

A ROLE FOR ATTENTIONAL BIAS IN COGNITIVE DEFICITS IN CHRONIC PAIN?

BY

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A thesis submitted to the Victoria University of Wellington in fulfilment of the requirements
for the degree of Doctor of Philosophy

Victoria University of Wellington

2017

Publications

Data and ideas from this thesis have been reported in the following formats:

Godfrey, H.K., & Grimshaw, G.M. (2016, August 25). *Attention to body-tissue threat in chronic pain: A pre-registered report*. Retrieved from osf.io/2mw7x

Godfrey, H.K. Robinson, K., Noble, S.N., & Grimshaw, G.M. (2015). *Assessing motivated attention to pain-relevant information: A series of studies using the attentional blink in pain-free participants*. Poster presented at ACNS, the Australasian Society for Cognitive Neuroscience Conference: Auckland, New Zealand.

Godfrey, H.K. (2015). *Testing motivated cognition in pain using the attentional blink*. Research presented at European Pain School 2015, Plasticity in Neural Processing as a Mechanism in Chronic Pain: Siena, Italy.

Godfrey, H.K. & Grimshaw, G.M. (2015). *Testing motivated cognition in pain using the attentional blink*. Poster presented at the 40th Annual Scientific Meeting of the New Zealand Pain Society: Auckland, New Zealand.

Godfrey, H.K. (2015). Does pain always lead to poorer cognitive performance? *Ngau Mamae: Quarterly Publication of the New Zealand Pain Society, Summer Issue*.

Godfrey, H.K. & Grimshaw, G.M. (2014). *Motivated cognition: Attention to pain-related information*. Research presented at KiwiCAM, the New Zealand Cognition and Memory conference: Auckland, New Zealand.

Abstract

This thesis extends current understanding of cognitive deficits in people with chronic pain, specifically those related to attention. Researchers have proposed that attentional capacity is allocated to pain sensation, and away from current tasks and goals, leading to broad cognitive deficits (deficit-view). However, an attentional bias to pain-related information has also been observed in people with chronic pain, suggesting that attention is motivated towards information in the environment that is pain-related, and away from information that is not. Such an attentional bias away from information not related to pain may contribute to the cognitive deficits observed on tasks using neutral stimuli (motivated attention hypothesis). In testing the deficit-view and motivated attention accounts of cognitive deficits in chronic pain, I focused on how people attend to rapidly presented information (temporal attention) and the ability to control attention in the face of distraction.

To assess how chronic pain affects temporal attention, I used a phenomenon known as the attentional blink, which is a failure to detect a second target that appears soon after a first. Participants viewed a stream of briefly displayed words in which two target words (indicated by their colour) were embedded, with a manipulation of the time between the first and second target. They were required to report the two targets. In one experiment the first target was either pain-related or neutral (to assess how pain-relatedness affects the *induction* of the blink), and in other experiments this manipulation was applied to the second target (to assess how pain-relatedness affects how targets *overcome* the blink).

In undergraduate participants, both induction and overcoming of the attentional blink was modulated by pain-relatedness. I then compared the effects of manipulating the second target in people with and without chronic pain. If people with chronic pain have general deficits in temporal attention, a deeper attentional blink (relative to control participants) for both kinds of targets should be observed. If motivated attention describes processing in people with chronic pain, a shallower attentional blink for pain-related targets than neutral targets (an attentional bias) should be observed. Critically, this bias should be larger in participants with chronic pain. Contrary to both the deficit and motivated attention views, the attentional blink in participants with chronic pain did not differ from that in controls for either pain-related or neutral targets. Furthermore, neither group showed an attentional bias for pain-related targets, and a follow-up experiment failed to replicate the attentional bias observed in undergraduate students as well. Collectively, these findings suggested that attentional bias, as assessed by modulation of the attentional blink, was not reliable. A

stronger test of the deficit-view and motivated attention hypothesis was needed. I shifted focus to another attentional domain, the control of distraction.

To assess how chronic pain affects attentional control, I used an emotional distraction task, in which participants identified a target letter in an array that flanked irrelevant distractor images that were either intact or scrambled. Intact images depicted either extreme threat to body-tissue, or benign scenes. Distraction is indicated by slowing on intact relative to scrambled distractor trials. If people with chronic pain have general deficits in attentional control, they should show greater distraction from both kinds of images (relative to controls). If motivated attention describes processing in people with chronic pain, greater distraction from body-threat images than neutral images (relative to controls) should be observed. While all participants were more distracted by images depicting extreme threat to body-tissue, people with chronic pain were not more distracted than control participants for either image type. Findings fail to support either a deficit or motivated attention view of attentional control in chronic pain.

Although these experiments do not provide evidence that chronic pain affects attentional processing, across experiments people with chronic pain reported that they experience deficits in attention, and they showed behavioural evidence of psychomotor slowing. These findings suggest that, as is repeatedly reported in the literature, people in chronic pain feel like they have attentional deficits, and that some aspects of cognitive and/or motor processing are impacted. Careful consideration is given to what specific cognitive functions might be impaired in chronic pain. The outcome of this discussion suggests pertinent research directions to further understanding of cognition in chronic pain experience.

Acknowledgements

Many people and organisations have contributed to the work in this thesis. My first thanks go to my primary supervisor, Gina Grimshaw. There are so many things to thank you for; you have been a huge part of my growth as a person and a scientist. Here I thank you for keeping a spot for me to return after I left university after my Masters, for supporting my development as a researcher, as a writer, as a thinker, and for showing me by example how to be an excellent mentor and scientist. I am very grateful for the times you encouraged me to be the best researcher I can be, and for the times you steered me back to reality when I was too immersed in the research or stuck in my own head. I will miss our discussions about our work, collaborating on other projects, and talking about science in general. To Ron Fischer, my secondary supervisor, thank you for coming on board to my project when my other secondary supervisor left. Thank you for the interest and support you have given to me and to my work. Your feedback has improved this thesis greatly.

Thank you to all the participants that contributed their time and energy. Especially to those who experience chronic pain for whom participation was tiring. I enjoyed meeting you all and am hugely appreciative of your contribution to the experiments in this thesis. Thank you to the organisations who provided funding for thank you vouchers, for taxi chits for participants who experience chronic pain, and for travel to meetings, in alphabetical order: The New Zealand Pain Society, The Rotary Club of Port Nicholson, and Victoria University of Wellington.

Thank you to the members of the Cognitive and Affective Neuroscience lab, past and present, for your feedback on my designs and data analyses, and for your friendship and support. Thank you to Kameron for developing the macro to help me score target responses, and to Claire, Kealagh, and Sophia for helping with data collection. Thank you to Amy, Daniel, Kealagh, Michael, Rebecca, Sophie, and Thomas for sitting in the lab with me when I ran participants outside of working hours. Thank you to Amy, Angus, Kealagh, Laura, Michael, Rosie, and Sophie for proofreading my thesis – any mouldy psychics are on me though!

Thank you to my family, Mum, Dad, Holly, Karyn, Kevin, Jordyn, and Lesley, and my friends for listening to me tell you the latest exciting thing I had learnt, for sitting through practice talks, and for giving me a boost when I needed it. Thank you for understanding when I was less present during busy times. Thank you to my Mum and Dad for encouraging my

curiosity about the world and how things work. Thank you to Dad for sharing software, and to Kev for providing a computer, iPad, and IT support for them.

There are many other people I have met in the New Zealand Pain community, and at European Pain School and other conferences who gave me encouragement, advice or lent an ear – thank you.

Last, but never least, thank you to my husband, Dave. When we met two and a half years ago, I am not sure that you understood what me being a PhD candidate entailed for our new relationship. Thank you for the practical support you have given: cooking dinner, picking me up on late nights, passing on your graphic design skills to help me improve my figures and diagrams (albeit with immense frustration at the design guidelines of APA format), and helping me to do the final reference check. Thank you for encouraging me when I was tired, reminding me why I was doing this PhD, reminding me of my abilities and strengths. You are my rock and I hope one day I can support you attaining in your goals with the patience and love you have shown me.

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Chapter 1

“A better understanding of how the brain responds in an adaptive and maladaptive way during the transition to, and maintenance of chronic pain is key if we are to target these mechanisms for better patient management, pain relief and wellbeing” (Wiech & Tracey, 2013).

Chronic pain, defined as pain lasting more than three (or sometimes six) months, is a significant individual and societal issue. The estimated prevalence in New Zealand is 20.8 %, meaning one fifth of the population currently endorse the statement that they experience “pain that is present almost every day and has lasted, or is expected to last, more than six months” (2015/2016 New Zealand Health Survey definition; Ministry of Health). There is a higher incidence of chronic pain in older people and those of lower socioeconomic status (2015/2016 New Zealand Health Survey, Ministry of Health), meaning that chronic pain disproportionately affects our most vulnerable people. Chronic pain affects quality of life: restricting the individual’s ability to work and study, to maintain a household, to raise children, and to participate in family, social, and community life (Pfizer, 2012). Chronic pain also has a significant economic cost. Chronic pain is estimated to the cost the US economy more than 100 billion US dollars each year (AAAOS Committee on Research, 2003; cited in Mollet & Harrison, 2006). Closer to New Zealand, pain costs Australia 34.30 billion AU dollars each year (Access Economics Australia Report, 2007). There are researchers working on the “problem of pain” at many levels – from sensory neuroscience research focusing on nociception, through to higher levels focusing on how government policy impacts those in chronic pain (Moon, 2003). In this thesis, I use cognitive psychology methodology to extend understanding of interactions between pain and mental processes.

What is Pain?

Although pain is very unpleasant, it is functional because it protects our body-tissue from injury and damage. Pain is multidimensional and complex, and theories of pain reflect this. Theories such as the Neuromatrix Theory (Melzack, 2001; Melzack & Katz, 2013) and Moseley’s (2007) reconceptualisation of pain describe a centralised system that detects threat to, or imbalance of, the body through input components; and responds via output components. In Moseley’s theory, pain, both acute (short-term) and chronic (long-term), is an experience generated when threat to body-tissue is perceived by a *threat protection system* (Moseley, 2007; Moseley & Jones, 2009; Tabor, Keogh, & Eccleston, 2017). The threat protection system receives input about the state of our tissues and produces output to protect

us. Nociceptors in the periphery respond to stimulation from pressure, heat and cold, and toxic chemicals (Moseley & Jones, 2009; Ringkamp, Raja, Campbell, & Meyer, 2013). When the nociceptors fire, signals are sent to the brain via the spinal cord. Multiple sources of information then interact to determine whether the system perceives threat to the body. Bodily sensory information is only one source of many. Other sources include cognitive factors such as the individual's previous experiences and expectations; their cultural practices; their beliefs, knowledge, and reasoning ability; their family and work context; the meaning they assign to their pain; and their current mood and emotions (Moseley, 2007; Moseley & Jones, 2009; Peters, 2015). If threat to body-tissue is perceived, a number of systems are then mobilised to protect us (Moseley, 2007). Most of these protective responses operate outside of our awareness. For example, activation of the sympathetic nervous system (mobilising flight or fight responses) and immune system changes (mobilising infection fighting) occur without conscious awareness (Butler & Moseley, 2003; Moseley & Jones, 2009). Pain itself is a conscious protective response associated with engagement of the threat protection system (Moseley, 2007); it acts as a warning, guiding protective behaviours and thoughts (de C Williams & Craig, 2016; Kruger, 2001).

Key to the description of pain are two components: the conscious experiential nature of pain, and the activation of behavioural, physiological, and cognitive responses that reduce threat (Melzack 2001; Melzack & Katz, 2013; Moseley, 2007). These two components make pain similar to other somatic experiences such as hunger, thirst, and itch. Some researchers (as early as Wall, 1979) see pain as resulting from, or interacting with, a protective system that serves to return the body to homeostasis, much like the process underlying hunger and thirst experience (Craig, 2002, 2003; Leknes & Tracey, 2008; Melzack & Katz, 2013; Morrison, Perini, & Dunham, 2013; Panerai, 2011; Paulus, 2007; Swanson, 1984; Tabor et al., 2017; Van Damme, Crombez, & Eccleston, 2008; Wall, 1979; Watt, 2004). Craig (2003) proposes that hunger, thirst, itch, and pain experiences are homeostatic emotions, or emotions that serve to motivate us to return to our optimal state. Hunger and thirst are the conscious experiences generated when the body is out of balance, needing food or water. Similarly, pain is the conscious experience of threat to body-tissue. Homeostatic emotions are also powerful motivators. When we are hungry, for example, it is difficult to focus on much except the need for food. Pain experience is similarly proposed to motivate actions that protect body-tissue (Craig, 2003; Tabor et al., 2017).

When pain is acute, the threat protection system is working optimally, protecting our body-tissue. Acute pain is commonly viewed as an adaptive response to an identifiable cause.

It may be very brief (on the order of seconds or minutes), or last up to thirty days (Marchland, 2012). Acute pain prompts actions to reduce pain, help-seeking behaviour for an illness or injury, and rest-seeking to allow healing time. Although we often think of pain as an extreme event arising from serious trauma, pain is also a daily experience that protects us in myriad ways. For example, sitting with crossed legs will, after a period of time, activate pressure nociceptors, which will start a cascade of processes that result in the feeling of being uncomfortable. Being uncomfortable motivates movement, which protects the tissue (Marchland, 2012).

In contrast to the adaptive functions of acute pain, chronic pain is commonly viewed as a maladaptive response to either on-going threat to body-tissues (for example, in arthritis), or to functional changes in the peripheral, spinal, or central nervous systems. It persists longer than three (Deodhar, Marcus, Cope, & Payne, 2009) or six months (Marchland, 2012). The distinction between acute pain and chronic pain has been compared to the distinction between fear and anxiety, and grief and depression (Swanson, 1984). Acute pain, fear, and grief are produced in response to an identifiable cause and result in protective cognitions and behaviours. Chronic pain, anxiety, and depression persist; the relationship between the original cause and current experience becomes decoupled, resulting in overprotective cognitions and behaviours, which can be maladaptive.

I focus on understanding cognitive processing in chronic pain. As such, in this thesis I mainly draw on literature that focuses on chronic pain. I do include discussion of literature that focuses on acute pain when it adds to understanding of cognition in chronic pain, or when the research has not been done in a chronic pain context. The relationship between pain and cognition is not unidirectional. Changes in cognition (for example distraction, attentional focus, and appraisal) can modulate the level of pain experienced (Bushnell, Čeko & Low, 2013; Jones, 2005; Moseley, 2007; Peters, 2015), but changes in pain experience can also modulate cognition. In order to develop effective pain management strategies, we need to understand both how cognitive functions are recruited to reduce pain experience (e.g., distraction; Frankenstein, Richter, McIntyre, & Rémy, 2001), and how activation of the threat protection system affects cognitive functioning. In this thesis, I focus on one direction – the impact of chronic pain experience on cognition.

Cognitive Deficits

Does pain experience affect the way we think? The general view is that chronic pain is associated with cognitive deficits. Deficits are part of the day-to-day experience of people

with chronic pain, who report 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, 2001¹). On a battery of tasks assessing executive function, people in chronic pain showed greater cognitive deficits than controls on nine aspects of executive control, particularly working memory and emotion regulation (Baker, Gibson, Georgiou-Karistianis, Roth & Giummarra, 2016²). They also show impairments on a recently developed scale, the Experience of Cognitive Intrusion of Pain (ECIP) scale. Participants in chronic pain were impaired on three factors – showing less control, more interruption, and greater rumination (Attridge, Crombez, Van Ryckeghem, Keogh & Eccleston, 2015³). These experienced impairments are thought to reflect poorer attentional control; poorer memory and executive functioning; as well as slowing in processing speed, psychomotor slowing (Moriarty & Finn, 2014; Moriarty, McGuire & Finn, 2011).

Chronic pain is also associated with functional and structural neurological changes. Frontal brain activation is reduced in chronic pain; in the anterior cingulate cortex (ACC), left and right superior parietal cortices, and right prefrontal cortex (Mao, Zhang, Bao, Liao, Yang, & Zhang, 2014). A meta-analysis suggests that the key areas that are associated with reduced activity in people with chronic pain, compared to control participants, are the left and right dorsolateral prefrontal cortex, the right parietal cortex, the left and right supplementary motor areas, and the left and right ventrolateral prefrontal cortex (Berryman, Stanton, Bowering, Tabor, McFarlane, & Moseley, 2013). Changes in white matter connectivity and grey matter density are also observed (Bushnell et al., 2013). Bushnell and colleagues' review of structural studies suggests that the dorsolateral and medial prefrontal cortices, the ACC, and the insula lose grey matter with chronic pain experience. Many of these areas that show changed function or structure in chronic pain are part of the frontoparietal network, important for attention and cognitive control (Ptak, 2012; Scolari, Seidl-Rathkopf, & Kastner, 2015).

¹ The full list of cognitive complaints are: "Forgetting a lot, recent things, appointments; minor accidents; not finishing things started; not keeping attention on activity; difficulty with concentration and thinking; making mistakes; difficulty reasoning and problem solving; confusion; reacting slowly; behaving confused or disoriented" (McCracken & Iverson, 2001; pp 394).

² The full list of impaired aspects of executive functioning are: "inhibit; shift; emotional control; self-monitor; initiate; working memory; plan/organise; task monitor; organisation of materials" (Baker et al., 2016; pp. 676).

³ The items loading on control were: "I can't stop thinking about pain; it is hard to think about anything else but pain; I can't push pain out of my thoughts". Interruption: "pain interrupts my thinking; pain easily captures my thinking; pain intrudes on my thoughts". Rumination: "pain goes around and around in my head; pain dominates my thinking; I keep thinking about pain; when my mind wanders it goes to pain" (Attridge et al., 2015; pp. 1980).

Researchers investigating the mechanisms underlying cognitive impairments associated with chronic pain have mostly taken a deficit-view. The deficit-view (or dyscognition) is based on a capacity model of cognitive resources (e.g., Desimone & Duncan, 1995; Dux & Marois, 2010; Kahneman, 1973; Marois & Ivanoff, 2005), and describes impaired cognition in pain (Crombez, Eccleston, Baeyens, & Eelen, 1996; Eccleston & Crombez, 1999; Keogh, Moore, Duggan, Payne, & Eccleston, 2013; Mercado, Barjola, Fernández-Sánchez, Guerra, & Gómez-Esquer, 2013a; Moore, Keogh, & Eccleston, 2012). According to the deficit-view, people have limited cognitive capacity and it can be consumed by pain, leading to disturbance of task-relevant cognitions and performance decrements on ongoing tasks (Crombez et al., 1996; Eccleston & Crombez, 1999).

Many of the studies that have assessed cognition in pain use self-reported deficits (e.g., Attridge, et al., 2015; McCracken & Iverson, 2001; Tesio et al., 2015), or neuropsychological batteries (e.g., Dick & Rashiq, 2007; Mifflin, Chorney & Dick, 2016; Tesio et al., 2015), which cannot isolate the specific cognitive mechanisms that are impaired. Experimental tasks are better able to target specific processes, such as interference (e.g., Stroop; Grisart & Plaghki, 1999), working memory, spatial attention, task switching and divided attention (e.g., Moore, Keogh & Eccleston, 2013a, 2013b), and linguistic processing (e.g., Leavitt & Katz, 2014) in chronic pain experience or acute pain models.

Experimental studies typically show that participants experiencing acute pain perform worse than controls. For example, performance for a tone discrimination task was worse when the task was completed during acute pain experience (electrically induced pain) than during a pain-free period (Crombez et al., 1996). Another example is provided by Moore and colleagues (2012), who found that acute pain experience (thermally induced pain) impaired performance on tasks assessing executive control of attention (an attention / task switching task), divided attention (a task requiring attention to both central and peripheral stimuli), and working memory (n-back task).

The few meta-analyses of cognitive deficits in people with chronic pain estimate the impact of chronic pain as moderate for working memory and executive control processes. Specifically, in working memory (Berryman et al., 2013), there was support for worse performance by people with chronic pain compared to people without chronic pain (effect sizes in the small to large range), in tasks measuring verbal and non-verbal working memory, attention and verbal working memory, immediate recall, continuous memory, and selective attention. In executive function (Berryman, Stanton, Bowering, Tabor, McFarlane, & Moseley, 2014), there was support for worse performance by people with chronic pain (effect

sizes in the small to moderate range) in tasks measuring complex executive function, set shifting, and response inhibition.

Many of the tasks that show pain-related impairments index processes that can be thought of as high-level, executive processes. Given the capacity view of cognition, higher-level executive processes require more cognitive resources than lower-level perceptual processes, and would therefore be more impaired when pain consumes some of an individual's capacity. Indeed, the boundary conditions for observing pain-related cognitive deficits that have been identified support the capacity view of executive function in pain. For example, some researchers report greater impairment only for those participants with chronic pain who are currently experiencing higher levels of pain (Eccleston, 1995; Grisart & Plaghki, 1999) or for the most difficult tasks (Eccleston, 1994; Mifflin et al., 2016).

Processing deficits in chronic pain are consistent with work by Engle, Kane, and colleagues (Engle & Kane, 2004; Kane, Conway, Hambrick, & Engle, 2007) on modelling cognitive control. Executive control processes rely on the integrity of the prefrontal cortex (Diamond, 2013; Miller & Cohen, 2001). In people with chronic pain, function of the prefrontal cortex has changed (areas in the frontoparietal network show less activation than in people without chronic pain; Berryman et al., 2013), and executive control is likely impaired. Engle, Kane, and colleagues suggest that attention is a key component of executive processing, and that *executive attention* is a process that controls allocation of capacity for current task completion in the face of distractions (Engle & Kane, 2004; Kane et al., 2007). Goals are important in Kane and Engle's description of executive attention – a key role of executive attention is to hold online the individual's current goals (Engle & Kane, 2004). This biasing of capacity ensures that perceptual processing and behavioural responses are congruent with the goal. Attentional deficits in chronic pain experience are especially prevalent in the literature for attention switching and attentional interference tasks, and the ability to focus on task-relevant information and ignore distracting information (attentional control) may be a potential factor underlying many cognitive deficits (Moriarty et al., 2011), including executive functioning.

In acute pain, safety goals are active and the attentional system seems to be engaged in monitoring for further danger, and for escape and pain relief opportunities. Tabor, Catley, Gandevia, Thacker, and Moseley (2013) demonstrated that acute pain experience and pain relief affordances affect distance perception. When a button could relieve pain, participants underestimated how far away the button was compared to when the button could not relieve pain. Tabor and colleagues (Tabor, Catley, Gandevia, Thacker, Spence, & Moseley, 2015)

further showed that participants underestimated how close a threatening stimulus was compared to a relief stimulus. These phenomena show that people in pain are motivated to avoid threat and seek relief, and that this motivation directs their attentional capacity and even changes their perceptual experience. Eccleston and Crombez (1999; see also Moore et al., 2012) proposed a theory with a similar protective motivation. They posit that pain experience automatically activates escape goals and behaviours; and that activation of these cognitions interrupts any other concurrent goals or behaviours. These authors suggest that chronic pain repetitively interrupts cognitions in order to redirect attention to the potential threat. This repeated interruption causes the cognitive deficits. In summary, attention may be a key process impaired in chronic pain experience.

Motivated Attention Hypothesis

While the deficit perspective on cognition in chronic pain recognises that pain consumes attentional capacity, it does not consider other ways the threat protection system may use attention to alter allocation of cognitive capacity to protect body-tissue. In their motivational account of attention to pain, Van Damme, Legrain, Vogt, and Crombez (2010) propose an assignment of attentional capacity in chronic pain experience that is based on goals. Homeostatic needs are important for assignment of attentional capacity, but so are individual goals. In addition to signaling an unaddressed threat to homeostasis (threat to body-tissue), pain also motivates goals, such as avoiding, terminating, or managing pain (Van Damme et al., 2010). The threat to homeostasis and the pain-relevant goals combine to enhance processing of pain-related information (and the pain experience) and to decrease processing of information unrelated to pain; resulting in an attentional bias to pain-related stimuli. Van Damme and colleagues mention that tasks unrelated to goals are likely to suffer (i.e., producing a deficit in performance), but do not apply their theory specifically to an explanation of cognitive deficits. Following Van Damme and colleagues' terminology, I refer to the theory that an attentional bias to pain-related information affects cognitive functioning in chronic pain experience as *the motivated attention hypothesis*.

The motivated attention hypothesis is consistent with the homeostatic role of pain (Craig, 2003). Pain serves to return us to a state of safety, and assignment of attentional capacity (to signals related to pain or threat to body-tissue) is one way to prompt such homeostatic behavior. Like the deficit-view, motivated attention builds on theories of limited resource capacity. In a seminal paper, Desimone and Duncan (1995) propose and present evidence for Biased Competition Theory. This theory describes how, in a limited capacity

system such as attention, bottom up (stimulus-driven) and top down (goal-driven) processes can bias capacity towards processing of certain stimuli. Desimone and Duncan (1995), like Engle and Kane (Engle & Kane, 2004; Kane et al., 2007), see attention as the mechanism that selects and prioritises processing within a limited capacity system. Certain kinds of stimuli consume more capacity than others and benefit from prioritised processing, resulting in an attentional bias. Threatening stimuli (e.g., biological threats: spiders, snakes; weapons; fire; mutilations; facial expressions signaling fear) are one kind of stimuli that ‘win’ capacity in such a system. Threatening stimuli often gain attentional capacity over other stimuli (Öhman, Flykt, & Esteves, 2001; Okon-Singer, Lichtenstein-Vidne, & Cohen, 2013; Pourtois, Schettino, Vuilleumier, 2013; Yiend, 2010), consistent with the existence of evolved cognitive systems that are sensitive to threat.

While the motivated attention perspective has not been explicitly applied to understanding cognitive deficits described in the chronic pain literature, in other literatures similar principles are being used to enhance understanding of cognitive deficits. Motivated attention may therefore be a broad principle that guides human behaviour. Humans seem to have been evolutionarily (Damasio & Carvalho, 2013; Lang, Bradley & Cuthbert, 1997) and developmentally (Frankenhuis, Panchanathan, & Nettle, 2016; Nettle & Bateson, 2015) shaped to be alert to things that may harm us; resulting in allocation of cognitive capacity to processing stimuli that signal threat.

For example, Frankenhuis and de Weerth (2013) and Frankenhuis and colleagues (2016) describe how the environment in which a child is raised may produce cognitive enhancements useful for that environment. Frankenhuis and de Weerth propose that biased 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 extreme stress (such as abuse). A deficit-view attributes the cognitive impairments to developmental insult to neural systems that support cognition. A motivated attention view suggests that attentional systems may be intact, but that environmental factors have driven a shift in attentional priorities. This molding of cognition explains why, compared to controls raised in non-stressful environments, people who were raised in stressful environments show deficits on tasks that require attention to neutral stimuli but enhancements on tasks that use threatening stimuli (Frankenhuis & de Weerth, 2013; Frankenhuis et al., 2016). Even with cognitive deficits in some domains, people raised in stressful environments are better able to adapt to the environmental context (Mittal, Griskevicius, Simpson, Sung, & Young, 2015) compared to non-stressed controls.

Even in adults, experience seems to mold cognition. Levy-Gigi, Richter-Levin, Okon-Singer, Kéri, and Bonanno (2016) describe a study in which police (who have been exposed in the past to distressing crime scenes) completed a letter discrimination task while ignoring concurrent distractors. Compared to a group of matched non-police participants, the police showed poorer performance on the letter discrimination task when presented with distracting low arousal negative images, suggesting that those images captured their attention. However, when presented with distracting high arousal negative images, the police performed better than matched controls on the letter discrimination task. Levy-Gigi and colleagues suggest that due to their crime scene experience police could ignore the high arousal negative stimuli more successfully than controls, even though cognitive deficits were observed in the presence of low arousal stimuli (Levy-Gigi et al; 2016).

It is clear from the work of Frankenhuys and colleagues' (Frankenhuys & De Weerth, 2013; Frankenhuys et al., 2016) and Levy-Gigi and colleagues (2016) that cognitive deficits and motivated attention are not mutually exclusive. The specific pattern of deficits depends on the stimuli or tasks that are used to assess performance. When neutral, non-threatening, stimuli are used to assess functioning, deficits should be observed because attention will be focused on the threat of the pain experience. Only when the background of the individuals is considered can contexts be identified in which motivated attention could be at play. In chronic pain research specifically, only by examining attention to pain-related stimuli can motivated effects be identified.

As well as predicting specific patterns of deficits and preserved processing in chronic pain experience, motivated attention would result in a negative relationship between cognitive performance and attentional biases, such that the greater the attentional bias, the poorer general cognitive functioning. In a limited capacity system, assignment of capacity to one type of stimuli would take away capacity from another. Although, there is no research with chronic pain that assesses this relationship, there are two studies that show that attentional biases to emotion are negatively correlated with cognitive functioning. Hakamata, Matsui, and Tagaya (2014) recruited healthy participants who completed a neuropsychological battery assessing functioning of five general processes (including attention), and a dot-probe task that measured attention to emotional stimuli. They reported that the greater the bias to attend to emotional stimuli on the dot-probe task (both negative and positive, compared to neutral), the poorer performance was on attention tasks that were part of the neuropsychological battery. Hsu and Davison (2017) followed Hakamata and colleagues' rationale and correlated deficits in attention with emotional biases (both negative

and positive) in currently-depressed, previously-depressed, and never-depressed participants. On an emotional Stroop task, currently depressed participants were slower to respond to emotional words (compared to neutral, an attentional bias) than previously- and never-depressed participants. Furthermore, Hsu and Davison reported that for currently-depressed participants, the greater the bias to attend to emotional stimuli, the poorer performance was on the other measures of selective attention.

Motivated attention in chronic pain experience.

The premise of motivated attention is not that experiencing chronic pain simply makes it hard to think; rather it is that pain (or the threat protection mechanisms producing pain) shifts our priorities, and thus processing capacity, towards information related to our pain experience and away from information unrelated to our pain experience. If pain biases assignment of cognitive capacity, then participants experiencing chronic pain may show altered performance on cognitive tasks that use pain- or threat-related stimuli compared to neutral stimuli. Whether performance is enhanced or impaired by prioritised processing of pain-related information will depend on the task used to assess performance.

Biased attention in chronic pain experience is theoretically consistent with the understanding of pain as being protective. Pain is just one way of protecting our body-tissue, as are regulation processes such as immune responses and preparing for flight or fight (Moseley, 2007; Moseley & Jones, 2009). An attentional bias can be thought of as another regulatory process, a way of anticipating threats in the environment so they can be responded to quickly. Such a bias would be protective but perhaps not suited to the context of everyday life. Hence, cognitive deficits are reported as part of chronic pain experience, and are observed by cognitive psychologists in experimental studies. In a model of adaptive and maladaptive response to threat in anxiety and other disorders with a stress component, Shechner and Bar Haim (2016) link anxiety and stress disorders to a mismatch between the context and the degree of attention to threat. They propose that individuals with these disorders over-attend to threats in the relatively safe context of modern life, leading to maladaptive outcomes. Similarly, biased attention to pain-related information in people with chronic pain is maladaptive for everyday functioning, and may contribute to the cognitive deficits observed.

Differences in developmental and lifetime experience are not the only factors that can motivate attention. Research on homeostatic emotions shows that there are processing biases towards relevant information that could return the individual to balance (e.g., in thirst, Aarts,

Dijksterhuis, & De Vries, 2001; and in hunger, Seibt, Häfner, & Deutsch, 2007). Thirsty and hungry people are hypervigilant to stimuli that are related to their need. Similarly, in chronic pain experience, people are hypervigilant to signals of pain and threat (Crombez, Van Damme, & Eccleston, 2005). People with chronic pain are faster to respond to stimuli in a location in which a pain-related stimulus was previously presented as compared to a neutral stimulus in dot-probe tasks (Asmundson, Wright & Hadjistavropoulos, 2005; Dehghani, Sharpe, & Nicholas, 2003). People with chronic pain are slower to name the colours of pain-related words than neutral words in Stroop tasks (Asmundson et al., 2005; Crombez, Hermans & Adriaensen, 2000; Duschek, Werner, Limbert, Winkelmann, & Montoya, 2014). And people with chronic pain are more likely to endorse the pain-related interpretation of ambiguous language than the neutral interpretation in ambiguity resolution tasks (Heathcote, Jacobs, Eccleston, Fox & Lau, 2017; Schoth & Liossi, 2016).

Importantly, these attentional biases in people with chronic pain are not always compared to biases in people without chronic pain. Sometimes interpretations are made solely on observed biases in chronic pain experience (Crombez et al., 2000; Dehghani et al., 2003; or, when a comparison is made, the magnitude of the attentional bias sometimes does not differ between participants with, and without, chronic pain (Asmundson, et al., 2005). While an attentional bias towards pain-related information might be a protective mechanism in all people, because protective processes are more engaged when pain is ongoing, a motivated attention view suggests that the bias would be enhanced in people who have chronic pain. Therefore, in order to test for evidence of motivated attention, biases in people with, and without, chronic pain must be compared; and people in chronic pain should show larger attentional biases.

A meta-analysis on attentional bias in chronic pain estimated the size of the attentional bias to pain-related stimuli compared to neutral stimuli in chronic pain experience to be small (Crombez, Van Ryckeghem, Eccleston & Van Damme, 2013a). For the difference in attentional bias between people with, and without, chronic pain, meta-analyses estimates range from non-significant to moderate. Crombez and colleagues (2013a) report that the comparison between the magnitude of the pain-related attentional bias in participants with, to those without, chronic pain (across studies using emotional Stroop, dot-probe, and spatial cueing tasks) is non-significant. Roelofs, Peters, Zeegers, & Vlaeyen, 2002 report larger biases in participants with chronic pain, than controls, on an emotional Stroop task, for sensory (e.g., *piercing*) and affective (e.g., *exhausting*) pain words. Schoth, Nunes, & Liossi (2012) report a moderately larger attentional bias in people with chronic pain than controls,

as assessed with visual probe tasks. Thus, although it appears that attentional biases to pain-related information are observed, it is not yet clear whether they are bigger in people with chronic pain, as the motivated attention hypothesis would lead one to predict.

Outline of the Thesis

In this thesis, I explore differences in attentional processing that are associated with chronic pain, using both a deficit-view and a motivated attention framework. I use both self-reported attentional processing and experimental tasks to compare attention in people with, and without, chronic pain. The experimental tasks allow me to directly compare processing of pain-related and neutral stimuli, and to isolate attentional, as opposed to other cognitive processes. I test the hypothesis that activation of the threat protection system guides attention to information that signals threat in all people; and that in people with chronic pain protective processing still drives patterns of attention. That is, not only is current processing interrupted (cognitive deficits), but cognitive capacity is motivated towards information processing and behaviours that are most adaptive for survival, more so in people with, than without, chronic pain (motivated attention).

I suggest that the theoretical perspective provided by the motivated attention hypothesis makes testable predictions about attentional biases *and* cognitive deficits in chronic pain experience, which can be observed within the same experiment. This can be done by observing attention to both pain-related and neutral stimuli in the same task in participants with, and without, chronic pain. Both the deficit-view and the motivated attention hypothesis describe a limited capacity attentional system, but differ in that the deficit-view posits that greater attention to pain experience contributes to poorer functioning; whereas the motivated attention hypothesis suggests that greater attention to pain-related information contributes to deficits for information unrelated to pain or threat. If processing biases in chronic pain are driven by motivated attention, then task performance should reflect enhanced processing of pain-related stimuli, and impaired processing of neutral stimuli. Alternatively, the deficit-view suggests that chronic pain should be associated with impaired processing of both pain-related and neutral stimuli.

The main tasks that have been used to assess attentional bias in people with chronic pain are the dot-probe and emotional Stroop tasks (e.g., Asmundson et al., 2005; Crombez et al., 2000; Dehghani et al., 2003; Duschek et al., 2014). However, there is not a reliable difference in attention bias between people with, and without, chronic pain using either task (Asmundson et al., 2005). A necessary condition of the motivated attention hypothesis is that

the attentional bias towards pain-related information is greater in people with chronic pain than controls. The dot-probe task may also not be a reliable measure of attentional bias (Chapman, Devue, & Grimshaw, 2017; Dear, Sharpe, Nicholas, & Refshauge, 2011; Schmukle, 2005). As mentioned above in the review of cognitive deficits in people with chronic pain, attentional control (the ability to focus on task-relevant information and ignore irrelevant information) may be a key process impacted by chronic pain (Moriarty et al., 2011). I therefore focus on assessing how chronic pain experience motivates allocation of attentional capacity in two types of attentional processing that have not previously been closely examined in chronic pain experience: temporal attention and the control of distraction. Both types of attention rely on filtering of task-relevant from task-irrelevant information.

In Chapter 2, I use the rapid-serial-visual-presentation paradigm (RSVP) to compare temporal attention processes in people with, and without, chronic pain. In Chapter 3, I use an irrelevant-distractor paradigm to similarly compare the control of spatial attention. In the following sections I briefly review these two different types of attention. Specific methodological details and predictions are provided in the relevant chapters.

Temporal attention.

In everyday life, we encounter a lot of rapidly changing information. That is, we encounter many stimuli we need to perceive and respond to sequentially. 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 at the appropriate time (Lang et al., 1997; Lang & Bradley, 2010). One method for testing serial information processing (also called temporal attention) is to use RSVP to present a rapid stream of information (Raymond, Shapiro & Arnell, 1992). Embedded in the stream are two target stimuli. Processing of each target in the sequence takes time, so a bottleneck can arise when the two targets appear close together in time. When the time between the two targets is short, the processing of new information (the second target) can be delayed (or even skipped altogether); inducing an attentional blink (Asplund, Fougner, Zughni, Martin, & Marois, 2014; Chun & Potter, 1995; Dux & Marois, 2009; Marois & Ivanoff, 2005, McHugo, Olatunji, & Zald, 2013).

In Chapter 2, I assess temporal attention for neutral and pain-related stimuli in undergraduate participants to determine whether attention is biased towards pain-related information generally, and then explore whether attentional bias in temporal attention is

larger in those who have chronic pain. I manipulate whether the task-relevant stimuli in the stream are neutral or pain-related. This manipulation allows comparison of temporal processing of both types of stimuli in people with, and without, chronic pain. If activation of the threat protection system in chronic pain experience generally reduces attentional capacity (as predicted by the deficit-view), then I expect to observe impaired temporal attention for both neutral and threat-related stimuli in people with chronic pain, compared to controls. However, if threat protection acts to motivate attention to threatening and pain-related information (as predicted by the motivated attention view), then people in chronic pain should be better able to process pain-related stimuli, but at the expense of their ability to process stimuli unrelated to pain. It is also possible that people in chronic pain will show an overall deficit in temporal attention in the context of an enhanced attentional bias to pain-related stimuli (providing evidence for both the deficit-view and the motivated attention hypothesis).

Attentional control of distraction.

In everyday life, we have to focus on information relevant to our goals and filter out irrelevant information. For example, we have to ignore the chocolate cake in the café in order to concentrate on what our friend is saying. Arousing stimuli, both positive and negative (including threat-related stimuli) are hard to ignore. In the emotional distraction paradigm (e.g., Grimshaw, Kranz, Carmel, Moody, & Devue, 2017; Gupta, Hur, & Lavie, 2016; Levy-Gigi et al. 2016), which builds on work in general control of distraction (Forster & Lavie, 2008), participants perform a simple and emotionally-neutral perceptual task while attempting to ignore task-irrelevant emotional and neutral images. Emotional distraction is reflected in slower response times when distractors are emotional than neutral.

In Chapter 3, I compare distraction by neutral and pain-related stimuli in participants with, and without, chronic pain. If activation of the threat protection system in chronic pain experience generally reduces attention capacity (as predicted by the deficit-view), then participants with chronic pain will show greater distraction by both neutral and pain-related stimuli. However, if threat protection acts to motivate attention to threatening and pain-related information (as predicted by the motivated attention view), then compared to controls, people with chronic pain will show relatively greater distraction by pain-related compared to neutral stimuli. It is also possible that people in chronic pain will show an overall deficit in control of attention in the context of an enhanced attentional bias to

pain-related stimuli (evidence for both the deficit-view and the motivated attention hypothesis).

Other cognitive processes.

In both experiments investigating attentional processing in chronic pain experience, I also examined processing efficiency in the three attention networks tapped by the Attention Network Test (ANT; Fan, McCandliss, Sommer, Raz, & Posner, 2002; Fan & Posner, 2004). The *alerting* network underlies vigilance to cues in the environment; the *orienting* network underlies shifts in spatial attention in response to cues; and the *executive* network underlies the ability to inhibit distracting information, and to select relevant information or responses (Fan et al., 2002). In the ANT paradigm (Fan et al., 2002) participants perform a flanker task with arrow stimuli pointing left or right. Their task is to report the direction of the centre arrow, while flanking arrows are congruent or incongruent with the centre arrow. Non-spatial cues are used to signal that the stimuli are coming soon (used in calculation of the alerting network measure). Spatial cues are used to signal where the stimuli will appear (used in calculation of the orienting network measure). Comparisons of reaction times on different trial types give indices of processing efficiency in the networks.

As the ANT paradigm uses neutral (i.e., not pain-related) stimuli, if activation of the threat protection system generally reduces capacity (as predicted by the deficit-view), then participants with chronic pain will show poorer efficiency in the networks. As the frontoparietal network seems to be impaired in chronic pain experience (Berryman et al., 2013; Bushnell et al., 2013; Mao et al., 2014), executive control of attention may be especially impaired in people with chronic pain. If so, the executive network (indexed by slowing on incongruent compared to congruent trials) will show especially poor efficiency in chronic pain experience, relative to the alerting and orienting networks. The attention network scores will also be used in correlational analyses with measures of temporal attention (Chapter 2) and the control of distraction (Chapter 3). These correlational analyses will be used to test for negative relationships between attentional biases and cognitive performance (as in Hakamata et al. 2014; and Hsu & Davison, 2017).

It is important to examine the laboratory assessed attentional processes in the context of people's subjective experience of their pain and attentional functioning. I also asked participants to report on their pain, control of attention, and vigilance to changes in their pain experience using standard questionnaires. The behavioural tasks provide indices of

attentional deficits and / or motivated attention, whereas the questionnaires provide indices of subjective experience.

Hypotheses & predictions.

Deficit-view: Because there is competition for cognitive capacity, greater attention to pain experience means impaired, or interrupted, attention to the environment.

Prediction 1: participants with chronic pain will show cognitive deficits compared to performance of participants without chronic pain. The RSVP, the emotional distraction, and the ANT paradigms allow for assessment of deficits in performance.

Motivated attention hypothesis: Even in chronic pain experience, protective processing still drives patterns of attention. That is, threat protection motivates assignment of attentional capacity towards information and behaviours most adaptive for survival, such as signals of threat to bodily tissue, signals of pain, or signals of relief of pain; and away from other kinds of information and behaviours.

Prediction 2: participants with chronic pain will show a larger attentional bias to pain-related stimuli compared to any bias observed in participants without chronic pain. A larger attentional bias in chronic pain experience can be observed with the RSVP and the emotional distraction paradigms.

Prediction 3: in chronic pain experience, the magnitude of the attentional bias in RSVP and emotional distraction will be negatively correlated with performance on the ANT.

Clinical Implications

While the main objective of this thesis is to develop, and test, theoretical perspectives on the interaction between pain and cognition, there are potentially more practical applications of this work. Clinically, the motivated attention perspective 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, 2013). Further elucidation of how chronic pain and cognition modulate each other may have similar clinical implications for people with chronic pain.

The motivated attention hypothesis predicts an enhanced attentional bias to pain-related information in chronic pain experience. This biases may play a causal or maintaining role in the experience of cognitive deficits. If so, modifying the bias may be a

successful way to ameliorate the deficits. There is a similar line of research in the alcohol addiction (McGeary, Meadows, Amir, & Gibb, 2014; Schoenmakers, de Bruin, Lux, Goertz, Van Kerkhof, & Wiers, 2010), depression, anxiety, and stress literatures (Shechner & Bar-Haim, 2016), using a treatment model called attentional bias modification (ABM: MacLeod & Clarke, 2015). ABM involves training people to attend away from disorder-related stimuli. For example, in a dot-probe training task, the probe always appears in the location of the neutral or positive cue, not the threat cue (MacLeod & Clarke, 2015).

ABM may also be a useful treatment model for people with chronic pain, as suggested by experiments with experimentally induced acute pain (Bowler, Bartholomew, Kellar, Mackintosh, Hoppitt, & Bayliss, 2017), by experiments with participants experiencing chronic pain (Schoth, Georgallis, & Liossi, 2013; Sharpe, Ianiello, Dear, Nicholson Perry, Refshauge, & Nicholas, 2012), by a review of the use ABM in pain (Sharpe, 2012), and by a theory of attentional bias in pain (The Threat Interpretation Model: Todd, Sharpe, Johnson, Nicholson Perry, Colagiuri, & Dear, 2015). However, ABM will only be useful if there is an underlying bias to retrain. Indeed, when there is an attentional bias to stimuli that cue that an acute pain is upcoming, distraction is a less effective pain management technique (Van Ryckeghem, Crombez, Van Hulle, & Van Damme, 2012).

A related treatment model is proposed by Dick and Rashid (2007) who suggest that training attention and executive functioning, as in Attention Process Training (APT), which is used in brain-damage rehabilitation (training which focuses on improving “sustained, selective, alternating, and divided attention”; Sohlberg, McLaughlin, Pavese, Heidrich, & Posner, 2000, pp. 658), is an option that should be explored to determine if it aids cognitive functioning in people with chronic pain. Dick and Rashid also point out that if cognitive functioning can be bolstered, the ability of patients to use other management techniques may be improved. Importantly, before APT and ABM programmes are used in management of chronic pain and associated deficits, we need to understand if, and how, attentional bias impacts cognitive functioning. Support for a deficit-view of attention in chronic pain would suggest that APT is a useful treatment model to explore, whereas support for the motivated attention hypothesis would suggest ABM.

Chapter 2

To directly test whether motivated attention contributes to cognitive deficits in chronic pain experience, it is necessary to compare attention to pain-related and neutral stimuli. Specifically, the motivated attention hypothesis predicts that people experiencing chronic pain should show enhanced attention to pain-related information, and diminished attention to neutral information. A necessary step in testing the motivated attention hypothesis is to assess if there is a bias to pain-related stimuli in temporal attention in people without chronic pain, by observing temporal attention for stimuli that are pain-related or neutral. Then this bias in people without chronic pain can be compared to the bias in people with chronic pain. There are four experiments in this chapter: Experiments 1, 2, and 4 assess attentional bias to pain-related words in participants without chronic pain, Experiment 3 tests the deficit-view and the motivated attention hypothesis by comparing performance for pain-related words compared to neutral words in people with chronic pain, to those without, chronic pain.

Temporal attention can be examined by using tasks that employ rapid-serial-visual-presentation (RSVP). In the most basic version of a task using RSVP, 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; and reporting accuracy of T2 is low. The resource capacity limit explanation of this attentional blink (Asplund et al., 2014; Chun & Potter, 1995; Dux & Marois, 2009; Marois & Ivanoff, 2005, McHugo et al., 2013) is that when attention is consumed with processing of T1, a bottleneck is created that prevents processing of T2. That is, there is not enough capacity left over to process T2. The more attention we pay to the first word, the greater the magnitude of the attentional blink is (reflected in lower accuracy is for reporting T2).

In RSVP, the targets can be neutral stimuli like letters and numbers, allowing temporal attention to neutral items to be assessed. A clinical study using the RSVP paradigm suggests that in chronic pain experience, temporal attention for neutral stimuli is impaired. Harker, Klein, Dick, Verrier, and Rashiq (2011) recruited participants with fibromyalgia (a chronic pain condition) and controls. All participants completed a target-reporting task with

letter targets and pattern masks. The duration of the targets and masks were varied to manipulate difficulty in detecting T1. Participants with chronic pain detected fewer T2s compared to controls when T1 detection was difficult. This group difference indicates that when attentional capacity is limited (in this case by a combination of chronic pain and task difficulty), temporal attention is impaired (Harker et al., 2011).

However, to test for attentional bias an emotional variant of RSVP is needed. The letters and numbers can be replaced with words or pictures that have emotional value. Emotional RSVP allows assessment of two different types of attention to emotion, depending on whether the emotional nature of T1 or T2 is manipulated. In one version of the task, T2 is always a neutral stimulus, but T1 can be either emotional or neutral. This version allows comparison of how processing capacity is consumed by emotional versus neutral stimuli. A deeper attentional blink (lower accuracy when the time between T1 and T2 is short) for emotional compared to neutral T1s reflects lesser processing of the T2 stimuli following an emotional T1. Emotional stimuli at T1 such as images, facial expressions, and words induce a deeper attentional blink than neutral stimuli (Arnell, Killman, & Fijavz, 2007; McHugo et al., 2013), indicating prolonged processing of the emotional stimulus. If pain-related stimuli undergo prolonged processing at T1, they will induce a deeper attentional blink than neutral stimuli. 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 attentional blink is a measure of attentional capture of T2. A shallower attentional blink for emotional compared to neutral T2s reflects greater capture of processing capacity by the emotional T2 stimuli. Emotional stimuli at T2 are more likely to overcome the attentional blink than neutral stimuli (Anderson, 2005). If pain-related stimuli attract attention, they will ‘overcome’ the attentional blink more easily, resulting in a shallower attentional blink than for neutral stimuli.

In other literatures, it has been demonstrated that participants’ experiences can modulate RSVP performance for both emotional T1 and T2 stimuli. Relative to controls, participants with clinical conditions e.g., schizophrenia (Strauss, Catalano, Llerena, & Gold, 2013) and alexithymia (Grynberg, Vermeulen, & Luminet, 2014) show changes in processing of T1 and T2 for emotional as compared to non-emotional stimuli. Negative words at T1 induced a deeper attentional blink, and negative words at T2 overcame the attentional blink more than neutral words, in patients with low negative symptoms of schizophrenia as compared to controls (Strauss et al., 2013). The attentional blink induced by fearful and angry facial expressions at T1 was deeper the higher participants scored on measures of alexithymia

(Grynberg et al., 2014). Together these studies demonstrate that temporal attention is affected by people's experience with clinical disorders, such that the pattern of attentional bias in the attentional blink is affected by disorder-relevance of the stimuli. Furthermore, MacLean, Arnell, and Busseri (2010) measured trait positive and negative affect and reported that there were relationships between these individual differences and the magnitude of the attentional blink for neutral stimuli, such that the greater positive affect, the shallower the blink, and the greater the negative affect, the deeper the blink. Thus, temporal attention is also affected by individual differences in non-clinical characteristics.

Notably, the motivational relevance of stimuli seems to be important in modulation of temporal attention. Smokers (Waters, Heishman, Lerman, & Pickworth, 2007) and heavy social drinkers (Tibboel, De Houwer, & Field, 2010) showed shallower attentional blinks for smoking and alcohol related stimuli, respectively, when they were presented at T2. The sensitivity of the attentional blink to emotional stimuli and to individual differences suggests that induction of an attentional blink with RSVP is a promising avenue for investigating attentional bias in temporal attention for pain-related information. However, at present there is limited evidence of whether sequential information that is pain-related is processed differently than neutral information.

Two studies are particularly relevant here. First, Schwabe and Wolf (2010) used a cold-pressor test (which is known to be painful, Graven-Nielsen, Sergerdahl, Svensson, & Arendt-Nielsen, 2001) to induce stress before participants completed a RSVP task with both aversive and neutral T1 and T2 words. Processing of an aversive T1 (versus neutral T1) resulted in lower accuracy (a deeper attentional blink) for neutral T2, and processing of an aversive T2 (versus neutral T2) resulted in higher accuracy (shallower attentional blink), but only following a neutral T1. Thus, aversive stimuli both induced a deeper attentional blink and more effectively overcame the attentional blink induced by a neutral stimulus. This modulation of temporal attention is presumably due to increased attention directed to processing the potential threat and attention capture by potential threat, respectively. Cold-pressor also slightly increased T2 detection generally, perhaps suggesting that recent pain or stress increases vigilance.

Second, Zheng, Wang, and Luo (2015) manipulated the nature of T1; they compared the size of the attentional blink induced by images of facial expressions depicting pain experience (including images of people with their hands placed on their forehead) and neutral images. Zheng and colleagues observed a deeper attentional blink following painful than neutral stimuli. Furthermore, the modulation of the attentional blink was related to individual

differences in pain catastrophisation. Greater pain catastrophising was associated with poorer accuracy on the RSVP task, suggesting that the more participants viewed pain as a signal of danger, the more attentional capacity was devoted to processing pain-relevant stimuli.

The findings of both Schwabe and Wolf (2010) and Zheng and colleagues (2015) suggest that there is a bias towards aversive stimuli in temporal attention. In Schwabe and Wolf the stimuli were general aversive words not related to pain or body-tissue threat, and Zheng and colleagues used stimuli which depicted people in pain. In the following experiments, reporting of T1 and T2 is compared for pain-related and neutral words.

Undergraduate psychology students, not currently or previously receiving treatment for depression or anxiety, were recruited for Experiments 1, 2, and 4. By excluding participants with these experiences, the scope of individual differences in motivation is reduced, allowing for better assessment of any differences in the attentional blink due to the stimuli themselves in a population without chronic pain. In Experiment 3, performance by people with, and without, chronic pain was compared to test for evidence of deficits in temporal attention and motivated attention in temporal attention.

In Experiment 1, the nature of the first target was manipulated: T2 was always neutral but T1 was either aversive pain-related (as opposed to positive pain-related words, like healing or relief⁴), or neutral. Thus, Experiment 1 assesses the relative induction of the attentional blink by pain-related and neutral stimuli. Any difference in the magnitude of the attentional blink for pain-related or neutral stimuli reflects prolonged engagement with the material at T1. If, as part of the threat protection system, there is a bias in temporal attention to pain-related information, then a deeper attentional blink will be observed following presentation of pain-related T1 than neutral T1 stimuli. In Experiments 2 – 4, the nature of the second target was manipulated: T1 was always neutral but T2 was either pain-related, or neutral. Thus, Experiment 2, 3, and 4 assess the relative overcoming of the attentional blink by pain-related and neutral stimuli. Any difference in the magnitude of the attentional blink for pain-related or neutral stimuli reflects capture by the material with a shallower blink at T2. If, as part of the threat protection system, there is a bias in temporal attention to pain-related information, then a shallower attentional blink will be observed with presentation of pain-related T2 than neutral T2 stimuli in Experiments 2 – 4.

⁴ In all the experiments in Chapter 2, pain-related words are aversive (e.g., *cruel*) and not pleasant (e.g., *heal*).

Expectation is one factor that can modulate activity in the threat protection system (Moseley, 2007). Indeed, in their meta-analysis of attentional bias to pain-related information, Crombez and colleagues (2013a) observed that bias to pain-related stimuli was greater in people with chronic pain when presentation of stimuli was blocked by stimulus type rather than intermixed. Therefore, to manipulate attentional expectation and arousal in the threat protection system, two modes of presentation were compared in Experiments 1, 2, and 4: blocked and mixed. In blocked presentation, whether the manipulated target was pain-related or neutral varied by block; participants completed two blocks of trials with pain-related targets and two sets with neutral targets. Using blocked presentation thus allows assessment of the modulating effect of raising the participants' general arousal level (pain-related blocks) and expectations on induction (when manipulating T1) and overcoming (when manipulating T2) of the attentional blink. In mixed presentation, pain-related and neutral targets varied randomly; participants did not know trial-to-trial which type of manipulated target would be present. In daily life, you do not always get prior warning of threat. Using mixed presentation thus allows assessment of the baseline attention to pain-related and neutral stimuli without block level expectation. If blocking the presentation engages the threat protection system and activates an attention set for pain-related information, then modulation of the attentional blink by pain-related stimuli will be greater under blocked than mixed presentation.

Together the experiments in this chapter allow for: 1) assessment of an attentional bias to pain-related information in people without chronic pain; and 2) for comparison between attentional biases in people with, and without, chronic pain, to test the deficit-view and the motivated attentional hypothesis of cognition in chronic pain experience.

Experiment 1

In Experiment 1, with undergraduate participants, the nature of T1 was manipulated to determine whether pain-related words engage more cognitive capacity and therefore produce a deeper attentional blink. To assess if expectation, and therefore activation of the threat protection system, plays a role in bias, the presentation of the target words was either blocked or mixed. In blocked presentation, the first target was either pain-related or neutral, presented in alternating blocks. In mixed presentation, the first target was pain-related or neutral, varied from trial to trial. In both conditions, the second target was always neutral. If a relatively deeper attentional blink is observed for pain-related words than neutral words in

blocked as compared to mixed presentation, this would be support for activation of a threat-protection system biasing attention in people without chronic pain experience.

Method

Participants

Forty-four first year Victoria University of Wellington psychology students, participating in an introduction to psychology research programme, were recruited. One participant did not complete the RSVP task and six participants' data was removed from the analyses for having low accuracy on the RSVP task (see the data processing section). The final sample was comprised of 37 participants; 33 women and four men (*mean age* = 18.57 years, *SD* = 1.42 years); all naïve to the purpose of the experiment. Eighteen participants completed the blocked presentation version of the experiment and 19 the mixed presentation version. All participants spoke English as a first language, had normal or corrected-to-normal eyesight, and were comfortable using a computer keyboard and mouse. All participants had not previously and were not currently receiving treatment for depression or anxiety. These participant characteristics also apply to the participants in Experiments 2 and 4 in this chapter. Experiments 1, 2, and 4 in this chapter were approved by the School of Psychology ethics committee, under the authority of the Human Ethics Committee at Victoria University of Wellington. All participants gave informed consent and were debriefed at the end of the session as to the purpose of the study.

Stimuli & Apparatus

The stimuli were presented using a Dell PC running Psychology Software Tools' E-Prime Suite version 2.0 (Psychology Software Tools, Pittsburgh, PA). Visual stimuli appeared on a 527 mm x 297 mm Alienware screen with a vertical refresh rate of 60 Hz, at an approximate viewing distance of 60 cm. No chin rest was used to achieve consistency across experiments: no chin rest was used in Experiment 3 in order to minimise the potential discomfort experienced by the participants with chronic pain.

Procedure

Participants completed the experiment individually or in groups of two to four in separate booths. Participants also completed the Attention Network Test (ANT) and questionnaire measures to guide development of Experiment 3 but these scores are not reported for Experiments 1, 2, or 4. In a group without pain there is too little variation in

ANT performance and questionnaire scores to observe clear relationships between the experimental tasks and measures of individual differences. The RSVP task was always completed first (and took approximately 30 – 40 minutes), followed in fixed order by the ANT task (approximately 10 minutes), and the post session questionnaires⁵ (approximately five minutes).

RSVP.

See Figure 1 for a pictorial representation of a trial. Each trial began with a 3 mm x 3 mm fixation cross for 1500 ms. A stream consisted of 20 items, presented for 110 ms each, with no interval between items. All items were presented in the centre of the screen in Arial, bold, size 36-point font on a grey background. The words were approximately 1 cm high by maximum 4.5 cm subtending from fixation (9 cm maximum length). Most words were within 3 cm subtending from fixation (6 cm maximum length). The letters were approximately 5 mm wide (not including spacing). Each stream contained two green target words embedded within 18 filler words in black text. At the end of the stream participants were prompted to enter the words in green ink they saw, in order, in two response boxes (one word in each box) – they were informed that there were always two green words present, but they could enter fewer if they did not see or remember them. There was no limit on response time, though participants were instructed not to think about their response for too long. The typed responses to T1 and T2 were recorded. Participants saw an example stream and completed 12 practice trials with pain-related T1 / neutral T2 targets (pairs not used in the experimental trials) before starting the four experimental trial blocks. Participants could take a self-timed break after each block.

⁵ Participants filled out a set of questionnaires via Qualtrics survey software. Participants completed, in fixed order, the McGill Pain Questionnaire – Short Form (Melzack, 1987), 21 item - DASS (Depression, Anxiety, Stress Scale: Lovibond & Lovibond, 1995a & 1995b), and the ACS (Attentional Control Scale: Derryberry & Reed, 2002). These questionnaires were administered as exploratory measures to test their use for research with participants experiencing chronic pain and the data are not reported in Experiments 1, 2, and 4. They will be described in Experiment 3.

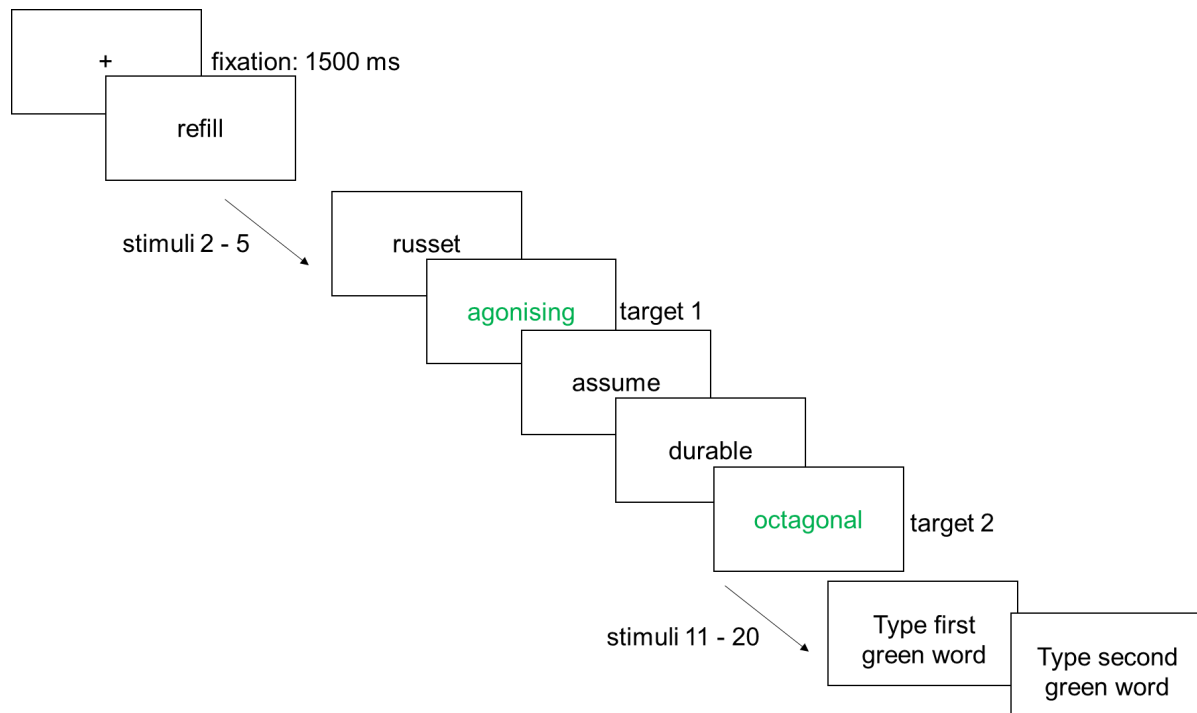


Figure 1. Trial diagram for the RSVP stream.

Each stimulus is presented for 110 ms with no inter-stimulus-interval. In this example, the TOA is 330 ms.

The nature of T1 was manipulated; T2 was always neutral. One-hundred and ninety-two target pairs (96 pain-related / neutral; 96 neutral / neutral) were collated. See Appendix A for the full list of target pairs. For the pain-related – neutral pairs, 96 pain-related target words were selected from previous attentional bias papers using emotional Stroop or dot-probe methods (Asmundson, et al., 2005; Dehghani et al., 2003; Kaur, Butow & Sharpe, 2013; Keogh, Ellery, Hunt, & Hannent, 2001; Sharpe et al., 2012) and included words defined by previous researchers as being associated with sensory pain (e.g., *burning*), affective pain (e.g., *despair*), health threat (e.g., *collapse*), disability (e.g., *helpless*), and general threat (e.g., *harmful*) themes. Neutral words were generated to match the pain-related words on frequency (no greater than 50 words per million difference) and length (no greater than one letter difference), using the MRC Psycholinguistic database (Wilson, 1988). For the neutral / neutral pairs, 96 neutral words (that were taken from those paired with the pain-related words in the previous attentional bias papers) were used as neutral target words, and were paired with neutral target words generated to match them on frequency and length. See Table 1 for descriptives. For half of the participants, the order of the neutral T1 / neutral T2 pairs was reversed.

Paired-samples *t*-tests with a Bonferroni corrected alpha level of .0125 for the matched target word pairs (pain-related – neutral, and neutral – neutral, see Tables 1 and 2) demonstrate: 1) that independent raters evaluated the pain-related words as significantly more negative in valence and higher in arousal than their matched neutral words; 2) that the pain-related words and matched neutral words do not differ significantly on length; 3) that the pain-related words are higher in word frequency than their matched neutral words; and 4) that the generated neutral words do not differ significantly from their matched neutral words on valence, arousal, length or frequency of use. Although the pain-related target words are higher on frequency than their matched neutral targets, frequency of use has been shown not to be a factor in the attentional blink (Anderson, 2005).

Table 1. *Frequency and length measures and valence and arousal ratings of the two target pair types.*

Type (96 words of each)	Valence	Arousal	Frequency	Length
<i>M (SD)</i>				
Pain-related – neutral match	2.51 (0.98)	6.02 (1.40)	15.35 (18.09)	7.75 (1.92)
neutral – neutral match	5.17 (0.91)	3.05 (1.15)	9.59 (13.77)	7.66 (1.91)
neutral – neutral match	5.31 (0.77)	2.79 (1.06)	15.02 (18.41)	7.74 (1.92)
neutral match	5.19 (0.95)	3.10 (1.36)	11.40 (14.74)	7.52 (2.10)

Note. the valence and arousal ratings were collected from a separate ratings experiment.

Table 2. *Inferential statistics for comparison of the targets within the two target pair types.*

	Pain-related – neutral match pairs				Neutral – neutral match pairs			
	Valence	Arousal	Frequency	Length	Valence	Arousal	Frequency	Length
<i>t</i>	19.966	15.463	2.584	0.366	0.953	1.653	1.691	0.744
<i>p</i>	< .001	< .001	.011	.738	.343	.102	.094	.459
<i>d_z</i>	2.039	1.583	0.264	0.033	0.102	0.169	0.172	0.076

Note. df is always 95.

A set of 864 filler items was collated as well, populated using the MRC psycholinguistic database (Wilson, 1988). The items were checked for emotional words, and any found were removed. A trial list for the filler words was made by randomly sorting the words and assigning them to a filler position (one of 18) and trial (one of 48). The same filler word list was used for each block of 48 trials. The full RSVP stream was generated on each trial in E-prime by randomly pairing a target pair with a filler item set.

For counterbalancing purposes the target pairs were split across four lists of 48 trials, each list containing 12 trials at four Target Onset Asynchronies (TOA: 110, 330, 550, and 770 ms). TOA 110 ms corresponds to no intervening fillers between the two targets (Lag 1), 330 ms to two intervening fillers (Lag 3), 550 ms to four intervening fillers (Lag 5), and 770 ms to six intervening fillers (Lag 7). For every TOA, Target 1 appeared twice at each of six positions (6 – 11) within the stream to make it unpredictable where in the stream the targets would appear. T1 and T2 were always presented somewhere between positions six and 18 in the stream.

There were two versions of presentation format, manipulated between-subjects. For the blocked version, the order of the trial sets was counterbalanced in an ABBA fashion. For two of the four blocks, 48 pain-related – neutral target pairs were presented, and for the other two blocks, 48 neutral – neutral pairs were presented. Half of the participants started with a pain-related – neutral block, and half with a neutral – neutral block. For the mixed version, there were still four trial blocks, to match the experiment protocol of the blocked version, but the first half of the experiment (96 trials) presented trials randomly from one of the pain-related / neutral trials sets and one of the neutral / neutral trial sets, and for the second half of the experiment (96 trials) from the other sets. The order of trial set presentation was counterbalanced between participants, and trials within each set were randomly presented.

Data processing

When sphericity was violated, the original degrees of freedom are reported, with the corrected p and ϵ (epsilon; Greenhouse-Geisser). For follow-up paired samples t -tests, d_z for dependent t -tests were calculated (Lakens, 2013). d_z is the mean difference divided by the standard deviation of the mean difference.

The raw responses for T1 and T2 were processed through a macro that calculates the percentage match between the typed response and the target word. If the percentage match was less than 50%, then the trial was recorded as incorrect. If the percentage match was greater or equal to 80% then the trial was recorded as correct. The trials with percentage matches of 50% – 79% were checked manually. If the core of the response matched the target, or if only tenses or number were inconsistent, then those trials were recoded as correct. The trials were sorted so batches of targets were processed at the same time for consistency. Responses to the first and second target were processed separately. T1 and conditional T2 proportion accuracy were calculated for each type by TOA cell for each participant. Conditional T2 (CT2) accuracy was used as a dependent measure because on trials where T1 was not reported correctly it cannot be inferred that T1 was processed. The attentional blink is a phenomenon that occurs following processing of T1, thus it is important to know that T1 was processed when interpreting T2 results.

Six participants were excluded as they had fewer than 50% correct trials at the 770 lag in the pain-related or neutral cell for T1 report accuracy. Less than 50% accuracy suggests that participants cannot reliably see the target words, even with a long interval between T1 and T2.

Results & Discussion

Separate 2 (type: pain-related, neutral) x 4 (TOA: 110, 330, 550, 770) x 2 (presentation: blocked v. mixed) mixed model ANOVAs were run for T1 accuracy and Conditional T2 accuracy, with type and TOA as within-subjects factors, and presentation as a between-subjects factor. See Table 8 for the RSVP task data by TOA and type.

RSVP T1 Accuracy

There was a main effect of type, such that reporting of pain-related T1s ($M = 74\%$, $SD = 13\%$) was more accurate than reporting of neutral T1s ($M = 65\%$, $SD = 16\%$), $F(1, 35) = 46.640$, $p < .001$, $\eta_p^2 = .571$ (Figure 2, left panel). Given that pain-related words were rated as higher in arousal and lower in valence than their matched neutral targets, this

relative better reporting of pain-related T1s is likely due to an arousal- or valence-related attention or memory boosting effect. This effect does not interact with TOA, and alone is not informative for inferring a modulation of temporal attention. There was no main effect of TOA, of presentation, nor any interactions between factors in the T1 data.

RSVP Conditional T2 Accuracy

There was a main effect of TOA (reflecting the attentional blink and subsequent recovery), $F(3, 105) = 202.348, p < .001, \varepsilon = .733, \eta_p^2 = .853$, qualified by a marginal interaction between type and TOA, $F(3, 105) = 2.818, p = .059, \varepsilon = .757, \eta_p^2 = .075$ (Figure 2, right panel). This interaction reflects enhanced reporting of T2s (which were always neutral) when paired with pain-related T1s ($M = 14\%, SD = 14\%$), relative to neutral T1s ($M = 10\%, SD = 12\%$) at the 110 ms TOA, $t(36) = 2.129, p = .040, d_z = 0.26$, and enhancement of reporting of T2s when paired with neutral T1s, ($M = 62\%, SD = 20\%$) relative to pain-related T1s, ($M = 57\%, SD = 19\%$) at the 770 ms TOA, $t(36) = 2.201, p = .034, d_z = 0.27$. This interaction is contrary to the hypothesis that a pain-related T1 will generally capture attention and produce a deeper attentional blink. Rather, the influence of TOA on the patterns of reporting neutral words following pain-related, as compared to neutral words, suggests a more complicated picture. When there is a short time between T1 and T2, perhaps the enhanced processing of pain-related words at T1 (reflected by the T1 accuracy data) extends into to the presentation of T2, thus greater reporting of T2 neutral words following pain-related words at 110 ms is observed. When there is a longer time between T1 and T2, the enhanced processing of pain-related words at T1 may lead to interference in processing relative to neutral T1s, as T2 occurs at a distant time from T1. There was no main effect of type or presentation, nor any other significant interactions between factors in the CT2 data.

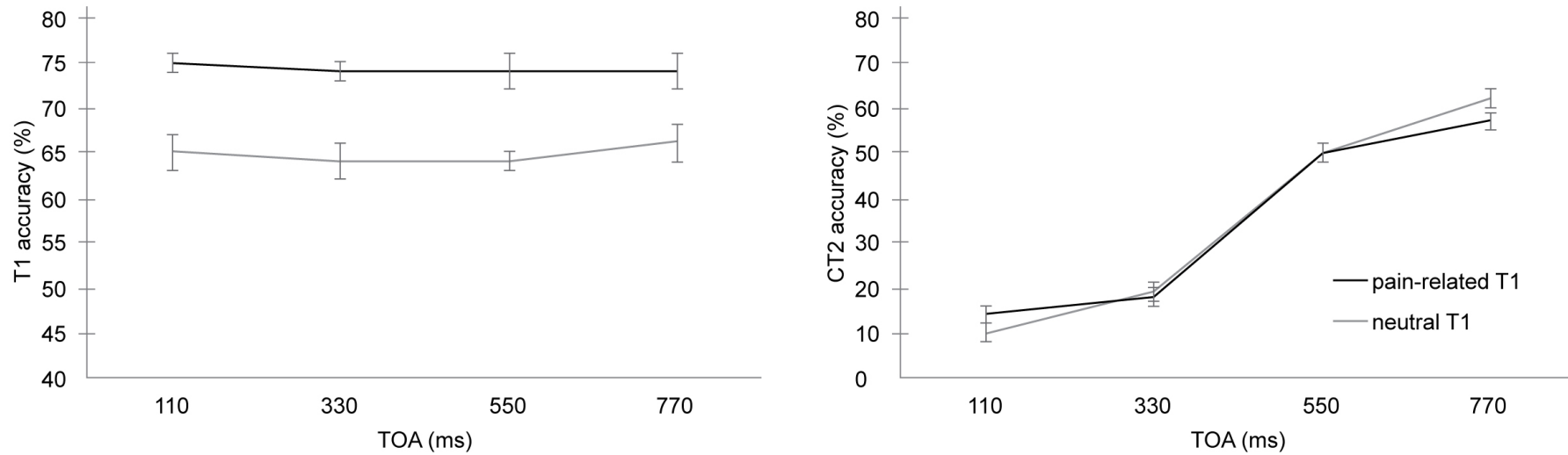


Figure 2. T1 (left panel) and conditional T2 (CT2, right panel) accuracy as a function of TOA and word type for Manipulating T1 in Experiment 1. Error bars are within-subjects standard errors with Morey's (2008) correction.

In Experiment 1, the nature of T1 was manipulated. Participants were better able to report pain-related than neutral T1s, indicating that the word types are processed differently. This additional processing of pain-related T1s modulated the early period of the attentional blink such that neutral words following pain-related words were more likely to be reported than neutral words following neutral words. In contrast, after recovery from the attentional blink, participants were less able to report neutral words that followed pain-related words, than neutral words following neutral words. These findings are contrary to the hypothesis that pain-related T1 may produce a deeper blink in people not experiencing chronic pain, rather the attentional bias depended on the time between T1 and T2. There was also no effect of, or interaction with, presentation, suggesting that induction of the attentional blink by pain-related information is not affected by engagement of the threat-protection system.

Experiment 2

In Experiment 2, undergraduate participants were recruited and the nature of T2 was manipulated (T1 is neutral) to determine whether pain-related words capture more cognitive capacity and therefore overcome the attentional blink more than neutral words. As in Experiment 1, to assess if expectation (and therefore activation of the threat protection system) plays a role in bias, the presentation of the target words was either blocked (pain-related and neutral words appear in different blocks as T2s), or mixed (pain-related or neutral words are selected randomly to be T2s). If the bias towards pain-related words (a shallower attentional blink is observed for pain-related words than neutral words) is greater in blocked as compared to mixed presentation, then this would be support for activation of a threat-protection system biasing attention in people without chronic pain experience.

Method

Participants

Sixty-six first year Victoria University of Wellington psychology students, participating in an introduction to psychology research programme, were recruited. Participants had not participated in Experiment 1. One participant did not complete the RSVP task and 15 participants were removed from the analyses for having low accuracy on the RSVP task. The final sample was made up of 40 participants; 29 women and 11 men (*mean age* = 18.43 years, *SD* = 1.43 years). Twenty-one participants completed the blocked presentation version of the experiment, and 19 the mixed presentation version.

Stimuli, Apparatus, Procedure & Data Processing

Experiment details were identical to those in Experiment 1, with the exception that the nature of T2 was manipulated; T1 was always neutral. The pain-related T1 / neutral T2 pairings used in Experiment 1 were reversed.

Results & Discussion

Separate 2 (Type: pain-related, neutral) x 4 (TOA: 110, 330, 550, 770) x 2 (Presentation: blocked v. mixed) mixed model ANOVAs were run for T1 accuracy and Conditional T2 accuracy, with Type and TOA as within-subjects factors and Presentation as a between-subjects factor. See Table 8 for the RSVP data by TOA and type.

RSVP T1 Accuracy

There was a main effect of type, such that reporting of the neutral T1s was better when they were followed by a neutral T2 ($M = 64\%$, $SD = 14\%$) than by a pain-related T2 ($M = 58\%$, $SD = 17\%$), $F(1, 38) = 24.117$, $p < .001$, $\eta_p^2 = .388$ (Figure 3, left panel).

RSVP Conditional T2 Accuracy

There were main effects of TOA (reflecting the attentional blink and subsequent recovery), $F(3, 114) = 199.061$, $p < .001$, $\eta_p^2 = .840$, and type, demonstrating enhancement for reporting of pain-related T2s ($M = 34\%$, $SD = 14\%$) relative to neutral T2s ($M = 31\%$, $SD = 14\%$), $F(1, 38) = 6.682$, $p = .014$, $\eta_p^2 = .150$, qualified by a TOA x type x presentation interaction, $F(3, 114) = 4.238$, $p = .007$, $\eta_p^2 = .100$. See Figure 3, right panel, and Figure 4.

To unpack the effect of presentation on the interaction between TOA and type, separate 2 (type: pain-related, neutral) x 4 (TOA: 110, 330, 550, 770) repeated measures ANOVAs were run for the two presentation types. For blocked presentation, there was a main effect of TOA, $F(3, 60) = 122.946$, $p < .001$, $\epsilon = .699$, $\eta_p^2 = .860$ (reflecting the attentional blink and subsequent recovery), a marginal effect of type, $F(1, 20) = 4.093$, $p = .057$, $\eta_p^2 = .170$ (relatively better accuracy for pain-related T2s than for neutral T2s), and a marginal interaction between TOA and type, $F(3, 60) = 2.372$, $p = .079$, $\eta_p^2 = .106$. The marginal TOA by type interaction was driven by enhancement of reporting pain-related T2s ($M = 63\%$, $SD = 17\%$) relative to neutral T2s ($M = 53\%$, $SD = 18\%$) at the 770 ms TOA with

blocked presentation, $t(20) = 3.679$, $p = .001$, $d_z = 0.56$. As the relative enhancement for pain-related words (the attentional bias) is at the longer TOA, this could reflect the impact of a more controlled expectation strategy to be alert for pain-related information (a pain-related attentional set engaged by the threat protection system), driven by the blocking of T2 type. Also, given that the difference is at the longest TOA, it cannot reflect pain-related words overcoming the attentional blink earlier than neutral words.

For mixed presentation, there was a main effect of TOA, $F(3, 54) = 80.865$, $p < .001$, $\eta_p^2 = .818$, and a marginal interaction between TOA and type, $F(3, 54) = 2.535$, $p = .066$, $\eta_p^2 = .123$. The marginal interaction was driven by significant enhancement of reporting pain-related T2s relative to neutral T2 at the 110 TOA with mixed presentation: pain-related ($M = 19\%$, $SD = 17\%$), neutral ($M = 10\%$, $SD = 10\%$), $t(18) = 3.225$, $p = .005$, $d_z = 0.50$. This same effect was marginally significant at the 330 ms TOA: pain-related, ($M = 19\%$, $SD = 17\%$), neutral, ($M = 13\%$, $SD = 17\%$), $t(18) = 1.743$, $p = .098$, $d_z = 0.33$. As the relative enhancement for pain-related words (the attentional bias) is at the shorter TOAs (less than 500 ms), this likely reflects a classic attentional blink effect. As predicted, pain-related stimuli overcome the limited processing period following T1 more effectively than neutral stimuli.

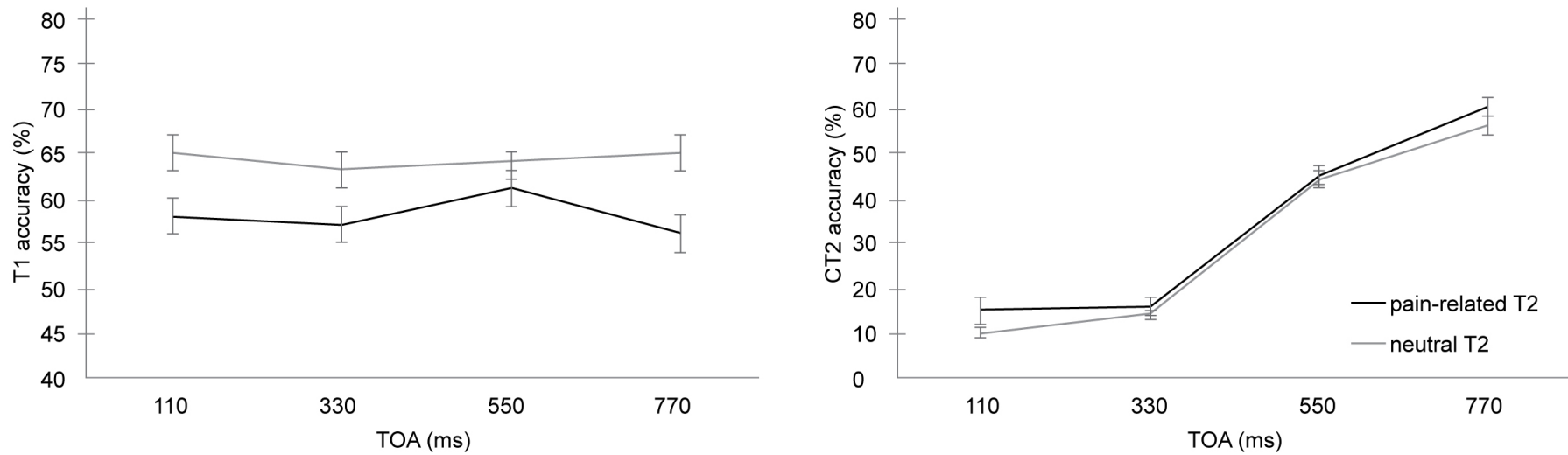


Figure 3. T1 (left panel) and conditional T2 (CT2, right panel) accuracy as a function of TOA and word type for Manipulating T2 in Experiment 2. Error bars are within-subjects standard errors with Morey's (2008) correction.

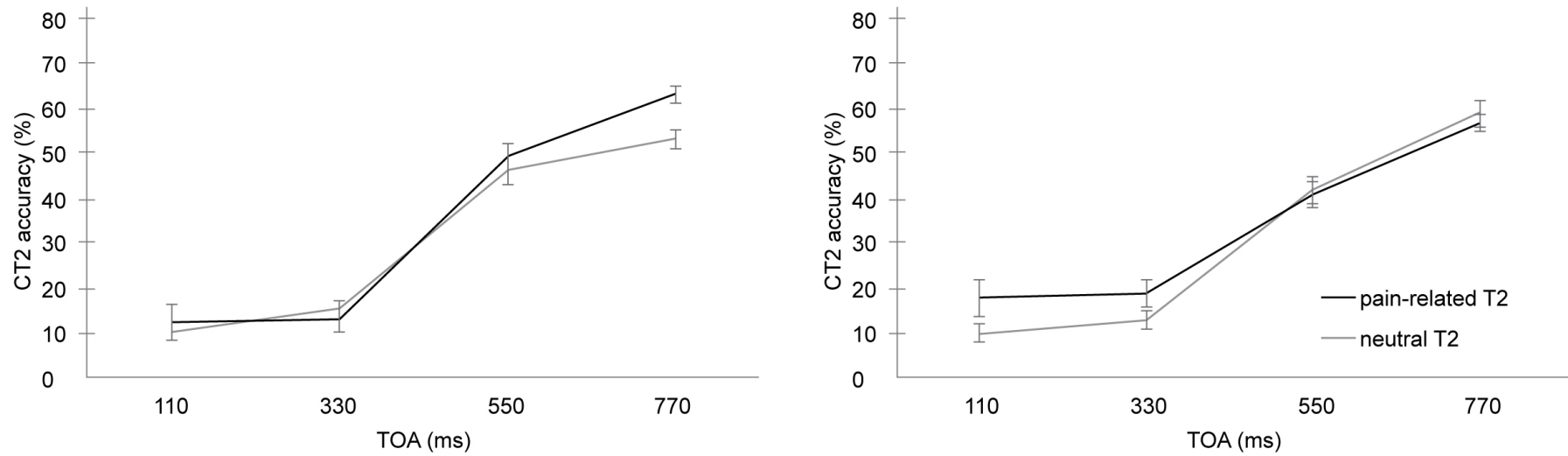


Figure 4. Conditional T2 accuracy (CT2) as a function of TOA and word type for Manipulating T2 blocked (left panel) and mixed (right panel) presentation in Experiment 2. Error bars are within-subjects standard errors with Morey's (2008) correction.

In Experiment 2 the nature of T2 was manipulated and there were differences in both reporting of, and in temporal attention for, pain-related as compared to neutral words. Participants were better able to report pain-related than neutral T2s. This enhanced processing of pain-related T2s affected processing of T1s. Participants were less accurate at reporting neutral T1s when a pain-related T2 followed, compared to when a neutral T2 followed. This effect in T1 performance could be due to the pain-related T2s diverting attention or memory away from processing of the neutral T1s. This effect is in T1 data, and does not interact with TOA, and alone is not informative for interpreting any attentional blink effect.

In the T2 data, the additional processing of pain-related T2s was dependent on presentation of the stimuli types, and mixed and blocked presentation modulated processing differently so that the impact of presentation on processing of pain-related and neutral words was observed at different TOAs. Under blocked presentation, the threat protection system seemed to be engaged by expectation and at long TOAs participants were better able to report pain-related than neutral T2s. However, this enhancement was not observed in the attentional blink period so is not an effect of pain-related stimuli on temporal attention, but rather a later bias in memory or reporting. In contrast, under mixed presentation, participants could not set an expectation for a trial set, and the predicted bias in temporal attention was observed such that the attentional blink was shallower for pain-related than neutral words.

Focusing on modulation of processing during the attentional blink period, Experiments 1 and 2 together show that the pain-related and neutral target stimuli selected are differently processed. Most importantly, there is evidence of differential processing of pain-related information during the attentional blink in Experiment 1 when the nature of T1 is varied at the 110ms TOA *and* in Experiment 2 when the nature of T2 is varied at the short TOAs with mixed presentation. In Experiment 1, at the 110 ms TOA, pain-related words at T1 induced a shallower attentional blink than neutral words at T1. In Experiment 2, there was a bias to pain-related information at the short TOAs, such that under mixed presentation pain-related words overcame the attentional blink more than neutral words in the attentional blink.

Experiment 3

Experiments 1 and 2 established that there is differential processing of pain-related information in people without chronic pain, as indicated by both a shallower attentional blink following pain-related T1 than neutral T1 in Experiment 1 at the 110 ms TOA, and a shallower attentional blink for pain-related T2 than neutral T2 at the short TOAs with mixed

presentation (randomly intermixing pain-related and neutral targets) in Experiment 2. This means that RSVP with a target-reporting task provides a potentially useful means of testing the deficit-view and motivated attention hypothesis in chronic pain experience. In Experiment 3, RSVP was used to compare patterns of attention to pain-related and neutral words in a community sample experiencing chronic-pain and a community sample not experiencing chronic-pain. Although stimuli type affected the magnitude of the attentional blink when it appeared at both T1 and T2, in Experiment 2 there was a larger attentional blink effect when manipulating T2 with mixed presentation, than when manipulating T1 in Experiment 1 ($\eta_p^2 = .123$ compared to $\eta_p^2 = .075$). Thus, to maximize observation of an enhanced attentional bias between people with, and without, chronic pain, in Experiment 3 the nature of T2 was manipulated, while keeping T1 neutral. Manipulating T2 allows for assessment of the overcoming of the attentional blink by pain-related as compared to neutral words in participants with, and without, chronic pain. Mixed presentation is also more ecologically valid and a closer representation of day to day processing of environmental information, as participants do not know trial by trial if pain-related words are going to be presented or not.

Additional behavioural and self-report measures were also included. After the RSVP, participants completed the Attention Network Test (ANT, Fan et al., 2002) and questionnaire measures of self-reported pain, depression, anxiety, stress, attentional control, and vigilance to changes in pain experience. The ANT was used to provide a measure of attention to neutral information in a task that has no emotional component, and allows calculation of indices of the effectiveness of three attention networks: the alerting network, which underlies vigilance to cues in the environment; the orienting network, which underlies voluntary shifts in spatial attention in response to cues; and the executive network, which underlies the ability to inhibit distracting information, or to select relevant information or responses (Fan et al, 2002).

Regardless of whether chronic pain experience is associated with deficits, enhancements, or no difference in ANT performance compared to controls, the ANT task is useful for teasing apart how chronic pain impacts attention by examining relationships between the efficiency of the networks and the magnitude of the attentional blink for pain-related and neutral stimuli. How chronic pain impacts these attention networks is not yet clear. As there is no pain-related information in the ANT task, according to the deficit-view a decrement in performance for the participants with chronic pain should be observed, and there will be a deficit on alerting, orienting, and executive efficiency for participants with

chronic pain compared to controls. Indeed, there are two papers that assess functioning of the attention networks in chronic pain experience. Miró, Lupiáñez, Hita, Martínez, Sánchez, and Buela-Casal (2011) and Miró, Martínez, Sánchez, Prados and Lupiáñez (2015) assessed the three attentional networks in participants with fibromyalgia and controls. Miró and colleagues (2011) observed that in fibromyalgia responding was slower overall, there was poorer control in the executive network, and higher efficiency in the alerting network compared to participants without fibromyalgia. Miro and colleagues (2015) observed similar patterns to the 2011 work in general response speed and greater interference in the executive network, however, the difference in alerting between participants with fibromyalgia and controls was not replicated.

If there is a difference in the magnitude of the attentional blink for pain-related and neutral words between participants experiencing chronic pain and controls, then correlational analyses between the attentional blink indices and ANT indices will be used to test the prediction of motivated attention that deficits in performance should be related to attentional bias. After the ANT, participants then completed a set of questionnaires to assess current pain, current depression, anxiety or stress symptoms, self-reported attentional control, and vigilance to pain. Exploratory correlational analyses between behavioural performance and questionnaire measures were performed to look for other clues as to mechanisms by which attentional biases may modulate cognitive performance.

In chronic-pain experience, a deficit-view predicts that attentional capacity is taken up with the pain experience and this translates to poorer performance on cognitive tasks. In Experiment 3, the deficit-view would be supported by a deeper attentional blink for neutral and pain-related stimuli, and poorer attention network scores in participants with chronic pain compared to participants without chronic pain.

The motivated attention hypothesis predicts that in chronic pain experience, assignment of attentional capacity is motivated towards pain-related information, but away from unrelated information. This hypothesis translates to a prediction that an attentional blink will be observed in participants both with, and without, chronic pain, but compared to control participants, participants with chronic pain may show worse performance for neutral T2s but will show enhancements for pain-related T2s. This performance difference will result in a greater relative difference between the magnitude of the attentional blink for neutral and pain-related T2s in participants with chronic pain (a larger attentional bias). Furthermore, in participants with chronic pain the magnitude of the attentional bias in the attentional blink will be positively correlated with the magnitude of the cognitive deficits (the blink for neutral

stimuli and performance on the attention network task). An alternative outcome that would provide support for the deficit-view and the motivated attention hypothesis is that in participants with chronic pain, there will be both an overall larger attentional blink for pain-related and neutral information, and a larger attentional bias towards pain-related information.

Method

Participants

The study was approved by the New Zealand Health and Disability Ethics Committee. All participants gave informed consent, were debriefed at the end of the session as to the purpose of the study, and were offered a thank you voucher. Fifty participants with chronic pain and 45 controls were recruited; all naïve to the purpose of the experiment. As an indication of being able to report any targets in the RSVP task, participants were excluded from the analyses if they had less than 50% correct trials at the 770 lag in the pain or neutral cell for target 1 report. Once participants were removed from the data set for not meeting this criterion, there were 41 participants with chronic pain and 41 matched controls. It is important to note from a deficit perspective that more participants with chronic pain ($n = 9$) did not reach the RSVP task performance criteria than control participants ($n = 4$); though this difference in the proportion of participants not meeting the performance criteria was not significant, $z = 1.29$, $\alpha < .05$.

Practical constraints on recruitment meant that I could not recruit more participants. Forty-one participants in each group gives me the ability to detect a between-group effect size of $d = 0.55$ (one tailed-test with 80% power; calculated using G*Power 3: Faul, Erdfelder, Lang, & Buchner, 2007). A Cohen's d of 0.55 is within the higher range of effect sizes in the executive function domain (Berryman et al., 2014) and attentional bias literature (Schoth et al., 2012) comparing performance by participants with, and without, chronic pain.

Participants were recruited from the community via posters and social media posts. When participants contacted the researcher, a screening-survey was sent via Qualtrics that assessed recruitment criteria information (first language English, comfortable using a computer keyboard and mouse, normal or corrected-to-normal vision), chronic pain cause (if known), duration of chronic pain, any co-morbidities (current injury, current or previous treatment for depression and / or anxiety, neurological disorder), and medication use. At the time they completed the study, the participants recruited with chronic pain reported experiencing pain for at least three months. Control participants did not currently experience

chronic pain. All participants had English as a first-language, had corrected or corrected-to-normal vision, and were able to use a mouse and keyboard. Demographic information was collected at the start of the laboratory session. In order to have groups matched on age, sex, and education level, I aimed to recruit a control participant to match each participant with chronic pain on sex, age (± 10 years), and education level (\pm one level). Where matching on all three variables was not possible, I matched on two.

Neither controls nor participants with chronic pain were excluded from the sample based on previous or current treatment for depression or anxiety, nor for having co-morbid neurological disorders or chronic fatigue, nor for medication use. However, these characteristics were recorded. There is a high co-morbidity between chronic pain and depression and anxiety (Burri, Ogata, Vehof, & Williams, 2015; Dominick, Blyth, & Nicholas, 2012; Gureje et al., 2008; Shipton, Ponnampuruma, Wells, & Trewin, 2013). By not having a co-morbidity restriction, the sample size was not unduly limited (important in a small community like Wellington, New Zealand). Variability in the participants with chronic pain and controls on depression, anxiety, and stress scores were expected; meaning that any differences in performance overall will be difficult to attribute to one mechanism or another. Indeed, cognitive deficits are observed in depression (Austin, Mitchell, & Goodwin, 2001; Ravnkilde, Videbech, Clemmensen, Egander, Rasmussen, & Rosenberg, 2002). The focus of this thesis though is on testing the deficit-view as well as the validity of the motivated attention hypothesis. The motivated attention hypothesis is specific to predictions about comparisons between overcoming of the attentional blink by pain-related and neutral stimuli. Differences in reporting accuracy between stimulus types is unlikely to be influenced by deficits in performance due to co-morbid neurological disorders or chronic fatigue, nor by medication use. In contrast, depression, anxiety and stress levels are likely to contribute to attentional biases (Cisler & Koster, 2010; Crocker, Heller, Warren, O'Hare, Infantolino, & Miller, 2013). If there are group differences in temporal attention to pain-related and neutral words that correlate with these individual differences, then depression, anxiety, and stress will be used as covariates in ANCOVAs.

The participants with chronic pain were 29 women and 12 men with a mean age of 37 years (16 – 64 years; excluding one participant who did not provide their age; $SD = 14$ years). The control participants were 32 women and 9 men with a mean age of 33 years (19 – 57 years; $SD = 12$ years). The groups did not significantly differ on sex, $\chi^2(1) = .576$, $p = .448$, $\Phi = .084$, or age distribution $t(79) = 1.273$, $p = .207$, $d = 0.287$. For education level,

participants selected either their current level of study, or last completed level, whichever was highest. The education levels of the two groups did significantly differ, $\chi^2(3) = 10.848$, $p = .013$, $\Phi = .364$, and are summarised in Table 3. Participants in the control group had studied to higher levels than in the group with chronic pain. Education should not affect reporting of pain-related and neutral words differently, as the target types are similar in frequency of use and length. Any difference in performance between pain-related and neutral words therefore cannot be attributed to education level, so it is possible to test for evidence of motivated attention in the context of an education level difference between the groups. However, any deficits in overall performance will be difficult to attribute to pain specifically, given the education level difference.

Table 3. *Education level of participants with chronic pain, and control participants, in Experiment 3, Chapter 3.*

	High school level 1, 2, or 3	Undergraduate	Honours	Postgraduate
Chronic Pain	8	19	10	4
Control	2	20	5	14

The participants with chronic pain were heterogeneous in cause of chronic pain, duration of chronic pain, and co-morbidities. See Appendix B for a summary of the duration and cause information given by the 41 participants with chronic pain. Thirty-five participants provided numerical duration information. The average was 11 years ($SD = 9$ years, range 3 months – 35 years). Fourteen participants reported having a current injury causing pain. Five reported current treatment for depression and / or anxiety, 12 reported receiving previous treatment for depression and / or anxiety, and seven reported both having received in the past and current treatment for depression and / or anxiety. Five participants reported having a neurological condition. Four reported experiencing chronic fatigue. Thirty participants were taking medication for their pain, depression and / or anxiety. For the 30 participants taking medication the average number of medications was three ($SD = 2$, range 1 – 9).

Of the 41 control participants, two reported receiving previous treatment for depression and / or anxiety, two reported receiving current treatment for depression and / or anxiety, and one reported both having received in the past and current treatment for depression and / or anxiety. To address the co-morbidities and group differences, if there are

group differences in the magnitude of the attentional blink for neutral and pain-related words, then depression, anxiety, and stress (assessed via questionnaire measures), co-morbidities, and number of medications will be used as covariates.

Stimuli & Apparatus

The stimuli and apparatus were identical to those in Experiment 1 and 2 for the RSVP. The ANT and questionnaires are described below.

Procedure

Participants completed the experiment individually, after instruction from the experimenter. I facilitated all participant recruitment and scheduling of appointments. Also, all participants with chronic pain were offered a taxi chit for getting to their next destination after the experiment; and I organised the taxis. Therefore, it was not possible for me, also the experimenter, to be blind to which participants had chronic pain and who were the controls. I had step-by-step written prompts, which I followed to facilitate explaining the tasks, and I used these prompts for all participants. While it is not ideal that I was not blind to group membership, I controlled for this as much as possible with the script.

RSVP.

The procedure was identical to Experiment 2, with mixed presentation only.

Attention Network Test (ANT).

The ANT version used was the ANT short version (Fan et al., 2002; E-Prime script downloaded from https://www.sacklerinstitute.org/cornell/assays_and_tools/ant/jin.fan/). The ANT uses several variants on the Eriksen and Eriksen (1974) letter flanker task that is commonly used to assess executive control. In the ANT, participants view a set of five black arrows against a white background on each trial. The participants' task is to report the direction that a centre arrow points (left or right); it is surrounded by arrows which match (congruent) or do not match (incongruent) its direction. On some trials an asterisk cue precedes the arrows that either indicates that the arrows will immediately follow the cue (centre cue) or indicates that the arrows will immediately follow the cue *and* where the arrows will appear (upper or lower location cue). The ANT provides measures for three aspects of attention. Alerting indicates the extent to which a warning cue facilitates performance on the upcoming task; orienting indicates the participant's ability to voluntarily shift their attention in anticipation of an upcoming target; executive control indicates the

ability to process goal-relevant information (the target arrow) while suppressing the flanking distractors (Fan et al., 2002).

On each trial, a fixation cross (0.5 cm x 0.5 cm) appeared in the centre of the screen for a random duration between 400 and 1600 ms then, on no cue trials, the arrows appeared in either the upper or lower visual field, 1 cm above the fixation cross. The fixation cross was present throughout the trial. See Figure 5, panel A. On trials with a central or location cue, an asterisk (0.5 cm x 0.5 cm) was presented immediately before the arrows for 100 ms. On central cue trials, the asterisk was presented in the centre (replacing the fixation for 100 ms). On location cue trials, the asterisk was presented in the centre of the upper or lower position (always where the arrows appeared). See Figure 5 panel B, for two examples of location cue trials. The arrowhead was 0.3 cm high, and each arrow was 0.9 cm long. There was a 0.1 cm gap between each arrow (the arrow display was thus 5 cm long). The arrows stayed on screen for up to 1700 ms. Within this response period, participants pressed the left arrow key with their left index finger if the centre arrow pointed left, and pressed the right arrow key with their right index finger if the centre arrow pointed right. Then the fixation cross stayed on screen during the ITI for a period of time, determined by response time and the fixation time, so that the trials were equated for length. See Figure 5, panel C, for a summary of a centre cue trial.

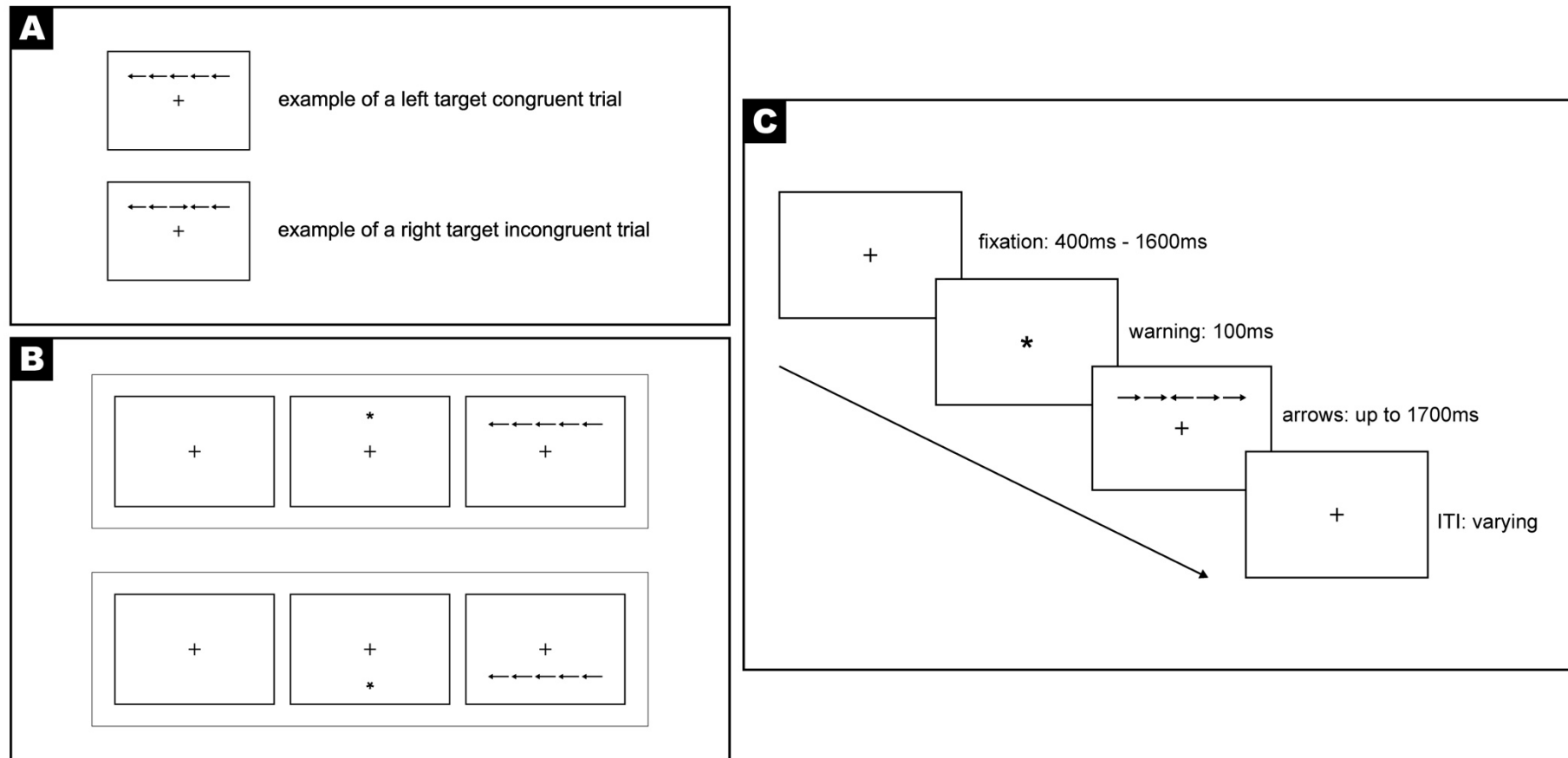


Figure 5. ANT stimuli and procedure. A. Examples of target displays in the ANT. B. Examples of location cue trials with an upper target and a lower target. C. Trial procedure for ANT-short task, the example displayed is for a centre cue trial.

The alerting score is the RT difference between trials with no cue and trials with a centre cue. The orienting score is the RT difference between trials with an upper or lower cue and centre cue trials. The executive score is the RT difference between all incongruent and congruent trials.

Participants completed six practice trials, without prompts to respond quickly and accurately, so they could see the range of cue and arrow pairings. Then they completed 12 practice trials with accuracy feedback, and were prompted to respond as quickly as they could without making mistakes. Participants then completed two blocks of 48 experimental trials (with no accuracy feedback). Participants could take a self-timed break between the two blocks. Each block consisted of 16 trials with no cue, 16 with a centre cue, and 16 with a location cue (eight above and eight below) in a random order. Each of the location-cued trials had the target in the upper position eight times (target left x four, target right x four) and in the lower position eight times (target left x four, target right x four). Participants were instructed to respond as quickly as they could without making mistakes. Accuracy and reaction time were recorded.

Questionnaires.

Participants completed, in fixed order, the McGill Pain Questionnaire – Short Form (Melzack, 1987) to assess current pain level, the 21 item Depression, Anxiety, Stress Scale (DASS; Lovibond & Lovibond, 1995a, 1995b) to assess symptoms of depression, anxiety and stress, the Attentional Control Scale (ACS; Derryberry & Reed, 2002) to assess subjective level of attentional control, and the Pain Vigilance and Awareness Questionnaire (PVAQ; McCracken, 1997) to assess subjective levels of attention to pain. The questionnaires were run via Qualtrics survey software and scored in their standard ways. See Appendix D for the questionnaire items.

The McGill Pain Questionnaire – Short Form (Melzack, 1987) was developed for use in time-limited pain research and is made up of three parts: ratings of how 10 sensory (e.g., *throbbing*) and five affective (e.g., *sickening*) adjectives match the participant's current pain experience on a scale from 0 – 3 (none, mild, moderate, severe), a Visual Analogue Scale (VAS) with the anchors *no pain* and *worst possible pain* (scored from 0 – 100), and a present pain index (0 – 5). Scores thus range from 0 – 150, with higher scores reflecting higher current pain. Melzack reported that scores on the short form are strongly convergent with scores on the long form, and that scores on the short form shift as expected during manipulations of pain experience (before and after pain medication in post-surgery pain, before and after epidural in labour pain, and before and after administration of TENS in musculoskeletal pain).

The DASS 21 is a shortened form of the 42 item DASS (Lovibond & Lovibond, 1995a, 1995b). Doubling the scale scores of the 21 item DASS gives scale scores for

depression, anxiety, and stress that are equivalent to scores obtained with the 42 item DASS (Lovibond & Lovibond, 1995b). An example of a question loading on the depression subscale is, *I couldn't seem to experience any positive feeling at all*; anxiety subscale, *I felt scared without any good reason*; stress subscale, *I was intolerant of anything that kept me from getting on with what I was doing*. Participants respond to the questions with a 0 – 3 response, where 0 = “did not apply to me at all”, and 3 = “applied to me very much, or most of the time” over the past week. Higher scores on each of the three subscales indicates greater experience of symptoms of depression, anxiety, or stress over the past week. The DASS 21 has high internal consistency (Antony, Bieling, Cox, Enns, & Swinson, 1998; Henry & Crawford, 2005, Lovibond & Lovibond, 1995a) and good convergent (Antony et al., 1998; Henry & Crawford, 2005; Lovibond & Lovibond, 1995a) and discriminant validity (Henry & Crawford, 2005).

The ACS (Derryberry & Reed, 2002) is a self-report measure of attentional control. An example question is, *when a distracting thought comes to mind, it is easy for me to shift my attention away from it*. Participants respond with a 1 – 4 response, where 1 = “almost never”, and 4 = “always”. Higher scores indicate greater self-reported ability to control attention. Depression and anxiety are associated with lower self-reported attentional control using the ACS (Ólafsson, Smári, Guðmundsdóttir, Ólafsdóttir, Harðardóttir, & Einarsson, 2011), and poor attentional control may be a boundary condition for observing attentional biases (Derryberry & Reed, 2002).

The PVAQ (McCracken, 1997, pp. 271) is a self-report measure of “awareness, vigilance, preoccupation, and observation of pain”. An example question is: *I am quick to notice changes in pain intensity*. Participants respond with a 0 – 5 response, where 0 = “never”, and 5 = “always”. Higher scores indicated greater attention to changes in pain experience. The PVAQ has high internal consistency and good convergent validity (McCracken, 1997; Roelofs, Peters, McCracken, & Vlaeyen, 2003).

Measure of current physical discomfort.

As a measure of current pain, in addition to the McGill Pain questionnaire, participants were asked to rate their current physical discomfort⁶ with a number between 0 and 100 at the end of the practice trials, at the end of each of the four trial blocks in the

⁶ “Physical discomfort” rather than “pain” was assessed to be consistent with an experiment not reported in this thesis, which assessed the impact of sustained physical discomfort induced by pressure on temporal attention for neutral stimuli.

RSVP task, and at the end of each of the two trial blocks in the ANT. The anchors were *no physical discomfort* (0) and *worst possible physical discomfort* (100). The physical discomfort ratings were recorded via key entry in E-prime.

Data Processing

When sphericity was violated, the original degrees of freedom are reported, with the Greenhouse-Geisser corrected p and epsilon (ϵ). For independent t -tests, Levene's test for equal variances was used and when significant, the corrected t and df are reported. For independent t -tests Cohen's d was calculated as a measure of effect size, and for dependent t -tests, d_z was calculated (Lakens, 2013). d_z is the mean difference divided by the standard deviation of the mean difference.

RSVP.

Identical to Experiment 2.

ANT.

The *alerting score* was calculated as the median RT of the correct trials with no cue minus the median RT of the correct trials with a centre cue. Larger scores on the alerting index reflect faster responding on cue trials relative to no cue trials; that is, greater vigilance to warning cues in the environment (Fan & Posner, 2004).

The *orienting score* was calculated as the median RT of the correct trials with a location cue minus the median RT of the correct trials with a centre cue. Larger scores on the orienting index reflect faster responding on the location cue trials relative to the centre cue trials; that is, greater ability to orient attention in response to a location cue above the advantage conferred by an alerting cue in general (Fan & Posner, 2004).

The *executive score* was calculated as the median RT of the correct incongruent trials minus the median RT of the correct congruent trials. Larger scores on the executive index reflect slower responding on the incongruent trials relative to the congruent trials; that is, a poorer ability to control distracting information or conflict (Fan & Posner, 2004).

Results

First, I present a comparison between participants with, and those without, chronic pain on the self-reported measures of pain, physical discomfort, depression, anxiety, stress, attentional control, and pain vigilance. Next, I present the results from the experimental tasks: the target-reporting task with RSVP, and the ANT. Last, I present additional analyses of the data by current pain-level rather than recruited group, and exploratory correlation analyses.

Recruitment Validity

McGill Pain Questionnaire and Physical Discomfort ratings.

There was a lot of missing data for the physical discomfort ratings taken at the end of each RSVP and ANT block. The missing data is likely due to a design choice, in that the key-press to submit RSVP target-reporting responses was also used to submit physical discomfort ratings. The physical discomfort rating screen followed the last RSVP reporting screen, and participants may have missed it by pressing the submit key twice. Only the rating taken at the end of the practice trials was examined. Participants in the control group reported much less physical discomfort on the scale from 0, *no physical discomfort*, to 100, *worst possible physical discomfort*, ($M = 5.76$, $SD = 11.90$), than those with chronic pain ($M = 28.66$, $SD = 23.88$), $t(58.711) = 5.497$, $p < .001$, $d = 1.229$.

One participant with chronic pain did not fill in the questionnaires due to time restrictions. The questionnaire results exclude that participant. The difference between groups in the physical discomfort ratings was mirrored in the McGill pain questionnaire total score (completed at the end of the experimental tasks), with those in the control group reporting much less current pain ($M = 11.71$, $SD = 21.85$) than those with chronic pain ($M = 56.23$, $SD = 28.09$), $t(73.621) = 7.948$, $p < .001$, $d = 1.794$.

DASS, ACS, and PVAQ.

See Table 4 for means and standard deviations. Participants with chronic pain reported more symptoms of depression, $t(53.229) = 3.775$, $p < .001$, $d = .857$, anxiety, $t(58.524) = 4.325$, $p < .001$, $d = 0.980$, and stress, $t(79) = 4.473$, $p < .001$, $d = .994$, than control participants, as measured by the DASS-21. They also reported being more vigilant to changes in pain experience, as measured by the PVAQ, $t(79) = 4.253$, $p < .001$, $d = 0.957$. There was a marginal difference on the ACS, with controls reporting greater self-reported attentional control than participants with chronic pain, $t(79) = 1.911$, $p = .06$, $d = 0.430$.

Table 4. *Questionnaire measures.*

	Chronic Pain	Control
	<i>M (SD)</i>	
McGill Pain Total**	56.23 (28.09)	11.71 (21.85)
Physical Discomfort**	28.66 (23.88)	5.76 (11.90)
DASS Depression**	5.88 (5.47)	2.32 (2.40)
DASS Anxiety**	5.10 (4.41)	1.71 (2.30)
DASS Stress**	8.60 (4.90)	4.37 (3.52)
ACS Total	49.70 (9.54)	53.44 (8.02)
PVAQ Total**	41.80 (15.13)	28.10 (13.85)

** indicates a significant group difference, $p < .001$.

The self-report questionnaire and physical discomfort data show that the groups differ in several ways. The group with chronic pain reported a higher current pain experience, more depression, anxiety, and stress, less attentional control, and more awareness of changes in pain, than the control group.

RSVP

Separate 2 (group: chronic pain, control) x 4 (TOA: 110, 330, 550, 770) x 2 (type: pain-related, neutral) mixed model ANOVAs were run for T1 accuracy and Conditional T2 accuracy, with group as a between-subjects factor and TOA and type as within-subjects factors.

RSVP T1 accuracy.

See Table 5 for means and standard deviations by TOA, type, and group. There was a main effect of TOA, $F(3, 240) = 5.181$, $\varepsilon = .898$, $p = .003$, $\eta_p^2 = .061$. Follow up paired-samples t -tests with a Bonferroni corrected alpha level of .008 indicated that accuracy to report T1 was lower at the 330ms TOA ($M = 72\%$, $SD = 17\%$) than the 110ms TOA ($M = 76\%$, $SD = 15\%$), $t(81) = 3.791$, $p < .001$, $d_z = 0.419$, but there was no difference between any of the other TOAs.

Table 5. *Accuracy for reporting T1 and T2 for participants with, and without, chronic pain in Experiment 3.*

	Chronic Pain		Control		Overall	
	T1 %	Conditional T2 %	T1 %	Conditional T2 %	T1 %	Conditional T2 %
TOA (ms)	Pain-related words					
	<i>M (SD)</i>					
110	73 (17)	19 (20)	76 (17)	15 (19)	74 (17)	17 (19)
330	70 (19)	16 (19)	70 (19)	14 (14)	70 (19)	15 (17)
550	73 (19)	51 (29)	74 (17)	53 (25)	73 (18)	52 (27)
770	69 (20)	64 (24)	73 (19)	67 (22)	71 (20)	65 (23)
	Neutral words					
	<i>M (SD)</i>					
110	75 (16)	17 (18)	79 (15)	13 (17)	77 (15)	15 (18)
330	74 (18)	21 (22)	73 (16)	20 (18)	74 (17)	21 (20)
550	73 (16)	50 (26)	74 (18)	54 (24)	74 (17)	52 (25)
770	75 (16)	62 (24)	76 (15)	66 (20)	76 (15)	64 (22)

There was a main effect of type, $F(1, 80) = 13.145, p = .001, \eta_p^2 = .141$, such that accuracy to report T1 in the context of a pain-related T2 ($M = 72\%, SD = 16\%$) was worse than in the context of a neutral T2 ($M = 75\%, SD = 14\%$).

See Figure 6 for T1 accuracy by TOA, type, and group. There was no type x TOA interaction, $F(3, 240) = 1.556, \epsilon = .889, p = .201, \eta_p^2 = .019$, nor any interactions with group: type x group, $F(1, 80) = .152, p = .697, \eta_p^2 = .002$, TOA x type x group, $F(3, 240) = .313, p = .816, \eta_p^2 = .004$. There was also no main effect of group, $F(1, 80) = .173, p = .678, \eta_p^2 = .002$. Chronic pain experience did not impact reporting of T1 overall (as would be predicted by a deficit-view), nor as a function of whether T2 was pain-related or neutral.

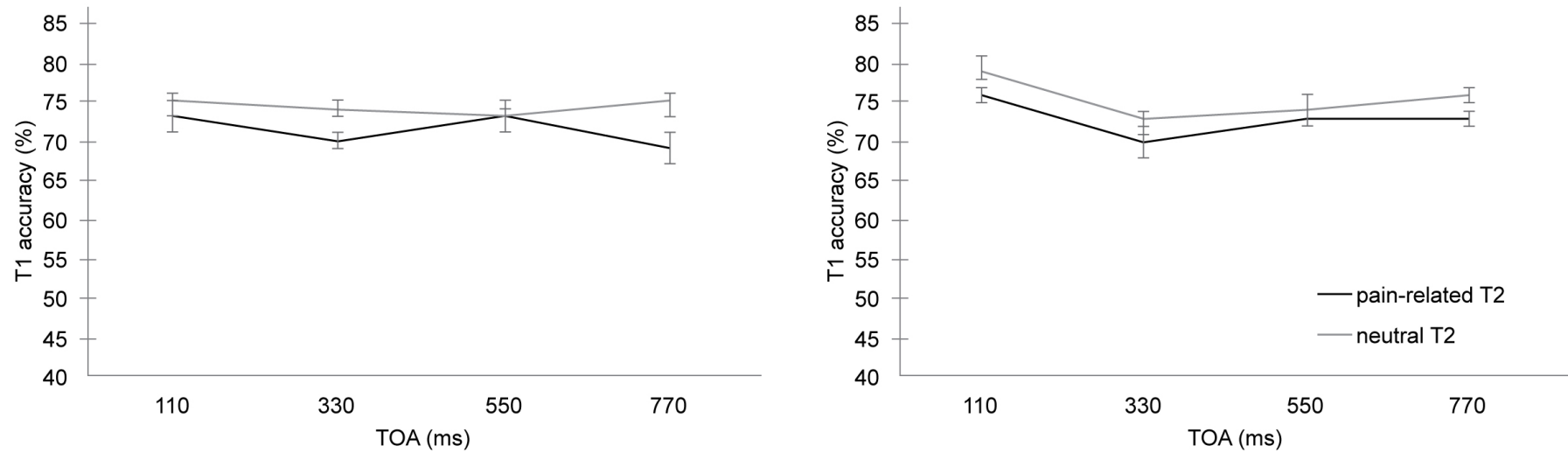


Figure 6. T1 accuracy as a function of TOA and word type for participants with (left panel) and without chronic pain (right panel). Error bars are within-subjects standard errors with Morey's (2008) correction.

RSVP conditional T2 accuracy.

The analyses of CT2 accuracy allow for the deficit-view and the motivated attention hypothesis of attention in chronic pain to be tested. If participants with chronic pain have a deeper blink across word type than participants without chronic pain (an interaction between group and TOA), then the deficit-view would be supported. If participants with chronic pain show a shallower blink for pain-related than neutral stimuli, and this attentional bias is larger than in participants without chronic pain (an interaction between group, TOA, and type), then the motivated attention hypothesis would be supported.

See Table 5 for means and standard deviations by TOA, type, and group. There was a main effect of TOA, $F(3, 240) = 324.050$, $\varepsilon = .658$, $p < .001$, $\eta_p^2 = .802$, reflecting an attentional blink and subsequent recovery. Follow-up paired-samples t -tests with a Bonferroni corrected alpha level of .008 indicated that CT2 accuracy at 110 ms TOA and 330 ms TOA were not significantly different from each other, $t(81) = 1.102$, $p = .274$, $d_z = 0.112$, but that all the rest of the TOAs differed significantly from each other, such that performance at the later TOAs was better than at the earlier TOAs.

There was also a TOA x type interaction, $F(3, 240) = 4.426$, $p = .005$, $\eta_p^2 = .052$, reflecting an attentional bias. Follow up paired-samples t -tests, with a Bonferroni corrected alpha level of .0125, indicated that there was no difference between accuracy to pain-related and neutral T2s at the 110 ms, 550 ms, or 770 ms TOA, but at the 330 ms TOA, pain-related T2s ($M = 15\%$, $SD = 17\%$) were reported less accurately than neutral T2s ($M = 21\%$, $SD = 20\%$), $t(81) = 3.629$, $p < .001$, $d_z = 0.401$. This effect is opposite to that reported in Experiment 2. The direction of the attentional bias suggests that the bias is away from pain-related information.

There was not a main effect of type in the CT2 data, $F(1, 80) = .774$, $p = .382$, $\eta_p^2 = .010$, nor any main effect of group, $F(1, 80) = .001$, $p = .971$, $\eta_p^2 < .001$, or interactions between group and TOA $F(3, 240) = 1.652$, $p = .178$, $\eta_p^2 = .020$, group x type, $F(1, 80) = .518$, $p = .474$, $\eta_p^2 = .006$, nor between group x TOA x type, $F(3, 240) = .053$, $p = .984$, $\eta_p^2 = .001$. See Figure 7 for CT2 accuracy by TOA, type, and group. The lack of a group x TOA interaction suggests that chronic pain does not modulate the magnitude of the attentional blink – there is no deficit in temporal attention associated with chronic pain. The

lack of a group x TOA x type interaction suggests that the bias away from pain-related information in the attentional blink period is no different for people with, than without, chronic pain – there is no motivated attention associated with chronic pain.

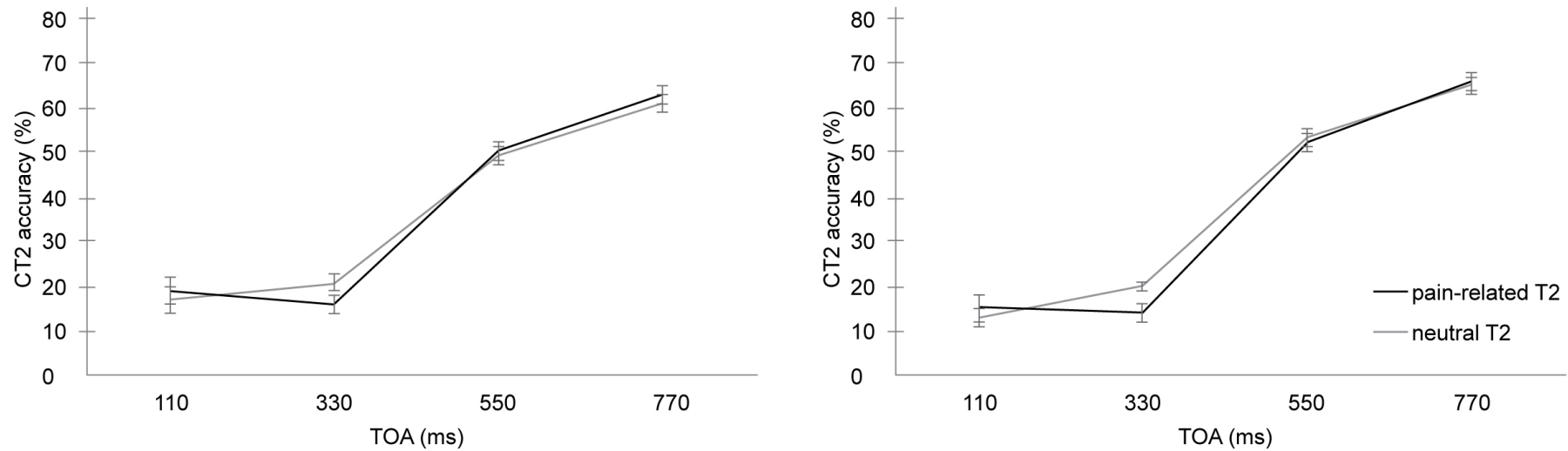


Figure 7. Conditional T2 (CT2) accuracy as a function of TOA and word type for participants with (left panel) and without chronic pain (right panel). Error bars are within-subjects standard errors with Morey's (2008) correction.

Both groups of participants demonstrated an attentional blink. However, the attentional bias was away from, rather than towards, pain-related information. In the attentional blink period, pain-related T2s were reported less often than neutral T2s. This pattern of reporting could suggest that during the attentional blink period, attentional capacity was directed away from pain-related information. This pattern directly contradicts the bias observed in Experiment 2, in which attention in participants without chronic pain was directed towards pain-related information, assessed using the same experimental design. Furthermore, neither the deficit-view or the motivated hypothesis of attention in chronic pain were supported. Contrary to a deficit-view of cognition in chronic pain, there was no difference between the magnitude of the attentional blink in the two groups. Contrary to a motivated view of temporal attention, the attentional bias was away from pain-related information, and the magnitude of the bias did not differ between participants with, and without, chronic pain.

ANT

Due to time restrictions, one participant did not complete the ANT. The ANT analyses are based on the 81 participants who completed the ANT (40 participants with chronic pain and 41 controls). See Table 6 for the mean network scores for participants with chronic pain and controls. There were no significant differences between participants with chronic pain and control participants on the ANT alerting, $t(79) = .050$, $p = .960$, $d = 0.011$, orienting, $t(79) = .145$, $p = .885$, $d = 0.032$, or executive scores, $t(67.762) = .636$, $p = .527$, $d = 0.146$; chronic pain was not associated with deficits in the attention networks.

Table 6. *Attention network scores for participants with, and without, chronic pain.*

	Chronic Pain	Control
	<i>M (SD)</i>	<i>M (SD)</i>
Alerting network	17 ms (30 ms)	17 ms (28 ms)
Orienting network	39 ms (34 ms)	40 ms (34 ms)
Executive network	96 ms (49 ms)	90 ms (33 ms)

As well as using the derived executive score, a 2 (congruency: congruent, incongruent) x 2 (group: chronic pain, control) mixed model ANOVA was conducted on the

responses to all congruent and incongruent trials, to see if chronic pain was associated with psychomotor slowing. There was only a main effect of congruency, $F(1, 79) = 400.920$, $p < .001$, $\eta_p^2 = .835$, with participants responding as is expected in a flanker task: faster to congruent trials ($M = 541$ ms, $SD = 97$ ms) than incongruent trials ($M = 634$ ms, $SD = 112$ ms). There was no main effect of group, $F(1, 79) = 2.474$, $p = .120$, $\eta_p^2 = .030$; chronic pain was not associated with psychomotor slowing. There was no interaction between group and congruency, $F(1, 79) = .409$, $p = .524$, $\eta_p^2 = .005$; chronic pain was not associated with deficits in executive control of attention.

Additional Analyses

Current pain level.

The participants in the two groups were clearly experiencing different levels of pain, depression, anxiety, and stress. Furthermore, the groups reported different levels in self-reported vigilance to pain. People with chronic pain feel like they pay a lot of attention to their pain. However, these differences do not translate to performance differences on the RSVP or ANT tasks. Some of the participants with chronic pain mentioned to the experimenter that they were not experiencing pain when they completed the tasks, and some of the control group reported that they were experiencing pain. In order to assess whether current pain is associated with performance, a median split was conducted on the McGill questionnaire total scores for the 81 participants who completed the questionnaires. Participants who scored 18 or less on the McGill total score were classified as low pain experiencers ($n = 41$), and those who scored greater than 18 as high pain experiencers ($n = 40$). Six participants with chronic pain were low pain experiencers and six control participants were high pain experiencers. The same analyses as above were performed on the questionnaire, RSVP, and ANT data.

For the most part, the same main effects and interactions were reported. Current pain does not impact temporal attention differently than chronic pain. However, there was one difference in the ANT data analyses. When the groups were defined by current pain experience (low or high), on the test of psychomotor slowing, there was a main effect of group, $F(1,79) = 5.837$, $p = .018$, $\eta_p^2 = .069$, such that participants experiencing more pain were slower to respond to both congruent ($M = 564$ ms, $SD = 109$ ms) and incongruent ($M = 665$ ms, $SD = 126$ ms) trials than participants experiencing less pain (congruent:

$M = 518$ ms, $SD = 79$ ms; incongruent: $M = 604$ ms, $SD = 88$ ms). This main effect is reflective of general psychomotor slowing in the group experiencing higher pain.

Correlations.

As group did not impact the magnitude of the attentional blink, or functionality of the attentional networks, exploratory correlational analyses amongst behavioural and questionnaire measures were conducted with the full participant set. An indication of the magnitude of the attentional blink for pain-related and neutral stimuli was calculated as CT2 accuracy at the 770 ms TOA minus the CT2 accuracy at the 330 ms TOA for pain-related and neutral words separately. This calculation takes the lag into account and uses the TOA at which the difference between stimuli was isolated (330 ms), and thus reflects the magnitude of the attentional blink, rather than solely reflecting accuracy for type overall (MacLean & Arnell, 2012). These two attentional blink magnitudes, and the difference between them (as a measure of bias: neutral attentional blink – pain-related attentional blink) were tested for correlations with the three attention network indices. Relationships between the questionnaire measures and the behavioural measures were also examined. There were two significant relationships between domains. The greater the attentional bias, the less efficient the alerting network was, $r(79) = -.224$, $p = .044$. The higher self-reported stress the less efficient the executive network was, $r(79) = .246$, $p = .027^7$.

The other significant relationships were within the same cognitive domain⁸, and within the questionnaire measures. The efficiency of the orienting network was significantly, negatively correlated with both the efficiency of the alerting network, $r(79) = -.377$, $p = .001$, such that the more efficient the orienting network, the less efficient the alerting network; and with the executive network, $r(79) = -.222$, $p = .046$, such that the more efficient the executive network, the more efficient the orienting network.

⁷ Higher scores on the executive network reflect poorer efficiency to control conflict between incongruent distracting flankers and the centre target.

⁸ The magnitude of the attentional blink for pain-related and neutral T2s were significantly, positively, correlated, $r(79) = .402$, $p < .001$, such that the deeper the blink for pain-related words the deeper the blink for neutral words. While these measures are not independent, so these correlations are not clearly interpretable, the magnitude of the bias was significantly, positively, correlated with the magnitude of the blink for pain T2s, $r(79) = .638$, $p < .001$, such that the greater the bias towards neutral words, the deeper the blink for pain-related words was; and the magnitude of the bias was significantly, negatively, correlated with the magnitude of the blink for neutral T2s, $r(79) = -.449$, $p < .001$, such that the greater the bias towards neutral words, the shallower the blink for neutral words was. These relationships reflect that as the attentional bias is towards neutral information, neutral words overcome the attentional blink more, whereas pain-related words are “blinked” more often.

See Table 7 for the relationships between questionnaire measures. Pain, depression, anxiety, and stress were positively related. As one factor increases so do the others, which reflects the co-morbidity of these symptoms. Interestingly, though not reflected in the behavioural measures of attention, higher self-reported pain, depression, anxiety, stress and vigilance to pain are associated with lower self-reported attentional control. And higher self-reported pain, depression, anxiety, and stress are associated with higher vigilance to pain.

Table 7. *Correlations between questionnaire measures.*

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Pain-related blink	-	.402**	.638**	-.171	-.001	-.121	-.095	-.039	-.007	-.008	-.040	.111	-.070
2. Neutral blink		-	-.449**	.068	-.068	-.114	.014	.032	-.001	-.054	-.010	.086	-.070
3. Neutral bias			-	-.224*	.056	-.022	-.078	-.065	-.006	.037	-.031	.036	-.009
4. ANT alerting				-	-.377**	.014	.067	-.043	-.057	.094	-.001	-.056	-.038
5. ANT orienting					-	-.222*	-.017	-.019	-.004	-.026	-.039	-.033	-.187
6. ANT executive						-	.005	.098	.157	.210	.246*	-.067	.184
7. Physical Discomfort							-	.773**	.327**	.337**	.339**	-.164	.295**
8. McGill Pain Total								-	.522**	.528**	.623**	-.362**	.429**
9. D-Depression									-	.656**	.778**	-.519**	.381**
10. D-Anxiety										-	.800**	-.539**	.336**
11. D-Stress											-	-.577**	.303**
12. ACS Total												-	-.384**
13. PVAQ Total													-

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

Note. D=DASS: Depression, Anxiety, Stress Scale.

Discussion

According to the motivated attention hypothesis, an overactive threat-protection mechanism in people with chronic pain directs attention towards pain-related information and away from neutral information. This view of processing led to the prediction that deficits would be observed on tasks using neutral stimuli, and prioritised attention to pain-related information would be observed, in participants with chronic pain as compared to control participants. To test the deficit-view and the motivated attention hypothesis, I extended Experiment 2 by recruiting participants who were currently experiencing chronic pain and matched controls (without chronic pain). All participants completed an RSVP task in which the nature of T2 was manipulated; the magnitude of the attentional blink for pain-related and neutral words was compared. Participants also completed the ANT task to measure alerting, orienting, and executive control aspects of attention, and questionnaires to measure current pain, depression, anxiety, stress, self-reported attentional control, and awareness of pain sensations.

The hypothesis derived from the deficit-view was that because there is competition for cognitive capacity, greater attention to pain experience means impaired, or interrupted, attention to the environment, contributing to production of the deficits. Prediction 1 was that participants with chronic pain will show cognitive deficits compared to performance of participants without chronic pain. Participants with chronic pain did report marginally poorer attentional control than control participants, and significantly more vigilance to their pain. So, participants with chronic pain do consider their attention to be impaired. Furthermore, when the data from the two groups of participants were combined, attentional control was negatively related to vigilance to pain. The poorer self-perceived attentional control was, the greater attention to changes in pain experience were reported. However, the patterns of processing T1 and T2 in the RSVP task and the efficiency of the attention networks in the ANT task were no different between participants with chronic pain and control participants, contrary to prediction 1, and evidence against the deficit-view of attention in chronic pain.

The motivated attention hypothesis was that even in chronic pain experience, protective processing still drives patterns of attention. That is, threat protection motivates assignment of attentional capacity towards information and behaviours most adaptive for survival, such as signals of threat to bodily tissue, signals of pain, or signals of relief of pain; and away from other kinds of information and behaviours. Prediction 2 was that participants with chronic pain will show a larger attentional bias to pain-related stimuli compared to any

bias observed in participants without chronic pain. Prediction 3 was that in chronic pain experience, the magnitude of the attentional bias in RSVP and emotional distraction will be positively correlated with the magnitude of the cognitive deficits in the ANT. The attentional blink for pain-related words was deeper than for neutral words in people with, and without, chronic pain. Participants were less likely to report the pain-related words than the neutral words in the attentional blink period at the 300 ms TOA. Against prediction 2, and contrary to the motivated attention hypothesis, this attentional bias was away from pain-related words and did not differ in magnitude between those with, and those without chronic pain.

The pattern of attentional bias away from pain-related words was unexpected and opposite to the bias observed in Experiment 2. In Experiment 3, the RSVP target-reporting task used mixed presentation with a manipulation of the word type used as T2s. These design choices were based on results from Experiment 2, in which participants without chronic pain showed an attentional bias towards pain-related stimuli in the attentional blink period, with mixed presentation. However, in Experiment 3, both participants with chronic pain and matched controls showed a bias away from pain-related stimuli. Although it is tempting to speculate on explanations for this discrepancy, it is also possible that the attentional bias observed in Experiment 2 is not robust. It therefore seems wise to attempt to replicate the attentional bias patterns from Experiment 2 before drawing strong conclusions. I will return to the primary finding of Experiment 3 – the complete lack of behavioural differences between participants with chronic pain and controls – following the report of the replication study.

Experiment 4

In Experiment 3, there was no difference in processing between participants with chronic pain and controls, and there was a bias away from pain-related words in temporal attention across the groups. Therefore, Experiment 2 was replicated with a different group of undergraduate participants to see if the biases towards pain-related words observed under blocked and mixed presentation in Experiment 2 were reliable.

Method

Participants

In Experiment 2, the bias towards pain-related words at T2 was observed at early TOAs (in the attentional blink period) with mixed presentation but at later TOAs (outside of the attentional blink period) with blocked presentation. For Experiment 4, participants were

58 Victoria University of Wellington psychology students, participating in an introduction to psychology research programme. Five participants with low accuracy on the RSVP task, one participant for whom the experiment crashed part way through the RSVP task, and one participant who did not complete the RSVP task were removed from the analyses. There were 51 participants in the final analysis (41 women, 10 men; *mean age* = 18.90, *SD* = 2.74; 27 in blocked and 24 in mixed presentation).

Stimuli, Apparatus, Procedure & Data Processing

The stimuli, apparatus, procedure, data processing were identical to those described for Experiment 2.

Results & Discussion

Separate 2 (type: pain-related, neutral) x 4 (TOA: 110, 330, 550, 770) x 2 (presentation: blocked v. mixed) mixed model ANOVAs were run for T1 and conditional T2 accuracy, with type and TOA as within-subjects factors and presentation as a between-subjects factor. See Table 8 for the RSVP data by TOA and type.

RSVP T1 Accuracy

Replicating Experiment 2, there was a main effect of type, $F(1, 49) = 22.520, p < .001, \eta_p^2 = .315$; such that reporting of the neutral T1s was higher when they were followed by a neutral T2 ($M = 72\%, SD = 15\%$) than by a pain-related T2 ($M = 68\%, SD = 15\%$). Unlike Experiment 2, there was also a trending main effect of TOA, $F(3, 147) = 2.455, p = .065, \eta_p^2 = .048$ (Figure 8, left panel). Both main effects were qualified by a type x TOA interaction, $F(3, 147) = 3.692, p = .013, \eta_p^2 = .070$. This interaction reflects that the difference, between reporting of neutral T1s that preceded neutral and pain-related T2s, was largest at the 110 ms and 770 ms TOA; 110 ms: pain-related ($M = 65\%, SD = 17\%$), neutral ($M = 73\%, SD = 15\%$), $t(50) = 5.205, p < .001, d_z = 0.49$, 770 ms: pain-related ($M = 69\%, SD = 17\%$), neutral ($M = 74\%, SD = 16\%$), $t(50) = 2.216, p = .031, d_z = 0.25$.

RSVP Conditional T2 Accuracy

See Figure 8, right panel, for the overall CT2 data and Figure 9 for the CT2 data by presentation. Replicating Experiment 2, there was a main effect of TOA (reflecting the attentional blink and subsequent recovery), $F(3, 147) = 228.529, p < .001, \epsilon = .677$,

$\eta_p^2 = .823$. Unlike Experiment 2, there was a trending main effect of presentation, reflecting lower accuracy for blocked ($M = 34\%$, $SD = 9\%$) as compared to mixed presentation ($M = 41\%$, $SD = 17\%$), $F(1, 49) = 3.741$, $p = .059$, $\eta_p^2 = .071$. Importantly, there was no type x TOA x presentation interaction, $F(3, 147) = .570$, $p = .636$, $\eta_p^2 = .011$. Note that there was also no type x TOA interaction, $F(3, 147) = .552$, $p = .647$, $\eta_p^2 = .011$, meaning that there is no modulation of the attentional blink by stimulus type (no attentional bias).

Table 8. Accuracy for reporting T1 and T2 in the three experiments in Chapter 2 by undergraduate research participants.

	Experiment 1 – Manipulating T1, $n = 37$		Experiment 2 – Manipulating T2 blocked, $n = 21$		Experiment 2 – Manipulating T2 mixed, $n = 19$		Experiment 4 – Manipulating T2, blocked $n = 27$		Experiment 4 – Manipulating T2, mixed $n = 24$	
	T1 %	Conditional T2 %	T1 %	Conditional T2 %	T1 %	Conditional T2 %	T1 %	Conditional T2 %	T1 %	Conditional T2 %
TOA (ms)	Pain-related words $M (SD)$									
110	75 (14)	14 (14)	57 (20)	12 (19)	60 (18)	18 (17)	63 (13)	10 (13)	67 (21)	20 (23)
330	74 (14)	18 (14)	56 (22)	13 (18)	58 (16)	19 (17)	64 (16)	12 (9)	72 (19)	23 (21)
550	74 (16)	50 (22)	59 (21)	49 (20)	62 (16)	41 (20)	67 (15)	45 (18)	73 (17)	55 (24)
770	74 (13)	57 (19)	58 (18)	63 (17)	54 (22)	57 (18)	66 (17)	67 (16)	74 (17)	70 (20)
	Neutral words $M (SD)$									
110	65 (18)	10 (12)	66 (20)	10 (13)	64 (16)	10 (10)	71 (14)	12 (12)	75 (16)	18 (21)
330	64 (17)	19 (15)	63 (17)	15 (16)	64 (16)	13 (17)	69 (14)	15 (12)	73 (17)	23 (20)
550	64 (17)	50 (19)	62 (18)	46 (20)	67 (16)	42 (22)	69 (16)	45 (17)	73 (19)	52 (24)
770	66 (19)	62 (20)	63 (17)	53 (18)	66 (14)	59 (16)	70 (15)	64 (17)	78 (18)	69 (20)

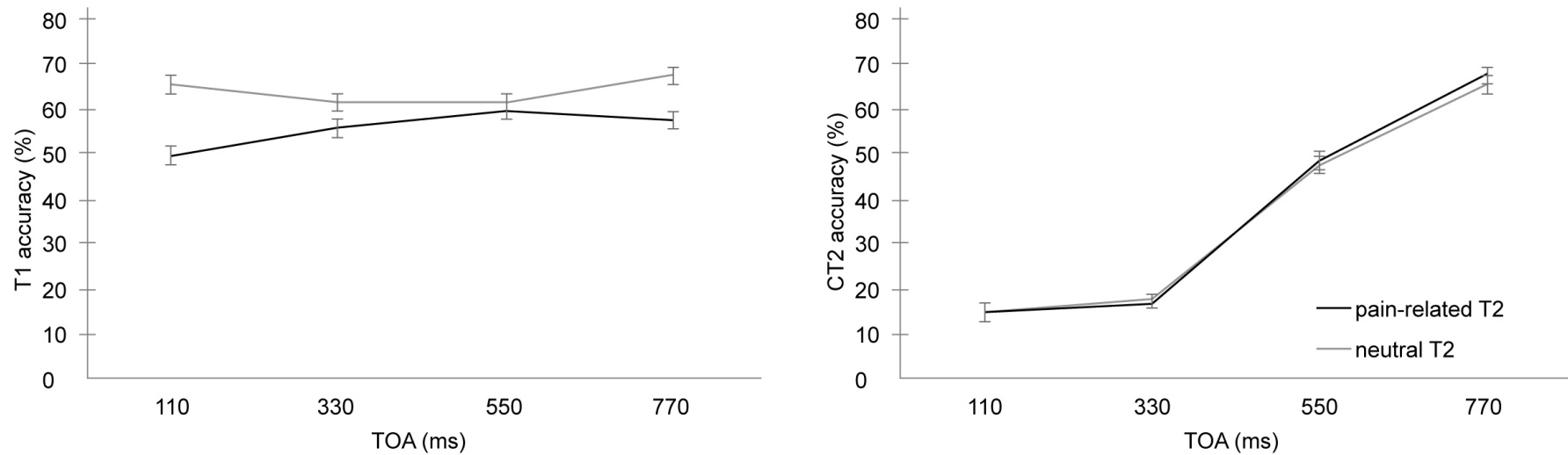


Figure 8. T1 (left panel) and conditional T2 (CT2, right panel) accuracy as a function of TOA and word type for Manipulating T2 in Experiment 4. Error bars are within-subjects standard errors with Morey's (2008) correction.

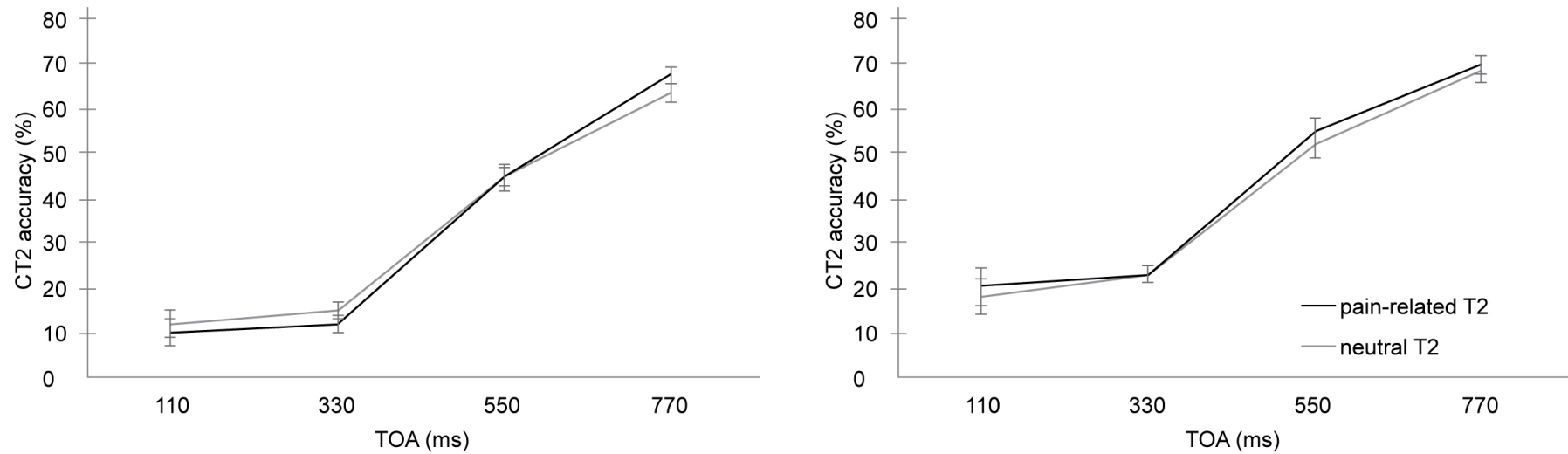


Figure 9. Type x TOA for conditional T2 (CT2) accuracy for Manipulating T2 blocked (left panel) and mixed (right panel) presentation in Experiment 4. Error bars are within-subjects standard errors with Morey's (2008) correction.

In Experiment 2, temporal attention was biased towards pain-related information. When the threat protection mechanism was activated by using blocked stimulus presentation, more pain-related T2s were reported than neutral T2s after the attentional blink period. Under mixed presentation, more pain-related T2s were reported than neutral T2s during the attentional blink. Neither of these effects were observed in the exact replication experiment. The effect of engagement of the threat protection system, by making threat predictable, on the attentional blink is either hard to detect, or it does not have a big effect on temporal processing of pain-related information in participants without chronic pain. Most importantly, there was no bias towards or away from pain-related information observed in Experiment 4.

General Discussion

Pain experience is one way our brain and body protect us from harm to our body-tissue. An attentional bias to pain-related information in the environment could confer additional protection above the experience of pain. In this series of experiments, I tested whether pain-related stimuli are processed differently than neutral stimuli in participants with, and without, chronic pain. To test whether individuals without chronic pain have an attentional bias to pain-related stimuli and away from neutral stimuli, I used RSVP to assess whether pain-related words induce a deeper attentional blink (Experiment 1), and / or overcome the attentional blink more effectively, relative to neutral words (Experiments 2, 3, and 4). Processing of pain-related words affected temporal attention. In Experiment 1, a shallower attentional blink was induced by pain-related words than neutral words at the 110ms TOA. In Experiment 2, pain-related words overcame the attentional blink *more* effectively than neutral words at the early TOAs with mixed presentation. However, the attentional biases observed were small and when I used Experiment 2's design with mixed presentation in Experiment 3 to compare biases in participants with, and without, chronic pain, pain-related words overcame the attentional blink *less* effectively than neutral information. And when I replicated Experiment 2 in Experiment 4, I did *not* observe a bias at all. These findings lead to two main conclusions. First, the RSVP task does not produce a reliable attentional bias to pain-related information, at least in undergraduate students. And secondly, people in chronic pain do not differ from controls in their temporal attention to pain-related or neutral information, contrary to both the deficit-view and the motivated attention hypothesis of attention in chronic pain.

In Experiment 1, in which the nature of T1 was manipulated to assess induction of the attentional blink by pain-related and neutral words, attention to pain-related T1s seemed to

have a different effect on the blink at shorter and longer TOAs. At shorter TOAs, there was a shallower attentional blink following a pain-related T1 than a neutral T1. When time between T1 and T2 was short and there was competition for attentional capacity, processing of a pain-related T1 seemed to enhance processing of following words, compared to processing a neutral T1. Processing of a pain-related word may have temporarily activated the threat protection system and more attentional capacity was allocated to closely following stimuli. In contrast, at longer TOAs, reporting of T2 was better following a neutral T1 than a pain-related T1. When time between T1 and T2 was longer, then processing of a pain-related T1 seemed to take processing capacity away from stimuli temporally further away from the threat. The longer TOAs are not in the attentional blink period. This enhancement of temporally-close stimuli and inhibition of temporally-distant stimuli from a threat could reflect reallocation of attentional capacity to stimuli temporally proximate to something threatening.

In Experiment 2, in which the nature of T2 was manipulated to assess overcoming of the attentional blink by pain-related and neutral words, engagement of the threat protection system by blocking T2 type seemed to affect the expectation of participants and there was enhanced reporting of pain-related T2s at the longer TOAs, in the recovery period of the attentional blink. With mixed presentation, in which participants had no set expectation and thus no activation of the threat protection system, there was greater reporting of pain-related T2s than neutral T2s at the short TOAs, in the attentional blink. When upcoming information was unpredictable, there seemed to be an early bias in temporal attention towards pain-related information.

In Experiment 3, the nature of T2 was manipulated under mixed presentation. Participants with chronic pain and matched controls were recruited. The participants with chronic pain, although heterogeneous in the nature of their pain condition and co-morbidities, were in more pain and had more self-reported symptoms of depression, anxiety, and stress than the control participants. Participants with chronic pain also reported being more vigilant to changes in their pain experience, and marginally poorer attentional control than participants without chronic pain. Yet these differences in subjective experience did not extend to observable differences in cognitive functioning. Aside from psychomotor slowing on responses to the ANT (only observable in a post-hoc comparison of participants experiencing more current pain to participants experiencing less current pain), contrary to a deficit-view of attention, no attentional deficits were observed in target-reporting for RSVP or ANT performance. A bias was observed in temporal attention; however, it did not differ

between participants with, and without, chronic pain, contrary to the motivated attention hypothesis. Furthermore, directly opposite to the bias observed in undergraduate participants in Experiment 2, in which pain-related words overcame the attentional blink *more* effectively than neutral words, in Experiment 3 pain-related words overcame the attentional blink *less* effectively than neutral words.

The inconsistency in the direction of the bias observed is concerning, and so to determine whether the effect of presentation on engagement of the threat protection system observed in Experiment 2 was reliable, I replicated the design in Experiment 4. The early bias towards pain-related words with mixed presentation, and the late bias towards pain-related words with blocked presentation did not replicate. In fact, there was no difference in the magnitude of the attentional blink for pain-related and neutral T2s in either presentation format.

The current studies suggest that in undergraduate students and a community sample without chronic pain, there is not a reliable bias in temporal attention to pain-related information and that engagement of the threat protection system by blocking stimuli type does not reliably modulate processing of pain-related information. These experiments suggest that pain-related words are not threatening words in people without chronic pain. In participants with chronic pain, the attentional bias away from pain-related words did not differ from the bias observed in control participants; this is contrary to a motivated view of cognition in chronic pain experience.

There are two points that need to be considered when interpreting the results. Firstly, as with the dot-probe task, the target-reporting task for RSVP may not be reliable enough to assess attentional bias (Dear et al., 2011; Schmukle, 2005), or, like the dot-probe and emotional Stroop tasks, the target-reporting task for RSVP may not be sensitive enough to detect differences in attentional bias between groups (Asmundson et al., 2005). Focusing on the experiments in which the nature of T2 was manipulated, I have observed an attentional bias towards pain-related stimuli, an attentional bias away from pain-related stimuli, and then no difference between processing of pain-related and neutral stimuli. Inconsistent findings across replications are not uncommon in this literature. Attridge, Eccleston, Noonan, Wainwright, and Keogh (2017) reported an attempted replication of cognitive functioning observations in people with chronic headache pain (Moore et al., 2013b). Both articles report cognitive deficits but in different tasks. Attridge and colleagues describe the variation in observation of cognitive deficits as “dynamic”. The variation in findings around cognitive deficits is likely due to a combination of noise in the data, difficulty in estimating effect sizes

to conduct a power analyses, and a lack of sensitivity of the tasks used to assess cognitive functioning, as well a lack of understanding of the mechanisms underlying the occurrence of cognitive deficits in chronic pain experience. These factors combine to mean that it is difficult to know which cognitive processes to target, or which tasks to use, in our research.

Alternatively, it could be that the motivated attention hypothesis does not describe information processing in chronic pain experience. Attention is not motivated towards pain and threat-related information as part of an over-active threat protection, and an attentional bias does not contribute to cognitive deficits observed in chronic pain experience. However, there are also limitations of the current experiments to consider. These issues mean that the experiments in this chapter cannot give a decisive answer as to whether there is a bias to pain-related information in people without chronic pain, or if there is motivated attention in people with chronic pain.

First, the stimuli were words. Words related to pain are symbolic, describing a subjective experience. As such, words are less potent and less biologically relevant as threat stimuli than images. Whether emotional content is conveyed via words or images affects processing (e.g., Hinojosa, Carretié, Valcárcel, Méndez-Bértolo, & Pozo, 2009). In a study comparing attentional biases for pain-related words and pictures, biases were only observed with picture stimuli (Dear et al., 2011). Thus, the use of words would have made it more difficult to observe any attentional differences between stimulus types. Using pictures of people in which body-tissue threat is present (e.g., mutilations or injuries) or not (e.g., daily activities) may allow for a better assessment of any attentional bias driven by an overactive threat protection system.

Second, to avoid repeating stimuli, pain-related words were selected that cover a range of subtypes, labeled by previous researchers as: affective ($n = 18$), disability ($n = 17$), health-related negative ($n = 8$), pain ($n = 15$), sensory ($n = 19$) and threat ($n = 19$). These categories were taken from previous research and could be re-categorised (e.g., the ‘pain’ words could be relabeled as ‘sensory’ words). It may be that pain words are not threatening stimuli generally, but certain subtypes are.

Third, in these experiments, the assignment of pain-related word subtype to TOA was random, so the magnitude of the attentional blink cannot be assessed for different subtypes. If attentional biases are specific, the subtype limitation would make it harder to observe effects of stimulus type on processing during the attentional blink and recovery. Furthermore, the neutral words were also not restricted to one category (e.g., tools), but were any word without affective connotation. By having pain-related words that were all in a category (pain) and

neutral words that were not, I inadvertently gave pain-related words a semantic processing advantage. Over the course of the experiment, participants could come to expect what kind of word T1 or T2 might be for the pain-related stimuli, but not for the neutral stimuli. This category limitation would make it easier to observe a bias towards pain-related information. However, given that I observe an attentional bias to pain-related words in Experiment 2, to neutral words in Experiment 3, and no bias in Experiment 4, the category limitation cannot be having a very large effect on processing of pain-related or neutral words.

There are also two issues that pertain specifically to Experiment 3, when comparing attention in people with, and without, chronic pain. First, I recruited a heterogeneous participant population, which is another source of noise in the data. The participants with chronic pain match the control participants on age and sex, but not education level. Furthermore, there are many differences in the cause, duration and severity of chronic pain, within the group with chronic pain. In a small city, wide recruitment was necessary to find participants experiencing chronic pain. However, chronic pain encompasses many experiences. In addition, because of the requirement that people travel to the university to participate, I likely recruited participants who are functioning relatively well. It is important to note that, although not a significant difference in proportion, consistent with the deficit-view, more participants with chronic pain did not meet target-reporting task performance criteria for RSVP than participants without chronic pain (nine in the group with chronic pain vs. four in the control group). The target-reporting task produces accuracy as the dependent measure; perhaps using a task that measures reaction time differences will allow clearer inferences to be made about cognitive functioning in chronic pain experience. More data may be able to be included in the analyses when using an RT task.

Second, in the RSVP task used with participants with chronic pain in Experiment 3, I chose to mix presentation of pain-related and neutral T2s randomly, instead of presenting T2 type in blocks. I chose to use mixed presentation as it is more ecologically valid and a closer representation of day to day processing of environmental information, and it produced an attentional bias towards pain-related information in Experiment 2. However, it is possible that choosing to block T2 type instead may have more strongly activated the threat protection system in chronic pain experience, and evidence for motivated attention may have been observed. In the next chapter, I block stimuli presentation to maximize observation of attentional biases in chronic pain.

There are many positive aspects of the design of the experiments in this chapter though. Unlike many studies that aim to assess cognitive functioning in chronic pain

experience (research guidelines by Ojeda, Failde, Dueñas, Salazar, & Eccleston, 2016), I included a matched control group, and collated information about co-morbidities and medication use. I also tested for attentional bias in participants without pain and thus observed the variability in attentional bias in several groups of undergraduate students and a community sample, which was important for interpreting the results of Experiment 3 comparing performance by participants with, and without, chronic pain. Furthermore, in the course of conducting these experiments, improvements for the next experiment with participants in chronic pain were identified, such as using a task with reaction time as a dependent measure.

It is important not to forget though, that the main outcome of this study is that contrary to the deficit-view and the motivated attention hypothesis, participants experiencing chronic pain did not differ in performance from controls on measures of temporal attention (for pain-related and neutral stimuli) or on different aspects of attention (alerting, orienting, or executive control). Taking on board the lessons learnt from the series of experiments using RSVP, in the next chapter I describe a stronger test of the deficit-view and the motivated attention hypothesis.

Chapter 3

Parts of this chapter have been published on Open Science Framework as a registered report: Godfrey, H. K., & Grimshaw, G. M. (2016, August 25). *Attention to body-tissue threat in chronic pain: A pre-registered report*. Retrieved from <https://osf.io/2mw7x/>

The current experiment uses the theoretical perspectives provided by the deficit-view and the motivated attention hypothesis to make predictions about attentional biases and cognitive deficits in the same study, this time focusing on control of distraction. In Experiment 3 in Chapter 2, an attentional blink was induced in participants with, and without, chronic pain. Contrary to the deficit-view, the overall magnitude of the attentional blink did not differ between participants with, and without, chronic pain. Contrary to the motivated attention hypothesis, the patterns of reporting pain-related and neutral words were the same in participants with, and without, chronic pain, and overall there was a bias away from pain-related stimuli. Although these findings do not provide support for either the deficit-view or the motivated attention hypothesis, the target-reporting task with RSVP, like the Stroop and dot-probe tasks, may not be sensitive enough to detect reliable attentional biases to threat-relevant information, and therefore not able to reveal larger biases in people who have chronic pain (as in Asmundson et al., 2005). Therefore, the deficit-view and the motivated attention hypothesis will be tested again with a spatial attention task that shows strong emotional modulation – the emotional distraction paradigm.

In the version of the paradigm presented here, the task involves presentation of intact and scrambled task-irrelevant images presented centrally, to maximally capture attention. Concurrent with presentation of the image, letters are presented. Participants indicate the identity of a target letter. Distraction is inferred by relative slowing on the identity task on intact as compared to scrambled image trials. The nature of the images in previous studies of emotional distraction (Grimshaw et al., 2017; Gupta et al., 2016; Levy-Gigi et al., 2016) has been neutral (images depicting people in every day scenarios), negative (images depicting body mutilations, sadness, disgust, threat, fear; angry faces; faces associated with loss in a gambling paradigm), or positive (images depicting erotica; happy faces; faces associated with winning in a gambling paradigm). Distraction is greater for both negative and positive images than neutral images, when the valenced images are matched on arousal (Grimshaw et al., 2017; Gupta et al., 2016). The magnitude of emotional distraction can be modulated by varying the proportion of trials on which intact images occur. When they are relatively rare (appearing on 25% of trials, as they do in this experiment) emotional images are much more

distracting than neutral images. Rare intact distractors encourage the use of reactive control (Braver, Grey, & Burgess, 2008; Geng, 2014), a set of attentional mechanisms that are initiated when conflict is detected, signaling the need to shift cognitive capacity back to task-relevant stimuli. The emotional distraction task with rare distractors thus gives a measure of the extent to which the distracting information competes with task-relevant information (Desimone & Duncan, 1995).

Previous research (Grimshaw et al., 2017; Gupta et al., 2016) has used high arousal negative images, which were generally mutilations of body-tissue. In the current experiment, distraction for neutral images is compared to distraction for images that all depict damage to body-tissue. These body-threat images should be especially relevant for participants with chronic pain, as pain is an experience generated when threat to body-tissue is detected by a threat protection system (Moseley, 2007).

In chronic pain experience, a deficit account predicts that attentional capacity is taken up with the pain experience, and current task performance is impaired. Thus, when rare intact images are presented, participants with chronic pain will find it harder to disengage from the image and therefore demonstrate more distraction overall (for both body-threat and neutral images) than controls. According to a motivated attention view, people with chronic pain will have a strong attentional bias that is driven by their overactive threat protection system. That is, their attentional system will be on alert for threatening information (especially body-threat information) taking attentional capacity away from task-relevant neutral information. Motivated attention would result in greater attentional capture by body-threat images than by neutral images in participants with chronic pain, compared to controls, leading to a specific impairment in control of distraction for body-threat images in people with chronic pain. Furthermore, in chronic pain experience the magnitude of the attentional bias in distraction will be positively correlated with the magnitude of the cognitive deficits (performance on the attention network task). An alternative outcome that would provide support for both the deficit-view and the motivated attention hypothesis is that in participants with chronic pain, there will be larger overall distraction for both body-threat and neutral images, and a larger attentional bias towards body-threat information.

The study design was published on Open Science Framework as a registered report on 25 August 2016. Data collection started in September 2016 and finished in May 2017.

Method

Participants

As described in the preregistration, I first attempted to recruit participants with chronic pain and controls who had not participated in previous RSVP experiments. Once I had exhausted the new participant pool, I expanded recruitment to those who participated in RSVP experiments, as the tasks are very different. At least six months had passed between participation in the two studies. I recruited 77 participants with chronic pain and 72 controls. As indicated in the pre-registration, participants were excluded from the analyses if they scored less than 75 percent correct overall on the letter identification task. It is important to note from a deficit perspective that only participants with chronic pain ($n = 5$) did not reach this performance criteria. This difference in the proportion of participants meeting the performance criteria is significant, $z = 2.00$, $\alpha < .05$.

A distraction task that used the same images as the current study was run with a different group of participants (Victoria University of Wellington undergraduate psychology students, participating in an introduction to psychology research programme; $n = 24$) as a pilot experiment. Mean distraction (RT intact – RT scrambled image) was 83 ms ($SD = 93$ ms) for body-threat images and 32 ms ($SD = 62$ ms) for neutral images, $t(23) = 2.175$, $p = .040$, $d_z = 0.437$. The difference between distraction indices for body-threat threat and neutral images was 52 ms ($SD = 116$ ms). The task therefore shows a robust attentional bias to body-threat information in this undergraduate population.

It was difficult to predict the effect size for the potential difference in distraction between the groups with, and without, chronic pain in order to do a sample size estimation. Effect sizes from meta-analyses of cognitive deficits (Berryman et al., 2014) and attentional biases (Schoth et al., 2012) in chronic pain experience were used as approximations for the effect size for the group difference (between chronic pain and controls). DeFife's (2009) conversions were used to calculate Cohen's d from Hedges' g . Berryman and colleagues (2014) reported a group difference of $d = 0.31$ for response inhibition, and of $d = 0.57$ for set shifting. Schoth and colleagues (2012) reported a group difference of $d = 0.45$ for pain-related attentional bias, and of $d = 0.38$ for an initial orienting bias. The average of these group effects is 0.43, which equates to approximately an additional 50 ms of distraction in the group with chronic pain (mean body-threat – neutral distraction difference of 102 ms) compared to the control group (mean body-threat – neutral distraction difference of 52 ms).

Sample size estimation calculations (using G*Power 3: Faul et al., 2007), to detect an independent groups difference of $d = 0.43$ with a one-tailed test with 80% power, as stated in the preregistration, suggested 69 participants per group. As there were four counterbalances in the design, 72 participants with chronic pain and 72 matched controls were needed to complete the counterbalance. The same exclusion criteria applied as for Experiment 3 in Chapter 2, and these criteria were stated in the preregistration. In addition, all participants indicated that they were willing to view gory images (such as might be seen in an R18 movie).

The 72 participants with chronic pain included in the analyses were 59 women and 13 men with a mean age of 37 years (18 – 73 years, $SD = 14$ years). The control participants were 55 women and 17 men with a mean age of 35 years (18 – 73 years, $SD = 13$ years). The groups did not differ significantly on sex, $\chi^2(1) = .674, p = .412, \Phi = .068$, age distribution $t(142) = .901, p = .369, d = 0.149$, or education distribution, $\chi^2(3) = 2.350, p = .503, \Phi = .128$. The groups were also balanced exactly for whether individual participants had previously completed an RSVP task ($n = 16$ in each group).

At the time they completed the study, participants with chronic pain had experienced pain for at least five months. As in Experiment 3, Chapter 2, these participants were heterogeneous in cause of chronic pain, duration of chronic pain, and co-morbidities. See Appendix C for a summary of the duration and cause information given by the 72 participants with chronic pain. Sixty-eight participants provided numerical duration information. The average was 11 years ($SD = 9$ years, range 5 months – 35 years). Twenty-four participants reported having a current injury causing pain. Nineteen reported current treatment for depression and / or anxiety, 10 reported receiving previous treatment for depression and / or anxiety, and 10 reported both having received in the past and currently receiving treatment for depression and / or anxiety. Six participants reported having a neurological condition. Ten reported experiencing chronic fatigue. Forty-two participants were taking medication for their pain, depression and / or anxiety. For the 42 participants taking medication the average number of medications was three ($SD = 2$, range 1 – 11).

Of the 72 control participants, two identified having a current injury causing pain, eight identified receiving previous treatment for depression and / or anxiety, three identified receiving current treatment for depression and / or anxiety, two identified both having received in the past and were currently receiving treatment for depression and / or anxiety, two reported having a neurological condition, and one reported experiencing chronic fatigue.

To address the co-morbidity and group difference issues, if there are group differences in the amount of distraction by body-threat and neutral images, then depression, anxiety, and stress (assessed via questionnaire measures), co-morbidities, and number of medications will be used as covariates.

Stimuli, Apparatus, & Procedure

The experiments were run on a Dell PC running Psychology Software Tools' E-Prime Suite version 2.0 (Psychology Software Tools, Pittsburgh, PA) on a 527 by 297 cm Alienware screen (1920 x 1080 pixels) with a vertical refresh rate of 120 Hz. Participants were seated approximately 60 cm from the screen. No chin rest was used, in order to minimise the discomfort experienced by the participants with chronic pain. Accuracy and response time (RT) were recorded by E-Prime.

Participants completed the experiment individually. They first completed the emotional distraction task, then the short version of the ANT (Fan et al., 2002), and the questionnaires last. Participation took approximately 1 hour.

As in Experiment 3, Chapter 2, I facilitated all participant recruitment and scheduling of appointments. Also, all participants with chronic pain were offered a taxi chit for getting to their next destination after the experiment; and I organised the taxis. Therefore, it was not possible for me, also the experimenter, to be blind to which participants had chronic pain and who were the controls. I had step-by-step prompts, which I followed to explain the tasks, and I used these written prompts for all participants. While it is not ideal that I was not blind to group membership, I controlled for this as much as possible with the script.

Emotional distraction task.

The stimuli, apparatus, and procedure are as described in the preregistration.

Stimuli & Apparatus. The background was black. The images were all centrally presented 396 x 298 pixels (10.5 cm x 7.8 cm) in the display. The letters appeared in six positions, three immediately above and three immediately below the image, spaced equidistantly. The letters were 31 x 33 pixels (O = 0.8 cm x 0.8 cm; K and N = 0.8 cm x 0.6 cm), size 26, white Arial font (see Figure 10). K or N appeared equally often at each of the positions.



Figure 10. An example of an intact image and letter display⁹.

The participants task was to indicate whether the target letter was a K or an N. A K or N was always present.

The 12 intact images in each category were selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008), the Nencki Affective Picture System (NAPS; Marchewka, Żurawski, Jednoróg, & Grabowska, 2014) and the Price, Dieckman, and Harmon-Jones (2012) database. All the images were selected from a subset of images that were rated for how much they depict pain and harm, and their valence and arousal level. In the ratings experiment, 81 participants rated the images (as part of a wider ratings experiment) on four dimensions: valence, arousal, pain, and harm. Valence was rated on 9-point self-assessment manikins with the left most anchor described by the terms: happy, pleased, satisfied, contented, hopeful; and with the right most anchor described by the terms: unhappy, annoyed, unsatisfied, melancholic, despaired. Arousal was rated on 9-point self-assessment manikins with the left most anchor described by the terms: stimulated, excited, frenzied, jittery, wide awake, aroused; and with the right most anchor described by the terms: relaxed, calm, sluggish, dull, sleepy, unaroused. Pain was rated on a 9-point scale (does this

⁹ For copyright reasons, an example of an image used in the experiment could not be used. This image was retrieved from <http://janetroper.com/dancing-with-your-intuition/> on 17 August 2016.

picture depict pain: 1 = not at all, 9 = very much), as was harm (does this picture depict harm: 1 = not at all, 9 = very much).

All neutral images depicted two people completing day-to-day activities to control for body presence and interest in the image (from Price et al. 2012: neutperson43, neutperson3, neutperson12, neutperson22, neutperson8, neutperson61, neutperson59, neutperson52, neutperson13, neutperson46, neutperson40; from IAPS: 2579). These neutral images form a more cohesive category than the neutral words used as stimuli in Chapter 2.

The body-threat images (from NAPS: People_211_v, People_222_h, Faces_364_v; from IAPS: 3131, 3150, 3064, 3000, 3080, 3053, 3063, 3069, 3071) depicted mutilations and were rated higher on depicting pain and harm than the neutral images. The body-threat images were rated higher in arousal and lower on valence than the neutral pictures. See Table 9 for the ratings data. All comparisons were significant at $p < .001$, with effect sizes of at least 9 (Cohen's d). The intact images were matched for luminance and reduced to 40% contrast using the SHINE Toolbox (MATLAB; Willenbockel, Sadr, Fiset, Horne, Gosselin, & Tanaka, 2010).

Table 9. *Image ratings on 9-point scales (n = 81).*

	Valence (range)	Arousal (range)	Pain (range)	Harm (range)
	<i>M (SD)</i>			
Neutral	5.50 (0.56)	2.21 (0.48)	1.11 (0.04)	1.10 (0.04)
Body-threat	1.58 (0.16)	7.41 (0.30)	8.32 (0.16)	7.74 (0.20)

Scrambled images were created from the intact images. The luminance and contrast matched intact images were each cut into 36 x 36 segments and scrambled using PhotoScape v 3.7. Scrambled images of each type were included in the block of that type (i.e., scrambled body-threat images appeared in the same block as intact body-threat images).

Procedure. See Figure 11 for a summary of the trial procedure. Each trial began with a fixation cross. The duration of the fixation was a random function of the refresh rate (120 Hz), ranging between 417 ms and 813 ms. The distractor and letter target display were presented for 200 ms; participants then had up to 1800 ms to respond. Visual feedback

(correct / incorrect, presented for 100 ms) was given only on practice trials. The length of the inter-trial-interval (ITI) was a random function of the refresh rate, and ranged between 207 ms and 623 ms (depending on the length of the fixation at the start of the trial).

Participants were instructed to focus on the fixation cross and that, in order to complete the task, they should ignore the intact and scrambled images that appear. The task was to press the 1 or 2 key, on the number pad of a keyboard, with their middle or index finger of their dominant hand for a K or N target letter on every trial, as quickly as possible without making mistakes. Assignment of response keys was counterbalanced across participants. Six participants with chronic pain and eight control participants were left handed but elected to use their right fingers to respond. One participant with chronic pain had an injured index finger so used their middle and ring finger to respond.

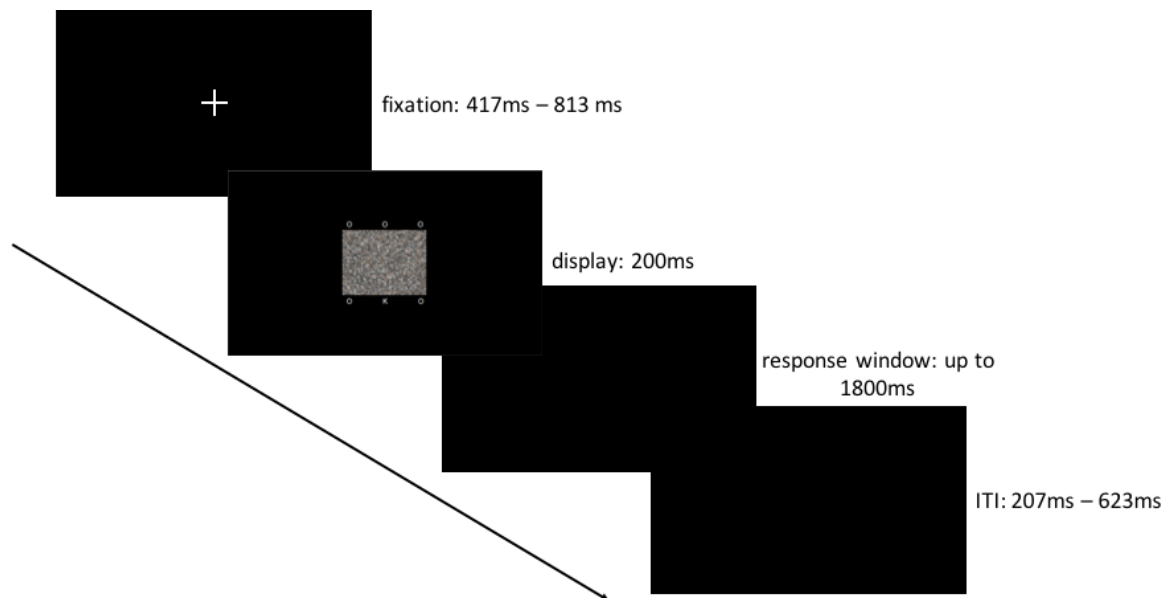


Figure 11. Trial procedure for emotional distraction task.

Participants completed 24 practice trials (with six intact neutral images not used in the experimental trials: luminance and contrast matched for just the six pictures, scrambles made as for the experiment scrambles) with a reminder of the instructions after 12 trials. Then participants completed four blocks of 48 trials each, with a self-paced break in between each block. Seven participants with chronic pain and two control participants did not understand the task on first presentation and so completed an extra block of practice trials.

Image type (body-threat or neutral) was blocked to maximally engage the threat protection system and presented in an alternating, counterbalanced order, repeated to make

four blocks. Within each block, intact images were randomly presented on 25% of trials, and scrambles on 75%.

ANT.

The ANT stimuli and procedure were identical to those used Experiment 3 in Chapter 2, and are as described in the preregistration. One participant used their middle fingers (rather than index fingers) to respond.

Questionnaires.

The questionnaires were identical to those used in Experiment 3 in Chapter 2, and are as described in the preregistration. One participant was fatigued and filled out the questionnaires on paper instead of via Qualtrics.

Planned Analyses

As outlined in the registered report (Godfrey & Grimshaw, 2016) the following planned analyses were made before the start of data collection. Any changes to the preregistration are noted.

Emotional distraction task.

As set in the preregistration, accuracy for the KN task was checked and if participants' accuracy was above 75% (the level previously used in Dr. Gina Grimshaw's lab), they were included in the final analyses.

First, mean RTs for accurate responses (excluding responses < 200 ms as anticipatory) for each participant were entered into a 2 (group: chronic pain, control) x 2 (presentation type: intact, scrambled) x 2 (image type: direct-threat, neutral) mixed model ANOVA, with group as a between-subjects factor and presentation and image type as within-subjects factors. When there was a difference between groups in overall RT, corrected distraction indices ($[RT \text{ intact} - RT \text{ scrambled}] / RT \text{ scrambled}$ for each image type) were used in follow-up analyses. When there was no difference between groups uncorrected distraction indices ($[RT \text{ intact} - RT \text{ scrambled}]$ for each image type) were used.

Any interactions in the first ANOVA were followed up with additional ANOVAs. For example, if there was a group x presentation type x image type interaction, it would be followed up by conducting separate 2 (presentation) x 2 (image type) ANOVAs for each group. Significant main effects were followed up with paired- or independent-samples *t*-tests.

As a manipulation check, there should be a main effect of type. The body-threat images are rated as being higher in arousal, less pleasant, and as depicting more pain and harm than the neutral images. Therefore, participants should be more distracted by the body-threat images than the neutral images. If chronic pain is associated with deficits in attentional control, there should be a main effect of group. Participants experiencing chronic pain should be more distracted overall by the images than controls. That is, there should be a deficit in control of distraction. If the motivated attention hypothesis reflects processing in people experiencing chronic pain, then there should also be an interaction between image type and group. Participants experiencing chronic pain should show much greater distraction for the body-threat images compared to controls. There are two possibilities for observing motivated attention: motivated attention with, or without, a concurrent deficit in control of distraction. In both situations supporting the motivated attention hypothesis, the relative difference between distraction by neutral and body-threat images should be greater in the group with chronic pain than controls.

ANT.

In the preregistration, I said that accuracy for the ANT task will be screened for outliers, and any two standard deviations from the mean will not be included in the final analyses. As the ANT has a maximum response time of 1700 ms, the data were processed as in Experiment 3, Chapter 2, as median RTs. The alerting score was calculated as the median RT of the accurate trials with no cue minus the median RT of the accurate trials with a centre cue. The orienting score was calculated as the median of the accurate trials with a centre cue minus the median of the accurate trials with a location cue. The alerting and orienting scores for each participant were entered into an independent-samples *t*-test with group as the independent factor. The executive score was calculated as the median RT of the accurate incongruent trials minus the median RT of the accurate congruent trials. The executive score for each participant was entered into an independent-samples *t*-test with group as the independent factor. The deficit-view predicts that participants with chronic pain should have poorer efficiency in the attention networks than control participants.

As well as using the derived executive score, median correct RTs for each participant were entered into a 2 (group: chronic pain, control) x 2 (congruency: congruent, incongruent) mixed model ANOVA with group as a between-subjects factor and congruency as a within-subjects factor. Main effects were followed up by paired- or independent-samples *t*-tests. If there was a significant interaction, it would be followed up with one-tailed paired-samples *t*-

tests for each group. These tests allow comparison of the effect size for congruent and incongruent RTs for each group, expecting incongruent trials to be slower than congruent trials. The deficit-view predicts that participants with chronic pain will be impaired at restraining prepotent responses to incongruent stimuli and thus should show greater interference than controls. Note however, that in Experiment 3 in Chapter 2, the efficiency of the attention networks did not differ between participants with, and without, chronic pain. Psychomotor slowing was observed in Experiment 3 in Chapter 2 when the participants were split by current pain level.

Questionnaires.

The scale totals for the McGill Pain Questionnaire, the 21 item - DASS (the depression, anxiety, stress subscales), the ACS, and the PVAQ will be calculated for each participant. Only full responses were included in the final analyses. Independent-samples *t*-tests were conducted for all the subscales with group (chronic pain, control) as the independent-samples factor. If significant differences were observed between groups that were related to performance on the distraction or ANT task, those variables would serve as covariates in further analyses of the emotional distraction and ANT data.

It was expected that participants in chronic pain would score higher on the McGill Pain Questionnaire, the subscale scores of the DASS, and the PVAQ than controls. Participants in chronic pain may score lower on the ACS than controls.

Exploratory Analyses

Exploratory correlation analyses were conducted to examine the relationships between body-threat distraction, neutral distraction, and the relative amount of distraction for the two image types (attentional bias); self-reported current pain level (McGill Pain Questionnaire); self-reported depression, anxiety, and stress symptoms (DASS); self-reported attentional control (ACS); self-reported pain vigilance (PVAQ); and the alerting, orienting, and executive network scores (ANT-short version). If any of the questionnaire scores correlated significantly with distraction or the ANT networks, then the score would be used as a covariate in an ANCOVA and potentially as a mediator in a mediational analysis.

Based on the relationships observed in Experiment 3 in Chapter 2, significant positive correlations were expected between depression, anxiety, and stress scores, such that higher scores on one would be associated with higher scores on the others. Significant positive correlations were also expected between pain level and depression, anxiety, stress, and pain

vigilance, such that, higher pain scores would be associated with higher depression, anxiety, stress, and vigilance to pain. Significant negative correlations were expected between self-reported attentional control and pain, depression, anxiety, stress, and pain vigilance, such that higher pain, depression, anxiety, stress, and vigilance to pain would be associated with lower reported attentional control.

In the preregistration, I predicted that there would be a significant positive correlation between the executive score on the ANT and reported anxiety and stress. This prediction was based on an error in my original analyses of the results in Experiment 3, Chapter 2. In the re-conduction of the correlational analyses for Experiment 3 in Chapter 2, there were significant negative relationships between the magnitude of the attentional bias and the efficiency of the alerting network, and between reported stress and the efficiency of the executive network. There were no relationships between anxiety and the attention networks.

Data Processing

When sphericity was violated, the original degrees of freedom are reported, with the Greenhouse-Geisser corrected p and epsilon (ϵ). For independent t -tests, Levene's test for equal variances was used and when significant, the corrected t and df are reported. For independent t -tests, Cohen's d was calculated as a measure of effect size, and for dependent t -tests, d_z was calculated (Lakens, 2013). d_z is the mean difference divided by the standard deviation of the mean difference.

Results

Recruitment Validity: Questionnaires

Due to a computer error, one participant did not complete the PVAQ, and due to time restrictions one participant did not complete the ACS and PVAQ. The analyses including the ACS use data from 143 participants, and the PVAQ, 142 participants. See Table 10 for means and standard deviations by group.

Participants in the chronic pain group reported more current pain than control participants, $t(84.657) = 12.980$, $p < .001$, $d = 2.179$. Participants with chronic pain had more symptoms of depression, $t(108.578) = 4.940$, $p < .001$, $d = 0.829$, anxiety, $t(120.136) = 6.831$, $p < .001$, $d = 1.147$, and stress, $t(142) = 5.802$, $p < .001$, $d = 0.974$, than control participants. They also reported being more vigilant to changes in pain experience, $t(140) = 6.279$, $p < .001$, $d = 1.062$, and having less self-reported attentional control than control participants, $t(141) = 3.233$, $p = .002$, $d = 0.543$.

Table 10. *Questionnaire measures.*

	Chronic Pain	Control
	<i>M (SD)</i>	
McGill Pain Total**	51.81 (29.47)	4.58 (9.18)
DASS Depression**	10.06 (9.68)	3.67 (5.18)
DASS Anxiety**	9.81 (6.73)	3.39 (4.27)
DASS Stress**	15.28 (7.99)	8.00 (7.03)
ACS Total*	49.66 (9.01)	54.33 (8.31)
PVAQ Total**	43.73 (13.18)	29.68 (13.47)

** indicates a significant group difference $p < .001$. * = .002.

The self-reported questionnaire data show that the groups differ in experience. The group with chronic pain reported a higher current pain experience, more depression, anxiety, and stress, less attentional control, and more awareness of changes in pain than the control group. Only 16 participants from each group of 72 had participated in Experiment 3 in Chapter 2, meaning that these subjective increases in pain, depression, anxiety, and stress are likely present in more than these 16 individuals with chronic pain.

Distraction

Accuracy.

Accuracy for each participant (excluding trials on which participants responded in less than 200 ms; less than one percent of all trials) was entered into a 2 (group: chronic pain, control) x 2 (presentation Type: intact, scrambled) x 2 (image type: body-threat, neutral) mixed model ANOVA, with group as a between-subjects factor, and presentation and image type as within-subjects factors. See Table 11 and Figure 12 for accuracy by presentation and image type.

There was a main effect of presentation type, $F(1, 142) = 7.526, p = .007, \eta_p^2 = .050$, such that participants were slightly less accurate on the KN task when an intact image was presented ($M = 93.89\%$, $SD = 5.44\%$), than when a scrambled image was presented ($M = 94.82\%$, $SD = 4.30\%$). That is, participants were distracted by intact images. There were no other main effects or interactions in the accuracy data, reflecting the high accuracy (and thus low variance) in the accuracy data.

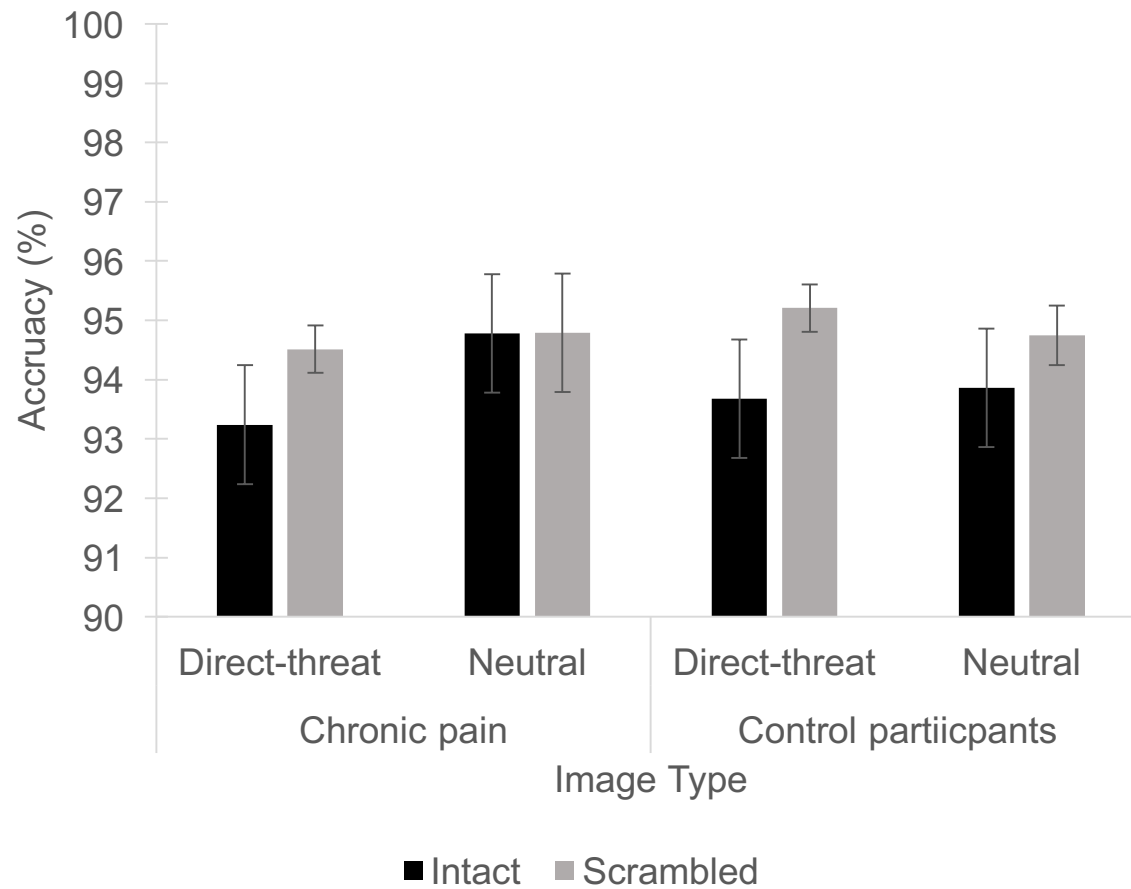


Figure 12. Accuracy for the KN task on trials containing intact and scrambled images in the body-threat and neutral blocks for participants with, and without, chronic pain. Error bars are within-subjects standard errors with Morey's (2008) correction.

Reaction time.

Mean reaction times for correct responses (excluding anticipatory responses of less than 200 ms) for each participant were entered into a 2 (group: chronic pain, control) x 2 (presentation Type: intact, scrambled) x 2 (image type: body-threat, neutral) mixed model ANOVA, with group as a between-subjects factor, and presentation and image type as within-subjects factors. See Table 11 and Figure 13 for the mean response time by presentation and image type. Six percent of the total data was excluded for being inaccurate responses or too fast. On average, each participant had four percent of their total trials excluded.

There were main effects of image type, $F(1, 142) = 44.676, p < .001, \eta_p^2 = .239$, and presentation type, $F(1, 142) = 39.801, p < .001, \eta_p^2 = .219$, qualified by an interaction between image type and presentation type, $F(1, 142) = 28.716, p < .001, \eta_p^2 = .168$. The interaction reflects a greater difference between RTs for intact and scrambled images (distraction), for body-threat images, $t(143) = 6.349, p < .001, d_z = 0.520$, than for neutral images, $t(143) = 2.863, p = .005, d_z = 0.243$. There was also a main effect of group, $F(1, 142) = 4.033, p = .047, \eta_p^2 = .028$, reflecting slower responding overall in the group with chronic pain ($M = 730$ ms, $SD = 118$ ms) compared to the control group ($M = 688$ ms, $SD = 133$ ms); evidence for psychomotor slowing in chronic pain experience. There was no interaction between group and image type, $F(1, 142) = .056, p = .813, \eta_p^2 < .001$, or between group and presentation type, $F(1, 142) = .329, p = .567, \eta_p^2 = .002$, nor a group x image type x presentation type interaction, $F(1, 142) = .043, p = .837, \eta_p^2 < .001$.

As there is a difference in RT between the two groups, corrected distraction indices were calculated. Corrected distraction provides an indication of the percentage of slowing on intact to scrambled trials. This correction allows for a clearer comparison between distraction by the two images types. A 2 (group: chronic pain, control) x 2 (image type: body-threat, neutral) mixed model ANOVA was run. There was a main effect of image type, $F(1, 142) = 30.220, p < .001, \eta_p^2 = .175$, such that participants were more distracted by body-threat ($M = 7.67\%$, $SD = 13.95\%$) than neutral images ($M = 1.49\%$, $SD = 5.68\%$), but no main effect of group, $F(1, 142) = .174, p = .677, \eta_p^2 = .001$, which is contrary to a

deficit-view – participants with chronic pain were not more distracted overall than control participants. There was no interaction between image type and group either, $F(1, 142) = .040$, $p = .842$, $\eta_p^2 < .001$, contrary to a motivated attention view. There are no interactions with group, whether I use corrected or uncorrected distraction indices (as indicated in the ANOVA above). There is no evidence that biased attention to threat underlies poorer control of distraction in people with chronic pain.

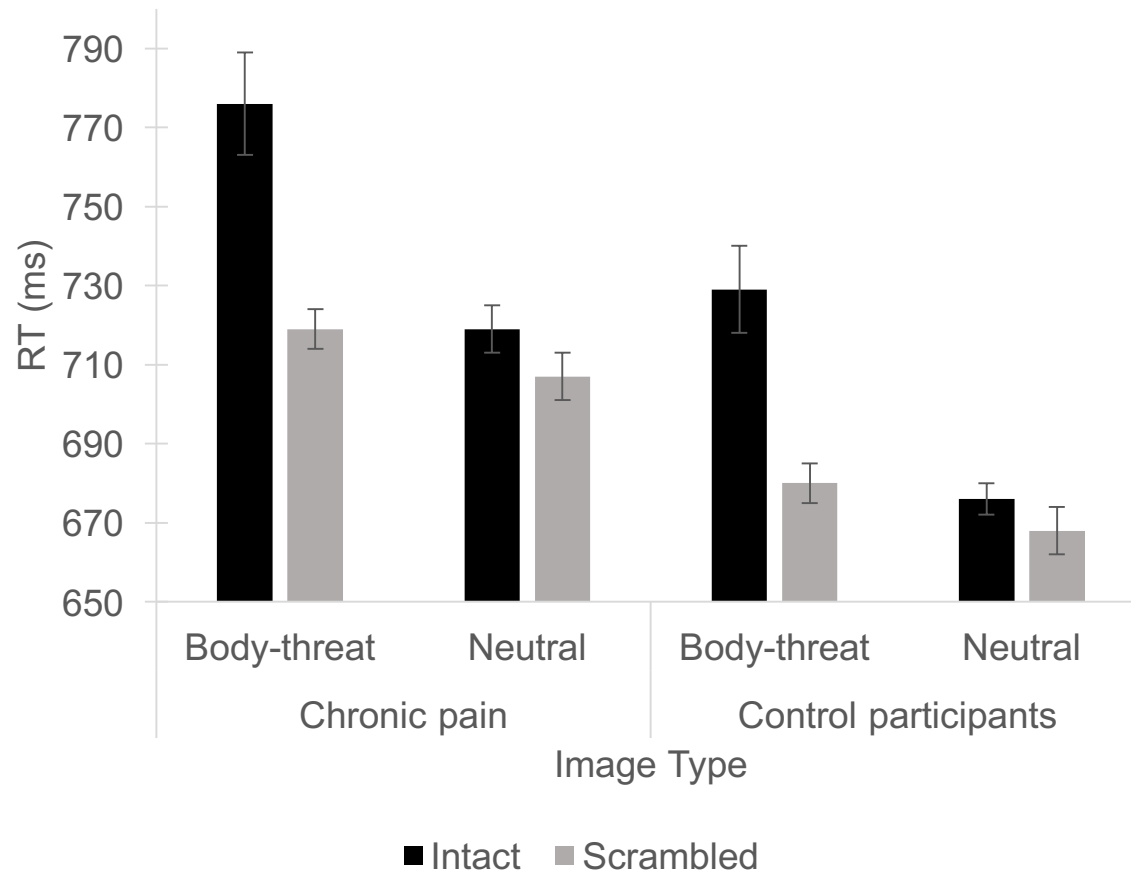


Figure 13. Reaction time for the KN task on trials containing intact and scrambled images in the body-threat and neutral blocks for participants with, and without, chronic pain. Error bars are within-subjects standard errors with Morey's (2008) correction.

Table 11. *Accuracy and RT by presentation type, image type and group.*

	Chronic Pain		Control	
	Body-threat	Neutral	Body-threat	Neutral
Accuracy <i>M (SD)</i>				
Intact %	93.24 (7.65)	94.78 (5.91)	93.68 (6.17)	93.86 (5.77)
Scrambled %	94.51 (4.49)	94.79 (4.97)	95.21 (4.32)	94.75 (5.13)
Distraction (uncorrected) %	1.28 (6.47)	.01 (5.15)	1.53 (5.02)	.89 (4.98)
Reaction Time <i>M (SD)</i>				
Intact ms	776 (154)	719 (124)	729 (169)	676 (127)
Scrambled ms	719 (121)	707 (114)	680 (135)	668 (126)
Distraction (corrected) %	8.07 (14.73)	1.66 (5.83)	7.27 (13.22)	1.31 (5.56)

Note. Uncorrected distraction indices are calculated as (intact –scrambled) for each image type / dependent variable. Corrected distraction indices are calculated as ([intact –scrambled] / RT scrambled) for each image type / dependent variable.

ANT

See Table 12 for mean network scores for participants with chronic pain and controls. Participants with chronic pain did not differ from controls on the ANT alerting, $t(109.35) = .170, p = .865, d = 0.028$, orienting, $t(142) = .899, p = .370, d = 0.150$, or executive scores, $t(130.200) = .178, p = .859, d = 0.030$.

Table 12. *Attention network scores for participants with, and without, chronic pain.*

	Chronic pain	Controls
	<i>M (SD)</i>	<i>M (SD)</i>
Alerting network	19 ms (41 ms)	19 ms (22 ms)
Orienting network	48 ms (31 ms)	44 ms (27 ms)
Executive network	93 ms (44 ms)	92 ms (32 ms)

To determine whether chronic pain experience was associated with psychomotor slowing, a 2 (congruency: congruent, incongruent) x 2 (group: chronic pain, control) mixed model ANOVA was conducted on the responses to all congruent and incongruent trials. There was a main effect of congruency, $F(1, 142) = 837.338, p < .001, \eta_p^2 = .855$, with participants responding as is expected in a flanker task: faster to congruent trials ($M = 532$ ms, $SD = 100$ ms) than incongruent trials ($M = 625$ ms, $SD = 113$ ms). There was also main effect of group, $F(1, 142) = 7.517, p = .007, \eta_p^2 = .050$, reflecting slower responding overall in the group with chronic pain ($M = 602$ ms, $SD = 117$ ms) compared to the control group ($M = 555$ ms, $SD = 86$ ms); evidence for psychomotor slowing on this task, as on the distraction task. There was no interaction between group and congruency, $F(1, 142) = .032, p = .859, \eta_p^2 < .001$; chronic pain was not associated with deficits in executive control.

Exploratory analyses**Current pain level.**

The participants in the two groups were clearly experiencing different levels of pain, depression, anxiety, and stress. Furthermore, the groups reported different levels of self-

reported attentional control and vigilance to pain. People with chronic pain feel like they are unable to concentrate, and they feel like they pay a lot of attention to their pain. However, in task performance, this perceived deficit only translates into psychomotor slowing. Control of distraction and the efficiency of the attention networks are not impacted by chronic pain experience. As in Chapter 2, Experiment 3, some of the participants with chronic pain mentioned to the researcher that they were not experiencing pain when they completed the tasks, and some of the control group reported that they were experiencing pain. In order to assess whether current pain is associated with performance, a median split was conducted on the McGill questionnaire total scores. Participants who scored 17 or less on the McGill total score were classified as low pain experiencers ($n = 71$), and those who scored 18 or greater as high pain experiencers ($n = 73$). Five participants with chronic pain were low pain experiencers, and six control participants were high pain experiencers. The same analyses as above were performed on distraction and ANT data.

For the distraction task, the patterns in the accuracy data were the same as when analysed by recruited group. For the most part the same patterns in the reaction time data were observed, except there was no main effect of group, $F(1, 142) = 2.629, p = .107$, $\eta_p^2 = .018$. When the reaction time analyses include current pain level (instead of chronic pain status) as the grouping factor, there is no evidence for psychomotor slowing. Thus, the ongoing nature of chronic pain, not the current intensity of pain, seems to contribute to psychomotor slowing in this task. However, for the ANT, psychomotor slowing was still observed when the data were analysed by current pain level. The patterns in the ANT networks were the same when analysed by current pain level as when analysed by recruited group.

Correlations.

First, correlations between performance on the distraction task (RT overall, uncorrected distraction indices for accuracy for body-threat and neutral images, corrected distraction indices for RT for body-threat and neutral images, and the relative amount of distraction for the corrected RT indices: corrected body-threat distraction – corrected neutral distraction) and the ANT (RT overall, alerting, orienting, and executive networks) were examined. There was a significant, positive relationship between RT on the distraction task and ANT, $r(142) = .592, p < .001$, such that the slower participants were on one task, the slower they were on the other, providing converging evidence for psychomotor slowing.

There were no other significant relationships between performance on the distraction task and ANT.

Second, exploratory correlations between performance on the distraction task and ANT, and the questionnaire measures (McGill total; DASS depression, anxiety, and stress; ACS; and PVAQ) were examined. Pain level was significantly, positively related to overall RT on the distraction task, $r(142) = .196, p = .018$, and the ANT, $r(142) = .232, p = .005$, such that higher pain was associated with slower RT. Interestingly, overall RT on the distraction task and ANT were not related to depression, anxiety or stress, suggesting a unique contribution of pain to psychomotor slowing on these tasks. Also, the faster participants were to respond on the ANT, the more biased they were to body-threat information (relative to neutral information), $r(142) = -.189, p = .023$. There were no other significant relationships between performance on the distraction task and ANT, and the questionnaire measures. Unlike in Experiment 3, Chapter 2, there was no relationship between stress and the executive network, $r(142) = .047, p = .579$. As there was no difference between performance in the groups with, and without, chronic pain for distraction or the ANT networks, no ANCOVAs on distraction or the ANT networks were conducted.

Third, exploratory correlations between the different questionnaire scales (McGill, DASS, ACS, and PVAQ) were examined, see Table 13. Pain, depression, anxiety, and stress were positively related. As one factor increases so do the others which reflects the co-morbidity of these symptoms. The higher current pain was, the more depression, $r(142) = .404, p < .001$, anxiety, $r(142) = .486, p < .001$, and stress symptoms, $r(142) = .411, p < .001$, were reported. As in Experiment 3, Chapter 2, the higher current pain, the less attentional control participants reported, $r(141) = -.234, p = .005$, and the higher current pain, the greater vigilance to pain was, $r(140) = .436, p < .001$. Attentional control and vigilance to pain were themselves negatively related, $r(140) = -.232, p = .005$, such that the greater vigilance to pain reported, the less attentional control reported. The higher current depression, anxiety, and stress, the less attentional control and the greater vigilance to pain.

Table 13. *Correlations between questionnaire measures.*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Corrected distraction – body-threat RT	-	.289*	.915**	.163	.022	.011	-.060	-.161	-.019	-.033	.029	.112	-.130	-.075
2. Corrected distraction – neutral RT		-	-.123	.130	.023	-.139	.055	.054	-.078	.036	-.035	.065	.049	-.102
3. Corrected Distraction – bias RT			-	.114	.013	.070	-.085	-.189*	.013	-.050	.045	.088	-.156	-.034
4. Distraction RT				-	.063	.114	.075	.592**	.196*	.007	-.013	.011	.036	.053
5. ANT alerting					-	-.154	-.173*	.210*	-.010	.065	.090	.052	-.158	.051
6. ANT orienting						-	-.067	.077	.161	.062	.007	.066	.037	-.054
7. ANT executive							-	.343**	.034	.105	-.013	.047	.033	-.002
8. ANT RT								-	.232**	.142	.036	.087	.007	.045
9. McGill Pain Total									-	.404**	.486**	.411**	-.234**	.436**
10. D-Depression										-	.583**	.663**	-.382**	.204*
11. D-Anxiety											-	.695**	-.375**	.322**
12. D-Stress												-	-.441**	.262**
13. ACS Total													-	-.232**
14. PVAQ														-

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

Note. D = DASS: Depression, Anxiety, Stress Scale.

Discussion

The purpose of the current experiment was to test both the deficit-view and the motivated attention hypothesis of attentional deficits in chronic pain experience. The two groups of participants completed an emotional distraction task to assess attentional bias to body-threat information, the ANT to assess the functioning of the attentional networks, and questionnaires assessing self-reported current pain, depression, anxiety, stress, attentional control, and attention to pain. The group with chronic pain reported higher current pain, depression, anxiety, and stress. Yet the differences in these experiences only translated to psychomotor slowing on the behavioural tasks, and poorer self-reported attentional control and greater vigilance to pain, in chronic pain experience. Findings do not provide evidence for either the deficit-view, beyond psychomotor slowing, or the motivated attention view of cognition in chronic pain experience.

The deficit-view of attention in chronic pain is that because there is competition for cognitive capacity, greater attention to pain experience means impaired, or interrupted, attention to the environment or to the current task. If the deficit-view is descriptive of processing, participants with chronic pain will show cognitive deficits, compared to performance of participants without chronic pain. Participants with chronic pain did report poorer attentional control, and greater attention to changes in pain experience, than control participants. So, participants with chronic pain do consider their attention to be impaired. Furthermore, participants with chronic pain were slower to respond on the distraction and ANT tasks; evidence for psychomotor slowing. However, the patterns of distraction in the emotional distraction task, and the efficiency of the attention networks in the ANT task, were no different between participants with chronic pain and control participants; evidence against the deficit-view of attention in chronic pain.

The motivated attention hypothesis of attention in chronic pain is that in chronic pain experience, protective processing still drives patterns of attention. That is, threat protection motivates assignment of attentional capacity towards information and behaviours most adaptive for survival, such as signals of threat to bodily tissue, signals of pain, or signals of relief of pain; and away from other kinds of information and behaviours. If motivated attention is descriptive of processing, participants with chronic pain will show a larger attentional bias to pain-related stimuli compared to any bias observed in participants without chronic pain. Also, in chronic pain experience, the magnitude of the attentional bias in distraction will be positively correlated with the magnitude of the cognitive deficits in the

ANT. Participants in both groups were more distracted by body-threat than neutral images. Participants were slower to respond K or N in the presence of a body-threat image than a neutral image. However, this attentional bias towards body threat information did not differ in magnitude between those with, and those without, chronic pain. There was no relationship between the attentional bias in distraction and performance on the ANT.

The same pattern of behavioural results was observed when analysing the data by current pain level rather than recruited group. Furthermore, the only relationships between performance on the behavioural tasks (both distraction and ANT) and the questionnaires indexing current experience were between current pain experience and overall RT on the distraction task, and between current pain experience and overall RT on the ANT. The psychomotor slowing seems to be specifically linked to current pain such that the more current pain experienced, the greater the slowing.

The magnitude of distraction observed was small compared to the pilot experiment in which distraction of 83 ms was observed for body-threat images and 32 ms for neutral images. In the main experiment, across the two groups, corrected distraction of 7.67% was observed for body-threat images and 1.49% for neutral images. These corrected indices translate to 53 ms distraction for body-threat and 10 ms for neutral images. Distraction was much smaller in the experiment with community participants (both participants with, and without, chronic pain) than in the experiment with first year undergraduate students. The pilot and experiment were run under the same conditions, with participants completing the protocol individually. However, it may be that participants recruited via the community are more invested in participation than those recruited from an undergraduate programme. Other research shows that the magnitude of distraction can be modulated by motivation, for example by providing financial incentives for fast and accurate performance (Walsh, Harper, Carmel, & Grimshaw, 2017). Regardless of the cause, the pilot experiment demonstrates that distraction can be much greater than observed in the current experiment. As distraction can be greater, if there was a cognitive deficit, or motivated attention, in distraction, it could have been observed with the current task.

There were many strengths in this design for testing for evidence of motivated attention and cognitive deficits. First, analyses of the experiments in Chapter 2 suggested that activation of the threat-protection system may be a necessary condition for observing motivated attention in chronic pain experience. I chose task parameters for the current experiment that were most likely to reveal a difference between groups, should one exist: image type was presented in blocks rather than randomly, and cohesive sets of stimuli were

used. Second, I based my sample size on a pilot study and previous studies of the effect size of cognitive deficits in chronic pain experience. I recruited 72 participants in each group, which should have been enough for me to observe differences in attention, if they were there.

Third, the two groups were carefully balanced on age, sex, and education level. Furthermore, the groups had the same number of participants who had previously participated in one of the other studies reported in this thesis. These factors should have therefore not impacted observing deficits or motivated attentional effects. The groups also differed in key ways which indicate that the group with chronic pain was experiencing maladaptive outcomes from their ongoing pain. The group with chronic pain reported experiencing higher levels of pain; more symptoms of depression, anxiety, and stress; poorer attentional control; and greater vigilance to changes in pain than the control group. Importantly, pain level was related to response speed, with the group with chronic pain showing psychomotor slowing. However, in this context, there was no evidence for motivated attention or cognitive deficits in attention. In two experiments now, I have not observed cognitive deficits in attention, or motivated attention.

Despite these strengths, the experiment was affected by some practical limitations and design choices. First, as in Experiment 3 in Chapter 2, I was limited by the population of the local area to recruiting a heterogeneous group of participants with chronic pain. As discussed, in a small city, wide recruitment was necessary to find participants experiencing chronic pain. However, chronic pain encompasses many experiences. The individuals differed widely in cause, duration, and severity of their pain, and number of medications. Deficits in attention in the current experiment were not observed, but it cannot be ruled out that the noise in the data – due to the wide variation in experience – is masking observation. Also, as noted in for the experiment in which participants with chronic pain completed the RSVP task, in this experiment, only participants with chronic pain did not meet the performance criteria for the emotional distraction task. The performance criteria may well have led to the exclusion of participants who most suffer from attentional deficits. I suggested in Chapter 2 that using a reaction time task may allow clearer conclusions to be made about cognitive functioning in chronic pain experience as more data could be preserved. Using a task with RT as the dependent variable did reduce the number of participants excluded from the final analyses from 13 (across participants with chronic pain and controls) to five (with chronic pain). It is likely that I was able to include data from more participants in the current study using RT tasks, than if I had used an accuracy task.

Second, I may not have observed evidence for deficits or motivated attention in the chronic pain group because I used attention to other people's body-tissue as the behavioural indicator of attentional bias. As threat protection is about preventing threat to your body-tissue (Moseley, 2007), it may be that threat to your own body-tissue is needed to observe reliable differences in attention in chronic pain experience. Furthermore, the images were all of acute injuries that differed widely in location of the damage on the body. It may be that threat to the area in the individual's body that is perceived as vulnerable is necessary to observe reliable differences in attention in people with chronic pain. In future tests of mechanisms that underlie the cognitive deficits observed in chronic pain experience, it will be important to make hypotheses about specific pain conditions and specific stimuli. For example, in a test of the specificity of motivated attention, the design in the current chapter could be repeated with participants only experiencing lower back pain, with images of damage to back tissue or the spine as distractions.

Third, perhaps by focusing on distraction with an intact image probability of 25% (prompting reactive control), evidence for motivated attention in proactive control was missed. Under reactive control conditions, participants must engage control after distraction occurs, for example by disengaging attention from the distractor (Braver et al., 2008; Braver, 2012; Geng, 2014). Reactive control is generally at play when there is a low proportion of distractors, as it is not an efficient use of cognitive capacity to use a more effortful control strategy. Under proactive control conditions, distraction is controlled before presentation of a distractor (Braver et al., 2008; Braver, 2012; Geng, 2014). Proactive control is an effortful mechanism which is useful when there is a high proportion of distractors, as it is an efficient use of cognitive capacity to use a more effortful control strategy.

In this experiment, intact distractors were relatively rare (present on 25% of trials) and it is likely that reactive control was used to direct attention away from the central distractor to the peripheral letters. Twenty-five percent distractor presence was chosen as distraction is larger than for higher distractor frequencies (Grimshaw et al., 2017). Greater distraction allows for movement in distraction up or down, important when testing for differences in distraction between participants with, and without, chronic pain. However, in selecting a design likely to evoke reactive control, any cognitive deficits or motivated attention in proactive control of distraction was not tested.

Proactive control is a process key to day-to-day functioning which may be changed in depression and anxiety (Braver et al., 2008; Braver, 2012; Grimshaw et al., 2017; Pessoa, 2009). If chronic pain is similar to depression and anxiety (in being a maladaptive form of an

adaptive process; Swanson, 1984), chronic pain may also involve an impairment of proactive control. Proactive control, unlike reactive control, is more taxing and thus requires more cognitive capacity than reactive control (Braver, 2012). Being a process which takes a lot of cognitive capacity, proactive control may be more impaired or subject to bias (than reactive control) when functional and structural changes have occurred in chronic pain experience. Although no other research has looked at impairment of reactive or proactive control of emotional (or neutral) distraction in chronic pain, age does impact control of distraction. Most studies show that proactive control is impaired in older adults but reactive control is not (e.g., Braver, Satpute, Rush, Racine, & Barch, 2005; Paxton, Barch, Racine, & Braver, 2008) – a finding which corresponds with the theory that proactive control takes more capacity than reactive control, and in older adults cognitive capacity is reduced. However, in one report reactive control was impaired in older adults but proactive control was not (Xiang, Zhang, Wang, Jiang, Zhang, & Hu, 2016). Future research should directly compare reactive and proactive control mechanisms in chronic pain experience for neutral and pain-related stimuli.

There are still more questions to answer about control of distraction in chronic pain experience. Here in the strongest test of the deficit-view and motivated attention hypothesis in this thesis, psychomotor slowing was present in participants with chronic pain, and there were self-reported deficits in attentional control, and greater attention to changes in pain in participants with chronic pain. However, there were no deficits in control of distraction or efficiency of the attention networks, and no evidence for motivated attention was observed.

Chapter 4

People with chronic pain report difficulties in thinking, and they show impairments in performance on tasks assessing cognitive functioning. It is not yet clear what cognitive changes underlie these deficits in functioning. The deficit-view of the impairments in chronic pain describes a pattern of processing in which attention is directed towards the pain experience, and pain interrupts the current task (Crombez et al., 1996; Eccleston & Crombez, 1999). In an alternative view, motivated attention describes a pattern of processing in which attention is directed towards information in the environment that is related to current concerns, and away from information that is not (Frankenhuis & de Weerth, 2013; Frankenhuis et al., 2016). Motivated attention had not been applied to understanding cognition in chronic pain before this thesis. In chronic pain experience, motivated attention describes a pattern of processing in which attention is directed towards information in the environment that is related to pain or body-tissue threat, and away from neutral information (similar to Van Damme et al., 2010). This allocation of capacity would result in deficits on tasks with neutral stimuli and enhanced processing of pain-related or threat-related stimuli. These two hypotheses are not mutually exclusive. For example, an individual could have overall deficits in attentional capacity (perhaps through atrophy of the frontoparietal network) but have remaining capacity heavily biased to pain-relevant information.

Attentional biases towards pain-related or threatening stimuli have been reported in people with chronic pain (e.g., Asmundson et al., 2005; Crombez et al., 2000; Dehghani, et al., 2003; Duschek et al., 2014). An attentional bias to pain-related stimuli is a necessary feature of the motivated attention hypothesis, but not of the deficit-view; as in the deficit-view attention to pain experience interrupts current task performance, whereas in the motivated attention hypothesis, an attentional bias directs attention to pain-related or neutral information. Therefore, testing predictions derived from the deficit-view and the motivated attention hypothesis in the same experiments was a useful research direction to take to advance understanding of the underlying cognitive mechanisms by which chronic pain impairs functioning. I focused on the attentional processes of temporal attention and attentional control of distraction, as well as the alerting, orienting, and executive attention networks. The hypothesis derived from the deficit-view, was that because there is competition for cognitive capacity, greater attention to the pain experience means impaired, or interrupted, attention to the environment or to the current task. This allocation of capacity

contributes to production of the deficits that are observed in chronic pain. Therefore, participants with chronic pain should show deficits compared to matched controls.

The motivated attention hypothesis holds that even in chronic pain experience, protective processing still drives patterns of attention. That is, threat protection motivates assignment of attentional capacity towards information and behaviours that are most adaptive for survival, such as signals of threat to bodily tissue, signals of pain, or signals of relief of pain; and away from other kinds of information and behaviours. Therefore, participants with chronic pain should show a greater attentional bias to stimuli that are pain-related or that depict threat to body-tissue compared to any bias observed in controls. Also in chronic pain experience, the magnitude of the attentional bias should be related to the magnitude of the cognitive deficits in the attention networks. As the deficit-view and motivated attention hypothesis are not mutually exclusive, another potential outcome was observation of both overall deficits in temporal attention and control of distraction, and enhanced attentional biases to pain-related than neutral information in chronic pain experience.

Major Findings

Cognitive deficits in temporal attention (as assessed with the attentional blink in Chapter 2), distraction (as assessed with the emotional distraction paradigm in Chapter 3), and the attention networks (as assessed with the ANT in Chapters 2 and 3) were not observed in people with chronic pain, nor was there any support for the motivated attention hypothesis. Indeed, the observations in Chapters 2 and 3 suggest that cognitive deficits in people with chronic pain are not ubiquitous, nor are patterns of attentional biases that differ from those in people without chronic pain.

Although these experiments provide no evidence for attentional deficits or motivated attention in people with chronic pain, they do show that chronic pain is associated with subjective concerns about attentional processing; in attentional control (as assessed with the Attentional Control Scale) and awareness of changes in pain experience (as assessed with the Pain Vigilance and Awareness Questionnaire). In participants with chronic pain, self-reported attentional control is poorer, and vigilance to pain is greater, than in control participants. These two domains of self-reported attention were also related to each other in the full participant sample (combining participants with chronic pain and controls). The poorer self-reported attentional control was, the greater self-reported vigilance to changes in pain. Furthermore, participants with chronic pain did show one cognitive deficit not specifically

related to temporal attention or control of distraction; psychomotor slowing on the distraction task and the ANT.

The aim of the experiments in Chapter 2 was twofold. The first goal was to test for the existence of an attentional bias to pain-related information in undergraduate participants. The second goal was to test for deficits and motivated attention in participants with chronic pain using RSVP. In Experiments 1, 2, and 4, I examined performance by participants without chronic pain. In Experiment 3, I compared performance in participants with, to those without, chronic pain. The focus of all experiments was on temporal attention, but in Experiment 3, I also assessed the efficiency of the attentional networks (alerting, orienting, and executive), and self-reported attentional control and vigilance to pain.

The stimuli were two coloured target words embedded in a rapid stream of black neutral words. The participants' task was to report the two targets. Reporting of the second target drops when the two targets appear close together in time, and recovers as the time between the two targets is extended. The magnitude of this attentional blink can be shifted by changing the nature of the stimuli used at target 1 and target 2. In Experiment 1, the second target (T2) was always neutral but target 1 (T1) could be pain-related or neutral, allowing for assessment of induction of the attentional blink by pain-related or neutral information. In Experiments 2 – 4, T1 was always neutral but T2 could be pain-related or neutral, allowing for assessment of the ability of pain-related and neutral information to overcome (or break through) the attentional blink. In Experiments 1, 2, and 4, the presentation of word types (pain-related or neutral) were either blocked or randomly mixed between-subjects. It was hypothesised that blocking stimuli type would activate the threat protection system in the blocks in which T1 was always pain-related, whereas mixed presentation was more ecologically valid in terms of approximating the unpredictable nature of threat in day-to-day life. Experiment 3 (comparing those with chronic pain to controls) used mixed presentation only.

In Experiment 1, the nature of T1 was manipulated and induction of the attentional blink was assessed. Undergraduate participants reported more pain-related T1s than neutral T1s. This additional processing resulted in a shallower attentional blink following pain-related than neutral words at the 110ms TOA. Presentation of the stimuli (blocked or mixed) did not affect this modulation of the attentional blink. In Experiment 2, the nature of T2 was manipulated to assess overcoming of the attentional blink. Under blocked presentation, there was a bias towards pain-related words at long target onset asynchronies (TOAs) only, after the attentional blink period. However, under mixed presentation, there was a bias towards

pain-related words at the shorter TOAs only – during the attentional blink period. These two experiments suggested that people do have an attentional bias towards pain-related information, and that it might be better observed when occurrence of pain-related words cannot be predicted.

In Experiment 3, I compared temporal attention between participants with, and those without, chronic pain when the nature of T2 was manipulated, and presentation was mixed. Participants with chronic pain experienced higher self-reported pain, depression, anxiety, and stress. However, these differences in current experience did not impact temporal attention, nor functioning of the alerting, orienting, or executive attention networks. Thus, there was no evidence supporting the deficit-view of attentional functioning in chronic pain, in either the temporal attention domain or the attention networks. There was an attentional bias *away* from pain-related stimuli during the attentional blink period; however, this bias did not differ in magnitude between participants with, and without, chronic pain. Moreover, neither the magnitude of the attentional blink for pain-related words or neutral words, nor the magnitude of the bias, were related to performance on the ANT. Thus, there was no evidence supporting the motivated attention hypothesis.

Experiment 4 was a direct replication of Experiment 2, with a different sample of undergraduate participants. The modulating effect of presentation (blocked v. mixed) on the timing of the observed attentional bias in Experiment 2 did not replicate. In fact, no attentional bias was observed in the attentional blink at all. Across this series of experiments with RSVP, I observed a bias *towards* pain-related information (in Experiments 1 and 2), *away* from pain-related information (in Experiment 3), and *no bias* (in Experiment 4). These inconsistencies in the attentional bias itself suggest that it is not robust. Other researchers report that it is difficult to observe reliable changes in attention in chronic pain experience (e.g. Attridge et al., 2017). The inconsistency in the direction of attentional bias in the experiments reported in Chapter 2 highlights that this difficulty may be partly due to the low reliability of tasks used, or to variability in people or attentional processes generally; not just in people with chronic pain.

The aim of the experiment described in Chapter 3 was to conduct a stronger test (than in Experiment 3, Chapter 2) for the deficit-view and motivated attention hypothesis in chronic pain experience, focusing on attentional control in the face of distraction. The task required participants to report whether a K or an N was present in an array of letters. The letter array spread above and below a central image. The images were intact or scrambled pictures of people doing day-to-day activities (neutral) or of mutilations to body-tissue

(body-tissue threat). Distraction by the intact images is inferred by slowing on the letter identification task relative to the response times for trials with scrambled images. I used blocked presentation of image type to activate the threat-protection system; participants could anticipate what type of distractor they would need to ignore. However, within a block, participants did not know on which trials intact and scrambled images would be presented. As intact images were only present 25% of the time, this design allowed for both activation of the threat protection system to be induced (by blocking type) and for the presence of an image to be unpredictable (intact v. scrambled). These design choices were made to maximise observation of deficits and attentional bias, if the deficit-view and / or the motivated attention hypothesis do describe processing in people with chronic pain. Distraction was compared between participants with, and without, chronic pain.

As in Experiment 3 in Chapter 2, the chronic pain group had higher levels of pain, depression, anxiety, and stress experience. However, these differences in experience did not impact control of distraction, nor functioning of the alerting, orienting, or executive attention networks. For the emotional distraction task, presence of images depicting threat to body-tissue did induce significantly greater distraction than neutral images in both groups of participants, meaning that the task produced an attentional bias to body-threat. However, overall distraction was not larger in participants with chronic pain. The efficiency of the attention networks was also not related to chronic pain. Thus, there was no evidence of deficits in control of distraction or the attentional networks. Moreover, the magnitude of the attentional bias towards body-tissue threat did not differ between the two groups, and the magnitude of the attentional bias was not related to performance on the ANT. Thus, there was no evidence supporting the motivated attention hypothesis.

In contrast to the behavioural task performance, on subjective measures in both experiments (in Chapter 2 and Chapter 3), participants with chronic pain reported poorer attentional control than control participants (this effect was marginally significant in Experiment 3, Chapter 2), and greater attention to changes in pain experience. Self-reported attentional control was related to vigilance to changes in pain experience in the full data set, such that the less attentional control participants thought they had, the more they thought that they were focused on changes in pain experience.

People with chronic pain also showed some differences in task performance. Specifically, in the more statistically powerful experiment in Chapter 3, psychomotor slowing was observed in both the distraction task and the ANT. Participants with chronic pain were slower to respond than matched controls. This slowing is not attentional as the group with

chronic pain are equally slowed whether the distractor was intact or scrambled – so it cannot be attributed to attention to the images. Critically, this slowing was related to current pain intensity, such that the greater current pain experience was, the slower responding was. Psychomotor slowing was uniquely related to pain and not to depression, anxiety, or stress, suggesting that it was the pain aspect of chronic pain (or some factor associated with pain), and not related psychopathology, that drove this psychomotor slowing.

Taken together, the experiments reported here suggest that while chronic pain is associated with psychomotor slowing and subjective impressions of attentional deficits, it does not impact temporal attention, control of distraction, or the attention networks. That is, no cognitive deficits were observed in these domains, and the hypothesis derived from the deficit-view was not supported. In undergraduate participants, attentional biases to pain-related words and images of body-tissue threat were observed. However, the biases to pain-related words were not reliable. Importantly, the biases observed in the attentional blink and in distraction did not differ between participants with, and without, chronic pain, and there was no relationship between the degree of bias and cognitive performance; the motivated attention hypothesis was not supported.

The experiments reported in this thesis have many features that provide for a strong test of the effect of chronic pain on attention. First, tasks that do not induce overall processing deficits may not have the sensitivity to detect either global cognitive deficits or motivated attentional effects in chronic pain experience. However, both the RSVP and emotional distraction tasks did induce deficits in processing. Attentional blinks and distraction were observed in participants with, and without, chronic pain.

Second, in Chapter 2, I carefully explored the attentional bias in the attentional blink observed in three groups of undergraduate participants, and in a group of community participants without chronic pain. This means that I observed the inconsistency in attentional bias direction across experiments. If I had not manipulated the nature of T2 in more than one experiment, I would not have observed an attentional bias towards pain-related words, away from pain-related words, and no bias. Observation of the inconsistency in the direction of the attentional bias was important for interpreting the results of Experiment 3 comparing performance by participants with, and without, chronic pain. The inconsistency meant that I could not come to a strong conclusion about attentional bias in temporal attention. I used interpretation of the earlier experiments (in Chapter 2) to drive the design of the later experiment (Chapter 3). The emotional distraction task produces a reliable attentional bias to high arousal negative images (Grimshaw et al., 2017; Walsh et al., submitted). Grimshaw and

colleagues used similar images to those in Chapter 3, meaning that I can be more confident in the attentional bias towards body-threat that I observed.

Third, I examined the effects of chronic pain in two different domains (temporal attention and control of distraction). I kept some consistency between experiments though, by using the ANT and the same questionnaires in both experiments. Consistent subjective impairments in control of attention and attention to pain, allow me to be confident that people with chronic pain feel that their attention is impaired. A consistent lack of differences between people with, and without, chronic pain in two attentional domains and in the attention networks, in two experiments with (mostly) different participants, allow me to be more confident in my conclusions that, the deficit-view and the motivated attention hypothesis are not descriptive of attentional processing in chronic pain experience. The subjective deficits must be driven by other factors or by non-attentional impairments.

Reflection on Theory and Methods

Cognitive deficits in chronic pain experience seem to be widely evidenced by self-report data (e.g., Attridge et al., 2015; McCracken & Iverson, 2001; Tesio et al., 2015), by performance on neurological batteries (e.g., Dick & Rashiq, 2007; Mifflin et al., 2016; Tesio et al., 2015), by changes in structure, activity, and connectivity in the brain (e.g., Bushnell et al., 2013; Mao et al., 2014), by performance on cognitive tasks (e.g., Grisart & Plaghki, 1999; Moore et al., 2013a, 2013b), by meta-analyses (Berryman et al., 2013; 2014), and by reviews of the literature (Moriarty et al., 2011). Yet in this thesis, although participants with chronic pain reported poorer attentional control and greater vigilance to pain than control participants, the only deficit observed was psychomotor slowing on the emotional distraction task and the ANT. Furthermore, the biases observed in participants without chronic pain on the RSVP task were not in a consistent direction.

I now consider why attentional deficits and motivated attention might not have been observed. I first focus on psychomotor slowing. Second, I discuss whether I might have focused on processes that are not impaired in chronic pain. Next, I identify design issues that may have masked or added noise to the observation of deficits, attentional biases, and motivated attention. Then, I consider what the current understanding of pain suggests about why there was no evidence seen for motivated attention. Lastly, I reflect on wider issues in pain research and scientific research generally. Throughout, I draw on the research presented in this thesis and the wider literature, to make recommendations for future research to elucidate what mechanisms underlie cognitive deficits in chronic pain experience.

Psychomotor slowing.

A capacity-limited, information processing, approach to cognitive functioning suggests that reaction time can be an index of processing speed; how quickly specific processing can occur in the mind (Cohen, 2014). Psychomotor slowing reflects impaired processing speed. As a group, people with chronic pain do show at least one marker of a cognitive deficit, a slowing in processing speed (psychomotor slowing). Deficits in attention and executive control domains are related to slowing in processing speed in other clinical conditions (Cohen, 2014), and in chronic pain psychomotor slowing seems to be a deficit evident with many cognitive tasks (Moriarty et al., 2011). However, we do not know what this psychomotor slowing reflects. Going back to Donders (1869/1969; Goldstein, 2005), cognitive psychologists have used the method of subtraction to isolate cognitive processes. Using the same logic, in the attentional blink I used the *difference* between reporting of T2 at short TOAs and long TOAs as a way of isolating the attentional bottleneck, and used the *difference* in the size of this attentional blink for pain-related and neutral words to isolate the attentional bias. Similarly, I used the *difference* between response times when distractors were intact versus scrambled to isolate the processes required to disengage attention from a meaningful stimulus, and the *difference* in distraction indices for body-threat and neural images to isolate the attentional bias. In the ANT, I also used the *difference* between trial types to isolate the alerting, orienting, and executive functions of attention. This cognitive subtraction approach is what allows me to conclude that the chronic pain is not associated with deficits in *attention*. However, all of these tasks include a range of processes that are not attentional – encoding, identification, lexical access (on RSVP), decision-making, response programming, and response execution. These processes are all present in both conditions (pain-related / body-threat, and neutral). Psychomotor slowing means that people with chronic pain have deficits in some of these other processes.

Psychomotor slowing in chronic pain could be a result of neurological dysfunction, but could also be a protective behavioural response (taking things slowly and carefully to protect body-tissue). The design of the experiments in Chapter 2 and Chapter 3 cannot be used to infer which non-attentional processes are affected. Further experiments that use subtraction are required to isolate those components. Regardless of the cause, psychomotor slowing could contribute to the subjective deficits reported by people with chronic pain. If people feel that their thinking or responses are slow, they may generalize this to all cognitive processing (e.g., attention). Interestingly, the psychomotor slowing observed in the group

with chronic pain for the emotional distraction and ANT tasks was uniquely related to current pain intensity, and not to levels of depression, anxiety, or stress. Similarly, Jongsma and colleagues (2011) reported that psychomotor slowing in chronic pain experience associated with chronic pancreatitis was best predicted by models that included pain duration and pain status (chronic pain v. controls) as factors. However, for some of the seven tasks Jongsma and colleagues used, depression, sleep disturbance, use of opioid pain control, and alcohol abuse added explanatory power to their findings. Future exploration of psychomotor slowing as a contributing factor to self-reported cognitive deficits should include factors known to be comorbid with chronic pain.

As there is psychomotor slowing in chronic pain experience, interpretation of impaired attentional functioning in people with chronic pain requires the comparison of two conditions with different attentional demands. The difference between these two conditions can be used to isolate the attentional component of the task while controlling for possible differences in response time, which reflect differences in the other processes involved in task performance. In cognitive tasks without such within-subjects comparisons, psychomotor slowing may account for the deficits observed. For example, in a Stroop task, it is the difference between congruent and incongruent trials, and not overall response time, that can be used as a measure of attentional processing. Attentional interference, assessed with a Stroop task, is reported to be greater in participants with a high level of chronic pain than in those with lower levels of pain, or in pain-free controls (Grisart & Plaghki, 1999). However, in an alternative interpretation of pain-related performance deficits on a similar task, Leavitt and Katz (2014) report their observation of slower naming times in a colour naming task in participants with chronic pain as impaired lexical access. Grisart and Plaghki (1999) make inferences from interference scores, however Leavitt and Katz (2014) make inferences from overall naming speed (for colour words printed in black ink). Group differences in response speed alone could be driven by psychomotor slowing.

All research examining cognitive functioning in chronic pain experience should have at least one within-subjects comparison to isolate any deficit to a specific process, by controlling for deficits in other processes that will be reflected in response time. This comparison would allow pain-related differences in mean RT to be ruled out as a contributing factor to any deficits. A good example is provided by Moore and colleagues (2013b). They reported that general flanker task performance (reaction time, RT), n-back task performance (accuracy), and attentional switching task performance (RT) were impaired during headache relative to during pain free periods in the same participants. Only overall performance was

impaired. Neither the congruency effect in the flanker task, nor the difference between switch and same trials was affected by pain. In an extension of Moore and colleagues, Attridge and colleagues (2017) controlled for processing speed differences (psychomotor slowing), and on no tasks did the effect of headache extend to attentional deficits. As in the experiments in Chapter 2 and 3, rather than attention per se being impacted by headache, there was impairment in RT (psychomotor slowing).

Although care must be taken when interpreting deficits in task performance, a careful examination of studies using the Stroop, and of the seven tasks identified by Moore, Keogh, and Eccleston (2009) as being good candidates for measuring the impact of pain on attention (continuous performance, flanker, endogenous precuing, n-back, inhibition, and dual tasks), suggests that interpretation of cognitive deficits in performance is not often confounded by psychomotor slowing.

It is important to acknowledge that I cannot rule out that the slowing I observed was not purely reflecting slowing in the peripheral motor system, as opposed to central slowing. In Chapter 3, two of the participants with chronic pain reported having arthritis, and one reported having repetitive strain injury. Both conditions can affect the hands, and may have physically slowed responding. One participant with chronic pain had an injured index finger so used their middle and ring finger to respond. They may have been slower on the experimental tasks, as the middle and ring finger is a less common mode of response than the index and middle finger. These physical changes only affected four out of the 72 participants with chronic pain. So, while I cannot conclusively rule out slowing in the peripheral motor system as a cause of the difference in response times between groups, motor slowing in four participants is unlikely to be driving the significant difference in response speed between the 72 participants with chronic pain and the 72 controls. Future research could include a task that involves a manipulation of motor movement, to isolate if slower responding observed on cognitive tasks has a purely motor component.

It is also relevant to note that six participants with chronic pain reported having another co-morbid neurological condition. Psychomotor slowing may result from such conditions (Cohen, 2014). Importantly, even with nine participants out of the 72 with chronic pain having conditions that may slow processing or motor speed, there was still a relationship between current pain level and the slowing on both the emotional distraction task and ANT. These relationships suggest that pain is likely accounting for some of the response slowing.

Target processes.

A reason that I may not have observed deficits, beyond psychomotor slowing, is that I targeted cognitive processes that are not affected by chronic pain. There are few meta-analyses that assess cognitive deficits in chronic pain experience. Two that do, suggest that working memory (Berryman et al., 2013) and executive function (Berryman et al., 2014) are impaired in people with chronic pain. Some of the specific process explored could be related to attentional functioning: e.g., “set-shifting” and “attention and working memory” (Berryman et al., 2014). A literature review by Moriarty and colleagues (2011), suggests that more domains (than working memory and executive function) are impaired in chronic pain experience: “attention; learning, memory, and general cognition; speed of information processing and psychomotor ability; and executive functioning”. However, perhaps attentional processes are not reliably impaired in chronic pain, but later processing (memory and executive function) is. Indeed, impairment in later processes could be reflected in the psychomotor slowing. By focusing on early attentional processes, it is possible that I missed impairments in the participants with chronic pain in other domains. A meta-analysis of *attentional* deficits in people with chronic pain is needed. Then we can make more reliable conclusions as to whether there is a significant effect of chronic pain on attention.

The deficits in people with chronic pain may also not be widespread across a whole domain. For example, it may be too broad to say “attention” is impaired. Researchers can make inferences about executive attention from different kinds of conflict resolution (Egner, 2008): two are perceptual and response level conflict. Perceptual level conflict describes how stimuli can compete with each other for processing resources (Egner, 2008; Kim, Chung, Kim, 2010; Kim, Chung, Kim, 2012). Stimuli can compete for processing resources in the RSVP stream, where the two targets compete for limited processing capacity; in the emotional distraction task, where the letter targets compete for limited processing capacity with the image distractor; and in the ANT, where the centre arrow competes for limited processing capacity with the surrounding arrows. Response level conflict describes how stimuli can lead to two competing responses, only one of which can be selected for the response (Egner, 2008; Kim et al., 2010; Kim et al., 2012). Stimuli can lead to response competition in the ANT, where the direction of the centre arrow (the required response) can be in conflict with the direction of the flanking arrows (the alternative response), and in the Stroop task, where the colour of the ink (the required response) can be in conflict with the meaning of the word (the alternative response).

In the RSVP and emotional distraction tasks described in this thesis, the conflict occurs at the perceptual level. In the ANT used in this thesis, the conflict is at both the perceptual and response level. Aside from psychomotor slowing, no deficits were observed in chronic pain experience. Tasks with response level conflict, e.g., flanker and Stroop tasks, are commonly used to assess performance in chronic pain (e.g., Berryman et al., 2014; Grisart & Plaghki, 1999; Moore et al., 2012). Perhaps resolution of response level conflict is more impaired in chronic pain experience than perceptual level conflict. By mainly using tasks that have only perceptual level conflict (the RSVP task and the emotional distraction task), it is possible that I missed impairments in the participants with chronic pain. Although, performance on the ANT (a task with response conflict) was not impaired. Indeed, conflict monitoring seems to involve the anterior cingulate cortex (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Cohen, 2014) which is also an area that shows grey matter loss and lower activation in chronic pain (Berryman et al., 2013; Mao et al., 2014). Future research should tease apart whether conflict resolution is impaired in chronic pain for both perceptual and response level conflict, or only response level conflict. Knowing at which levels chronic pain impairs cognition, will help to further understanding of how chronic pain modulates attention.

Design limitations.

It is possible that chronic pain experience does not impact the attentional processes assessed in this thesis. It is also possible that limitations in the design of the experiments may have masked cognitive deficits and motivated attention. Design limitations may also have contributed to the variability of attentional biases observed in participants without chronic pain. First, I did not want to increase fatigue in participants with chronic pain, so to limit the total running time I chose to use the short version of the ANT. The short version may not provide network scores (alerting, orienting, executive) that are as stable as those provided by the longer version. However, comparison of the network scores reported in this thesis with those reported by Fan and colleagues (2002) in participants without pain (age range = 20 – 40 years) using the full version shows that, although the alerting score is qualitatively smaller in the current experiments, the scores for the orienting and executive networks are comparable (Table 14). Further, the scores reported within this thesis are remarkably consistent across groups and experiments. Therefore, the choice to use the short form of the ANT is unlikely to have masked deficits in the attention networks in chronic pain experience.

A second design limitation was the number of participants for whom data was excluded. The RSVP task was difficult and the data from 13 participants (nine with chronic pain, four controls) were excluded due to insufficient correct target responses in Experiment 3, Chapter 2. In Chapter 3, with a task that used variation in RT rather than accuracy as an indication of impairment and attentional bias, data from five participants with chronic pain were excluded because of low accuracy. It is possible that these performance criteria mean that the data from the most impaired participants was not analysed. Excluding this data may have limited the variation in cognitive functioning in the data with which to observe deficits between groups. However, sufficient trials are necessary to produce stable means. This issue is not easy to solve. Importantly, the performance criteria should have affected observation of performance for pain-related, body-tissue threat, and neutral stimuli equally (as the stimuli were balanced for word length and frequency in Chapter 2, and for brightness and contrast in Chapter 3). Thus, while data loss impacts assessment of the deficit-view, within-subjects and between-group differences in attention to pain-related or body-threat, and neutral, stimuli could still be observed, allowing for motivated attention in chronic pain to be assessed.

Third, there were two issues with specific characteristics of the participants with chronic pain. More impaired participants with chronic pain would have struggled to get to the university. Therefore, I likely recruited participants who were at the higher functioning end of the continuum, limiting potential observation of deficits. The other issue is that in both experiments including participants with chronic pain, the pain cause, pain duration, pain intensity, co-morbidities, and medication use were highly variable. Such variability will have contributed to the noise in the data, and may have masked cognitive deficits or motivated attentional effects in participants with chronic pain. Future research is necessary to identify which individual characteristics contribute to variation in cognitive functioning and which do not. Researchers working in a larger community than Wellington, New Zealand, could recruit participants with similar pain profiles (in terms of cause and duration) to limit noise in the data, which would allow for more robust tests of the deficit-view and motivated attention hypothesis of functioning in chronic pain experience.

Fourth, attentional biases may depend on the personal relevance of the pain-related stimuli. The heterogeneous set of pain-related words used in Chapter 2, and the range of injuries to different body parts depicted by the images used in Chapter 3, mean that I may not have adequately targeted personally-relevant pain. Perhaps for motivated attention to be observed the threat depicted by the stimuli must not be to body-tissue generally, but to your specific body-tissue or to body-tissue that is damaged or painful in your pain experience.

Returning to the current understanding of pain adds to this discussion of personal relevance in threat, and stimulus specificity.

Threat protection: stimuli specificity and individual differences.

Pain is a protective conscious experience produced when threat to body-tissue is detected (de C Williams & Craig, 2016; Kruger, 2001; Moseley, 2007; Moseley & Jones, 2009; Tabor et al., 2017). Threatening stimuli may therefore only activate the threat protection system if the stimuli are related to your experience with your body-tissue. A recent review of the literature on the interruptive nature of chronic pain by Vlaeyen, Morley, and Crombez (2016; building on a theory proposed by Eccleston and Crombez, 1999) suggests that chronic pain has a greater impact on cognitive functioning when the pain is intense, novel, unpredictable, and threatening. Specific stimuli may increase the input to the threat protection system that suggests that *your* body-tissue is vulnerable to harm. Conversely, non-specific stimuli may not increase perceived threat to your body-tissue. Consequently, a larger attentional bias in people with chronic pain may only be observed with specific rather than general stimuli.

There is stimulus specificity in another clinical condition – post-traumatic-stress-disorder (PTSD). Chronic pain is the result of ongoing activation of the threat protection system (Deodhar et al., 2009). PTSD is the result of ongoing exposure to danger (Friedman, 2011). Like people with chronic pain, people with PTSD show cognitive deficits (e.g., Brandes, Ben-Schachar, Gilboa, Bonne, Freedman, & Shalev, 2002) and are hypervigilant to threat beyond the time of the trauma (Friedman, 2011). However, the threat that people with PTSD are biased to attend to is specific. When assessed with the emotional Stroop or visual search tasks, participants with PTSD show attentional biases to stimuli specific to the trauma they experienced, and not to generally arousing stimuli (Beck, Freeman, Shipherd, Hamblen, & Lackner, 2001; Dolcos, 2013; Pineles, Shipherd, Mostoufi, Abramovitz, & Yovel, 2009). PTSD shares some features with chronic pain. They are both maladaptive responses to ongoing threat, and people with PTSD and people with chronic pain show cognitive deficits and attentional biases. If attentional biases are specific in PTSD, they may also be in chronic pain.

The experiment Beck and colleagues (2001) conducted is especially relevant to a discussion of the importance of stimulus specificity when assessing cognitive functioning in chronic pain. All participants had been in a traumatic car accident. As a result of the accident, participants either had comorbid PTSD and chronic pain, or chronic pain alone, or neither

condition. Beck and colleagues compared processing of accident words, pain words, positive words, and neutral words using an emotional Stroop task in these participants. The stimuli thus matched the severe nature of the accident (accident words, e.g., *wreck*), or described the subsequent pain experience (pain-related words, e.g., *agony*), or matched the arousal level of the experience but not the valence (positive words, e.g., *delight*), or did not match anything about the participants' experience (neutral words, e.g., *table*). Participants with both PTSD and chronic pain showed psychomotor slowing compared to the other groups, and were slower to respond to the colour of words related to the trauma and pain; an attentional bias towards accident and pain-related words. Critically, participants with chronic pain and not PTSD showed an attentional bias to pain-related words but not trauma words. People with both PTSD and chronic pain seem to have threat protection systems that are alert for information that may signal further trauma and pain; whereas people with chronic pain alone, seem to have threat protection systems that are alert for information that may signal further pain (but not trauma). Beck and colleagues' experiment provides evidence that threat-protection is specific, not general, in PTSD and chronic pain.

In an experiment focused on chronic pain, Crombez and colleagues (2000) tested specificity of attentional bias in chronic back pain for five types of words. They compared interference on an emotional Stroop task induced by sensory-pain (e.g., *burning*), affective-pain (e.g., *cruel*), back-pain (e.g., *lumbago*), other health related (e.g., *asthma*), and negative (e.g., *spiteful*) words. Only an attentional bias towards sensory pain words was observed. In this thesis, I included both sensory and affective pain-related words (and disability, health-related, and general threat words) as stimuli in the RSVP experiments. It is possible that a bias towards one pain-related word type was diluted by no bias towards other pain-related word types. This would have restricted observation of attentional-biases and motivated attention.

Providing another example of stimulus specificity in chronic pain, Mercado and colleagues (2013b) presented participants with fibromyalgia with words describing fibromyalgia-specific symptoms, arousing-negative words, arousing-positive words, and neutral words. Participants completed an emotional Stroop task during electroencephalogram (EEG) recording. Only fibromyalgia-specific words had enhanced processing in the P450 event-related potential (ERP) component in participants with fibromyalgia compared to controls. While it is not yet clear what determines the specificity (Crombez et al., 2000), the stimulus specificity demonstrated in chronic pain (Beck et al., 2001; Crombez et al., 2000;

Mercado et al., 2013b) suggests that attentional bias in clinical conditions may be specific rather than general.

The research by Beck and colleagues (2001), Crombez and colleagues (2000), and Mercado and colleagues (2013b) suggests that stimulus specificity may be important for observing attentional biases in people with chronic pain. However, although an attentional bias may be specific, that does not mean that a specific bias must be greater in people with chronic pain. The motivated attention hypothesis leads to the prediction that attentional bias (specific or not) must be greater in people with chronic pain than controls. There are two studies that demonstrate specific attentional biases but not motivated attention in chronic pain. Andersson and Haldrup (2003) tested for a bias towards pain-related words that had been selected by each participant as matching their pain experience, and for a bias towards general threat words. Using the emotional Stroop task, they observed that participants with chronic pain showed an attentional bias towards pain-related and threat-related words, compared to neutral words. However, Andersson and Haldrup also observed similar attentional biases in control participants. The magnitude of the attentional biases in participants with chronic pain were not larger than the biases observed in participants without chronic pain. Another example is provided by Dear and colleagues (2011), who tested for specific, rather than general, attentional biases to threat. They used word stimuli that participants reported to be representative of their chronic pain experience, and picture stimuli depicting actions that participants endorsed as painful. Using the dot-probe task, they observed that participants with chronic pain showed a greater attentional bias to pictures that represented situations that would be painful to them. Again, as in Andersson and Haldrup, an attentional bias was also observed in control participants, but the magnitude of the attentional bias was not related to chronic pain. The results of Andersson and Haldrup, and Dear and colleagues, are similar to the results reported in Chapter 3. An attentional bias was observed towards body-tissue threat stimuli using the emotional distraction paradigm, but it was no greater in people with chronic pain experience.

There are also individual differences in activation of the threat protection system that could constrain the conditions under which cognitive deficits and motivated attention occur. For example, at times when participants with chronic pain reported that they were experiencing greater pain, less positive emotions, and pain-related fear, the greater was their attention to pain (assessed by one item of the PVAQ; Crombez, Viane, Eccleston, Devulder & Goubert (2013b). Furthermore, acceptance of pain was associated with less attention to pain (Crombez et al. 2013b).

Attempts to control chronic pain may also increase both pain experience and attentional biases to pain-related information (Eccleston and Crombez, 2007). Notebaert, Crombez, Vogt, De Houwer, Van Damme, and Theeuwes (2011) have in fact tested whether concerted attempts to control pain are associated with an attentional bias to pain-related stimuli. Participants without chronic pain were conditioned to associate a coloured ring display with an acute pain. Participants had to identify a target that might or might not appear in the conditioned coloured ring. Half of the participants were told that if they responded quickly on the target identification task, there was a 90% probability that they would not experience pain on that trial, and conversely if they responded slowly, a 90% probability that they would experience pain. The other half of the participants had no chance to control their pain experience. All participants responded faster to targets in the conditioned coloured ring, demonstrating an attentional bias towards the conditioned colour. Importantly, participants who had control over their pain experience had a larger attentional bias than participants who had no control. Extrapolating these observations from acute to chronic pain, the degree to which an individual with chronic pain attempts to control their pain may determine the magnitude of their attentional bias to pain-related information in the environment.

If research to test the motivated attention hypothesis continues, stimulus specificity and individual differences will be important variables to consider. Disorder-related stimuli, or stimuli signaling threat to the damaged or painful body area, should be used rather than general pain-related or body-tissue threat stimuli. Furthermore, a more restricted subset of people with chronic pain should be recruited, for example individuals with back pain (using images of back damage or pain inducing postures) or arthritis (using images of damaged joints). In addition, individual differences in acceptance of pain and attempts to control pain should be measured as potential covariates. If then no evidence for motivated attention is observed, inclusion of stimulus specificity and individual difference variables would allow for a stronger conclusion that motivated attention does not describe attention in chronic pain. If evidence for motivated attention was observed, then the degree of specificity of the bias and the individual differences that increase attentional bias should be explored. This refined experimental approach will be useful for both furthering understanding of the mechanisms underlying cognitive deficits in people with chronic pain, and for guiding clinical implementation of strategies to reduce the magnitude of the cognitive deficits.

Scientific issues in pain research.

Although design choices may have masked evidence for cognitive deficits or motivated attention, there is a more concerning explanation for the null effects. Cognitive deficits in attention may not be as widespread or reliable in chronic pain experience as presented in the literature. Indeed, as mentioned in Chapter 2, recent research shows that evidence for cognitive deficits in people with chronic pain is not definitive. For example, Attridge and colleagues (2017) attempted to replicate the findings of Moore and colleagues (2013b). Moore and colleagues reported that when experiencing headache pain, participants showed deficits in performance for processing speed (flanker task), working memory (n-back), and executive control (cued attention switching task). Attridge and colleagues used the same tasks as Moore and colleagues, plus two extra tasks (dual task, and uncued switching task), and did observe deficits associated with headache in processing speed on the flanker task, on switch speed and accuracy on the cued attention switching task, and on switch speed on the uncued attention task. However, some of the deficits reported by Moore and colleagues did not replicate. For example, Moore and colleagues observed impairment on n-back task performance (accuracy) during headache, but Attridge and colleagues did not.

Attridge and colleagues (2017) describe the consistent observations of deficits under pain but with different measures as “dynamic”. Indeed, Crombez and colleagues (2013b) also describe self-reported attention to pain as “dynamic” and not stable within- or between- individuals. As another example of dynamic cognitive deficits, Boselie, Vancleef and Peters (2016) could not replicate an effect observed in their own lab in 2014 (Boselie, Vancleef, Smeets, & Peters, 2014). In 2014, they observed decreased n-back performance following pain induced by cold-pressor. However, in 2016, they only observed decreased n-back performance only with concurrent pain induced by heat, not following pain induced by cold-pressor. The type of pain that induces cognitive deficits also seems to be dynamic.

Furthermore, in contrast to Tabor and colleagues (Tabor et al., 2013; Tabor et al., 2015), who demonstrated that acute pain affects judgments of distance, Tabor and colleagues (2016) did not observe a modulating effect of chronic pain on distance perception. Given that chronic pain entails engagement of protective cognitive, affective, and motivational states, and may prioritise protective goals, one would have expected acute and chronic experiences to have additive effects on distance perception. That is, chronic pain experience would have impacted distance estimates to a greater degree than acute pain experience (and thus be more readily observable). Tabor and colleagues’ (2016) findings go against that expectation.

These three examples demonstrate two important points. First, patterns of cognitive functioning may not always translate from acute to chronic pain experience. Chronic pain is different from acute pain. Chronic pain is associated with maladaptive outcomes, is of longer duration, and results from prolonged engagement of protective responses (Butler & Moseley, 2003; Marchland, 2012; Moseley, 2007). We should not assume that cognitive effects in acute pain models will reflect chronic pain experience. Second, there is variability in the conditions under which cognitive deficits are observed that do not seem to reliably transfer from one participant group to another, from one type of pain to another, or from one task to another. It seems that we do not yet understand the key contributors to cognitive deficits in pain. The recent work by Attridge and colleagues (2017), Boselie and colleagues (2016), and Tabor and colleagues (2016), shows that important first steps in testing the deficit-view and motivated attention hypothesis are to determine in which cognitive domains there are reliable deficits for neutral information in people with chronic pain, and reliable attentional biases in people with, and without, chronic pain.

It is also possible that the literature on chronic pain and cognition is subject to many of the same issues that affect other psychological research. These include low statistical power; publication bias; and bias in the choices made at the hypothesising, design, data collection, analysis, and reporting stages that can be used, consciously or unconsciously, to *p-hack* (Munafò et al., 2017). Publication bias and researcher degrees of freedom contribute to the generation of Type-I errors, leading to false-positive findings flooding the literature (Button, 2016; Cumming & Calin-Jageman, 2017; Ioannidis, 2005; Simmons, Nelson, & Simonsohn, 2011, 2012, and 2013; Simonsohn, Nelson, & Simmons, 2014; Wicherts, Veldkamp, Augusteijn, Bakker, van Aert, & van Assen, 2016). It is possible that there are unpublished observations that show no evidence for cognitive deficits or attentional biases in chronic pain experience. Unpublished studies bias meta-analyses, and add to the difficulty in conducting informative power analyses because they lead to inflated effect sizes.

Some of these considerations apply to my research as well. The experiments in Chapter 2, that recruited undergraduate participants, were conducted without power analyses to determine sample size. The power analysis for Experiment 3 in Chapter 2 was based on the number of participants I could recruit (final sample $n = 41$ in each group). That is, the power was constrained by the available sample size, and I conducted a power analysis to determine the smallest effect size that could be detected. It would have been better to have more participants in order to detect a smaller effect. The experiment in Chapter 3 comparing performance between participants with, and without, chronic pain was conducted *after*

sample-size was determined with a power analysis. This power analysis for Chapter 3 was based on an estimate of a moderate effect size, taken from the effect sizes reported in meta-analyses for cognitive deficits and biases in attention. However, given the discussion of how performance deficits associated with chronic pain may be dynamic, the true group difference effect may be smaller than estimated. Until the factors that make cognition in chronic pain dynamic are more clearly defined, future research should estimate sample-size based on effect sizes smaller than are reported in the literature.

One way to reduce researcher degrees of freedom, is to register your experiment before you conduct it. Preregistration sets the hypothesis you made, your methods, how you collected your data, and your analysis plan, before you collect the data. As preregistration becomes more widespread, it will also help to reduce publication bias. Sixty-five journals (see: https://docs.google.com/spreadsheets/d/1D4_k-8C_UENTRtbPzXfhjEyu3BfLxdOsn9j-otrO870/edit#gid=0) that publish psychology research (including, Attention, Perception, & Psychophysics; Cognition & Emotion, Psychological Science; and Royal Society Open Science), are now assessing submissions based on peer review of preregistrations, and provisionally accept for publication regardless of the results. The experiment reported in Chapter 3 was preregistered on Open Science Framework before data collection commenced. The preregistration described the background literature, the hypothesis, the method, and planned analyses. These were followed (except where noted in Chapter 3). Thus, by setting out what I planned to do before I collected the data and analysed it, I reduced the post hoc influence I could have on the analysis.

Clinical Recommendations

Given the findings from this thesis, what lessons can we apply to strategies for pain management? Attentional bias modification (ABM) has been proposed as a management technique for chronic pain (Bowler et al., 2017; Schoth et al., 2013; Sharpe, 2012; Sharpe et al., 2012; Todd, et al., 2015), specifically to reduce pain experience, and associated co-morbidities. ABM involves repeated pairings of neutral stimuli with the required task response. Attention is never directed towards the threat / negative / pain-related stimuli. For example, in a dot-probe task, the probe appears in the location of the neutral stimulus one hundred percent of the time (MacLeod & Clarke, 2015). If cognitive deficits can be minimised in people with chronic pain by reducing attentional bias to threat or stimuli related to pain, then people with chronic pain may have improved quality of life, and be able to take

advantage of pain management strategies with their freed cognitive capacity (Baker, Georgiou-Karistianis, Gibson, & Giummarra, 2017; Wiech & Tracey, 2013).

The attentional biases observed in people with chronic pain in the current experiments, were not bigger than those observed in controls. As there is not an enhanced attentional bias to modify in people with chronic pain, ABM may not be a suitable strategy for treatment of cognitive deficits. We first need to understand if, and for what kinds of stimuli, there are stronger attentional biases in chronic pain. However, ABM may still be a promising avenue for improving quality of life in people with chronic pain, even if its effects are not mediated by changes in attentional bias. Sharpe and colleagues (2012) report that in two studies, ABM with dot-probe (designed to shift attention away from pain-related words) did improve outcomes in participants with chronic pain, when compared to sham training (50 % of the probes followed the pain-related stimuli, 50 % the neutral stimuli). In the first study, at three-months post-training, participants with chronic pain who received ABM reported less pain than those who received sham training. In the second study, at six-months post-training, participants with chronic pain who received ABM reported lower relative anxiety and disability than those who received sham training. However, Sharpe and colleagues could not link these outcomes to a change in attentional bias, and they promote further research to uncover the mechanism by which ABM changed outcomes.

The mechanism by which ABM works is a concern in treatment of other disorders. A careful review of ABM studies in people with high levels of anxiety suggests that improvement in anxiety often occurs without a change in attentional bias (Mogg, Waters, & Bradley, 2017). Other training paradigms may also target the unknown mechanism by which ABM works, and as such be useful for alleviating cognitive deficits in chronic pain. Other training paradigms proposed for chronic pain include Attention Process Training (APT; Dick & Rashiq, 2007), attention management (Elomaa, de C Williams, & Kalso, 2009), and mindfulness (Day & Thorn, 2017; Garland & Howard, 2013). APT involves training to reduce brain-damage related deficits in sustained, selective, alternating, and divided attention domains (Sohlberg et al., 2000). Dick and Rashiq (2007) have proposed that APT may be suitable for improving functioning in people with chronic pain. The current experiments suggest that attentional processes are not impacted in chronic pain, but that processing speed is. Cognitive training, attention management, and mindfulness may be useful for reducing psychomotor slowing; in turn reducing psychomotor slowing may improve peoples' perceptions of their abilities. Indeed, future research should test whether bringing processing

speed up to unimpaired levels is the potential mechanism by which ABM improves quality of life for people in chronic pain.

When I began this thesis, I believed that it was important to understand the processes that underlie cognitive deficits in people with chronic pain. I still hold this belief because, if we understand the processes, we can develop effective treatments or management techniques. This belief is pertinent to the use of any treatment. We must understand the mechanism/s by which the technique works to optimise our use of it, and achieve the greatest improvement in quality of life for those in our communities who experience chronic pain.

Conclusions

In this thesis, I presented a series of experiments that explored attention to pain-related information in people with, and without chronic pain. It culminated in a strong test of the deficit-view and the motivated attention hypothesis of attention in chronic pain. I observed whether pain-related stimuli overcame the attentional blink (indexing temporal attention), or impaired control of distraction, more than neutral stimuli. In participants without chronic pain, there was not a reliable bias towards pain-related words, although there was a bias observed towards images depicting damage to body-tissue. Psychomotor slowing was observed in participants with chronic pain, which suggests that some aspect of cognitive or behavioural processing is affected in people with chronic pain. However, the aspect that was affected in these experiments was not attentional. There were no attentional deficits observed in participants with chronic pain, contrary to the deficit-view of attention in chronic pain. The magnitude of the attentional biases observed in people with, and without, chronic pain did not differ, contrary to the motivated attention hypothesis.

Importantly, the experiments in this thesis demonstrate that, even in the absence of observable impairment in attention, people with chronic pain did feel like they had less attentional control, and paid more attention to their pain, than control participants did. Furthermore, higher levels of current pain were associated with greater psychomotor slowing. We still do not understand the cognitive processes that underlie these subjective deficits and the psychomotor slowing associated with pain experience. To advance understanding of how chronic pain affects cognitive functioning, future research should next target cognitive processes in a more fine-grained manner by testing the roles of 1) psychomotor slowing, 2) perceptual and response conflict, 3) the specificity of pain-relatedness or of threat to body-tissue, and 4) of individual differences in cognitive functioning. As we gain greater

understanding of the processes underlying the cognitive deficits in chronic pain, we can make better recommendations for effective management techniques.

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Appendix A

Pain-related and neutral target pairs, used in Chapter 3

pain-related – neutral pairs			
T1	Type*	T2	Type
worry	affective	gritty	neutral
despair	affective	halting	neutral
troublesome	affective	yourselves	neutral
cruel	affective	yank	neutral
dreadful	affective	planting	neutral
miserable	affective	unclouded	neutral
angry	affective	plumb	neutral
discouraging	affective	supplementary	neutral
unbearable	affective	squinting	neutral
intense	affective	discover	neutral
devastating	affective	commonplace	neutral
hopeless	affective	activate	neutral
punishing	affective	deductive	neutral
agonising	affective	beforehand	neutral
exhausting	affective	upbringing	neutral
tiring	affective	rattle	neutral
frustrating	affective	contractual	neutral
vicious	affective	surpass	neutral
endless	disability	instinct	neutral
incurable	disability	disguise	neutral
sick	disability	soda	neutral
inactive	disability	stainless	neutral
injury	disability	sensor	neutral
suffering	disability	inhabited	neutral
unhealthy	disability	exception	neutral
chronic	disability	mending	neutral
debilitating	disability	commencement	neutral
dependent	disability	symbolism	neutral
mutilated	disability	jitterbug	neutral
vulnerable	disability	contender	neutral
inhibited	disability	spherical	neutral
tormenting	disability	exterior	neutral
paralysed	disability	emphasize	neutral
disease	disability	request	neutral
helpless	disability	shelled	neutral
paralysis	health related negative	memorial	neutral
nausea	health related negative	orange	neutral
casualty	health related negative	hatchway	neutral
ambulance	health related negative	implement	neutral
painful	health related negative	recruit	neutral
disabled	health related negative	resolved	neutral
collapse	health related negative	flutist	neutral
sickness	health related negative	outweigh	neutral
splitting	pain	palpable	neutral
tearing	pain	republic	neutral

choking	pain	avocado	neutral
tightness	pain	covering	neutral
inflamed	pain	consult	neutral
aching	pain	coyote	neutral
stabbing	pain	vinegar	neutral
pain	pain	maxim	neutral
gripping	pain	astute	neutral
tender	pain	leash	neutral
gasping	pain	beehive	neutral
spasm	pain	milky	neutral
squeezing	pain	animation	neutral
cramps	pain	plaque	neutral
pulsating	pain	crosswise	neutral
drilling	sensory	angling	neutral
sharp	sensory	meat	neutral
burning	sensory	manila	neutral
stings	sensory	filter	neutral
throbbing	sensory	computer	neutral
flickering	sensory	backwater	neutral
beating	sensory	shrunk	neutral
gnawing	sensory	paving	neutral
grinds	sensory	fortify	neutral
sore	sensory	champ	neutral
shooting	sensory	acoustic	neutral
piercing	sensory	teacher	neutral
boring	sensory	waiter	neutral
pinching	sensory	playable	neutral
penetrating	sensory	commodity	neutral
stiff	sensory	petty	neutral
pounding	sensory	fourteen	neutral
hurting	sensory	poplar	neutral
tugging	sensory	cottage	neutral
horrifying	general threat	absorption	neutral
threat	general threat	fiction	neutral
surgery	general threat	realist	neutral
harmful	general threat	matting	neutral
hazardous	general threat	undulated	neutral
menacing	general threat	traceable	neutral
wound	general threat	wobble	neutral
terrifying	general threat	travelling	neutral
fearful	general threat	explicit	neutral
lethal	general threat	eager	neutral
suffocating	general threat	inexperienced	neutral
killing	general threat	troupe	neutral
crushing	general threat	pendant	neutral
panic	general threat	scarf	neutral
uncontrollable	general threat	fundamentalist	neutral
danger	general threat	tweed	neutral
tortured	general threat	peerless	neutral
scared	general threat	softly	neutral

damage	general threat	broccoli	neutral
neutral – neutral pairs			
T1	type	T2	type
easel	neutral	cavern	neutral
painter	neutral	shining	neutral
restraining	neutral	incantation	neutral
whirling	neutral	haughty	neutral
administer	neutral	projection	neutral
corkscrew	neutral	underlie	neutral
trailer	neutral	verify	neutral
doorbell	neutral	disengage	neutral
golden	neutral	viewer	neutral
groceries	neutral	unglazed	neutral
brushed	neutral	reborn	neutral
reclaimed	neutral	perpetual	neutral
rear	neutral	gull	neutral
microwave	neutral	overboard	neutral
ornament	neutral	monthly	neutral
items	neutral	wholly	neutral
drums	neutral	script	neutral
sponge	neutral	rocky	neutral
banister	neutral	rebound	neutral
mention	neutral	hearing	neutral
king	neutral	rent	neutral
feather	neutral	chatty	neutral
flooring	neutral	cardboard	neutral
airmail	neutral	dessert	neutral
roofing	neutral	nowhere	neutral
gadget	neutral	cavalry	neutral
cobalt	neutral	rascal	neutral
dining	neutral	flung	neutral
flannel	neutral	dotted	neutral
mirror	neutral	invite	neutral
expensive	neutral	succeeding	neutral
sailboat	neutral	stamping	neutral
blooming	neutral	digital	neutral
waterfalls	neutral	proposition	neutral
appliance	neutral	waistcoat	neutral
undertake	neutral	formation	neutral
timetable	neutral	intuitive	neutral
gardener	neutral	frenchman	neutral
grasped	neutral	woodland	neutral
strawberries	neutral	unchallenged	neutral
gymnasts	neutral	specify	neutral
fireplace	neutral	carpenter	neutral
sheet	neutral	herd	neutral
diverse	neutral	coating	neutral
subcommittee	neutral	productivity	neutral
baggage	neutral	charcoal	neutral
dried	neutral	womb	neutral

hockey	neutral	mauve	neutral
knit	neutral	band	neutral
customers	neutral	peppered	neutral
innovative	neutral	accolade	neutral
decanted	neutral	lettering	neutral
drinking	neutral	eastern	neutral
imagines	neutral	courtship	neutral
metabolite	neutral	foundation	neutral
senator	neutral	aviator	neutral
tiles	neutral	depot	neutral
swivel	neutral	upshot	neutral
tunnel	neutral	nutmeg	neutral
postmark	neutral	commonly	neutral
afterwards	neutral	blackboard	neutral
embroidered	neutral	relativity	neutral
schooling	neutral	sincerity	neutral
ultraviolet	neutral	compensation	neutral
tribune	neutral	assess	neutral
donors	neutral	fame	neutral
neighbour	neutral	forestry	neutral
advertisers	neutral	consumption	neutral
waterproof	neutral	standpoint	neutral
wiring	neutral	yearn	neutral
annually	neutral	dappled	neutral
crystal	neutral	plate	neutral
advocates	neutral	schematic	neutral
elephant	neutral	pipng	neutral
resident	neutral	memorise	neutral
trousers	neutral	decorator	neutral
skirt	neutral	peach	neutral
octagonal	neutral	digestible	neutral
blackberry	neutral	excitement	neutral
cotton	neutral	flash	neutral
chart	neutral	slab	neutral
coaching	neutral	watchmen	neutral
cartoon	neutral	waning	neutral
sideboard	neutral	faculty	neutral
refresh	neutral	warping	neutral
detectors	neutral	vineyard	neutral
remarks	neutral	unearth	neutral
scaffolding	neutral	photographer	neutral
bibliographies	neutral	knowledgeable	neutral
waited	neutral	tepid	neutral
currents	neutral	mostly	neutral
lessons	neutral	reserve	neutral
garage	neutral	lapping	neutral
campus	neutral	skilful	neutral
postcard	neutral	coachmen	neutral
envelope	neutral	unrelated	neutral

Notes.

1. The pain-related / neutral, T1 / T2pairs, were reversed in Experiments 2, 3 and 4.

Depending on the counterbalance, half of the participants received the neutral / neutral, T1 / T2 pairs, in reversed order.

* The type label for the pain-related words are from the previous studies they were used in.

Appendix B**Duration and cause of chronic pain information for the 41 participants experiencing chronic pain in Chapter 2**

Duration	Duration number (years)	Cause
3 months	.25	Knee injury through ski accident which flares.
8 months	.67	Unsure of cause - possibly emotional/postural.
10 months	.83	Possibly from running or over training.
1 year 3 months	1.25	Pelvic (a year) and chest pain (3 months).
3 years	3	It's a hereditary condition which has been diagnosed.
3 years	3	Ankylosing spondylitis - an arthritic condition which causes the fusing of joints. I experience it currently in my back, hips, jaw.
3 – 4 years	3.5	Shoulder impingement and torn meniscus.
approx. 4 years	4	-
4 years	4	Complex Regional Pain Syndrome, lumbar lordosis, sacralisation of L5 vertebra.
almost 5 years	5	ankylosing spondylitis.
5 years	5	Spinal injury resulting in degeneration of intervertebral discs at L4-S1.
more than 5 years	5	-
5 – 6 years	5.5	Arthritis.
6 years	6	Many factors.
7 years	7	Caused by cervical spinal nerve irritation.
7 years	7	I have an artificial leg on my right side (since birth from PFFD), a hip replacement on my left side (from Perthes disease and it being out of alignment), and ongoing knee pain on the left side which I'm currently seeing specialists for (for around 4 years now).
8 years	8	Cervical spine injury in 2008 and surgery for that injury in 2009. Nerve damage has caused chronic pain
8 years	8	Endometriosis.
3 – 5 years / 8years	8	Lower back 8 years, just from working... old untreated contusions shoulders, wrists 3-5 years. old knees.
8 years	8	Autoimmune disease that manifests as arthritis and fatigue.
10 years or so	10	Endometriosis is the main cause of my chronic pain.

approx. 10 years	10	Severe endometriosis, adenomyosis, adhesions, ovarian cysts and nerve damage are the causes of my chronic pain.
10 years	10	Hand pain for 9 years (likely thoracic outlet syndrome); jaw pain for 10 years (initially from hyperextending, but later due to sensitisation from inappropriate treatment); hip pain due to labral tear (that I suppose is only chronic because the labrum can't heal).
10 years	10	-
approx. 12 years	12	No idea.
over 15 years	15	Diagnosis of spondyloarthritis to explain chronic pain in lower spine. But no diagnosis of pain and discomfort in other areas.
16 – 18 years	17	Fibromyalgia.
18 years	18	I was diagnosed with juvenile idiopathic arthritis at the age of 6 and a half. I have experienced this chronic pain for about 18 years.
18 years	18	I believe its caused by my hydrocephalus. Water on the brain.
20 plus years	20	Cause is psychosomatic, developmental trauma related.
20 years	20	Neck pain from possible injury AND/OR exacerbated by poor posture.
24 years	24	Regular headaches for 24 years (since age 13), became daily 8 or 9 years ago. Brain scan by neurologist showed no abnormalities at age 13, chiropractor has suggested excess pressure on the atlas vertebrae. Length of neck, and 'forward head posture' probably contribute to the issue. Headaches exacerbated by lack of sleep and stress.
30 years	30	Car accident (femur, low back and cervical neck injuries) combined with wind up/summation.
approx. 30 years	30	Since my 20s (I'm 51 now) from an injury, now I have some other issues some degenerative back pain, injuries and maybe a rheumatological condition that is being investigated.
35 years	35	Head Injury from birth - I took 36 hours to be born, also have gout, arthritis, OOS in joints.
number of years	-	I have suffered with back pain for a number of years and have never determined the cause.
-	-	Spine issues the biggest cause - cuada equine and arthritic stuff.

several years	-	I have joint pain from osteoporosis, gastrointestinal discomfort on an ongoing basis due to coeliac disease and have trouble with back pain from Scheuermann's Disease.
-	-	Cervical disk herniations and herniated / destroyed lumbar disks.
several years	-	Back pain and restless leg.

Appendix C

Duration and cause of chronic pain information for the 72 participants experiencing chronic pain in Chapter 3

Duration	Duration number (years)	Cause
5 months	0.42	Do not yet know the cause (ear pain), have appointment with Ear Nose Throat specialist in 3 weeks to investigate.
5 – 6 months	0.46	Injured both shoulders in different ways, and left arm while attempting 'down dog' at yoga.
6 months	0.5	Fibromyalgia.
approx. 11 months	0.92	Since around the start of the year the pain emerged in my back.
1 year	1	Over 1 year, caused by endometriosis and a digestive issue as well as asthma issues.
over a year	1	Osteoarthritis in knee for over a year
13 months	1.08	Mostly bad posture/incorrect heavy lifting for an extensive length of time which has affected the alignment of my neck.
1.5 years	1.5	Assume that it is due to lack of sleep and bad diet.
at least 2 years	2	Caused by repetitive use of knees (sliding with knee pads) on skateboard ramps.
approx. 2 years	2	Patellar injury on right knee.
2 years	2	1 year constant, over 1 year on and off. Was confirmed diagnosis 1 year ago with Lupus.
2 years	2	TMJ injury.
about 2 years	2	Lower back, and neck pain caused by a car crash and multiple sports related injuries.
2 years	2	Back pain - bulging L5 disc (gym injury 2 years ago). Knee pain - both left and right knee anterior cruciate and meniscal damage.
5 years	3	Prolapsed disks in lumbar spine impact ~1/3 of the nerves in my legs
3 years	3	Cervical neuralgia in my C6&7.
3 years	3	Three years ago, I got severe food poisoning and this developed into Chronic Fatigue Syndrome which developed into Fibromyalgia.
3 years	3	Psoriatic arthritis.
3 – 4 years	3.5	3-4 years for my Fibromyalgia, had ME for 7 years following a serious bout of glandular fever.
3 – 4 years	3.5	I have a dehydrated disc in my lower back, which causes nerve pain and localised aching, I also have nerve damage in my ankle which gets painful with exercise.
4 years	4	Undiagnosed.

5 years	5	Caused by multiple injuries to left shoulder, which is now artificial. Also, caused by fracture of right calcaneus, which was reduced and fixed.
many years	5	Many years (since around age 14); skeletal issues.
6 years	6	I have a condition called Interstitial Cystitis.
6 years	6	Lower back pain 6 years, fibromyalgia type symptoms for 17 months.
7 years	7	Congenital physical disability (PFFD) and ongoing issues on my left leg including a hip replacement.
7 years	7	Nerve damage from an injury in 2009.
8 years	8	Since 2008 - I have Undifferentiated Connective Tissue Disease.
8 years	8	3 – 5yrs. Both ankles injured in car accident and nothing is showing up on scans that there is anything medically wrong. Have been told because of my ME/CFS that it's turned into chronic pain. Also have CRPS or chronic pain in right shin area from injury 8+yrs ago.
8 years	8	Most of my life, however the last 8 years have been dominated by cancer, osteoarthritis, and rheumatoid arthritis.
8 years	8	I've had back pain for over 8 years - I think it is caused by muscle tightness, poor posture, excessive sitting, perhaps small injuries that have occurred over the course of my life.
8 years	8	I have experienced pain in my lower back for 8 years. A combination of growth spurts, a poor core, bad posture and physical stress such as cricket and working as a Gardener.
8 years	8	At least 8 years, though probably since childhood. I have Ehlers-Danlos Syndrome (Type: Classical) and fibromyalgia, as well as other conditions such as carpal tunnel that influence my on-going pain
9 years	9	I have had two surgeries for it. It is a rare condition in my shoulder joint.
9 years	9	Since July 2007. The nerve pain was caused by shingles, but the virus also 're-hardwired' my entire pain system so that every time I have an operation or injury, it compounds the pain. I am still recovering from a fall down the stairs three years ago - this caused me significant additional pain particularly back, hip and leg pain.

I had chronic pain from roughly 1981 to 1990 then in 2008 to now.	9	Experienced off and on for many years. Caused by cervical, lumbar, thoracic disk herniations, S-I joint pain.
9 years	9	No known cause.
10 years	10	Pain for a decade, chronic at least 4 years. It began with a back injury and the pain then spread to other areas, back, posture, and chronically tense muscles all play a role.
more than 10 years	10	It keeps returning. My back is causing it.
10 years	10	Since February 2006, as a result of a car accident, after the car accident I had acupuncture and the needle hit a tender spot and I've had pain in that site ever since. I've been to the pain clinic, had Botox and a range of medication. I had craniosacral therapy in 2008 and managed to get down to 2 pills a day, then they took indomethacin off the market and I've struggled to be pain free. I also have chronic sinusitis and despite having had 3 operations in 4 years. since my last surgery in August I've also been having sinus headaches which causes my "normal" headache to react.
10 years	10	did not respond
10 years	10	Little to no triggers. Dysautonomia may or may not be the cause of extreme chest pain. I've been experiencing serious fibromyalgia pain for the last 9-10 years. I was formally diagnosed 7 years ago. I was also diagnosed with Rheumatoid Arthritis about 4-5 years ago (also a source of pain, but not as severe or constant as fibromyalgia).
10 years	10	Knee injury, (multiple comminuted fracture of tibial plateau).
11 years	11	Fibromyalgia, CFS, TMJD, Costochondritis.
12 years	12	Chronic low back pain for about 12 years. I believe it is from a skiing accident I had as a child. A weak core contributes to it.
about 12 years	12	I was in a minor car accident in 2003, and experience back and/or neck pain regularly since then. My current daily pain episode lasts for 13 months and was caused by an injury in a gym. (I started weight training trying to strengthen my back and stop being in and out of pain). My trainer added some weight for my barbell assuring I was ready. One deadlift after we knew I was not ready at all...
13 years	13	

13 – 14 years	13.5	On and off for approx. 12 years but since September 2013 the pain got to the point where I could no longer keep my job. The pain has not ceased since then. My pain may be due to numerous UTI infections. The pattern has, over the years, been that I get a UTI infection and then the pain lingers, even when the infection has been successfully treated. With each infection, the pain got worse and lasted longer. It has got to the point now that the pain is constant. It was a UTI in 2013 that triggered this flare up that has persisted. I was in my teens when it first started. Doctors, therapists and I think the UTIs are triggered by stress and anxiety.
15 years	15	Ehlers-Danlos syndrome.
15 years	15	Chronic pain from psoriatic arthritis on and off for 15 years but been having an active flare up for 6+ months.
15 years	15	Back trouble.
15 years	15	Legg-Calve Perthes Disease.
17 years	17	Suspected psoriatic arthritis.
17 years	17	Diagnosed first in 1999 - spinal injuries. Today trigeminal neuralgia, Lymphoma and hip pain, right more severe, when bending and climbing hills, stairs.
18 years	18	Fibromyalgia.
20 years	20	Spondylosis + other unidentified pain like fibromyalgia.
20 years	20	I have fibromyalgia, which had onset around the time I contracted chronic fatigue syndrome post-virally.
20 years	20	I have a congenital condition and was diagnosed with CFS and fibromyalgia in my teens.
over 20 years	20	Over 20 years, shoulder and neck pain causing migraines (which have now gone away) but I have shoulder and neck pain each day. Causes multiple including falling out of a tree as a child, injury when gardening many years ago, computer use and posture.
22 years	22	Since my back injury in 1994 and most recently in 2014 when I injured my shoulder at work and the pain still continues. Sometimes it's 5/10, sometimes it's 11/10 where 10 is excruciating pain.
since birth	25	I have had health problems since birth, have been diagnosed with Fibromyalgia resulting from this.

whole life	25	I have endometriosis, which they believe has been present from a very early age.
28 years	28	I was born with congenital dislocation of both hips, correct by surgery at approx. 10 months. I was diagnosed with arthritis at 16 as a result of ongoing problems. I was diagnosed with medication induced ulcerative colitis in 2015 which causes ongoing pain. I have also multiple sclerosis which causes a variety of chronic pain depending on the area affected, the onset of this was 2012. My GP has recently also decided that I possibly have fibromyalgia.
30 years	30	Repetitive strain injury in upper limbs, affecting wrists, elbows, shoulders.
30 years	30	Diagnosed with Fibromyalgia three years ago, have suffered for some 30 years... also fall under ME as I also suffer CFS.
30+ years	30	IBS 30 + years; chronic back pain (have had surgery x 2) 5 years.
whole life	33	My whole life, probably due to the types of work I do (physical, hard work, long hours). Poor sleep habits, depression/ anxiety and various injuries.
35 years	35	Had a back operation in my late twenties (laminectomy) but have developed arachnoiditis and fibromyalgia resulting in pain all the time.
many years	-	Medications.
-	-	Chronic neck pain, probably from poor posture, possibly from teenage injury.
many years	-	No not really.
many years	-	Migraine. Stress, tiredness, hormones.

Appendix D

Questionnaires used in Experiment 3 in Chapter 2 and in Chapter 3

Administered via Qualtrics, expect where noted in text.

1. The Short-form McGill Pain Questionnaire (Melzack, 1984)

Please check the column to indicate the level of your current pain experience for each word.

	None (0)	Mild (1)	Moderate (2)	Severe (3)
Throbbing				
Shooting				
Stabbing				
Sharp				
Cramping				
Gnawing				
Hot-burning				
Aching				
Heavy				
Tender				
Splitting				
Tiring-exhausting				
Sickening				
Fearful				
Punishing-cruel				

What is the level of the pain you are currently experiencing?

No pain

Worst possible pain

What is the level of the pain you are currently experiencing? Please check one.

- 0 No pain ___
- 1 Mild ___
- 2 Discomforting ___
- 3 Distressing ___
- 4 Horrible ___
- 5 Excruciating ___

2. The Depression Anxiety Stress Scale (DASS21; Lovibond & Lovibond, 1996a, 1996b)

Scores are doubled to be comparable to the full DASS.

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

- 0 Did not apply to me at all
 - 1 Applied to me to some degree, or some of the time
 - 2 Applied to me to a considerable degree, or a good part of time
 - 3 Applied to me very much, or most of the time
-
- 1 I found it hard to wind down
 - 2 I was aware of dryness of my mouth
 - 3 I couldn't seem to experience any positive feeling at all
 - 4 I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)
 - 5 I found it difficult to work up the initiative to do things
 - 6 I tended to over-react to situations
 - 7 I experienced trembling (eg, in the hands)
 - 8 I felt that I was using a lot of nervous energy
 - 9 I was worried about situations in which I might panic and make a fool of myself
 - 10 I felt that I had nothing to look forward to
 - 11 I found myself getting agitated
 - 12 I found it difficult to relax
 - 13 I felt down-hearted and blue
 - 14 I was intolerant of anything that kept me from getting on with what I was doing
 - 15 I felt I was close to panic
 - 16 I was unable to become enthusiastic about anything
 - 17 I felt I wasn't worth much as a person
 - 18 I felt that I was rather touchy

- 19 I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)
- 20 I felt scared without any good reason
- 21 I felt that life was meaningless

3. The Attentional Control Scale (ACS; Derryberry & Reed, 2002)

Please indicate using the below scale how frequently each item is a true description of your thoughts/behaviours.

1 = almost never

2 = sometimes

3 = often

4 = always

- 1. ___ It's very hard for me to concentrate on a difficult task when there are noises around
- 2. ___ When I need to concentrate and solve a problem, I have trouble focusing my attention
- 3. ___ When I am working hard on something, I still get distracted by events around me
- 4. ___ My concentration is good even if there is music in the room around me
- 5. ___ When concentrating, I can focus my attention so that I become unaware of what's going on in the room around me
- 6. ___ When I am reading or studying, I am easily distracted if there are people talking in the same room
- 7. ___ When trying to focus my attention on something, I have difficulty blocking out distracting thoughts
- 8. ___ I have a hard time concentrating when I am excited about something
- 9. ___ When concentrating I ignore feelings of hunger or thirst
- 10. ___ I can quickly switch from one task to another
- 11. ___ It takes me a while to get really involved in a new task
- 12. ___ It is difficult for me to coordinate my attention between the listening and writing required when taking notes during lectures
- 13. ___ I can become interested in a new topic very quickly when I need to
- 14. ___ It is easy for me to read or write while I'm also talking on the phone
- 15. ___ I have trouble carrying on two conversations at once
- 16. ___ I have a hard time coming up with new ideas quickly
- 17. ___ After being interrupted or distracted, I can easily shift my attention back to what I was doing before
- 18. ___ When a distracting thought comes to mind, it is easy for me to shift my attention

away from it

19. ____ It is easy for me to alternate between two different tasks

20. ____ It is hard for me to break from one way of thinking about something and look at it
from another point of view

4. The Pain Vigilance and Awareness Questionnaire (PVAQ; McCracken, 1997)

Please consider any experiences of pain you have had over the past 2 weeks, and indicate how frequently each item is a true description of your thoughts/behaviours.

	Never					Always				
	0	1	2	3	4	5				
1. I am very sensitive to pain	0	1	2	3	4	5				
2. I am aware of sudden or temporary changes in pain	0	1	2	3	4	5				
3. I am quick to notice changes in pain intensity	0	1	2	3	4	5				
4. I am quick to notice effects of medication on pain	0	1	2	3	4	5				
5. I am quick to notice changes in location or extent of pain	0	1	2	3	4	5				
6. I focus on sensations of pain	0	1	2	3	4	5				
7. I notice pain even if I am busy with another activity	0	1	2	3	4	5				
8. I find it easy to ignore pain	0	1	2	3	4	5				
9. I know immediately when pain starts or increases	0	1	2	3	4	5				
10. When I do something that increases pain, the first thing I do is check to see how much pain was increased	0	1	2	3	4	5				
11. I know immediately when pain decreases	0	1	2	3	4	5				
12. I seem to be more conscious of pain than others	0	1	2	3	4	5				
13. I pay close attention to pain	0	1	2	3	4	5				
14. I keep track of my pain level	0	1	2	3	4	5				
15. I become preoccupied with pain	0	1	2	3	4	5				
16. I do not dwell on pain	0	1	2	3	4	5				