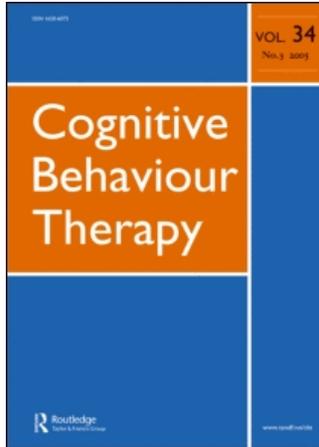


This article was downloaded by:[Watt, Margo]
On: 27 January 2007
Access Details: [subscription number 768566197]
Publisher: Routledge
Informa Ltd Registered in England and Wales Registered Number: 1072954
Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Cognitive Behaviour Therapy

Publication details, including instructions for authors and subscription information:
<http://www.informaworld.com/smpp/title-content=t713926011>

A Brief Cognitive-Behavioral Approach to Reducing Anxiety Sensitivity Decreases Pain-Related Anxiety

To link to this article: DOI: 10.1080/16506070600898553
URL: <http://dx.doi.org/10.1080/16506070600898553>

Full terms and conditions of use: <http://www.informaworld.com/terms-and-conditions-of-access.pdf>

This article maybe used for research, teaching and private study purposes. Any substantial or systematic reproduction, re-distribution, re-selling, loan or sub-licensing, systematic supply or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.

© Taylor and Francis 2007

A Brief Cognitive-Behavioral Approach to Reducing Anxiety Sensitivity Decreases Pain-Related Anxiety

Margo C. Watt^{1,2}, Sherry H. Stewart^{2,3}, Marie-Josée Lefavre² and Lindsay S. Uman²

¹*Department of Psychology, St Francis Xavier University, Antigonish, NS, Canada;*

²*Department of Psychology, Dalhousie University, Halifax, NS, Canada;* ³*Department of Psychiatry, Dalhousie University, Halifax, NS, Canada*

Abstract. Anxiety sensitivity (AS; fear of anxiety-related sensations) is a known risk factor for anxiety disorders and recently has been linked to pain disorders. The present study was guided by the hypothesis that a program designed to reduce AS levels might also result in a decrease in anxiety related to pain sensations. Female undergraduates, selected as either high or low in AS according to screening scores on the Anxiety Sensitivity Index (ASI), were randomly assigned to participate in 3 1-hour, small group sessions of either cognitive behavioral therapy (CBT; psycho-education, cognitive restructuring, and interoceptive exposure) or a non-specific treatment (NST). Immediately prior to and following the intervention, participants completed the 20-item Pain Anxiety Symptoms Scale (PASS-20). Consistent with hypothesis, results revealed a 3-way interaction between AS group, intervention condition, and time on PASS-20 total scores. Only participants with high pre-morbid levels of AS assigned to the CBT condition showed a significant reduction in scores on the PASS-20 from pre- to post-treatment. Implications for improving CBT approaches for pain disorders are discussed. *Key words:* anxiety sensitivity; fear of pain; pain anxiety; cognitive behavioral treatment

Received 24 February, 2006; Accepted 22 June, 2006

Correspondence address: Margo C. Watt, PhD, Department of Psychology, St Francis Xavier University, PO Box 5000, Antigonish, NS, B2G 2W5, Canada. Tel: +902 867 5215; Fax: +902 867 5189. E-mail: mwatt@stfx.ca

Anxiety sensitivity (AS) refers to the fear of arousal-related bodily sensations arising from beliefs that these sensations have harmful physical, psychological, and/or social consequences (Reiss, 1991). AS has been implicated in the development and maintenance of anxiety disorders, particularly panic disorder (Schmidt, Lerew, & Jackson, 1997). More recently, researchers have examined the role of AS in other disorders, including chronic pain (Asmundson, Wright, & Hadjistavropoulos, 2005).

Kuch, Cox, Woszczyna, Swinson, and Shulman (1991) found that panic disorder patients with chronic pain reported significantly higher levels of AS compared to panic disorder patients without chronic pain. Subsequently, among 55 motor vehicle

accident survivors with chronic pain, Kuch, Cox, Evans, and Shulman (1994) found that those with accident phobia reported higher AS and pain in more locations. Similarly, Asmundson and Norton (1995) found that chronic back pain patients with high AS scored higher on the Pain Anxiety Symptoms Scale (PASS: McCracken, Zayfert, & Gross, 1993) subscales that assessed cognitive anxiety and fearful appraisals about pain than patients with low AS. In contrast, the 2 AS groups did not differ in reported pain severity. Moreover, patients with high AS were twice as likely as patients with moderate or low AS to report use of analgesic medication to relieve pain.

Using structural equation modeling (SEM), Asmundson and Taylor (1996) found that AS

accounted for 30% of the variance in fear of pain which, in turn, accounted for 68% of the variance in pain escape/avoidance behavior in a sample of patients with chronic musculoskeletal pain. Whereas pain severity accounted for an additional 13% of the variance in fear of pain, it did not contribute significantly to the prediction of escape/avoidance behavior. Asmundson, Norton, and Veloso (1999) extended these findings to a sample of patients (85% female) with recurring headaches; high AS headache patients reported more cognitive anxiety, fearful appraisals, and escape/avoidance behavior on the PASS. There was no difference across AS groups in physiological response to pain, perceived headache severity, or use of prescribed analgesics. More recently, Norton and Asmundson (2004) applied SEM to data from a sample of patients with recurrent headaches and found that AS had a direct and significant loading on fear of pain, the magnitude of which exceeded the loading of pain severity.

To investigate the relationship between AS and acute pain, Keogh and Birkby (1999) used a cold pressor task (i.e. hand immersion in ice water) as a pain-inducing stimulus with a non-clinical sample. There was an interaction between gender and AS levels, such that high AS women reported greater sensory pain (e.g. more "throbbing," "stabbing," "aching") than low AS women. Keogh and Mansoor (2001) investigated the effect of AS and coping styles (focused attention vs avoidance) on young women's responses to experimentally induced pain. They found no differences between high and low AS women in pain threshold or tolerance (see also Uman et al., this issue), but did find that high AS women reported more sensory ("throbbing", "stabbing") and affective ("sickening", "fearful") pain than low AS women.

In summary, findings indicate that AS affects pain perception. Individuals with high AS perceive pain more negatively than those with low AS, and report higher levels of anxiety related to the pain experience (see also Conrod, this issue; Tsao et al., this issue). High AS may be a risk factor for psychopathological responses to pain in the same way that it is a risk factor for anxiety disorders (Keogh & Birkby, 1999). The tendency of high AS individuals to be more sensitive to arousal sensations, and to misinterpret and

catastrophize about these sensations, may predispose them to exacerbate a variety of pain-related conditions, such as headaches, gastrointestinal pain, musculoskeletal pain, and menstrual pain (Asmundson, Vlaeyen, & Crombez, 2004). If this is the case, efforts to reduce AS might also reduce pain anxiety.

Until recently, reductions in AS have emerged only as a positive side-effect of programs designed to target panic disorder. Telch et al. (1993) found a reduction in AS levels in a sample of panic disorder patients who participated in a 12-session group cognitive behavioral therapy (CBT) intervention designed to reduce panic symptoms, and no change in AS levels over a similar interval in a wait list control group. Treatment gains in the CBT group were maintained at follow-up assessment 6 weeks later. Similarly, Penava, Otto, Maki, and Pollack (1998) reported a reduction in AS for participants in a CBT program designed to reduce panic symptoms in panic disorder patients. Mean scores on the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992) dropped from a pre-program high of 35.0 (SD=12.5) to 21.5 (SD=12.9) at the completion of the CBT program. These results show that CBT can reduce AS levels from the clinical range for panic disorder with or without agoraphobia (30.5–46.7) into the normal range (14.2–22.5) according to normative sample means (Peterson & Reiss, 1992).

More recent intervention studies have targeted AS levels specifically. For example, Schmidt and colleagues (in press) conducted a study of young women with above average levels of AS (mean ASI score=26) who were randomly assigned to a risk reduction or control condition. Both conditions involved a 30-minute presentation delivered via computer followed by 10 minutes with an experimenter. In the risk reduction condition, participants reviewed information related to stress and arousal, and interoceptive exposure exercises were explained. In the control condition, participants did not discuss anxiety-related issues, but focused only on general health and nutrition. The risk reduction group showed a 30% reduction in AS levels at post-treatment – a reduction that was maintained at 1-year follow-up. The control group showed no reduction of AS over time.

We developed a brief CBT program designed specifically to target clinically elevated

levels of AS (see Watt, Stewart, Conrod, & Schmidt, in press-a for more detailed information). Female undergraduates who indicated a willingness to participate in a psychology experiment were eligible to participate if found to score high or low in AS in a mass screening (i.e. > 1 SD above or below screening sample ASI mean for women). Women only were selected as participants to reduce variability due to gender and because women have higher AS levels, especially physical concerns, than men (Stewart et al., 1997). Potential participants were told that the study was investigating the effectiveness of a new program for helping people with their sensitivity to anxiety. High AS and low AS participants were assigned by randomized number to either an active CBT or non-specific treatment (NST) condition. The NST condition was included to control for effects of group or therapist exposure that could influence results and, consistent with the approach used by others (e.g. Harrington & Telch, 1994), consisted of discussion about ethics in psychology. The CBT program included 3 50-minute sessions conducted in small group format (6–10 participants) over 3 consecutive days. During the first session, participants learned about anxiety, panic attacks, AS, and the anxiety cycle. They learned how their interpretations of arousal sensations could affect their reaction to the sensations (e.g. avoidance behavior). During the second session, participants were taught strategies to identify, challenge, and restructure their dysfunctional thoughts consistent with accepted cognitive therapy for panic disorder (Craske & Barlow, 2001). The third session included a novel interoceptive exposure component of aerobic exercise (running) intended to expose the individual to feared arousal-related sensations with the goal of habituation. Running was selected because it was an activity that could be easily performed in a group format and could be assigned as homework to be performed prior to follow-up. Outcome measures were taken immediately prior to and following the intervention, plus 10 weeks post-intervention. As predicted, results indicated a significant reduction in AS levels only among participants with high pre-morbid AS levels assigned to the CBT condition (see Watt et al., in press-a).

The primary objective of the present study was to test whether our brief CBT

intervention, found to reduce AS levels, could also reduce pain anxiety levels. It was first hypothesized that high AS participants would report more pain-related anxiety pre-intervention as compared to low AS participants. The second hypothesis was that high AS – CBT participants would reveal a greater reduction in pain-related anxiety from pre- to post-intervention as compared to the other 3 groups in the 2×2 between-subjects design. Finally, change in AS levels from pre- to post-treatment were hypothesized to mediate the expected effects of CBT in reducing pain anxiety in high AS women.

Method

Participants

A total of 221 first-year undergraduate women from 2 universities in eastern Canada participated in the brief CBT intervention. Participants were selected based on their ASI scores obtained during in-class screening. Women in the high AS and low AS groups scored at least 1 one SD above, or below, the mean ASI screening score for females (i.e. 17.9 ± 8.7). Consistent with other studies in the AS and pain area (e.g. Keogh & Mansoor, 2001), an extreme groups approach was chosen to increase the probability of detecting AS effects with a relatively small sample size, and to deal with the possibility that the intervention may only be relevant for those with relatively high (or "clinically significant") AS levels.

Of the 221 students who participated in the brief CBT intervention study, 186 were invited to participate in a separate post-treatment study regarding pain-related anxiety and completed the baseline pain anxiety measure. (Data collection for the pain anxiety study started in the second year of the intervention study; consequently, not all participants in the brief CBT intervention completed the pain anxiety measure.) Exclusion criteria included chronic health conditions (e.g. heart conditions) and medication to manage anxiety or pain. The mean (and SD) ASI scores at pre-treatment for the low AS and high AS groups in the present study were 8.3 (SD=3.6) and 34.2 (SD=6.4), respectively. The participants' mean age was 18.9 (SD=2.0) years (range 17–33 years). The majority were first year undergraduate students (94%) and Caucasian

(92%). A set of 2×2 (AS group \times Treatment condition) analyses of variance (ANOVAs) and χ^2 analyses revealed no significant effects for any demographic variables. Cell sizes were: high AS/CBT=42; low AS/CBT=50; high AS/NST=51; low AS/NST=43.

Measures

Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a 16-item self-report questionnaire. Each item inquires about the amount of fear the participant experiences in regard to bodily sensations commonly associated with anxiety. Participants are asked to rate each item on a 5-point Likert-like scale ranging from *very little* (0) to *very much* (4). The ratings on the 16 items are summed for a total ranging from 0 to 64. Studies have found support for test-retest reliability, criterion validity, and construct validity (e.g. support for the distinction between AS and trait anxiety) (Peterson & Reiss, 1992). The pre-treatment ASI score in the present study showed excellent internal consistency (Cronbach's $\alpha=0.91$).

Pain Anxiety Symptoms Scale-20 (PASS-20; McCracken & Dingshra, 2002). The PASS-20 is a short version of the 40-item PASS (McCracken et al., 1993) that measures fear and anxiety responses specific to pain (see also Carleton et al., this issue). The PASS-20 has 4 5-item subscales that measure Avoidance, Fearful Thinking, Cognitive Anxiety, and Physiological Responses to Pain, respectively. Participants rate each item on a 6-point scale ranging from *never* (0) to *always* (5). Reliability analyses with PASS-20 indicate good internal consistency akin to the PASS-40 (McCracken & Dingshra, 2002) with alphas ranging from 0.75 to 0.86 for the 4 subscales and a mean alpha of 0.81. Psychometric analyses reveal good convergent, discriminant, predictive and construct validity. Internal reliability analyses of the pre-treatment PASS-20 total scale in the present study yielded an excellent Cronbach's alpha of 0.95 with subscale alphas ranging from 0.76 to 0.91 indicating good to excellent reliability.

Procedure

Female undergraduates at 2 eastern Canadian universities participated. Testing the brief

CBT on women only allowed for the control of gender effects, but also fits with literature findings that women have higher AS levels than men (Stewart et al., 1997) and are particularly sensitive to pain (Keogh, Hatton, & Ellery, 2000), and that AS effects on pain may be specific to women (Keogh & Birkby, 1999). Participants were selected for the brief CBT study based on their ASI scores collected during an in-class mass screening. A battery of questionnaires was administered pre- and post-intervention. For the purposes of the present study, only PASS-20 scores collected immediately pre- and post-intervention, and ASI scores collected at screening and post-intervention were examined.

Results

A multivariate analyses of variance (MANOVA) was conducted to test the hypothesis that high AS participants would report more pain anxiety pre-intervention as compared to low AS participants. Cohen's d was used as an estimate of the effect size, with a value greater than 0.80 representing a large effect, values between 0.50 and 0.80 representing a moderate effect, and values between 0.20 and 0.50 representing a small effect (Cohen, 1988); anything smaller than 0.20 is regarded as inconsequential (Cohen, 1992). The results revealed a significant main effect of AS group in terms of PASS-Total and the 4 PASS-20 subscale scores at pre-treatment ($F(4,182)=34.36, p<0.001$). High AS participants ($n=93$) scored significantly higher than low AS participants ($n=93$) on the PASS-20 Total score as well as on the 4 subscales of Avoidance, Fearful Thinking, Cognitive Anxiety, and Physiological Anxiety (see Table 1). The scores of the high AS participants in the present study are comparable to those of a sample of chronic pain patients included in the PASS-20 validation study (McCracken & Dingshra, 2002; see Table 1).

A $2 \times 2 \times 2$ mixed model ANOVA with AS group (high AS vs low AS) and treatment condition (CBT vs NST) as the between-subjects factors, and time (pre- vs post-intervention) as the within-subjects factor, was conducted on PASS-20 Total scores to test the second hypothesis. There was a significant Treatment condition \times Time interaction ($F(1,160)=8.60, p<0.001$), which

Table 1. Means, standard deviations, and results of ANOVA with PASS-20 Total and subscale scores at pre-treatment.

	High AS <i>n</i> =40	Low AS <i>n</i> =50	ANOVA			McCracken & Dinghra (2002) <i>n</i> =282
	M (SD)	M (SD)	<i>F</i> (1,185)	<i>p</i>	<i>Cohen's d</i>	M (SD)
PASS-20 Total	46.60 (18.37)	21.18 (12.21)	123.47	0.001	1.63	38.62 (20.38)
Avoidance	11.82 (5.66)	6.71 (3.95)	51.12	0.001	1.05	12.84 (6.11)
Fearful Thinking	8.51 (4.89)	2.84 (2.76)	94.68	0.001	1.43	7.37 (6.38)
Cognitive Anxiety	15.58 (5.72)	7.42 (4.54)	116.27	0.001	1.58	12.27 (6.73)
Physiological Anxiety	10.69 (5.24)	4.22 (3.41)	99.60	0.001	1.46	6.15 (5.69)

PASS-20=20-item Pain Anxiety Symptoms Scale (PASS; McCracken & Dinghra, 2002).

was qualified by the hypothesized significant 3-way interaction (AS group \times Treatment condition \times Time) ($F(1,160)=4.15$, $p<0.05$; see Figure 1). Simple effects analysis revealed that the Treatment condition \times Time effect was specific to the high AS group ($F(1, 78)=8.72$, $p<0.01$) as opposed to the low AS group ($F(1,82)=0.64$, n.s.). Tests of simple main effects of Time among the high AS participants assigned to each Treatment condition confirmed that the significant reduction in PASS-20 Total scores from pre- to post-treatment for the high AS group was specific to the CBT condition ($F(1,33)=14.18$, $p<0.001$) as opposed to the NST/control condition ($F(1,45)=0.72$, n.s.) (see Figure 1). The magnitude of the effect of the CBT intervention in reducing pain anxiety among high AS participants from pre- to post-treatment ($d=0.45$) indicated a small-to-moderate effect.

To test whether a reduction in AS levels as a result of the brief CBT would mediate the effects of the intervention on pain anxiety, mediation analyses were performed (Baron &

Kenny, 1986). First, difference-scores (pre-minus post-intervention) were calculated for both the PASS-20 Total and the ASI to construct a single score reflecting the magnitude of the intervention effect for each construct. A set of 2×2 (AS group \times Treatment condition) between-subjects ANOVAs was subsequently conducted on these change scores. For the analysis of PASS-20 Total change scores, there was a significant effect of Treatment condition ($F(3,160)=8.60$, $p<0.01$), qualified by a significant AS group \times Treatment condition interaction ($F(3,160)=4.15$, $p<0.05$). Simple effects analyses showed that the Treatment condition effect was specific to high AS participants ($F(1,78)=8.71$, $p<0.01$) as opposed to the low AS participants ($F(1,82)=0.64$, n.s.). High AS participants in the CBT condition revealed a significantly greater change in PASS-20 Total scores from pre- to post-intervention as compared to high AS participants in the NST condition (see Table 2); this was a moderate-to-large effect of the intervention on pain anxiety ($d=0.65$).

A significant 2-way (AS group treatment condition) interaction also was found for ASI-change scores ($F(3,160)=3.54$, $p<0.05$). Analyses of simple effects revealed that the AS group effect was specific to the CBT condition ($F(1,79)=8.75$, $p<0.01$) as opposed to the NST/control condition [$F(1,81)=1.68$, n.s.]. High AS participants showed greater ASI change from pre- to post-treatment than low AS participants, but only in the CBT Treatment condition (see Table 2); again, this was a moderate-to-large effect ($d=0.67$).

Finally, a 2 (AS group) \times 2 (treatment condition) analysis of covariance (ANCOVA)

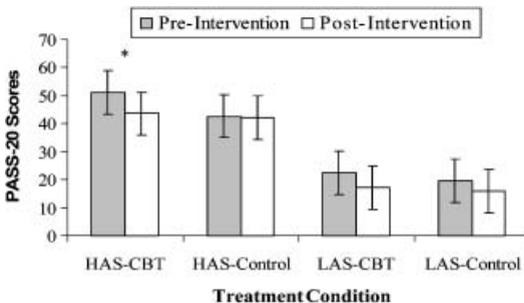


Figure 1. Changes in pain anxiety from pre- to post-intervention.

Table 2. *Mediational analyses: mean (and SE) PASS-20 total and ASI change scores as functions of AS group (High AS vs Low AS) and treatment condition (CBT vs NST).*

AS group	Treatment condition	PASS-20 – Change scores	ASI – Change	PASS-20 – Change scores
		without covariate	scores	with covariate
		M (SE)	M (SE)	M (SE)
HAS	CBT	8.1 (1.5) ^a	6.7 (0.6) ^b	7.4 (1.5)
	NST	1.1 (1.3) ^a	4.9 (0.7)	0.8 (1.3)
LAS	CBT	4.9 (1.3)	3.0 (0.7) ^b	5.3 (1.3)
	NST	3.7 (1.5)	3.9 (0.8)	3.9 (1.5)

PASS-20=20-item Pain Anxiety Symptoms Scale (McCracken & Dinghra, 2002); ASI=Anxiety Sensitivity Index (Peterson & Reiss, 1992). Change scores calculated as pre-treatment mean minus post-treatment mean. Greater scores indicate greater change over the course of the intervention. Means with similar superscripts are significantly different from one another ($p < 0.05$). AS=anxiety sensitivity; CBT=cognitive behavior therapy; NST=non-specific treatment; HAS=high AS; LAS=low AS; M=mean; SE=standard error.

was conducted using PASS-20 Total change scores as the dependent variable and ASI change scores as the covariate. This analysis continued to reveal a significant main effect of Treatment condition ($F(4,158)=8.40, p < 0.01$). However, the crucial AS group \times Treatment condition interaction for PASS-20 Total change scores was eliminated [$F(4,158)=3.63, n.s.$] when controlling for ASI change scores. This provides evidence of AS change as a mediator of the effect of CBT in reducing pain anxiety among high AS participants. Covariate adjusted means (i.e. PASS-20 Total change scores adjusted for change in ASI scores) are presented in the final column of Table 2.

Discussion

Consistent with predictions and the purported theoretical link between AS and pain, high AS young women reported significantly higher levels of overall pain anxiety pre-intervention, as compared to their low AS counterparts. High AS young women reported significantly more cognitive anxiety related to pain, more fearful appraisals of pain, more escape and avoidance responses intended to reduce pain, and more physiological anxiety symptoms related to the experience of pain as compared to low AS young women. The large effect sizes are consistent with other studies (e.g. Asmundson & Norton, 1995; Asmundson et al., 1999) which have found high AS individuals to report more cognitive anxiety, fearful appraisals of pain, and pain-related escape/avoidance behaviour than low AS individuals.

In contrast to previous findings, high AS participants in the present study also reported significantly higher levels of physiological anxiety as compared to low AS participants. It is possible that this finding is attributable to the gender composition of the present sample. Whereas most other studies have looked at both men and women (e.g. Asmundson & Norton, 1995; Asmundson et al., 1999), the present study included women only. Women, as compared to men, tend to report more AS physical concerns (Stewart et al., 1997). Keogh and Birkby (1999) found that high AS women reported greater sensory pain than high AS men and low AS individuals, as indicated by their selection of pain descriptors such as "throbbing," "stabbing," and "aching". It is also possible that the relatively larger sample size in our study (c.f. Asmundson & Norton, 1995; Asmundson et al., 1999) provided the power needed to detect a difference between high and low AS participants in physiological anxiety related to pain.

A primary objective of the present study was to determine whether a brief CBT intervention found to reduce AS levels could also reduce pain-related anxiety. A moderate effect of treatment was found (average $d=0.55$). High AS individuals who were randomly assigned to participate in the CBT program showed a significant reduction in overall pain anxiety scores as compared to high AS individuals in the control (NST) condition or low AS individuals in either treatment condition. Moreover, mediator analyses supported the intervening role of

AS in explaining fear of pain: CBT effects on fear of pain among high AS participants were eliminated when controlling for AS change due to the intervention. This finding supports Asmundson et al.'s (1999) contention that high AS may increase the risk of developing high levels of pain-related fear because high AS individuals are more apt to fear the consequences of the pain sensations. Indeed, AS appears to be one of the factors that mediate fear of pain and ultimately contribute to the etiology of chronic pain (see Asmundson et al., 2005).

Studies have found that regardless of pain severity, ASI scores of chronic pain patients correlate positively with cognitive anxiety related to pain, escape/avoidance tendencies, and fear of the negative consequences that may accompany pain (see Asmundson & Norton, 1995). Given the findings of the present study and the fact that high AS individuals seem predisposed to report higher levels of sensory and affective pain (see Keogh & Mansoor, 2001), CBT targeting AS would seem to be advisable as a pain management approach. Targeting cognitions that can exacerbate AS could work to prevent the development of chronic and persistent pain. Moreover, reducing AS levels may yield positive benefits in terms of the use of analgesics given that chronic pain patients with high AS report more continued use of analgesics as compared to chronic pain patients with moderate and low AS (Asmundson & Norton, 1995).

Perhaps, if the educational component of the present intervention had focused more on the relationship between AS and pain sensations, and pain-related cognitions had been targeted for restructuring, treatment effects might have been even more pronounced. With regards to the exposure component of the intervention, it remains to be determined whether the more effective strategy would involve exposure to avoided physical activity or the anxiety sensations presumed to underlie the pain anxiety. Certainly, some success has been reported in relieving pain-related cognitions and fear of pain when patients have been gradually exposed to feared activity (Vlaeyen, de Jong, Geilen, Heuts, & Breukelen, 2001). Greenberg and Burns (2003), however, caution against attempting to treat pain anxiety as if it was a specific phobia rather than a

manifestation of AS. Assuming that high AS people fear pain because it triggers the physiological sensations (e.g. dizziness) about which they form catastrophic appraisals, then reducing pain anxiety will require a treatment similar to the present brief CBT involving deliberate exposure to symptoms of anxiety vs avoided activity. Future research could directly compare the efficacy of direct treatment of pain anxiety (via graded *in vivo* exposure to feared pain-related activities; Vlaeyen et al., 2001) vs our indirect approach (via treatment of AS). Studies could also examine the efficacy of combining these 2 approaches to determine if the combination has any incremental benefits.

The present study was not without its limitations. The first limitation was the lack of follow-up, which means we do not know the durability of the intervention effects. Because the larger project in which this study was embedded was interested in the effects of our intervention on a variety of outcome measures (see Watt, Stewart, Birch, & Bernier, *in press-b*) and because participants' time was limited, follow-up data for each of the outcome measures was not collected at all time points. The outcome of interest in this investigation (i.e. PASS-20 scores) was collected only at pre- and post-treatment, not at follow-up. A second limitation is the homogeneity of the sample. Given that the participants in the present study were exclusively female undergraduates and predominantly Caucasian, it is possible that the findings may not be generalizable to other populations. The magnitude of the treatment effect might be more muted with men who tend to report lower AS (Stewart et al., 1997) and less pain (Keogh et al., 2000).

Another study limitation involves the use of self-report measures; people may not be as good at assessing their own behavior as they would like to think. For example, high AS individuals may not be truly aware of the extent of their pain-related avoidance behavior. Future research would benefit by accessing cross-validated data (e.g. observer data), as well as using other types of methodology to better assess the theoretically-relevant domains of cognitive, physiological, and overt behavioral responding. These could include the use of implicit cognitive processing tasks to assess the cognitive domain without

contamination by various self-report biases (Keogh, Thompson, & Hannent, 2003). Furthermore, it is possible that the association between AS and pain anxiety is inflated by contextual overlap between the measures. For example, Lilienfeld, Turner, and Jacob (1996) have proposed that AS and illness/injury sensitivity (IS), both "fundamental fears", are lower-order factors nested within the higher-order factor of trait anxiety. In turn, Keogh and Asmundson (2004) have suggested that IS acts as a higher-order factor of pain-related constructs, including pain anxiety. Certainly, future research would benefit by investigating the links between IS and pain anxiety, and comparing the relative degrees of relation between IS and AS with pain anxiety. A final limitation concerns the lack of data pertaining to participants' medical and psychiatric diagnostic status. We did not exclude individuals with panic disorder or chronic pain, for example, so the extent to which the promising results are due to the presence of such individuals cannot be determined.

In conclusion, the findings from this study support the purported association between AS and pain anxiety. Moreover, this study provides evidence for the efficacy of a brief CBT program designed specifically to reduce AS (Watt et al., in press-a) in lowering pain anxiety. Results hold implications for the prevention and treatment of chronic pain given that research has shown that anxiety related to pain symptoms is associated with the suffering and disability of chronic pain (see Asmundson, 1999a; 1999b). Participants in this brief CBT intervention learned strategies for reducing tendencies to misinterpret and catastrophize about the consequences of arousal-related sensations, not pain sensations specifically. Nonetheless, they exhibited a significant reduction in anxiety related to pain. The positive effects of this intervention on pain anxiety could be the result of participants generalizing their learning to pain, or because the reduced fear of arousal sensations impacts on fear of pain. The skills-based nature of this program would seem to be amenable for use in a variety of settings, including outpatient clinics or hospitals. A theory-driven approach to pain treatment permits matching to specific patient characteristics (e.g. high AS). Finding positive effects

from such a brief (3-hour) intervention is especially appealing given the increasing demands for more time- and cost-efficient provision of healthcare.

Acknowledgements

The authors are grateful to Kerry MacSwain and James Brazeau for their research assistance. This research was supported by a grant from the Nova Scotia Health Research Foundation (NSHRF) to the first and second authors. The second author is supported through an Investigator Award from the Canadian Institutes of Health Research and a Killam Professorship from the Dalhousie University Faculty of Science. The third and fourth authors are supported by graduate studentships from the NSHRF.

References

- Asmundson, G. J. G. (1999a). Anxiety sensitivity and chronic pain: Empirical findings, clinical implications, and future directions. In S. Taylor (Ed.), *Anxiety Sensitivity: Theory, Research, and Treatment of the Fear of Anxiety* (pp. 269–285). Mahwah, NJ: Erlbaum.
- Asmundson, J. G. J. (1999b). Beyond pain: The role of fear and avoidance in chronicity. *Clinical Psychology Review*, *19*, 97–119.
- Asmundson, J. G. J., & Norton, R. (1995). Anxiety sensitivity in patients with physically unexplained chronic back pain: A preliminary report. *Behaviour Research and Therapy*, *33*, 771–777.
- Asmundson, J. G. J., Norton, P. J., & Veloso, F. (1999). Anxiety sensitivity and fear of pain in patients with recurring headaches. *Behaviour Research and Therapy*, *37*, 703–713.
- Asmundson, J. G. J., & Taylor, S. (1996). Role of anxiety sensitivity in pain-related fear and avoidance. *Journal of Behavioural Medicine*, *19*, 577–586.
- Asmundson, J. G. J., Vlaeyen, J. W. S., & Crombez, G. (2004). *Understanding and Treating Fear of Pain*. Oxford: Oxford University Press.
- Asmundson, G. J. G., Wright, K. D., & Hadjistavropoulos, H. D. (2005). Hyper-vigilance and attentional fixedness in chronic musculoskeletal pain: Consistency of findings across modified Stroop and dot-probe tasks. *Journal of Pain*, *6*, 497–506.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.

- Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112, (1), 155–159.
- Craske, M., & Barlow, D. H. (2001). Panic disorder and agoraphobia. In D. H. Barlow (Ed.), *Clinical Handbook for Psychological Disorders: A Step-by-Step Treatment Manual* (3rd edn) (pp. 1–59). New York, NY: Guilford Press.
- Greenberg, J., & Burns, J. W. (2003). Pain anxiety among chronic pain patients: Specific phobia or manifestation of anxiety sensitivity? *Behaviour Research and Therapy*, 41, 223–240.
- Harrington, P. J., & Telch, M. J. (1994). Lowering anxiety sensitivity in non-clinical subjects [Summary]. *Convention Proceedings for the 28th Annual Convention of the Association for Advancement of Behavior Therapy (AABT)*, 23.
- Keogh, E., & Asmundson, G. J. G. (2004). Negative affectivity, catastrophizing, and anxiety sensitivity. In G. Asmundson, J. Vlaeyen, & G. Crombez (Eds), *Understanding and Treating Fear of Pain* (pp. 91–115). New York: Oxford University Press.
- Keogh, E., & Birkby, J. (1999). The effect of anxiety sensitivity and gender on the experience of pain. *Cognition and Emotion*, 13, 813–829.
- Keogh, E., & Mansoor, L. (2001). Investigating the effects of anxiety sensitivity and coping on the perception of cold pressor pain in healthy women. *European Journal of Pain*, 5, 11–25.
- Keogh, E., Hatton, K., & Ellery, D. (2000). Avoidance versus focused attention and the perception of pain: differential effects for men and women. *Pain*, 85, 225–230.
- Keogh, E., Thompson, T., & Hannent, I. (2003). Selective attentional bias, conscious awareness and the fear of pain. *Pain*, 104, 85–91.
- Kuch, K., Cox, B. J., Evans, R., & Shulman, I. (1994). Phobias, panic, and pain in 55 survivors of road vehicle accidents. *Journal of Anxiety Disorders*, 8, 181–187.
- Kuch, K., Cox, B. J., Woszczyzna, C. B., Swinson, R. P., & Shulman, I. (1991). Chronic pain in panic disorder. *Journal of Behaviour Therapy and Experimental Psychiatry*, 22, 255–259.
- Lilienfeld, S. O., Turner, S. M., & Jacob, R. G. (1996). Further comments on the nature and measurement of anxiety sensitivity: A reply to Taylor (1995b). *Journal of Anxiety Disorders*, 10, 411–424.
- McCracken, L. M., & Dhingra, L. (2002). A short version of the Pain Anxiety Symptoms Scale (PASS-20): Preliminary development and validity. *Pain Research and Management*, 7, 45–50.
- McCracken, L. M., Zayfert, C., & Gross, R. T. (1993). The Pain Anxiety Symptoms Scale: Development and validation of a scale to measure the fear of pain. *Pain*, 50, 67–73.
- Norton, P. J., & Asmundson, G. J. G. (2004). Anxiety sensitivity, fear, and avoidance behaviour in headache pain. *Pain*, 111, 218–223.
- Penava, S. J., Otto, M. W., Maki, K. M., & Pollack, M. H. (1998). Rate of improvement during cognitive-behavioural group treatment for panic disorder. *Behaviour Research and Therapy*, 36, 665–673.
- Peterson, R. A., & Reiss, S. (1992). *Anxiety Sensitivity Index Manual* (2nd edn). Worthington, OH: International Diagnostic Systems.
- Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review*, 11, 141–153.
- Schmidt, N. B., Lerew, D. R., & Jackson, R. J. (1997). The role of anxiety sensitivity in the pathogenesis of panic: Prospective evaluation of spontaneous panic attacks during acute stress. *Journal of Abnormal Psychology*, 106, 355–364.
- Schmidt, N. B., Eggleston, A. M., Woolaway-Bickel, K., Fitzpatrick, K. K., Vasey, M. W., & Richey, J. A. (in press). Anxiety sensitivity amelioration training (ASAT): A longitudinal primary prevention program targeting cognitive vulnerability. *Journal of Anxiety Disorders*.
- Stewart, S., Taylor, S., & Baker, J. (1997). Gender differences in dimensions of anxiety sensitivity. *Journal of Anxiety Disorders*, 11, 179–200.
- Telch, M. J., Lucas, J. A., Schmidt, N. B., Hanna, H. H., Jaimex, T. L., & Lucas, R. A. (1993). Group cognitive-behavioural treatment of panic disorder. *Behaviour Research and Therapy*, 31, 279–297.
- Vlaeyen, J. W. S., de Jong, J., Geilen, M., Heuts, P. H. T. G., & Breukelen, G. (2001). Graded exposure in vivo in the treatment of pain-related fear: A replicated single-case experimental design in four patients with chronic low back pain. *Behaviour Research and Therapy*, 39, 151–166.
- Watt, M. C., Stewart, S. H., Conrod, P., & Schmidt, N. B. (in press-a). Personality-based approaches to treatment of co-morbid anxiety and substance use disorder. In S. H. Stewart, & P. Conrod (Eds), *Co-morbid Anxiety and Substance Use Disorders: Theoretical and Treatment Issues*. New York, NY: Springer.
- Watt, M. C., Stewart, S. H., Birch, C. D., & Bernier, D. B. (in press-b). Brief CBT for high anxiety sensitivity decreases drinking and drinking problems: Evidence from a randomized controlled trial. *Journal of Mental Health*.