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Anxiety sensitivity and fear of pain in patients with recurring headaches

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Abstract

Anxiety sensitivity (AS) plays an important role in the cognitive, affective and behavioral profiles of patients with chronic pain related to musculoskeletal injury. However, investigators have not considered whether these findings extend to patients with other classes of chronic pain. The primary purpose of this investigation was to address this issue in 72 patients with recurring headaches who completed a self-report questionnaire inventory during a treatment visit to an outpatient neurology clinic. The mean ASI score for the group (mean = 24; SD = 11) was relatively high. When patients were classified on the basis of ASI scores, 20 (28%) met criteria for high, 41 (57%) for medium and 11 (15%) for low AS. Multivariate analysis of variance confirmed that these groups differed on specific aspects of their cognitive, affective, and behavioral profiles. High AS patients reported greater depression, trait anxiety, pain-related escape/avoidance behavior and fearful appraisals of pain than did patients with medium or low AS. High AS patients also indicated greater cognitive disruption in response to pain than did patients with low AS. Groups did not differ in headache severity, physiological reactivity, change in lifestyle, anger, nor did they differ in use of over-the-counter or prescribed analgesics. Multiple regression analysis identified AS, pain-related cognitive disruption, and sensory pain experience as significant predictors of fear of pain. Lifestyle changes attributed to headache were, on the other hand, predicted by headache severity, physiological and cognitive anxiety and escape/avoidance behavior. These results provide further evidence of the important association between AS and fear responses of patients with chronic pain syndromes. Implications and future directions are discussed.

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1. Introduction

Avoidance behavior (Fordyce, 1976) and fear of pain (Lethem et al., 1983; Philips, 1987; McCracken et al., 1992; Vlaeyen et al., 1995) have been proposed as a mechanism through which pain may be maintained over time. The basic fear and avoidance model can be conceptualized as a vicious cycle in which negative expectancies about the harmfulness of pain initiate avoidance behavior which, in turn, contributes to deconditioning (e.g. muscular atrophy, decreased mobility, weight gain), further pain experiences, negative expectancies and strengthened avoidance. Rachman and Arntz (1991) have suggested that, much like generally anxious and phobic patients, the avoidance behavior of patients with chronic pain is related to inaccurate expectancies.

Numerous empirical investigations have addressed the postulates of the fear and avoidance models and, for the most part, have provided confirming evidence (for review see Asmundson et al., 1998). To illustrate, McCracken et al. (1992) have indicated that fear of pain is a strong predictor of disability and interference with activities of daily living. These investigators have also shown fear of pain to predict restricted range of motion during passive straight leg raise testing (McCracken et al., 1993). In a similar vein, Waddell et al. (1993) have reported that pain-related fear specific to work activities is a stronger predictor of disability than biomedical indices (e.g. anatomical pattern, time course and severity). Recent investigations (e.g. Vlaeyen et al., 1995; Crombez et al., 1998) are in accord with this general pattern of findings.

It has recently been proposed that fear of pain may be mediated, at least in part, by a predispositional tendency to fear somatic perturbations and the negative expectancies associated with these perturbations (see Asmundson, 1998). More specifically, people may come to fear pain because they fear the consequences of the affective and somatic arousal that it produces (Asmundson & Taylor, 1996). This tendency to be(come) fearful has been operationalized by the anxiety sensitivity (AS) construct (Reiss & McNally, 1985; Reiss, 1991) and is measured by the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI is a 16-item self-report questionnaire comprising questions such as "It scares me when I feel faint", "It is important for me to stay in control of my emotions" and "Unusual body sensations scare me". Items are scored using a scale ranging from 0 = very little to 4 = very much.

Several studies provide preliminary evidence indicating that AS does influence fear and other dimensions of the pain experience in patients with chronic musculoskeletal pain. In the first such study, Asmundson and Norton (1995) assessed 70 patients with chronic back pain using a self-report battery comprising a number of questionnaires including the ASI and the Pain Anxiety Symptoms Scale (PASS; McCracken et al., 1992). The latter is a 40-item self-report measure designed to elicit information regarding cognitive and physiological anxiety associated with pain, pain-specific escape and avoidance behaviors and general fearful appraisals of pain. Results indicated that, despite similar pain severity ratings, patients with high AS scored higher on measures of negative affect as well as on pain-specific cognitive anxiety and fearful appraisals than did those with medium or low levels. It was also observed that 71% of patients with high AS, compared to 34 and 25% of patients with medium and low AS, reported current use of analgesic medication to relieve pain symptoms.

Most recently, Asmundson and Taylor (1996) used structural equation modeling to test the predictions that (a) AS directly exacerbates fear of pain and (b) AS indirectly exacerbates pain-

related avoidance via its effects on fear of pain. Additional analyses were conducted to assess the impact of pain severity on these variables. Participants in this study were 259 patients with complaints of chronic pain related to musculoskeletal injury. The results supported both predictions, indicating that AS accounted for 30% of the variance in fear of pain which, in turn, accounted for 68% of the variance in escape/avoidance behavior. Pain severity accounted for an additional 13% of the variance in fear of pain, but did not contribute significantly to the prediction of escape/avoidance. This finding, combined with the preliminary observations of Asmundson and Norton (1995), provide foundational support for the hypothesis that high AS exacerbates fear of pain and thereby promotes avoidance behavior.

The study of the relationship between AS and fear of pain is, nonetheless, in its infancy. Among numerous unanswered questions, is whether the findings obtained from samples of patients with chronic pain stemming from musculoskeletal injury will generalize to patients with other chronic pain syndromes, such as recurring headache (Asmundson, 1998). The literature indicates that fear of pain is elevated in patients with recurrent and persistent headaches (Hursey & Jacks, 1992), that it disrupts cognition and behavior independent of headache frequency, severity or duration (Hursey & Jacks, 1992) and that it may play a role in perpetuating headache pain (Philips & Jahanshahi, 1986). To date, however, there have been no investigations of the role of AS in pain syndromes other than that associated with musculoskeletal injury.

The purpose of the present investigation was three-fold. First, in an effort to extend the limited knowledge of fear and avoidance in patients with headache, we sought to replicate the findings of Asmundson and Norton (1995) in a sample of patients with recurrent headaches. As such, our primary hypothesis was that headache patients with high AS would report more adverse cognitive, affective, and behavioral experiences when compared to patients with medium and low AS. We also hypothesized that patients with high AS would be more likely to use analgesic medications than those with medium and low AS. This is based on the profile of medication use observed by Asmundson and Norton (1995) and indications that high AS is associated with the use of substances that reduce arousal (McNally, 1996; Norton et al., 1997). Finally, we assessed the contribution of various cognitive, affective and behavioral variables in predicting fear of pain and lifestyle changes attributed to headache. Our assumption here was that the identified predictors may represent appropriate and specific targets towards which to direct cognitive-behavioral and other interventions.

2. Method

2.1. Subjects and subject classification

Participants in the investigation were 72 patients with frequent and recurring headaches who completed a self-report questionnaire inventory following a treatment visit to an outpatient neurology clinic. The majority of participants (85%) were female. They ranged in age from 17 to 77 years (mean age = 43.1 years; S.D. = 11.9 years) and reported being bothered by frequent and recurrent headaches for a mean duration of 193.1 months (S.D. = 159.4 months; range = 9–600 months). Their headaches were primarily common migraine (n = 55; 76%),

followed by chronic daily (n = 9; 13%), cluster (n = 5; 7%), tension-type (n = 2; 3%) and psychogenic (n = 1; 1%). Typical headache severity was 3.8 (S.D. = 1.0; range = 1 to 5), as reported on a standard headache intensity scale ranging from 0 = no headache to 5 = extremely painful. 67 (93.1%) of the patients reported taking medications for their headaches and, of these, 18 were taking over-the-counter analgesics, 20 were taking prescribed medications and 29 were taking both.

To address our primary hypothesis, we classified patients on the basis of their scores on the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992), a 16-item measure of the fear of anxiety signs and symptoms that has demonstrated reliability and validity (see McNally, 1996). The literature contains several procedures for designating individuals as having high, medium or low AS. For example, some investigators have divided groups on the basis of the mean plus or minus one standard deviation for high and low AS groups, respectively (Carr et al., 1994; Schmidt et al., 1997). Others have used normative data (Donnell & McNally, 1990; Asmundson et al., 1994). As our mean score for the group was relatively high and was accompanied by a large standard deviation (mean = 23.8; S.D. = 11.4) we chose the later approach, defining our upper bound by ASI scores of $\geq$30, our lower bound by scores of $\leq$12 and our medium bound as scores of 13 to 29. 20 patients (28%) met criteria for high AS, 41 (57%) for medium AS and 11 (15%) for low AS.

2.2. Materials and procedure

During a scheduled treatment visit, participants were provided a verbal and written explanation of the investigation and its purposes. They then provided written informed consent. Subsequently, they were given a questionnaire inventory to complete. This inventory included the following self-report questionnaires.

2.2.1. Headache questionnaire (HQ)

The HQ was developed for use in this investigation. It includes questions regarding the amount of time patients have been bothered by recurrent headaches, the severity of their typical headache (0 = no headache to 5 = extremely painful), past and current prescription and over-the-counter medication use, other bodily pain and its duration, the disturbing/distressing nature of headaches (0 = not at all to 4 = extremely) and the degree to which headaches have restricted or changed ones lifestyle (0 = no change to 4 = extreme change).

2.2.2. Anxiety sensitivity index (ASI)

As previously described, the ASI (Peterson & Reiss, 1992) comprises 16 items designed to measure the fear of anxiety signs and symptoms. Recently, some investigators have suggested that the ASI may not only assess fear of anxiety per se, but other arousal states such as those associated with atypical, unpredictable or aversive somatic sensations (Asmundson, 1998; Cox et al., 1998). The ASI has demonstrated excellent reliability and validity (see Peterson & Reiss, 1992; McNally, 1996).

2.2.3. McGill pain questionnaire, short form (MPQ-SF)

The MPQ-SF (Melzack, 1987) comprises a 15-item adjective checklist and two scales for rating pain severity. Melzack (1987) has shown that the MPQ-SF possess good reliability and validity. The sensory pain subscale of the adjective checklist was of interest in the present investigation.

2.2.4. Pain anxiety symptoms scale (PASS)

The PASS (McCracken et al., 1992, 1993) is a 40-item questionnaire comprising 3 subscales that assess pain-related cognitive anxiety, physiologic anxiety, escape/avoidance behaviors, as well as a subscale assessing general fear of pain. Although some changes have been suggested to improve factorial validity (Larsen et al., 1997), the PASS possesses good reliability and validity (McCracken et al., 1992, 1993).

2.2.5. Beck depression inventory (BDI)

The BDI (Beck et al., 1961) is a 21-item measure of depressive symptoms. The psychometric properties of the BDI have been extensively evaluated (Beck, Steer, & Garbin, 1988) and have shown excellent reliability and validity.

2.2.6. State-trait anxiety inventory, trait form (STAI-T)

The STAI-T (Spielberger, Gorsuch, & Luschene, 1970) comprises 20-items designed to assess trait anxiety. The STAI-T possess strong psychometric properties (Spielberger et al., 1993).

2.2.7. State-trait anger expression inventory, trait form (STAXI-T)

The STAXI-T (Spielberger et al., 1985; Spielberger, 1988) is a 10 item questionnaire designed to assess trait anger. The available data suggest that the STAXI-T has good reliability and validity (Spielberger et al., 1985).

3. Results

3.1. ASI and self-report data

To address our primary hypothesis, we conducted a one way multivariate analysis of variance (MANOVA) with ASI classification (high versus medium versus low) as the independent variable and the self-report measures of cognition, affect, behavior and pain as the dependent variables. The MANOVA indicated a significant overall difference between groups, $F(22, 110) = 2.77$, Wilks $\lambda = 0.42$, $p < 0.000$. Follow-up univariate analysis of variance (ANOVA) was used to examine each of the self-report measures individually. These analyses, presented in Table 1, were conducted with alpha conservatively adjusted to 0.01. As illustrated, the groups differed significantly on the BDI, $F(2, 68) = 5.83$, $p < 0.005$; STAI-T, $F(2, 68) = 5.05$, $p < 0.01$; PASS pain-related cognitive anxiety, $F(2, 68) = 5.41$, $p < 0.01$; PASS escape/avoidance behavior, $F(2, 68) = 5.27$, $p < 0.01$ and PASS fearful appraisals of pain, $F(2, 68) = 26.48$, $p < 0.000$. Groups did not differ significantly with regard to trait anger,

Table 1
Means and standard deviations for self-report data provided by headache patients with high, medium and low anxiety sensitivity

Measure	Group				
	high AS	medium AS	low AS	univariate F	p
CA	35.4 ± 9.6	29.7 ± 8.6	24.1 ± 9.6	5.41	0.007
PA	24.1 ± 13.2	18.9 ± 10.2	12.5 ± 10.5	3.61	0.033
EA	32.3 ± 8.9	25.1 ± 7.5	26.0 ± 8.1	5.27	0.008
FP	32.5 ± 8.7	17.0 ± 9.1	10.8 ± 8.2	26.49	0.000
BDI	16.6 ± 10.2	11.2 ± 6.8	6.8 ± 4.4	5.83	0.005
STAI-T	48.0 ± 13.2	39.5 ± 11.5	35.1 ± 8.5	5.05	0.009
STAXI-T	20.4 ± 6.2	17.3 ± 4.3	16.1 ± 3.9	3.55	0.035
MPQ-SF sensory	18.1 ± 6.2	15.4 ± 6.2	11.4 ± 5.8	3.83	0.027
Lifestyle change[a]	3.8 ± 1.1	3.5 ± 1.0	4.1 ± 0.7	1.72	0.187
Disturbing nature[a]	4.2 ± 0.8	3.9 ± 0.8	3.9 ± 0.7	0.84	0.436
Headache severity[a]	3.8 ± 1.1	3.8 ± 0.9	3.8 ± 0.8	0.01	0.993

CA = Pain Anxiety Symptom Scale (PASS), cognitive anxiety; PA = PASS, physiological anxiety; EA = PASS, escape/avoidance; FP = PASS fearful appraisals subscale; AS = anxiety sensitivity; BDI = Beck Depression Inventory; STAI-T = Spielberger State-Trait Anxiety Inventory, Trait Form; STAXI-T = Spielberger State-Trait Anger Expression Inventory; MPQ-SF = McGill Pain Questionnaire, Short Form. [a]Measures from the Headache Questionnaire.

physiological responsivity to pain, pain-related lifestyle disruption, pain-related distress, sensory pain experience or severity of headache pain.

Post hoc analyses, using Tukey's honestly significant difference, were conducted on the significant between-group effects to determine whether the high AS group differed from the medium and low AS groups. These analyses revealed that high AS patients had significantly higher scores on the BDI, STAI, PASS escape/avoidance subscale and PASS fearful appraisals subscale than did patients with medium or low AS (all alphas < 0.05). The high AS group also scored higher on the PASS cognitive anxiety subscale than did patients with low AS ($p < 0.05$).

3.2. ASI and analgesic use

Patients provided information on the HQ as to whether they were currently using over-the-counter or prescription medications to control their headaches. In order to determine whether AS influenced analgesic intake, we conducted several χ^2 analyses. As illustrated in Table 2, the proportion of patients reporting use of over-the-counter medications did not differ between AS groups, $\chi^2(2) = 0.76$, $p = 0.68$. Similarly, the proportion of patients reporting use of prescription medications did not differ significantly between AS groups; although, there was a trend (in the direction opposite than expected) for greater use of prescribed analgesics in the low and medium AS groups, $\chi^2(2) = 3.94$, $p = 0.14$. We also considered the proportions of

Table 2
Proportion of subjects reporting use of over-the-counter and prescription analgesics

Group	*N*	Over-the-counter analgesics[a]	Prescription analgesics[b]
High AS	20	70.0% (14)	50.0% (10)
Medium AS	41	65.9 (27)	70.1% (29)
Low AS	11	54.5% (6)	81.8% (9)

[a] $\chi^2(2) = 0.76$, $p = 0.68$. [b] $\chi^2(2) = 3.94$, $p = 0.14$.

patients taking neither, one or both types of medication and found that these did not differ by AS group, $\chi^2(4) = 0.97$, $p = 0.92$.

3.3. Predicting fear of pain and lifestyle changes

As previously noted, we were also interested in evaluating predictors of fear of pain and lifestyle change attributable to headache. To do so, we performed a stepwise multiple regression analysis in which fear of pain, as assessed using the fearful appraisals subscale of the PASS, was used as the criterion variable and ASI, BDI, STAI, PASS cognitive anxiety, physiological anxiety and escape/avoidance subscales, MPQ-SF sensory pain and headache severity scores as the predictor variables. This analysis indicated that 56.6% of the variance in fear of pain was accounted for by AS, pain-related cognitive anxiety and sensory pain experience, $F(3, 64) = 27.87$, $p < 0.000$ (see Table 3). AS accounted for 39.8% of the variance while cognitive anxiety and sensory pain experience contributed an additional 12.7 and 4.1%, respectively. We also assessed the importance of the aforementioned predictor variables to the degree of lifestyle change that patients had attributed to their headaches. Interestingly, of the total variance in lifestyle change explained by the significant model, $F(4, 63) = 12.53$,

Table 3
Variables predictive of fear of pain

Variable	FP	ASI	CA	MPQ-SF	*B*	*β*	$R^{2\,a}$
ASI	0.629				0.470[b]	0.451	0.398
CA	0.560	0.365			0.391[b]	0.320	0.127
MPQ-SF	0.437	0.286	0.351		0.403[c]	0.221	0.041
Mean	20.49	23.81	30.44	15.54			
S.D.	11.77	11.28	9.63	6.44			
							$R^2 = 0.566$
							Adj. $R^2 = 0.546$
							$R = 0.753$

FP = Pain Anxiety Symptoms Scale, fear of pain; ASI = Anxiety Sensitivity Index; CA = Pain Anxiety Symptoms Scale, cognitive anxiety; MPQ-SF = McGill Pain Questionnaire, Short Form, sensory pain. [a]Incremental. [b] $p < 0.001$. [c] $p < 0.05$.

Table 4
Variables predictive of changes in lifestyle attributed to headache

Variable	Change	Severity	PA	CA	EA	*B*	*β*	$R^{2\,a}$
Severity	0.503					0.435	0.420[b]	0.253
PA	0.432	0.351				0.037	0.434[c]	0.075
CA	0.213	0.421	0.694			−0.049	−0.470[c]	0.052
EA	0.436	0.391	0.535	0.621		0.039	0.331[c]	0.063
Mean	3.69	3.82	19.43	30.44	27.24			
S.D.	1.00	0.96	11.57	9.63	8.49			
								$R^2 = 0.443$
								Adj. $R^2 = 0.408$
								$R = 0.666$

PA = Pain Anxiety Symptoms Scale, physiological anxiety; CA = Pain Anxiety Symptoms Scale, cognitive anxiety; EA = Pain Anxiety Symptoms Scale, escape/avoidance. [a]Incremental. [b]$p < 0.001$. [c]$p < 0.01$.

$p < 0.000$ (see Table 4), headache severity accounted for 25.3%, pain-related physiological anxiety for 7.5%, cognitive anxiety for 5.2% and escape/avoidance behaviors for 6.3%.

4. Discussion

Fear and avoidance models of chronic pain suggest, in general, that there is a reciprocal relationship between fear and avoidance that serves to maintain pain behavior and disability over a prolonged period. Confirming evidence of the postulates of the fear and avoidance models is mounting (see Asmundson et al., 1998). In patients with recurrent headaches, preliminary evidence suggests that fear of pain plays a significant role in psychological distress and disruption of pleasurable activities (Hursey & Jacks, 1992). We have been interested in determining variables which impact on ones propensity to be(come) fearful of pain and to engage in escape and avoidance behavior. Our initial findings in patients with chronic musculoskeletal pain suggest that AS, a predispositional tendency to fear somatic perturbations and associated negative expectancies, plays an important role in increasing fear of pain and, through its effect on fear of pain, exacerbating escape and avoidance (Asmundson & Norton, 1995; Asmundson & Taylor, 1996).

In the present investigation, we sought to extend the aforementioned findings to patients with recurrent headaches. Interestingly, and in contrast to patients with chronic pain related to musculoskeletal injury, the mean ASI score across all headache patients was relatively high (i.e. 23.8 ± 11.4), exceeding the normative mean, and approaching or exceeding means observed for obsessive–compulsive disorder, generalized anxiety disorder, social phobia, specific phobia and somatoform disorders (Taylor et al., 1992; Cox et al., 1998). This suggests, in general, that patients with persistent headache may have an increased probability to be(come) fearful and avoidant of a multitude of stimuli (e.g. Reiss & McNally, 1985; Reiss, 1991) and of experiencing emotional disorders (Cox et al., 1998). When classified on the basis of AS levels, between groups differences emerged on a number of variables and, as predicted, patients with

high AS were found to report more adverse effects related to their pain experience than did those with medium or low levels. Specifically, high AS patients reported greater depression, trait anxiety, pain-related escape/avoidance behavior and fear of pain than did patients with medium or low AS. High AS patients also exhibited greater cognitive disruption in response to pain than did patients with low AS. These differences were evident despite indications that groups did not differ in their headache severity or reported change in lifestyle attributed to headache.

The aforementioned findings are of importance in that they support our suggestion that AS plays an important role in exacerbating fear of pain and pain-related escape avoidance behavior in patients with chronic pain, including those with recurrent headaches. They are also important in that they provide support to observations that factors such as fear of pain warrant careful consideration in assessing the impact of recurrent headaches on quality of life (e.g. Hursey & Jacks, 1992). Additionally, however, the results of the present investigation hold some practical implications. With the goals of identifying specific targets for cognitive-behavioral intervention, we assessed variables predictive of fearful appraisals of pain and changes in lifestyle attributed to (headache) pain. Our assumption was that significant predictors may represent suitable treatment targets. Results indicated that AS, pain-related cognitive disruption and sensory pain experience were significant predictors of fear of pain, accounting for 39.8, 12.7 and 4.1% of the variance, respectively. We have previously argued that AS may represent a suitable treatment target in patients with chronic pain conditions (Asmundson, 1998; Asmundson et al., 1998). While not yet systematically evaluated in patients with chronic pain, available treatment protocols for elevated AS (e.g. Craske & Barlow, 1989), when combined within more traditional approaches to pain treatment and management, may serve to reduce fear and some aspects of behavioral disruption experienced by these patients. The present results support the application of AS interventions within the context of chronic and recurrent pain. However, at least for patients with recurrent headaches, some lifestyle changes attributed to pain appear to be mediated primarily by the severity of the pain experiences and, to a lesser extent by physiological anxiety, cognitive anxiety and escape/avoidance behavior. These variables, respectively, accounted for 23.5, 7.5, 5.2 and 6.3% of the variance in reported lifestyle change. Consequently, an effective means of dealing with pain severity, whether pharmaceutically or cognitive-behaviorally based, remains a target for treatment that is of immediate importance.

Our prediction that patients with high AS would be more likely to use analgesic medications than those with medium and low AS was not supported. Indeed, the patient groups did not differ on use of over-the-counter or prescription analgesics. Nor did they differ in the proportion using none, one or both general categories of analgesics. Given that the majority (i.e. 93.1%) of patients in the current sample were taking some form of analgesic to provide relief from their headaches, this pattern of results may not be particularly surprising. Notwithstanding, this finding is discrepant with previous observations that analgesics are more likely to be taken by chronic musculoskeletal pain patients with high AS than those without (Asmundson & Norton, 1995). Further investigation in other samples of pain patients will be necessary to reconcile these incompatible findings.

In summary, the results of the present investigation add to a growing literature indicating that AS plays an important role in increasing both fear of pain and pain-related escape and

avoidance behaviors. The present results are the first to show that this state of affairs applies not only to patients with chronic pain related to musculoskeletal injury, but also to those with recurrent headaches. Notwithstanding, there are several limitations and future directions that need to be pointed out. First, lifestyle changes were assessed with self-report only and were not confirmed by observations of behavior as might have been provided by a significant other. Second, our results are based primarily on female treatment seeking headache patients. Treatment seeking headache patients may differ in psychological characteristics when compared to those who do not seek treatment (Ziegler & Paolo, 1995) and gender differences may exist. Third, we did not formally assess the possibility of comorbid psychiatric diagnoses in our patients sample. Future investigations in this area should consider the addition of structured clinical interviews to determine psychiatric co-morbidity and, importantly, should attempt to evaluate differences that may exist in AS, fear of pain and escape/avoidance between recurrent headache patients with and without related trauma history (e.g. Chibnall & Duckro, 1994; Haas, 1996). Finally, additional attention needs to be given to determining the variables that account for the variance in fear of pain. To this end, we might consider examining a variety of chronic pain conditions with a goal of determining whether the AS model or some alternative explanation, such as general negativity of affect (e.g. Watson & Pennebaker, 1989), provides the most parsimonious account of factors influencing fear of pain and related behavioral manifestations.

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