

Pain correlates of depressed mood in young adults

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OBJECTIVE: To provide an initial indication of the intensity and quality of pain in young adults reporting depressed mood and to investigate possible underlying mechanisms.

DESIGN: Case-control study.

SETTING: University undergraduate subject pool.

PARTICIPANTS: Sixty introductory psychology undergraduates classified as either reporting high levels of depressed mood ($n=30$; age 18.7 ± 0.87 years, mean \pm SD) or reporting low levels of depressed mood ($n=30$; age 18.6 ± 0.81 years).

MAIN OUTCOME MEASURES: Beck Depression Inventory, Short-Form McGill Pain Questionnaire, Pain Catastrophizing Scale, Pittsburgh Sleep Quality Index and pressure dolorimeter pain thresholds.

RESULTS: Young adults reporting high levels of depressed mood had significantly higher pain intensity at testing time, as measured by a visual analogue scale ($P=0.015$) and a present pain index ($P=0.002$), affective pain intensity for the previous month ($P=0.000$), pain catastrophizing ($P=0.025$) and global sleep disturbance ($P=0.000$) than young adults reporting low levels of depressed mood. Within the group of young adults reporting high levels of depressed mood, significantly higher sleep disturbance scores ($P=0.020$) were identified in those reporting high levels of overall pain intensity.

CONCLUSIONS: The results are discussed in terms of their implications for research as well as for the assessment and treatment of pain in individuals with depression.

Key Words: *Depression, Pain, Pain catastrophizing, Pain threshold, Sleep disturbance*

La douleur est en corrélation avec l'humeur dépressive chez les jeunes adultes

OBJECTIF : Fournir dans un premier temps une indication de l'intensité et de la qualité de la douleur chez les jeunes adultes rapportant une humeur dépressive et rechercher des mécanismes sous-jacents potentiels.

MODÈLE : Étude cas/témoins.

CONTEXTE : Groupe d'étudiants du premier cycle universitaire.

PARTICIPANTS : Soixante étudiants du premier cycle universitaire suivant un cours d'introduction à la psychologie catégorisés soit dans un groupe rapportant des niveaux élevés d'humeur dépressive ($n=30$; âge $18,7 \pm 0,87$ ans, moyenne \pm EC) ou dans un groupe rapportant des niveaux peu élevés d'humeur dépressive ($n=30$; âge $18,6 \pm 0,81$ ans).

PRINCIPALES MESURES DES RÉSULTATS : Le *Beck Depression Inventory* (inventaire de Beck sur la dépression), la formule abrégée du *McGill Pain Questionnaire* (questionnaire McGill sur la douleur), la *Pain Catastrophizing Scale* (échelle de dramatisation de la douleur), le *Pittsburg Sleep Quality Index* (index de Pittsburgh sur la qualité du sommeil) et un dolorimètre mesurant le seuil nociceptif à la pression.

RÉSULTATS : Les jeunes adultes rapportant des niveaux élevés d'humeur dépressive ressentaient une douleur significativement plus élevée au moment de l'évaluation, telle que mesurés par une échelle analogue visuelle ($p=0,015$) et un index de la douleur présente ($p=0,002$), une plus grande douleur affective pendant le mois précédent ($p=0,000$), démontraient une plus grande dramatisation de la douleur ($p=0,025$) et accusaient plus de troubles généraux du sommeil ($p=0,000$) que les jeunes adultes rapportant des niveaux peu élevés d'humeur dépressive. Dans le groupe des jeunes adultes rapportant des niveaux élevés d'humeur dépressive, des scores nettement plus élevés concernant les troubles du sommeil ($p=0,020$) ont été identifiés chez ceux rapportant des niveaux élevés d'intensité de la douleur.

CONCLUSIONS : Les résultats sont discutés sur le plan de leurs implications pour la recherche mais également sur celui de l'évaluation et du traitement de la douleur chez les individus atteints de dépression.

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Pain and depressed mood are relatively independent constructs. However, there is evidence that the two are highly correlated in some situations. For instance, depression is frequently concomitant with both adult chronic pain (1,2), and child and adolescent chronic pain (3-5). Similarly, frequent complaints of pain have been noted in depressed adults (1,6). Pain as a significant symptom in depressed children and adolescents has been less well recognized. Some epidemiological data (7) suggest that a common concomitant symptom of childhood or adolescent depressed mood is frequent complaints of pain (eg, headache, abdominal pain). However, the relative susceptibility to pain in children and adolescents who have depressed mood has not been carefully evaluated.

Not much attention has been paid to the age group marking the transition from adolescence to adulthood (ie, young adults between 17 and 25 years of age). The majority of adult studies on the comorbidity of pain and depression have used participants ranging from 18 to 80 years of age. Similarly, the limited research on the comorbidity of pain and depression in children and adolescents has generally tended to use participants ranging from four to 18 years of age. While participants between 17 and 25 years of age have been included in these studies, the use of wide age ranges limits the specific conclusions that can be made to this particular age group.

The transitional age group is an important population in and of itself, especially given the results from studies that have found that the incidence of depressive disorders among adolescents appears to increase with age, peaking in the late teens and early 20s (8). Further, a sharp increase in suicide rates has been witnessed for older adolescents and young adults over the past 30 years, with rates of 11.2/100,000 and 17.7/100,000 for Canadians aged 15 to 19 and 20 to 24 years, respectively (9). Government statistics suggest that, in Canada, suicide is the second leading cause of death among adolescents 15 to 19 years of age (10). Moreover, in a majority of adolescent cases, suicide is associated with depression (11). Thus, studies that employ the best validated measures of pain and the most precise evaluation of the presence of depression in well defined samples in this late adolescent-young adult age range are warranted.

Understanding the pain-depression relation is further hampered by the lack of information regarding whether or how depression is related to the individual's pain experience itself. The experience of pain is a multidimensional construct, composed of a sensory aspect, an affective aspect and an evaluative aspect. The sensory aspect can be defined as the sensory quality of the pain experience in terms of temporal, spatial, pressure, thermal and other properties (12). The affective aspect reflects the emotional portion of the pain experience in terms of tension, fear and autonomic properties that accompany pain (12). The evaluative aspect reflects the subjective, overall intensity of the pain experience and is based, in part, on the sensory and affective components as well as on other factors such as prior experience and the meaning of the situation to the person (12).

The majority of past pain-depression research has only as-

essed pain in terms of description (eg, location, frequency) and duration (13), with an almost total absence of any subjective or objective measures of the pain problem itself. The few studies to date that have included a reliable and valid instrument such as the McGill Pain Questionnaire (14) and the Visual Analogue Scale (15) have produced mixed findings. Some investigators have found that depression is not related to the reported intensity or quality of the individual's pain (16). Others report that depression in adult chronic pain patients is associated with the sensory aspect of the pain experience (17,18) and greater pain intensity (17). Yet, some researchers report that depressed mood in adult chronic pain patients is associated with the affective aspect of the pain experience (19,20). Similarly, other researchers argue that the affective descriptors of pain are good predictors of psychological distress in chronic pain patients (21). Moreover, these studies have not examined the possible mechanisms underlying the comorbidity of depressed mood and pain that may mediate the relation between depressed mood and the individual's experience of pain.

The following mechanisms possibly underlie the comorbidity of depressed mood and pain.

1. Young adults with depressed mood may have increased pain sensitivity and, thus, may perceive stimulation as painful that others may not perceive as painful (22).
2. Young adults with depressed mood may tend to interpret negatively life events (ie, catastrophize) and, therefore, are more likely to interpret a given bodily sensation as more threatening than it actually is (23).
3. Young adults with depressed mood may have less restful sleep (24), which may exacerbate pain.

The present study was designed to investigate further the relation between depressed mood, and the sensory and affective aspects of the pain experience and to examine these possible underlying mechanisms. These mechanisms are not exhaustive nor are they mutually exclusive. Further, difficulties arise in elucidating cause from effect in several mechanisms. For example, poor sleep might cause pain and vice versa. Moreover, it is important to emphasize that the participants in the present study have not been diagnosed as having a depressive disorder. Rather, they are exhibiting depressed mood.

It has been suggested that the relation between depression and the sensory aspect of the pain experience is mediated by an increase in pain sensitivity (22,25), possibly due to increased somatic focus and subsequent activation of pain facilitation neurons (23). Pain sensitivity is quantified by a decrease in pain threshold, defined as the lowest intensity of a stimulus that induces pain (26). Various studies have been undertaken to study pain threshold in depressed adults, and the results have been contradictory. Some authors report a decrease in the pain threshold in depressed patients (27), yet others report an increase in pain threshold (25,28,29). In these studies, pain threshold was often studied under stress-inducing or fear-inducing conditions by using pulpar stimu-

lation, radiant heat or electrocutaneous stimulation, or by simultaneously determining pain tolerance levels, all of which may lead to inaccurate pain threshold measurement. For example, under fearful conditions, measurement of pain threshold may have been prematurely terminated, resulting in a deflated estimate of pain threshold.

Catastrophizing is a cognitive process characterized by excessive focus on negative aspects of situations, rumination and heightened levels of emotional and physical distress (30,31). Pain catastrophizing is considered to be a negative orientation towards pain, thought to intensify the experience of both pain and depression (32). There is debate, however, as to whether catastrophizing is a symptom of depression or a separate construct (33). In support of the latter position, the results from a regression analysis assessing the unique contribution of the Pain Catastrophizing Scale (PCS), the Beck Depression Index (BDI) and other measures of basic psychological constructs in the prediction of pain performed in Sullivan et al's (32) study did not support the position that pain catastrophizing was conceptually confounded with depressed mood.

The relation among pain catastrophizing, depression and pain is still unclear. Among adult chronic pain patients, catastrophizing has been found to be related to depression and pain severity (30,33). More specifically, catastrophizing has been found to mediate the relation between depression and the affective aspect of the pain experience in chronic pain patients (34). However, the relation between pain catastrophizing and pain severity in depressed patients has not been examined. It is possible that individuals with depressed mood may be more prone to having pain catastrophizing thoughts and, therefore, are more likely to interpret a given bodily sensation as more threatening than it actually is (23). Moreover, this negative appraisal is more likely to elicit the affective aspect of the pain experience (34) as opposed to the sensory aspect.

Sleep disturbance is qualified as disrupted sleep latency, variable sleep duration, alterations in habitual sleep efficiency and perceived sleep quality. Disturbed sleep is a commonly reported symptom in patients with depression; approximately 80% of depressed patients report poor sleep quality (24). In a recent review, Soldatos (35) reported that insomnia is more than twice as prevalent in depressed than in non-depressed individuals and more than three times as prevalent among depressed adolescents than nondepressed adolescents. Sleep problems have also been reported by chronic pain patients and are often described as an aspect of their pain that is one of the most disturbing (36).

Given that sleep disturbance is a symptom common to both pain and depressive conditions, sleep disturbance may play an underlying role in the relation between pain and depression. For instance, it may be that depressed individuals have less restful sleep (24) and that this sleep disturbance may exacerbate pain. On the other hand, it is possible that chronic pain itself disrupts restful sleep and that recurrent disruptions lead to depression. Likewise, restless and shortened sleep episodes may cause musculoskeletal discomfort,

leading to feelings of frustration, despair, hopelessness, helplessness, etc. While these relations may exist, the identification of them is not the focus here. However, understanding how the three constructs interact is of importance when trying to identify common aspects of sleep that are found in individuals with pain, depression or both.

There are four main goals of the present study. The first goal is to provide an initial indication of the sensory and affective pain intensities in the previous month and the pain intensity on the day of testing in males and females between 17 and 20 years of age reporting high levels of depressed mood, with a score of 9 and above on the BDI versus males and females between 17 and 20 years of age reporting low levels of depressed mood, with a score of 0 to 4 on the BDI (37). The second goal is to determine whether there is an association among young adults' depressed mood, pain thresholds, defined as the minimum amount of pressure (kg/cm^2) exerted by a pressure dolorimeter that produces pain, and the sensory aspect of the pain experience. The third goal is to determine whether there is an association among young adults' depressed mood, pain catastrophizing and the affective aspect of pain experience, and the fourth is to determine the relations among depressed mood, sleep disturbance (including both the overall quality and the specific aspects of sleep) and overall pain intensity.

PARTICIPANTS AND METHODS

Participants

Eighty-three participants were initially randomly selected from introductory psychology students at Dalhousie University who had previously completed the BDI as part of a screening procedure ($n=360$) approximately 12.5 weeks (range of eight to 17 weeks) before testing. Data from 23 of the participants were later excluded from data analysis because their BDI score range at the time of testing did not correspond with their screening BDI score range. The final sample consisted of 30 participants reporting high levels of depressed mood (23 females, seven males; age 18.7 ± 0.87 years [mean \pm SD]; BDI testing score 14.17 ± 5.34) and 30 participants reporting low levels of depressed mood (22 females, eight males; age 18.6 ± 0.81 years; BDI testing score 2.23 ± 1.33).

Measures

BDI: BDI is a self-report inventory developed by Beck et al (38) to evaluate cognitive, affective, somatic and behavioural dimensions of depressive symptoms. The BDI consists of 21 groups of statements. Each group has four response categories, which are weighted and scored 0, 1, 2 and 3 according to the severity of the response. A total BDI score is derived from the sum of the intensity rank values and can range from 0 to 63, with higher scores reflecting greater levels of depressive symptomatology. The BDI has been shown to have excellent psychometric characteristics (37), and its validity with university students has been demonstrated (39).

PCS: The PCS is a self-report inventory developed and validated by Sullivan et al (32) to evaluate the magnification, ru-

mination and helplessness dimensions of pain catastrophizing. The PCS consists of 13 statements describing different thoughts and feelings that may be associated with the experience of pain. The degree to which each statement applies when the individual is experiencing pain is rated on a scale of 0 to 4, where 0 = 'not at all', 1 = 'to a slight degree', 2 = 'to a moderate degree', 3 = 'to a great degree' and 4 = 'all the time'. A PCS score is derived from the sum of the rank values assigned to each statement and can range from 0 to 52.

Pittsburgh Sleep Quality Index: The Pittsburgh Sleep Quality Index (PSQI) is a self-report inventory developed and validated by Buysse et al (40) to assess the quality and disturbance of sleep for the past month. Seven component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction) are derived from 19 single items that each have weights of 0 to 3. The final global score is simply a summation of the seven component scores and can range from 0 (good quality of sleep) to 21 (very poor quality of sleep). An additional five questions (that are to be rated by a roommate or bed partner) are used solely for clinical information and were, therefore, not included in the present study.

Short Form McGill Pain Questionnaire: The Short Form McGill Pain Questionnaire (SF-MPQ), a self-report inventory, is a shortened version of the standard McGill Pain Questionnaire (14) developed by Melzack (41) to evaluate sensory, affective and qualitative dimensions of the pain experience. The main component consists of 15 descriptors (11 sensory and four affective) that are rated on an intensity scale of 0 to 3, where 0 = none, 1 = mild, 2 = moderate and 3 = severe. Two pain scores are derived from the sum of the intensity rank values of the words chosen for sensory and affective descriptors. The scores range from 0 to 33 for the sensory descriptors and 0 to 12 for the affective descriptors. A total descriptor score is derived by summing the sensory and affective descriptor scores. The SF-MPQ also includes a Visual Analogue Scale (VAS) of pain and a Present Pain Intensity Index (PPI). The VAS is a 83 mm horizontal line anchored by no pain and worst possible pain. The PPI is rated on a six-point scale and is scored from 0 to 5, where 0 = no pain, 1 = mild, 2 = discomforting, 3 = distressing, 4 = horrible and 5 = excruciating. The SF-MPQ correlates very highly with the major pain indexes of the Long Form McGill Pain Questionnaire (41,42). In addition, concurrent validity of the SF-MPQ was recently reported in a study of patients with chronic pain due to cancer (42).

Pressure dolorimetry: A pressure dolorimeter (Pain Diagnostics and Thermography Inc) has a force gauge fitted by a 1 cm² rubber disk, through which a known force (kg/cm²) can be applied on the body. Pressure dolorimetry is a reliable and valid method of assessing pain thresholds (26).

PROCEDURE

Participants were asked to complete the SF-MPQ and the PSQI. Following completion of the questionnaires, the participants underwent pain threshold measurements with the

use of a pressure dolorimeter at 11 anatomic sites (right occiput, right and left trapezius, right supraspinatus, right lateral epicondyle, right second rib, right and left medial knee, right and left thumbnail, and right mid-dorsal forearm). Pain threshold was measured according to the method employed by Fischer (26) and consisted of the following three steps.

1. The participant was instructed to say 'stop' or 'now' when he or she first felt pain.
2. Once the target site was located by brief preliminary thumb palpation, the rubber tip of the pressure dolorimeter was placed on the point to be examined, with the shaft vertical to the examined surface, and pressure was increased steadily at approximately 100 g/s.
3. When the participant said 'stop' or 'now' the pressure was stopped and the pressure dolorimeter was removed from the skin. All measurements were done with the participant in a sitting position, with the exception of the thumbnail measurements, which were recorded with the participant standing and resting his or her hand on the edge of a table.

The researchers performing the pain threshold measurements received training to establish reliability. After the pain threshold measurements were completed, the participant completed the PCS and the BDI. Total time required of each participant was approximately 1 h. The study was approved by the Dalhousie Department of Psychology Human Ethics Committee.

RESULTS

Descriptive statistics (mean \pm SD), MANOVA, univariate ANOVA and/or factorial ANOVA were performed for the two SF-MPQ scores, the VAS scores, the PPI indexes, the overall pain thresholds, the PCS scores and the PSQI scores for both the young adults reporting high levels of depressed mood and the young adults not reporting high levels of depressed mood. Multiple regression analyses were then used to determine the associations among depressed mood, pain intensity, pain threshold, pain catastrophizing and sleep disturbance. Two-tailed tests of significance and an alpha level of 0.05 were used to analyze the data. Because sex and age differences have been previously found for the constructs under investigation in the present study (25,43), sex and age effects were assessed in the analyses.

Pain intensity, sensory pain intensity, affective pain intensity and depressed mood

Descriptive statistics (mean \pm SD) for the two SF-MPQ scores (sensory and affective descriptors), the VAS scores and the PPI indexes for both the participants reporting high levels of depressed mood and the participants reporting low levels of depressed mood are presented in Table 1. There were no significant effects of sex or age; thus, the results presented across sex and age are collapsed.

A between-subjects MANOVA was performed on the four dependent variables: sensory descriptors, affective descriptors, VAS and PPI. The independent variable was level of depressed mood (reporting high levels of depressed mood and reporting low levels of depressed mood). The analysis revealed a significant overall difference between groups ($F_{4,55}=5.10$, $P=0.001$). Subsequent univariate F tests for each dependent variable revealed that young adults reporting high levels of depressed mood had significantly higher affective descriptor scores ($F_{1,58}=14.23$, $P=0.000$), VAS scores ($F_{1,58}=6.28$, $P=0.015$) and PPI indexes ($F_{1,58}=10.81$, $P=0.002$), but not sensory descriptor scores ($F_{1,58}=3.20$, $P=0.079$), than young adults reporting low levels of depressed mood.

To determine whether the elevated scores on the SF-MPQ subscales for the young adults reporting high levels of depressed mood were confounded with the four somatic items on the BDI (insomnia, tiredness, poor appetite and weight loss), these items were omitted and the BDI score was prorated. MANOVA revealed a significant overall difference between categories ($F_{4,50}=4.99$, $P=0.002$). Subsequent univariate F tests revealed that young adults reporting high levels of depressed mood had significantly higher affective descriptor scores ($F_{4,50}=13.33$, $P=0.001$), VAS scores ($F_{4,50}=4.90$, $P=0.031$) and PPI indexes ($F_{4,50}=11.22$, $P=0.001$), but not sensory descriptor scores ($F_{4,50}=2.33$, $P=0.133$), than young adults reporting low levels of depressed mood. The results suggest that greater pain intensity at the time of testing and higher affective pain intensity for the previous month, reported by individuals with high levels of depressed mood, were not confounded with the somatic items on the BDI.

Depressed mood and pain thresholds

A principle components analysis performed on the 11 anatomic points generated a two-component solution with eigenvalues greater than 1 (factor 1: eigenvalue 6.203, percentage of variance 56.4%; factor 2: eigenvalue 1.939, percentage of variance 17.6%). The factor matrix demonstrated that the majority of the points loaded heavily on factor 1, with the exception of the right trapezius. Thus, the measurements for the right trapezius were excluded from further analyses. The remaining 10 points were summed and averaged for each participant.

Pain threshold was analyzed as a 2x2 (sex versus level of depressed mood) factorial ANOVA, which revealed that males had significantly higher pain thresholds than females ($F_{1,56}=14.11$, $P=0.000$). Pain threshold was thus collapsed across sex in each category (ie, high or low level of depressed mood). Males and females reporting high levels of depressed mood had mean pain thresholds of 3.65 ± 1.27 kg/cm² and 2.64 ± 0.91 kg/cm², respectively. Males and females reporting low levels of depressed mood had mean pain thresholds of 3.42 ± 0.99 kg/cm² and 2.50 ± 0.56 kg/cm², respectively. There was no significant main effect of category ($F_{1,56}=0.546$, $P=0.463$) or interaction between sex and level of depressed mood type ($F_{1,56}=0.032$, $P=0.859$). These results indicate that regardless of mood, males had lower pain sensitivity than females.

TABLE 1
Descriptive statistics of the Short-Form McGill Pain Questionnaire (SF-MPQ) subscales for participants reporting high levels of depressed mood and participants reporting low levels of depressed mood

SF-MPQ subscale	High level of depressed mood (n=30) (mean \pm SD)	Low level of depressed mood (n=30) (mean \pm SD)
Sensory descriptors	8.87 \pm 4.36	6.6 \pm 5.4
Affective descriptors	3.7 \pm 2.32***	1.7 \pm 1.74
Visual Analogue Scale	1.96 \pm 2.24*	0.7 \pm 1.58
Present Pain Index	1.03 \pm 1.03**	0.3 \pm .65

* $P<0.05$; ** $P<0.01$; *** $P<0.001$

Depressed mood and pain catastrophizing

Young adults reporting high levels of depressed mood had a mean PCS score of 18.43 ± 9.26 , and young adults reporting low levels of depressed mood had a mean pain catastrophizing score of 13.37 ± 7.73 . There were no significant sex or age effects. A one-way ANOVA revealed that young adults reporting high levels of depressed mood had significantly higher PCS scores than those reporting low levels of depressed mood ($F_{1,58}=5.30$, $P=0.025$).

Depressed mood and sleep disturbance

The descriptive statistics (mean \pm SD) for the global and component PSQI scores for all categories are presented in Table 2. There were no significant age or sex differences.

An ANOVA revealed that young adults reporting high levels of depressed mood had significantly higher global PSQI scores than those reporting low levels of depressed mood ($F_{1,58}=19.62$, $P=0.000$). A MANOVA was performed to determine which components of the PSQI were significantly different between participants reporting high levels of depressed mood and participants reporting low levels of depressed mood. Significant differences were found on the components of subjective sleep quality ($F_{1,58}=12.22$, $P=0.001$), sleep latency ($F_{1,58}=13.80$, $P=0.000$) and daytime dysfunction ($F_{1,58}=15.43$, $P=0.000$).

Pain intensity and sleep disturbance

Because no standardized cutoff scores were available for the SF-MPQ to determine high and low overall pain intensity, a median split of the SF-MPQ total descriptor score was used to classify individuals as having high overall pain intensity (a score of 10 or greater; $n=30$) and low overall pain intensity (a score of 9 or less; $n=30$). An ANOVA revealed that participants with high overall pain intensity had significantly higher global PSQI scores than participants with low overall pain intensity ($F_{1,58}=11.76$, $P=0.001$). A MANOVA revealed that individuals with high overall pain intensity scored significantly higher on the components of subjective sleep quality ($F_{1,58}=7.05$, $P=0.010$), sleep latency ($F_{1,58}=4.06$, $P=0.049$), sleep disturbances ($F_{1,58}=4.92$, $P=0.030$) and daytime dysfunction ($F_{1,58}=12.04$, $P=0.001$) than those with low overall pain intensity (Table 2).

TABLE 2
Descriptive statistics for the Pittsburgh Sleep Quality Index (PSQI) subscales for all participant categories

Participant category	PSQI subscale scores (mean ± SD)							
	Global	C1	C2	C3	C4	C5	C6	C7
High level of depressed mood (n=30)	^a 7.5±2.87***	^a 1.4±0.86***	^a 1.77±0.9***	0.63±0.76	0.47±0.86	1.37±0.49	0.23±0.68	^a 1.67±0.80***
Plus high overall pain intensity (n=22)	^b 8.05±3.15*	1.55±0.91	1.77±0.97	0.77±0.81	0.59±0.96	1.41±0.5	0.18±0.66	1.77±0.19
Plus low overall pain intensity (n=8)	^b 6.125±1.13	1±0.53	1.75±0.71	0.25±0.46	0.13±0.35	1.25±0.46	0.36±0.74	1.36±0.52
Low level of depressed mood (n=30)	^a 4.6±2.1	^a 0.77±0.5	^a 0.97±0.76	0.37±0.67	0.3±0.6	1.13±0.57	0.13±0.43	^a 0.97±0.56
High overall pain intensity (n=30)	^c 7.3±3.2***	^c 1.33±0.88**	^c 1.6±0.93*	0.667±0.76	0.5±0.86	^c 1.4±0.56*	0.13±0.57	^c 1.633±0.81***
Plus high level of depressed mood (n=22)	^d 8.05±3.15*	^d 1.55±0.91*	1.77±0.97	0.77±0.81	0.59±0.96	1.41±0.5	0.18±0.66	1.77±0.19
Plus low level of depressed mood (n=8)	^d 5.13±2.03	^d 0.75±0.46	1.13±0.64	0.38±0.52	0.25±0.46	1.38±0.74	0±0	1.25±0.46
Low overall pain intensity (n=30)	^c 4.9±2.1	^c 0.833±0.53	^c 1.13±0.86	0.333±0.66	0.27±0.58	^c 1.1±0.48	0.2±0.57	^c 1±0.58

Rows with same subscripts indicate where comparisons were made. C1 Subjective sleep quality; C2 Sleep latency; C3 Sleep duration; C4 Habitual sleep efficiency; C5 Sleep disturbances; C6 Use of sleeping medication; C7 Daytime dysfunction. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

TABLE 3
Multiple regression of the Beck Depression Inventory (BDI), pain threshold and Pain Catastrophizing Scale (PCS) scores in the prediction of sensory pain intensity

Predictor	Unstandardized beta coefficient (mean ± SE)	Standardized beta coefficient	t	P
BDI	0.166±0.090	0.237	1.835	0.0718
Pain threshold	-0.271±0.664	-0.051	-0.408	0.6848
PCS	0.154±0.073	0.272	2.122	0.0383
Constant	4.685±2.304	-	2.034	0.0467

$n=60$, $R^2=0.168$

Depressed mood, high overall pain intensity and sleep disturbance

As can be seen in Table 2, 22 of 30 (73%) participants reporting high levels of depressed mood reported high overall pain intensity and had significantly higher global PSQI scores than the remaining eight participants reporting low overall pain intensity ($F_{1,27.99}=6.05$, $P=0.020$). A MANOVA revealed that there were no significant differences on the individual subscales of the PSQI between those who had high overall pain intensity and those who had low overall pain intensity (for all components, $P>0.05$).

Within the group of participants reporting high levels of overall pain intensity, there were significant differences in the global PSQI scores between participants who reported

TABLE 4
Multiple regression of the Beck Depression Inventory (BDI), pain threshold and Pain Catastrophizing Scale (PCS) scores in the prediction of affective pain intensity

Predictor	Unstandardized beta coefficient (mean ± SE)	Standardized beta coefficient	t	P
BDI	0.152±0.034	0.478	4.005	0.0002
Pain threshold	-0.081±0.278	-0.0330	-0.290	0.7731
PCS	0.041±0.0304	0.159	1.341	0.1854
Constant	1.033±0.966	-	1.070	0.2893

$n=60$, $R^2=0.292$

high levels of depressed mood (n=22) and participants reporting low levels of depressed mood (n=8; $F_{1,19.63}=5.90$, $P=0.022$). A MANOVA demonstrated that the only component of the PSQI upon which those reporting high levels of depressed mood and those reporting low levels of depressed mood differed significantly was subjective sleep quality ($F_{1,28}=5.48$, $P=0.027$).

Depressed mood, pain threshold, pain catastrophizing and sensory pain intensity

A multiple regression analysis was performed to assess the contribution of participants' BDI scores, pain thresholds and PCS scores in the prediction of sensory pain intensity. As can be seen in Table 3, the results of the regression analysis indi-

cated that 16.75% of the variance in sensory pain intensity could be predicted with the combination of the three predictor variables. With respect to the individual predictors, only the standardized beta coefficient for the PCS was significant ($\beta=0.272$, $P=0.038$).

Depressed mood, pain threshold, pain catastrophizing and affective pain intensity

A multiple regression analysis assessing the contribution of participants' BDI scores, pain thresholds and PCS scores in the prediction of affective pain intensity indicated that 29.2% of the variance in affective pain intensity could be predicted with the combination of the three predictor variables (Table 4). With respect to the individual predictors, only the standardized beta coefficient for the BDI was significant ($\beta=0.478$, $P=0.0002$).

Depressed mood, pain intensity and sleep disturbance

A stepwise regression analysis was performed to assess the contribution of level of depressed mood and overall pain intensity to the global PSQI score. The categorical level of depression variable was entered first and achieved a significant beta of 2.9 ($P<0.05$) in predicting the global PSQI score. With the addition of the SF-MPQ total descriptor score in the equation, the beta for level of depression decreased to 2.22 and remained significant ($P<0.05$). The beta for pain intensity was 0.157 ($P<0.05$) and accounted for 10% of the additional variance in the PSQI global scores (for summary see Table 5).

DISCUSSION

The present study was designed to provide an initial indication of the intensity of pain in young adults reporting high levels of depressed mood; to examine associations among depressed mood, pain thresholds and sensory pain intensity; to examine associations among depressed mood, pain catastrophizing and affective pain intensity; and to investigate the relations among depressed mood, overall pain intensity and sleep disturbance.

The results demonstrated that young adults reporting high levels of depressed mood had greater pain intensity on the day of testing and greater affective pain intensity during the previous month than young adults reporting low levels of depressed mood. Young adults reporting high levels of depressed mood did not, however, have significantly greater sensory pain intensity in the previous month than young adults reporting low levels of depressed mood. Removal of the somatic items from the BDI did not affect the significance of these findings. Thus, it can be concluded that the increases in present pain intensity and affective pain intensity were not due to overlapping symptomatology.

These findings are inconsistent with those from studies reporting no relation between depression and the quality of pain (16), a relation between depression and both sensory and affective pain intensities (34), or a relation between depression and sensory aspect of pain only (17,18). The results are more consistent with those suggesting that depression is

TABLE 5
Summary of stepwise regression analysis for variables predicting sleep disturbance

Variable	Unstandardized beta coefficient (mean \pm SE)	Standardized beta coefficient
Step 1		
Category of depression	2.9 \pm 0.654	0.503*
Step 2		
Category of depression	2.228 \pm 0.646	0.386
Level of pain intensity	0.157 \pm 0.050	0.352

$n=60$, $R^2=0.25$ for step 1; $\Delta R^2=0.36$ for step 2 ($P<0.05$). * $P=0.000$

associated with the affective aspect of the pain experience (19,20), and with those who argue that the affective descriptors of pain are good predictors of psychological distress in chronic pain patients (21).

The hypothesis that young adults reporting high levels of depressed mood have lower pain thresholds than young adults reporting low levels of depressed mood was not supported. The results demonstrated no significant difference in pain thresholds between young adults reporting either high or low levels of depressed mood. This is inconsistent with past research that reports either an increase or a decrease in pain threshold in depressed individuals. It is interesting to note, however, that the pain thresholds for the young adults reporting either high or low levels of depressed mood in the present study are lower, by inspection, than the pain thresholds found for 'normal or healthy controls' in other studies using pressure dolorimetry (26). This suggests that another factor may have affected pain threshold in the present study. In one study, pain threshold was found to be highly negatively correlated with anxiety (18). It is, therefore, plausible that the participants in both categories may have also had anxiety symptoms that equalized the pain thresholds across categories. Studies including a measure of anxiety are warranted and would enable researchers to establish whether the findings are due to depressed mood, anxiety or both.

Alternatively, the lower pain thresholds may also have been due to the possibility that some of the participants in either group may have had subclinical presentations of fibromyalgia, a form of nonarticular rheumatism characterized by diffuse musculoskeletal aching and tender points at multiple characteristic sites (44). Studies including a measure qualifying the type of pain experienced by the participants may explore this possibility.

The hypothesis that lower pain threshold mediates the relation between depressed mood and sensory pain intensity was not confirmed. Only PCS score significantly predicted sensory pain intensity. A possible explanation of this finding is that perhaps sensory pain intensity is influenced by the magnification dimension of pain catastrophizing. For example, it is possible that individuals who engage in more magnification-related thoughts, such as the PCS statement 'I think of other painful experiences', may tend to overestimate the actual occurrence of pain incidents. This possibility could

be explored by studies explicitly examining the influence of each pain catastrophizing dimension on the recall of pain incidents.

The results demonstrated that young adults reporting high levels of depressed mood had greater pain catastrophizing than young adults reporting low levels of depressed mood. There are several possible explanations for this finding. Pain catastrophizing may be limited to a particular time or situation (ie, state pain catastrophizing). In other words, there is something about being in a depressed mood that increases pain catastrophizing. Studies examining whether pain catastrophizing decreases with a decrease in depression could shed light on this possibility. If pain catastrophizing does not decrease, it may be that it is a relatively enduring characteristic of the individual that transcends the boundaries of place and time (ie, trait pain catastrophizing). This implies that there may be something inherent in the individual that makes the person more susceptible to both depressed mood and pain catastrophizing. For example, a person may not have acquired the ability to employ effective coping skills when subjected to a negative experience, which, in turn, may lead to both depressed mood and pain catastrophizing.

The role of catastrophizing in the mediation of depressed mood and affective pain intensity was not confirmed. This finding fails to support past research indicating that catastrophizing mediates the relation between depression and the affective aspect of the pain experience (34). Thus, another mechanism may be responsible for mediating depressed mood and affective pain intensity. For example, individuals with depressed mood may have learned to get attention with pain complaints (45). Thus, they may use pain to get the attention of others.

In the present study, sleep disturbance was significantly greater in young adults reporting high levels of depressed mood and even more pronounced in those reporting both high overall pain intensity and high levels of depressed mood. The finding that sleep disturbances are greater in individuals with high levels of depressed mood than in those reporting low levels of depressed mood is not surprising given the amount of literature that supports this notion (35). However, the significant differences between the two groups on the subjective sleep quality, sleep latency and daytime dysfunction components of the PSQI may help identify specific areas of sleep that are disrupted in individuals with high levels of depressed mood and, therefore, help target specific sleep factors for behavioural or cognitive modification. It is important to note, however, that the higher scores on the subjective sleep quality component of the PSQI found for individuals with high levels of depressed mood may not be a direct result of actual disrupted sleep per se but rather a consequence of negative perceptions that individuals with high levels of depressed mood may have.

The significant difference in PSQI global scores between individuals reporting high overall pain intensity and those reporting low overall pain intensity indicates that sleep disturbance is associated with pain. Sleep components of subjective sleep quality, sleep latency, sleep disturbances and

daytime dysfunction were worse in young adults with high levels of overall pain intensity. The differences in sleep latency found between individuals with high overall pain intensity and those with low overall pain intensity are partially supportive of earlier studies that found pain patients to have difficulty initiating sleep (36). Specifically, difficulty initiating sleep is a component that is considered when sleep problems are associated with the duration of sleep.

Within the group of young adults reporting high levels of depressed mood, those reporting high levels of overall pain intensity had significantly higher PSQI global scores than individuals reporting low levels of overall pain intensity, suggesting that sleep disturbances are pronounced when both high pain intensity and depressed mood exist simultaneously. Specifically, having high pain intensity exacerbates the disturbed sleep one would have if only a depressed mood existed.

That nonsignificant differences were found between PSQI component scores for these two pain intensity groups implies that no one aspect of sleep accounted for the differences between the groups' global PSQI scores. Instead, it appears that the combination of all seven sleep components serves to create the overall differences observed between the sleep of individuals expressing high levels of depressed mood and high overall pain intensity and those reporting high levels of depressed mood and low overall pain intensity. Whether sleep disturbance exists when pain is present before depressed mood, or vice versa, remains unclear. Because the SF-MPQ and the PSQI evaluate pain and sleep, respectively, within the previous month, and reports of mood pertain to the past two weeks, inferences about the co-occurrence of pain and poor sleep in the two-week period that mood is not assessed are possible.

Within the group reporting high overall pain intensity, those who expressed high levels of depressed mood had significantly higher global PSQI scores than those with low levels of depressed mood. This finding suggests that even if a person experiences high pain intensity, their level of depressed mood influences the perceived overall quality of their sleep. This hypothesis is supported by the finding that scores on subjective sleep quality for the two groups were significantly higher for individuals reporting high levels of depressed mood. Negative thoughts often expressed in depression may lead one to perceive that the quality of their sleep is poor although it may not be realistically different from the actual sleep quality of individuals who do not express these negative thoughts (for example, individuals with low levels of depressed mood).

The regression analysis identified the level of depressed mood as being more predictive of overall sleep quality than pain intensity level. However, when pain intensity exists in addition to high levels of depressed mood, an additional 10% of sleep problems reported by participants are accounted for. This finding concurs with those from past studies that claim that poor sleep is often attributed more to mood than to pain experience, although the experience of pain (and in this case, high pain intensity) influences how well an individual describes their sleep (36,46).

A number of limitations in the present study need to be considered. First, as previously mentioned, the sample of participants may not have had a 'purely' depressed mood or a 'purely' nondepressed mood (ie, may have also had anxiety symptoms), thus making generalizability difficult. Moreover, the results must be interpreted with caution because an elevated prevalence of pain has been noted among patients with an anxiety disorder (6). Second, the participants were not diagnosed with major depressive disorders. Rather, they were individuals who endorsed a higher number of symptoms indicative of a depressed mood on a self-report measure (ie, a subclinical population). Thus, the present study is limited in its ability to generalize to clinical populations. Third, the assessment of sensory and affective pain intensity required participants to recall the types of pain experienced in the previous month. Thus, the accuracy of memory may be questionable. Studies using a longitudinal pain assessment technique, such as a pain diary, may give a more accurate account of pain intensity. Finally, sleep was measured by self-report rather than by physiological methods, resulting in a limited amount of information regarding participants' sleep patterns.

Despite these limitations, the data have theoretical and practical implications. From a theoretical perspective, the results suggest that depressed mood in young adults may be associated with an increase in the affective and not the sensory dimension of pain; however, the mechanisms involved in this association were not determined. This finding further stresses the importance of assessing pain multidimensionally rather than using unidimensional scales when examining the relation between depression and pain. Moreover, the occurrence of higher pain intensity, pain catastrophization and sleep disturbance, albeit at the low end of the measures, in a subclinical population stresses the importance of examining these variables in a clinical population.

A practical implication of the present study is that if a patient presents with both depressive symptoms and pain complaints reflecting the affective aspect of the pain experience, a treatment program including cognitive therapy may help alleviate those pain symptoms. However, it is not pragmatic to implement a treatment program solely on the basis of one presenting symptom. Therefore, further studies examining other potential concomitant symptoms, such as increasing pain throughout the day and extreme pain levels in the late evening, need to be performed. Given that depressed mood in the present study was associated with greater pain catastrophizing scores, depressed mood may impair the individual's ability to tolerate and to cope with continuous pain throughout the day. The impairment may be sufficient to result in reports of greater discomfort by the end of the day. Longitudinal studies including multiple measurements (eg, morning, noon and bedtime) of pain intensity on a VAS may help explore this possibility.

Additionally, if nonclinic samples in future studies show that mood and pain intensity, among other factors, can affect the quality of sleep, then the implications for clinical populations are heightened. Ideally, a prospective study examining the relations among pain, sleep and depression may allow

firm conclusions concerning causal relations to be made. With this accomplished, allocation of treatment would be easier. It is possible that the poor sleep reported by pain patients is seen as a symptom of the pain experience rather than a separate construct, and if this is the case, treatment for a sleeping disorder may be overlooked. Similarly, an individual who reports depressed mood may seek treatment for the depression and assume that any poor sleep they have is a result of the depression. However, it is equally possible that treatment sought for sleeping problems may alleviate negative mood if, indeed, depressed mood is the result of poor sleep.

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