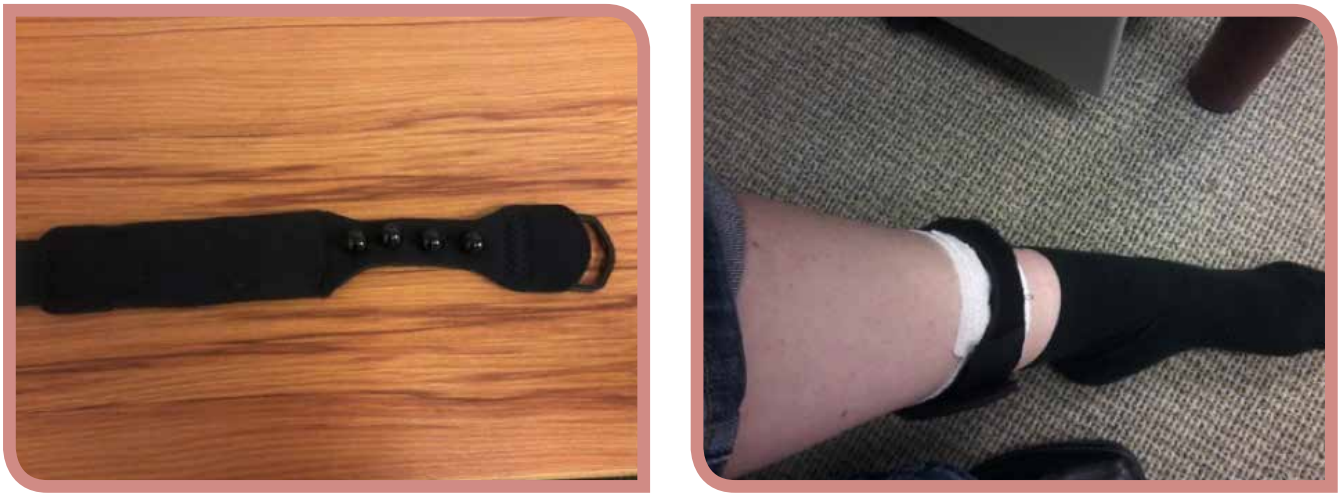

Figure 3. Pressure cuff used to induce tonic physical discomfort.

The intensity, initiation, and termination of the pressure are completely within the participants' control. The use of this pressure cuff means that participants can experience a tonic discomfort (rather than transitory sensation such as electric shock) throughout entire RSVP task blocks (rather than before the attention task as with cold pressor). Thus, this method of inducing discomfort produces results that are more comparable to the experience of chronic pain participants in terms of tonicity than electric shock or cold pressor. However, unlike the chronic pain experience, these participants know the discomfort will fade immediately on release of the cuff, and is thus less threatening. A further advantage of the cuff is that it can be moved between the left and right leg in alternating trial blocks, making habituation to the pressure unlikely. Participants reported mild discomfort and no lasting effects.

Phases 1, 2 and 3 (currently running Phase 1)

Phase 1 will involve running the three RSVP task variants while healthy, pain-free participants concurrently wear the pressure cuff. Phases 2 and 3 will involve participants who are already experiencing pain; as such, the physical discomfort induction will not be used. Phase 2 will involve recruiting participants experiencing chronic pain to complete the three RSVP tasks. Chronic pain will be defined as "pain that is present almost every day and has lasted, or is expected to last, more than six months" (New Zealand Health Survey definition; Ministry of Health, 2013). Performance of participants with chronic pain will be compared to age-matched controls. Phase 3 is not yet fully defined, but will involve recruiting participants experiencing lifestyle pain (current ideas include tattoo associated pain, sports injury associated pain, pain associated with extreme rituals) and appropriate control participants to complete the three RSVP tasks.

The reason for recruiting participants experiencing different kinds of pain is to allow me to get at different aspects of motivated cognition. First, participants in Phase 1, experiencing acute physical discomfort, might have less threat associated with their experience than chronic pain participants, and lifestyle pain participants may assign different meaning (outcome of something worthwhile) and valence (less negative) to their pain than acute and chronic pain participants. Second, participants with chronic pain may have more of a drive to reduce pain than people experiencing acute or lifestyle pain (Durnez & Van Damme, in press). Furthermore, inclusion of participants not experiencing chronic pain will help me to disentangle the role of depression and anxiety (which also affect cognition and have a high incidence in chronic pain patients) from the role of pain itself in any attentional blink effects. In summary, the use of three participant groups will permit examination of how different kinds of pain experience specifically motivate attention to aversive pain-related stimuli as compared to neutral stimuli. The main goal for each phase is to assess how the pain experience motivates allocation of attentional resources to neutral and aversive information. In future studies, it will be important to use healing words or words that signal relief from pain (as according to motivated cognition, attention should also be motivated to such information), and further to use words related to the patients' current pain experience compared to words that are not (e.g. surgical versus arthritic words).

Conclusion

This PhD thesis will give a fresh perspective on the relationship between pain and cognition, and may suggest fruitful avenues of research in developing pain management techniques for the 17% of New Zealanders in chronic pain (New Zealand Health Survey definition; Ministry of Health, 2013). Chronic pain is the result of either the protection system detecting threat where there is none (because of changes in the peripheral nociception system, the spinal cord, or the central nervous system), or a by-product of a condition where the ongoing threat level cannot be eliminated (as in arthritis or cancer pain). An increase in threat detection may therefore be a hallmark of cognition in chronic pain. If so, in future research it will be important to know if this is the result of changes in the protection system or if it precedes development of chronic pain. Testing if the motivated cognition proposal is descriptive of processing will get us further toward understanding the complex relationship between pain and cognition.

If any readers know of relevant previous research or have comments, these would be warmly welcomed.

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