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Pain moderates changes in psychological flexibility but not substance use symptoms during substance use disorder treatment

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Abstract

Pain-related problems frequently complicate substance use disorder (SUD) course and prognosis. However, it is unclear if the negative outcomes associated with co-occurring pain are due to its link with greater SUD severity, disruption of SUD treatment processes, or connection to a third psychological process. The current study modeled the longitudinal effects of pain during a 4-week intensive outpatient treatment (IOP) on SUD symptoms and limited psychological flexibility (PF), a common feature of psychological well being that is commonly restricted in both SUD and pain patients. After controlling for initial severity of SUD symptoms, current pain level at treatment intake moderated change in a subcomponent of PF, values commitment, but not SUD symptoms during the IOP. During the treatment, pain level also limited improvement in PF but not self-reported SUD symptoms. Targeting additional increases in psychological flexibility surrounding commitment to values during SUD treatment may help improve outcomes among patients who began treatment with significant pain symptoms.

Keywords

Substance use disorder treatment; Outcomes; Intensive outpatient; Pain; Psychological flexibility

1. Introduction

Pain-related problems frequently complicate substance use disorder (SUD) course, prognosis, and treatment. SUD patients with both acute and chronic pain problems report more extensive drug use histories, greater severity of drug and alcohol problems, and more co-morbid psychiatric symptom sequelae than those without significant pain (Jakubczyk et al., 2015; Potter et al., 2008; Witkiewitz et al., 2015a, 2015b). Further, those in pain are less likely to attend or complete SUD treatment and reductions in pain predict lower risk of relapse following treatment (Jakubczyk et al., 2016). Co-morbidity of these conditions is

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also problematic due to the increasing prevalence of prescription pain medication abuse (Dworkin et al., 2007; Huang et al., 2006). Despite these complications, mixed evidence has made it difficult to determine how pain operates as a risk during SUD treatment (Caldeiro et al., 2008). First, significant levels of pain may predict poor SUD treatment response indirectly via its association with severity of SUD symptoms (i.e., higher mean-levels of SUD behavior) and/or psychiatric distress rather than having a direct influence on the rate of SUD symptom remission during treatment. Alternatively, pain may both directly disrupt key change processes in effective reduction of SUD symptoms wherein pain attenuates the typical rate of symptom remission. Finally, a third, independent psychological process related to both pain and SUD symptom severity may potentiate poor SUD treatment response. Studying how pain moderates the effective reduction of psychological problems during SUD treatment will elucidate strategies for combating pain's role as a prognostic risk factor.

1.1. Pain and SUD treatment prognosis

Pain may have both direct and indirect effects on SUD symptoms during treatment. First, the immediate pain relief following substance use may both enhance the rewarding properties of a substance (Leknes et al., 2011) and compel ongoing use of substances to "self-medicate" states of physical and psychological distress (Khantzian, 1997). During treatment sessions, acute pain may co-opt cognitive resources vital for processing information (Attree et al., 2003; Crombez, 1998; Crombez et al., 1998; Dick et al., 2002) and impede the frequency and accuracy of behavioral practice (Crombez et al., 1997; Harman and Ruyak, 2005). Additionally, pain has been shown to amplify hopelessness and negative affect during the early phases of SUD recovery (Bair et al., 2003; Samwel et al., 2006; Witkiewitz et al., 2015a, 2015b). These proximal cognitive, behavioral and emotional effects of pain during treatment may underlie the poor outcomes associated with concurrent pain and SUD.

However, the established link between pain and severe substance use obscures the nature of these effects. While pain may play a direct role in attenuating treatment effects, the disparity may actually be due to the severity of SUD rather than any direct psychological effects of pain itself. Some studies have been unable to confirm a link between pain and SUD treatment outcomes (Ilgen et al., 2006). Studies that have established a link between pain and SUD treatment outcome (Caldeiro et al., 2008) typically focus on recovery of psychosocial functioning rather than SUD symptoms remission specifically. Controlling for the initial level of SUD symptom severity in order to identify how pain uniquely effects prospective adaptation during SUD treatment will be an important next step. Additionally, it will be vital to identify psychological factors that are common to both SUD and pain-related problems that may be implicated in treating their co-occurrence.

1.2. Pain and psychological processes during SUD treatment

One common psychological feature of SUD and pain-related problems that may independently account for negative clinical outcomes is psychological flexibility (PF). Generally, PF is described as the maintenance of contact with the present moment to facilitate behavior that is aligned with one's values (Hayes and Duckworth, 2006; Kashdan and Rottenberg, 2010). A high degree of PF related to SUD reflects an ability to manage the

psychological symptoms of pain or other distress while continuing to make behavioral choices in alignment with personal values (e.g., reducing substance use; Hayes and Duckworth, 2006). Alternatively, a low degree of PF leads to avoidance and/or rigidity regarding difficult psychological experiences like pain and a decrease in behavioral choices that are aligned with personal values (e.g., relapse in substance use; Hayes and Duckworth, 2006). As mitigation of distress and other negative experiences is a primary objective, those with low PF are more likely to engage in maladaptive coping behaviors in an effort to eliminate or reduce distress quickly (i.e., substance abuse) rather than practicing acceptance or alternative coping that supports personal values (Thompson and McCracken, 2011).

Numerous cross-sectional studies have confirmed independent links between limited PF, pain, and SUD. More importantly, longitudinal increases in PF have also been associated with effective management of pain, recovery from SUD and general increases in psychological well-being (Kashdan and Rottenberg, 2010; McCracken and Morley, 2014; McCracken and Vowles, 2014). However, research has yet to evaluate the extent to which pain and SUD co-morbidity influences improvement in PF during SUD treatment. Understanding this relationship will be a vital step toward first understanding pain's influence as a risk for poor treatment outcome and ultimately leveraging PF for the psychological treatment of SUD and its common co-morbidity with pain.

To investigate the influence of pain on treatment outcomes, we evaluated the relationship between self-reported pain along with SUD symptoms and PF during a 4-week IOP for SUD. Given the typical increases in PF during psychological intervention for SUD and that pain is associated with greater SUD severity, we hypothesized that pain would disrupt normative treatment processes by reducing typical gains in PF over and above the influences of initial SUD severity.

2. Methods

2.1. Treatment setting

Data were collected through outcome assessments completed at intake and discharge by patients of an IOP for SUDs at a VA hospital in the Midwest. The program consisted of intensive abstinence-based treatment over the course of 3–4 weeks to stabilize symptoms related to significant SUD. Patients were required to attend 9–11 h of a structured, evidence-based group therapy curriculum per week, in addition to routine medical and psychiatric care appointments and one hour per week of individual case management. All patients were provided a preset, structured group curriculum and patients did not have flexibility in the curriculum. Groups provided were open to continuous enrollment and adapted from manualized protocols of cognitive behavioral therapy (CBT; DeMarce et al., 2014), mindfulness-based relapse prevention (Bowen et al., 2010), motivational enhancement therapy (MET; Miller, 2014), and twelve-step facilitation (TSF; Nowinski et al., 1999). While these approaches were expected to generally improve PF, changes in PF were not a specific goal for the IOP. Groups were facilitated by licensed clinical social workers and a licensed clinical psychologist, as well as by supervised social work and psychology trainees. Medication was prescribed, as necessary, by psychiatric staff for both SUD and psychiatric conditions (comorbid symptoms detailed in Table 1).

2.2. Treatment sample

SUD patients with pain ratings (n=284) who received treatment in the IOP were included in the sample. Demographics and psychiatric details for the sample are presented in Table 1. All patients in the program met criteria for alcohol and/or substance dependence disorder (as per DSM-IV criteria for a range of substances) and agreed to attempt abstinence from all substances of abuse while in the program. Patients were assessed using American Society of Addiction Medicine Patient Placement Criteria (ASAM-PPC; Mee-Lee et al., 2001) and either met or exceeded criteria for placement in an intensive outpatient program. Rates of completion for the IOP did not differ by age, racial/ethnic category, gender, substance use, co-morbid psychiatric symptoms (i.e., PTSD, depression, or generalized anxiety), or initial levels of pain and psychological flexibility.

2.3. Measures

Individual differences and treatment outcomes in this sample were determined using self-report of substance use, pain level, and PF during the course of IOP.

2.3.1. Self-reported pain—At intake, current pain level was measured using the Numeric Pain Rating Scale (NPRS) during psychiatric and medical evaluation by verbal self-report via an 11-point scale with “0” indicating no pain and “10” indicating a severe and debilitating level of pain. Despite adequate psychometric sensitivity (Williamson and Hoggart, 2005), differences between single units expressed on the 11-point scale may not account for clinically equivalent differences in pain (Sloman et al., 2006). For instance, the difference between 0 and 3 is not necessarily psychologically equivalent to the difference between 5 and 8.

Pain groups: Pain scale ratings were used to derive a dichotomous categorical predictor comparing low-level pain (i.e., LLP, 0–2 rating; n=165) and high-level pain (i.e., HLP, 3–10 rating; n=142) to broadly estimate the role of pain. Numerous studies have confirmed the efficacy of a cut-off method for NPRS analysis (Hanley et al., 2006). While previous studies have identified mild pain as 0–3 on the NPRS scale, we used a median-split sample procedure to determine a cut-off relevant for our sample (i.e., 2 rating) to preserve power for group comparisons.

2.3.2. Substance use—The Brief Addiction Monitor (BAM; 17-items) was administered at treatment intake and discharge to track substance use and related problems over time (Cacciola et al., 2013). As a multidimensional instrument, the BAM has been validated for periodic re-administration via brief self-report to monitor multiple dimensions of SUD related to treatment needs, progress, and outcomes. (Full-scale $\alpha=0.70$; Substance Use (subscale), $\alpha=0.75$; Risk Factors (Subscale), $\alpha=0.78$; Protective Factors (Subscale), $\alpha=0.57$). Measurement reliability was consistent with estimates previously reported (Cacciola et al., 2013). These dimensions include both use patterns for multiple substances (e.g., alcohol, cocaine, and opiates) and other risk or protective factors pertinent to SUD patients (e.g., family/social conflict and support, risk taking, daily activity, community engagement, financial status, and co-morbid medical and psychiatric symptoms).

2.3.3. Psychological flexibility (PF)—The Acceptance and Action Questionnaire - Substance Abuse (AAQ-SA) was used at both intake and discharge to measure PF (Luoma et al., 2011). Derived from the original AAQ, the 18-item AAQ-SA evaluates the psychological factors of values and acceptance related to substance use (e.g., thoughts, urges and cravings) (Bond et al., 2011). Reliability for the full-scale score was adequate in the current sample ($\alpha=0.77$). Specifically, AAQ-SA factors load onto two principal subscale components: *Values Commitment* (VC) and *Defused Acceptance* (DA) (Luoma et al., 2011). VC items measure efforts to align action with goals and values, even in the presence of difficult thoughts, feelings, urges, memories, and sensations (VC subscale current sample, $\alpha=0.85$). Higher ratings on VC items indicate higher PF reflecting a pursuit of goal-directed action over and above the interference of substance use. Alternatively, the DA subscale indexes the willingness to allow unwanted private events to occur without unnecessary attempts to avoid or cope with them (DA subscale current sample, $\alpha=0.83$). Lower ratings on DA items indicate higher PF and low levels of experiential avoidance. When taken together, higher AAQ-SA scores indicate higher levels of PF due to (1) commitment to goal-directed action guided by principles even during temptation to use substances and (2) willingness to accept (rather than avoid) difficult experiences. Finally, previous research has confirmed that the AAQ-SA is a reliable and valid assessment tool lacking socio-cultural bias in the measurement of psychological flexibility among those with SUD (Luoma et al., 2011).

2.3.4. Comorbid psychiatric symptoms—Measures of comorbid psychiatric symptoms (summarized in Table 1) included: the 7-item Generalized Anxiety Disorder Scale (i.e., GAD-7, current sample $\alpha=0.94$), the 9-item Patient Health Questionnaire (i.e., PHQ-9, current sample $\alpha=0.88$), and the 15-item civilian version of the Post-traumatic Stress Disorder (PTSD) Checklist (i.e., PCL-C, current sample $\alpha=0.87$).

2.4. Statistical analysis

Descriptive statistics (e.g., means, standard deviations) were calculated for substance use, pain groups (i.e., LLP vs. HLP), and PF at each time point. First, to establish that PF is associated with positive SUD outcomes, BAM Use Subscale change scores (i.e., intake – discharge) as well as dual-diagnosis symptom measures (i.e., GAD, PHQ-9 and PCL) were regressed on intake AAQ-SA scores.

Next, a hierarchical mixed modeling (HLM) approach was used to estimate the effects of pain group and time on changes in BAM and AAQ-SA scores from IOP intake to discharge. To determine if change in substance use factors was related to pain, we fit the following model:

$$BAM = \beta_{0t} + \beta_1(PAIN) + \beta_2(TIME) + \beta_3(PAIN \times TIME) + e_1.$$

This model estimates the main effects for Pain group (β_1) and Time (β_2), along with their two-way interaction (β_3) on BAM items. The main effect of Pain group (β_1) estimated the increase in BAM score for the HLP vs. LLP groups at intake. The main effect of Time (β_2) estimated the increase in BAM item score during the IOP for the LLP group relative to the HLP group. Finally, the Pain group $\times$ Time interaction (β_3) estimated the moderating effects

of pain group on changes in BAM score during the IOP. As our sample was undergoing abstinence-based treatment at the time of data collection, the rate of change for BAM items specific to substance use were not expected to differ between pain groups but were included in the model to account for this possibility. A residual term (e_1) was also included to account for variation in the outcome not accounted for by the predictor variables. A maximum likelihood (ML) estimation approach was used to take full account of all available data without utilizing artificially imputed values or omitting data via list or pairwise procedures.

Next, to test the main hypothesis (i.e., pain modifies the rate of PF change during IOP) we then fit the following HLM model:

$$AAQ = \beta_0 + \beta_1(PAIN) + \beta_2(TIME) + \beta_3(PAIN \times TIME) + \beta_4(USE) + e_1$$

This model estimates the main effects for Pain level groups (β_1) and Time (β_2), along with a parameter for their two-way interaction (β_3) on the AAQ-SA full and subscale scores. The main effect of Pain group (β_1) estimated the increase in AAQ-SA scale score associated with significant pain as opposed to mild pain at intake. The main effect of Time (β_2) estimated the increase in AAQ-SA scale score during the IOP for the mild pain group relative to the significant pain group. Finally, the Pain group $\times$ Time interaction (β_3) estimated the moderating effects of pain group on changes in AAQ-SA score during the IOP. As prior literature suggests that pain is likely to be associated with higher levels of SUD risk (i.e., higher scores on BAM risk subscale items), use sub-scale BAM scores were also included in the model as (β_4) to adjust for the effect of substance use severity at the start of treatment on the relationships between pain and PF over time. The inclusion of this parameter accomplishes two things simultaneously: 1) direct estimation of the effect of SUD severity on the change in AAQ-SA during treatment and 2) adjustment of effects for Pain group, Time, and the Pain group $\times$ Time interaction parameters for the random effects of SUD severity. Therefore, inclusion of this parameter results in more accurate estimation of the influences of target effects in the model on treatment adaptation in AAQ-SA. A residual term (e_1) was included again to account for variation in the outcome not accounted for by the predictor variables. REML estimation was used to handle missingness for this model along with Tukey's HSD corrections to account for multiple tests. Cohen's d effect sizes were also calculated to index the relative magnitude of each effect in order to limit sole reliance on p-value significant testing methods. While BAM is a standard measure used to collect data for program evaluation, the AAQ-SA was collected for a short duration to assess the change in PF during treatment. Consequently, only the subsample of patients ($n=279$) completing this measure was included in analysis for this model.

3. Results

3.1. Sample characteristics and treatment outcomes

At IOP intake, our sample reported average pain scores of 2.7 (SD=2.8) and average psychological flexibility ratings on the AAQ-SA of 74.6 (SD=13.5). Other investigations providing the AAQ-SA in this SUD population report scale scores consistent with this estimate (McGaffin et al., 2013). Linear regression confirmed the positive association

between pre-treatment scores on the AAQ-SA and change in the substance use reported via the BAM during treatment ($R^2=0.060$; $p<0.05$) such that higher AAQ scores at intake predicted a small, but significant reduction in substance use during the brief IOP.

The model estimating pain and time effects for the BAM full scale and item criterion are presented in Table 2. A large main effect of time was present for full-scale BAM scores ($F(1, 282)=70.47$, $p<0.001$, $d=-1.54$). Namely, BAM scores significantly decreased during the course of IOP. A moderately sized main effect of pain group was also detected wherein HLP group exhibited significantly higher full-scale BAM scores ($F(1, 282)=13.31$, $p=0.001$, $d=0.37$) than the LLP group. No significant Pain group $\times$ Time interaction was present ($F(1, 282)=0.416$, $p=0.519$), suggesting that SUD symptoms decreased at the same rate for both pain groups. A main effect of time was present for BAM items such that both use and risk factors significantly reduced while protective factor items remained largely unchanged during the treatment. Additionally, a main effect of pain group was present for a number of BAM items such that the HLP group reported more cocaine use ($F(1, 282)=4.01$, $p=0.027$, $d=0.30$), health risks ($F(1, 282)=8.81$, $p=0.003$, $d=0.22$), insomnia ($F(1, 282)=11.53$, $p=0.001$, $d=0.28$), mood instability ($F(1, 282)=8.91$, $p=0.003$, $d=0.25$), and interpersonal conflict ($F(1, 282)=4.47$, $p=0.035$, $d=0.13$) than the LLP group. On average, the HLP group also engaged in more supportive spiritual practice than the LLP group ($F(1, 282)=6.91$, $p=0.009$, $d=0.26$).

3.2. Changes in psychological flexibility during IOP

Estimates of the main effects and interaction of time and pain level on full scale and subscale AAQ-SA scores (i.e., full, VC and DA) are presented in Table 3 and depicted in Fig. 1. LMM analysis detected a large, significant main effect of time on all AAQ scales such that PF increased from intake to discharge. No main effect of pain group was observed for any of the AAQ-SA scale criterion. However, a Pain group $\times$ Time interaction was detected for the VC subscale ($F(1, 276)=5.645$, $p=0.018$; Fig. 1) such that AAQ-SA adaptation was significantly slower in the HLP group ($d=0.33$) compared to the LLP group ($d=0.75$). Importantly, as the full-scale BAM score was included as a covariate in this model, these effects explain the variance in VC subscale scores over and above pain level differences in BAM scores. While no Pain group $\times$ Time interaction was observed for the DA subscale ($F(1, 276)=0.06$, $p=0.80$; Fig. 1), the BAM score covariate had a significant main effect on this subscale ($F(1, 276)=5.35$, $p=0.02$), suggesting that this component of PF may be uniquely related to SUD severity (Fig. 2).

4. Discussion

After controlling for initial severity of SUD symptoms, current pain level at treatment intake moderated change in a sub-component of PF, values commitment, but not SUD symptoms during the IOP. During the treatment, pain level also limited improvement in PF but not self-reported SUD symptoms. Poor SUD outcomes among those reporting significant pain may, then, be a product of intractable values surrounding substance use during treatment that cannot otherwise be explained by differences in SUD severity. Though PF generally increased in our sample over time, significant pain had a moderate, negative effect on the

rate of improvement in PF related to VC. That is, the values regarding substance use were more resistant to adaptation for the HLP group, even after accounting for the differences in SUD severity. While short-term improvements in substance use during IOP were evident in both pain groups, evidence from prior studies of the link between PF and psychological well-being suggests that those reporting moderate to severe pain (as the HLP group) may have a less positive long term prognosis in terms of SUD outcomes. As this effect was not similarly present for the DA subscale, the two components of PF appear to operate differently during early SUD recovery. In summary, these results suggest that the poor post-treatment SUD prognosis linked with pain may be due to both SUD severity and restriction in PF adaptation.

Our results also suggest that pain may have a different structural and temporal relationship with different principle components of PF during SUD treatment. While DA increased at the same rate for both pain groups, fewer gains in VC were detected for the significant pain group relative to the mild pain group during IOP. In the earliest stages of abstinence, pain may become more salient and unavoidable because substance use is no longer an available form of avoiding the physical and psychological elements of pain-related distress. Recent research has specifically confirmed that exposure to pain symptoms may reduce the disability and psychological effects of pain conditions (van de Meent et al., 2011). Therefore, ongoing exposure to any level of pain without using substances to mitigate the distress likely increases DA and decreases experiential avoidance. However, a similar shift in VC PF may not occur at the same rate for those who experienced significant pain because the commitment to reduce increasingly salient pain likely competes with the commitment to remain abstinent. Furthermore, higher levels of pain may increase additional risks for poor prognosis and co-opt the cognitive resources necessary to attend to group material and practice active commitment to deeply instantiated values.

Our study is the first to integrate previously separate lines of research to evaluate the relationship between pain and SUD co-morbidity on treatment changes with PF in a clinical sample. A particular benefit of this integration is that it identified that pain poses risk through its relationship with SUD severity and its attenuation of treatment effects for PF. As pain typically predicts poor treatment utilization, the focus of our analyses on a SUD treatment sample endorsing significant variation in pain level marks a relatively important opportunity to advance knowledge in this area and our use of a brief and simplistic measure of pain to discern these differences affords easy application in medical and clinical settings.

Despite these benefits, it is important to acknowledge a number of limitations for the current study. Our sample is comprised entirely of Veterans and included only a small number of females, limiting the generalizability of our findings for broader populations of women and civilians. Additionally, the 30-day duration and intensive format of treatment may uniquely underlie reported results and may not effectively describe a longer-term recovery period. Further, pain ratings indexed current levels of unmanaged pain at the time of intake and do not necessarily distinguish the acuity/chronicity or pain level change during the IOP. Use of a single self-report item for pain level that does not account for these factors likely underestimates the unique effects of protracted pain on SUD treatment and psychological flexibility. Detection of these effects without these additional considerations suggests that

these effects may be large and underlying mechanisms should be further explored in more controlled trials. Lack of a second pain measure at discharge also significantly limits our ability to interpret how changes in pain level during treatment likely influence our findings. Utilization of a broad measure of SUD symptoms (i.e., full-scale BAM) – rather than reliance on report of substance use behavior – allowed us to model differences in SUD-relevant problems that may be prognostic of long-term outcomes. However, it is important to note that these results did not directly measure *long-term* effects of pain on substance use behavior, specifically, as most patients successfully maintained abstinence during the short-term treatment.

Future work evaluating the mechanisms that underlie pain's long-term moderation of both SUD symptoms and PF adaptation will provide an important window into how to mitigate pain's negative effects on SUD prognosis. In particular, research exploring the role of instrumental variables not measured in the current study (e.g., circadian rhythms and attentional bias related to pain) will help elucidate the link between pain level and PF regarding substance use during treatment. Additionally, future research mapping out the similarities and differences between PF and other, similar constructs like cognitive flexibility, emotional intelligence, resilience, and personality will be advantageous for interpreting these effects and the . As our results highlight the unique role of PF in SUD treatment for those in pain, testing the efficacy of direct interventions designed to enhance PF (i.e., Acceptance and Commitment Therapy and Mindfulness-Based Relapse Prevention) in both SUD and pain populations may be a vital step in improving SUD treatment outcomes for those in pain. Future clinical trials designed to increase commitment to shifting value about substance use may provide unique benefits for those with significant pain in the early stages of SUD recovery. Additionally, initial screening for pain level in the context of SUD treatment may be fruitful for identifying those in significant pain who may benefit from behavioral pain treatment and additional support to bolster psychological flexibility. As this and other research on SUD treatment response illustrates, it is evident that pain may spur and maintain ongoing mental health problems that compromise well-being through numerous pathways. Further explication of the mechanisms through which pain alters the course and remission of psychiatric symptoms is vital for mitigating these effects in the SUD population.

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Fig. 1.
Significant main effects of time and pain group on full scale Brief Addiction Monitor (BAM).

Fig. 2.
AAQ-SA subscale scores by pain group over time.

Table 1

Demographic and clinical characteristics of the treatment sample.

	%
<i>Age</i>	
18–44	39.3%
45	60.7%
<i>Sex</i>	
Male	92.9%
<i>Race</i>	
African American	19.8%
Caucasian	78.1%
Other	2.1%
<i>Employment</i>	
Full-time	5.3%
Part-time	1.8%
Retired	8.8%
Disabled	44.9%
Unemployed (seeking work)	35.0%
Other	4.2%
<i>Marital Status</i>	
Single	42.4%
Married	15.2%
Divorced	32.2%
Other	10.2%
<i>Residential Status</i>	
Own Residence	42.4%
Cohabiting	29.7%
Transitional Housing	5.3%
Homeless	17.3%
Other	4.6%
Unknown	0.7%
<i>Referral Source</i>	
Inpatient (Mental Health)	27.4%
Outpatient Substance Use Clinic	28.8%
Emergency Department	2.8%
Self	0.8%
Other	40.2%
<i>Comorbidities</i>	
<i>(% in Moderate to Severe Range)</i>	
Generalized Anxiety	37.3%
Post-traumatic Stress Disorder	88.8%
Major Depression	52.9%

	%
<i>Average number of comorbidities</i>	1.83

Percentages for clinical symptoms reflect those in the moderate to severe range as designated by the measures used. Average number of comorbidities reflects an average of the sum of those meeting this clinical threshold for the measures included.

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Table 2

Means, standard deviations, and Cohen's *d* effect sizes for the main effects of pain and time on Brief Addiction Monitor items and full scale ratings.

	Pain groups		Cohen's <i>d</i> effect sizes	
	LLP group (n=165)	HLP group (n=142)	Pain effect	Time effect
Full Scale Score	31.78 (10.79)	35.22 (10.24)	0.33 **	-1.54 ***
<i>Substance Use Items</i>				
Any Alcohol Use (days)	1.83 (1.62)	1.99 (1.52)	0.10	-1.25 ***
Heavy Alcohol Use (days)	1.72 (1.61)	1.90 (1.57)	0.11	-1.21 ***
Drug Use (days)	1.31 (1.54)	1.42 (1.60)	0.07	-0.92 ***
Marijuana (days)	0.66 (1.22)	0.63 (1.20)	-0.02	-0.55 ***
Sedative (days)	0.40 (0.98)	0.64 (1.23)	0.22	-0.39 **
Cocaine (days)	0.55 (1.08)	0.93 (1.42)	0.30 **	-0.68 ***
Stimulants (days)	0.16 (0.71)	0.27 (0.90)	0.14	-0.35 **
Opiates (days)	0.68 (1.30)	0.78 (1.36)	0.08	-0.53 ***
Inhalants (days)	0.21 (0.70)	0.20 (0.68)	-0.01	-0.36 **
<i>Risk Factor Items</i>				
Physical Health	2.64 (0.93)	2.84 (0.87)	0.22 **	-0.62 ***
Insomnia	2.93 (1.26)	3.25 (1.04)	0.28 **	-0.37 **
Mood	2.92 (1.25)	3.36 (2.12)	0.25 **	-0.74 ***
Cravings (days)	2.45 (1.24)	2.56 (1.28)	0.09	-0.99
Risky Situations	1.75 (1.54)	1.88 (1.48)	0.09	-0.63
Interpersonal Conflict	1.63 (1.24)	1.80 (1.35)	0.13 *	-0.61
<i>Protective Factor Items</i>				
Self-efficacy	3.14 (0.97)	2.92 (0.98)	-0.23	0.11
Self-help behaviors	1.05 (1.33)	1.05 (1.25)	0.00	0.95
Supportive Spiritual Practice	2.12 (1.52)	2.50 (1.45)	0.26 *	0.18
Work/School Participation	0.74 (1.26)	0.68 (1.26)	-0.05	0.52
Adequate Income	1.06 (1.51)	1.18 (1.57)	0.08	0.05
Sober Support	2.50 (1.48)	2.47 (1.41)	-0.02	0.11
Treatment Satisfaction	2.30 (1.19)	2.33 (1.14)	0.03	0.68

Tukey's HSD Corrected.

*
p 0.05.

**
p 0.01.

p 0.001.

Table 3

F-statistics and Cohen's *d* effect for pain group differences in the Acceptance and Action Questionnaire – Substance Abuse (AAQ-SA) full and subscales before and after IOP treatment.

	AAQ-SA	AAQ-SA	AAQ-SA
	Full scale	Defused acceptance subscale	Values commitment subscale
LMM Effects			
Pain	F (1,276)=2.06	F (1,276)=1.42	F (1,276)=1.02
Time	F (1,276)=16.07***	F (1,276)=9.66**	F (1,276)=9.10**
Use	F (1,276)=2.01	F (1,276)=5.35*	F (1,276)=0.01
Time × Pain	F (1,276)=2.07	F (1,276)=0.06	F (1,276)=5.64*
Time × Use	F (1,276)=0.28	F (1,276)=0.62	F (1,276)=1.73
Cohen's <i>d</i> Effect Sizes			
<i>Time (Post vs. Pre)</i>			
LLP	0.89	0.70	0.75
HLP	0.75	0.73	0.33
<i>Pain Group (HLP vs. LLP)</i>			
Pre-treatment	-0.07	-0.14	0.03
Post-treatment	-0.35	-0.14	-0.43