

# Relationships between components of emotional intelligence and physical pain in alcohol-dependent patients

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**Purpose:** Chronic pain is a significant comorbidity in individuals with alcohol dependence (AD). Emotional processing deficits are a substantial component of both AD and chronic pain. The aim of this study was to analyze the interrelations between components of emotional intelligence and self-reported pain severity in AD patients.

**Patients and methods:** A sample of 103 participants was recruited from an alcohol treatment center in Warsaw, Poland. Information concerning pain level in the last 4 weeks, demographics, severity of current anxiety and depressive symptoms, as well as neuroticism was obtained. The study sample was divided into “mild or no pain” and “moderate or greater pain” groups.

**Results:** In the logistic regression model, across a set of sociodemographic, psychological, and clinical factors, higher emotion regulation and higher education predicted lower severity, whereas increased levels of anxiety predicted higher severity of self-reported pain during the previous 4 weeks. When the mediation models looking at the association between current severity of anxiety and depressive symptoms and pain severity with the mediating role of emotion regulation were tested, emotion regulation appeared to fully mediate the relationship between depression severity and pain, and partially the relationship between anxiety severity and pain.

**Conclusion:** The current findings extend previous results indicating that emotion regulation deficits are related to self-reported pain in AD subjects. Comprehensive strategies focusing on the improvement of mood regulation skills might be effective in the treatment of AD patients with comorbid pain symptoms.

**Keywords:** mood regulation, pain, alcoholism, emotional regulation

## Introduction

According to the American Society of Addiction Medicine, “dysfunctional emotional response,” along with cognitive function impairments, is a key feature of addiction. Deficits in emotional processing including emotion recognition, labeling,<sup>1,2</sup> and alexithymia<sup>3</sup> have been reported in various alcohol-dependent (AD) samples. Difficulties in the identification and description of one’s own emotional state, together with impairments in perception and labeling of emotion displayed by others, may represent a complex emotion-processing deficit in this clinical group. The aforementioned competencies are implied as one of the basic constituents of emotional intelligence (EI). EI has been defined<sup>4</sup> as a compound of emotional skills or traits that includes: “the ability to perceive accurately, appraise, and express emotion; the ability to access and/or generate feelings when they facilitate thought; the ability to understand emotion and emotional knowledge; and the ability to regulate emotions to promote emotional and intellectual growth”. Preliminary data suggest that individuals with AD also have

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lower EI; however, there is still not enough clinical data coming from AD population to make unequivocal conclusions.<sup>5,6</sup> Of importance, limited data show that emotion-processing deficits may be associated with adverse outcomes in AD samples. Deficits in utilization of emotions predicted poorer post-treatment outcomes in a recent study of those treated for AD.<sup>7</sup> In another study, poor emotion-regulation skills in AD samples predicted post-treatment alcohol use at 3-month follow-up.<sup>8</sup>

Pain is commonly comorbid with alcohol use disorders (AUDs).<sup>9,10</sup> National surveys have revealed that AD is approximately twice as likely to occur among individuals with chronic pain.<sup>9,10</sup> In clinical samples, more than 40% of patients treated for chronic pain met criteria for an AUD.<sup>11</sup> Conversely, between 18% and 38% of patients who participated in various addiction treatment programs in the USA reported at least moderately severe pain in the last 12 months.<sup>12</sup> Despite the importance of understanding the intersection between pain and alcohol use, only limited work has been carried out in this area and most of this has been done from a neurobiological perspective. Of clinical importance, pain is a predictor of poor drug- and alcohol-related outcomes in those treated for addictive disorders, including AUDs,<sup>12,13</sup> and self-reported decrease in pain following treatment for AD is associated with a lower risk of alcohol relapse.<sup>14</sup>

Several explanations for the prevalent co-occurrence of AUD and chronic pain have been proposed. It has been postulated that comorbidity of pain and AUD may be explained by recursive, partly shared, neural systems. In this context, pain is conceptualized as a disorder of reward function<sup>15</sup> sharing pathophysiological mechanisms with addictions.<sup>16</sup> Biologically, a strong evidence of overlapping neurocircuitry, underlying pain and addiction, led Egli et al<sup>17</sup> to argue that AUD could be conceptualized as either a chronic pain disorder or a type of chronic emotional pain syndrome (p. 449).<sup>18</sup>

Among the psychological mechanisms that influence pain,<sup>19</sup> a significant role for emotional processes has been affirmed.<sup>20–22</sup> It is acknowledged that pain can be derived solely from emotional or social sources in the absence of nociception.<sup>16</sup> Neurobiological data indicate reciprocal interactions between pain and emotions.<sup>23,24</sup> Numerous studies highlight interactions between pain and stress, pain and negative affect, pain and alexithymia, and broadly defined emotional regulation processes and pain.<sup>21,24,25</sup> Such studies also demonstrate the modulating effect of negative emotions on pain intensity.<sup>26–29</sup> Moreover, chronic pain can elicit ineffective coping strategies and physical and psychosocial disability, leading to reduced quality of life<sup>30</sup> and depressive

or anxiety symptoms. It has been consistently reported that more than 50% of chronic pain patients also manifest clinical symptoms of depression.<sup>31,32</sup> Wiech and Tracey<sup>33</sup> noted that the relationship between pain and emotions is bidirectional, in that the experiential component of pain aggravates concurrent negative emotions, which then fuels the experience of pain. Studies suggest that individual differences in emotional regulation intervene in this relationship. In a sample of women with rheumatoid arthritis, Hamilton et al<sup>34</sup> found that self-reported pain severity varied as a function of emotional intensity and regulation. Women who reported higher emotional intensity and lower ability to regulate their emotions suffered more after a week of increased pain. Likewise, Connelly et al<sup>35</sup> found that variability in regulating positive and negative affect was associated with the pain severity in a group of subjects with rheumatoid arthritis. Within the EI framework, Ruiz-Aranda et al<sup>36</sup> showed that healthy women, who rated themselves as being more skilled in “emotional repair” (i.e., the ability to use positive thinking to repair negative mood), perceived a standard pain stimulus – induced via the cold-pressor experimental paradigm (CPT) – as less painful than did women who self-reported lower skills. In another study from this group,<sup>37</sup> participants with higher behaviorally measured EI scores rated pain as both less intense and unpleasant.

Together, the current literature shows that emotional processing deficits are common in both AD and chronic pain, and may lead to significant adverse outcomes in AD subjects. Moreover, recent data reveal that the two might share a common neurobiological background.<sup>38</sup> As suggested by LeBlanc et al<sup>38</sup> chronic pain and nociceptive hypersensitivity may induce alcohol craving and relapse through alterations in synaptic plasticity within brain reinforcement circuitry. The authors further suggest that pain-induced affective dysregulation in vulnerable individuals may contribute to the transition to addiction. Accordingly, clinical data show that pain syndromes are most commonly diagnosed among psychiatric patients, when reward alterations are also noted.<sup>15</sup> To the best of our knowledge, there are no data on the relationship between EI and pain severity in a clinical sample of AD individuals. Hence, the purpose of the present study was to explore the relationship between components of EI and self-reported pain severity in AD patients. We also examined whether EI components mediate the relationship between components of negative affect (severity of depression and anxiety) and self-reported pain. We anticipated that EI would make an independent contribution to the prediction of pain severity in this treatment sample.

## Patients and methods

### Participants

The study group comprised 103 alcohol-dependent patients entering an 8-week inpatient treatment program in Warsaw, Poland. The study assessments were performed within the first week since admission. Subjects with comorbid severe medical illness, receiving or requiring opioid analgesic therapy, as well as those currently receiving pharmacotherapy for AD, were not admitted to the inpatient treatment program. Similarly, individuals with comorbid psychiatric disorders requiring medication were not admitted to the treatment center. A current diagnosis of AD was established according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed, Text Revision<sup>39</sup> criteria during admission and confirmed with the MINI International Neuropsychiatric Interview<sup>40</sup> by a trained member of the research team. Confirmed abuse or dependence on psychoactive substances other than nicotine and alcohol was exclusionary. All individuals with significant cognitive deficits, scoring <25 on the Mini-Mental State Examination,<sup>41</sup> as well as patients with a history of psychosis or presence of alcohol withdrawal symptoms, were not eligible to participate.

The study was approved by the Bioethics Committee at the Medical University of Warsaw and the Medical School Institutional Review Board at the University of Michigan. Written informed consent was obtained from all individuals who participated in the study.

### Measures

Sociodemographic characteristics (education, marital status, and employment) were obtained using a modified version of the University of Arkansas Substance Abuse Outcomes Module, a self-administered questionnaire.<sup>42</sup> A single item from the Polish version of Short Form Health Survey (SF-36)<sup>43</sup> was included as a measure of physical pain during the past 4 weeks. The question that was asked was, "During the last 4 weeks, how much physical pain did you experience on average?," with the possible responses: 1 – no pain, 2 – very mild pain, 3 – mild pain, 4 – moderate, 5 – strong, and 6 – very strong physical pain during the last 4 weeks. Consistent with prior work,<sup>12,44,45</sup> the responses 1–3 were subsequently re-coded into a categorical variable "mild or no pain," and responses 4–6 into "moderate or greater pain."

The subscale score for state anxiety from the brief symptom inventory,<sup>46</sup> depression severity,<sup>47</sup> and neuroticism (NEO Five-Factor Inventory)<sup>48,49</sup> were included as measures of negative affect.

The Schutte Self-Report Emotional Intelligence Test (SSEIT) was utilized as a measure of EI. The most consistently reported factor structure of SSEIT has four factors, which were applied in this study. Factors were calculated and named according to Saklofske et al<sup>50</sup> and matched those described by Petrides and Furnham<sup>51</sup> as follows: appraisal of emotions, mood regulation/optimism, utilization of emotions, and social skills.

### Statistical analysis

In the bivariate analyses, AD subjects dichotomized by the severity of pain ("mild or no pain" vs "moderate or greater pain") were compared in terms of basic demographic characteristics as well as severity of depressive and anxiety symptoms, neuroticism, and EI components. Analyses of variance (ANOVAs) and chi-square tests tested for differences between the two pain-level groups for continuous and dichotomous variables, respectively. Subsequently, a logistic regression model was applied in order to determine the strongest independent correlates of physical pain in the current AD sample. To avoid multicollinearity between the affect-related risk factors (EI, depression, anxiety, and neuroticism), a correlation matrix was performed, and intercorrelation values above 0.7 between variables excluded them from further analyses. Controlling for age and gender, all variables that were significantly associated with pain severity in the bivariate analyses were included in the model.

Since it was hypothesized that individual differences in EI would serve as a mediator in the relationship between depression/anxiety and pain severity, two sets of mediation analyses were conducted: one with depression and the other with anxiety being the independent variables. Mediation was tested using the Preacher and Hayes's bootstrapping method with 5,000 resamples with replacement. For this, the SPSS macro suggested by Preacher and Hayes was used. The criterion for statistical significance in all tests (two-tailed) was  $p < 0.05$ . The data were analyzed using statistical package SPSS® 23.0 for Windows.

### Results

The study comprised 80 men (77%) and 23 women (23%). Their mean ( $\pm$ SD) age was 43.57 ( $\pm$ 11.47) years and mean education level was 12.07 ( $\pm$ 2.82) years (the last level of secondary school in Poland). All patients were Caucasian.

ANOVAs looking at associations with self-reported pain levels revealed that patients with moderate/severe physical pain had higher neuroticism scores ( $F=6.79$ ;  $p=0.01$ ) and

**Table 1** Comparison of alcohol-dependent patients with moderate/severe and without or mild physical pain

	No/mild physical pain (n=80)	Moderate/severe physical pain (n=23)	p-value
Age (years)	43.71 (12.48)	45.0 (9.3)	0.89
Gender (males/females)	59/19	18/4	0.38
Education (number of classes completed)	12.39 (2.74)	10.65 (2.68)	<b>0.01</b>
Severity of depressive symptoms (BDI)	16.92 (10.62)	22.91 (11.10)	<b>0.02</b>
Severity of anxiety symptoms	0.69 (0.62)	1.26 (0.96)	<b>0.001</b>
Neuroticism	25.22 (7.89)	29.91 (5.59)	<b>0.01</b>
SSEIT <sub>R</sub>	37.78 (5.34)	33.77 (5.37)	<b>0.003</b>
SSEIT <sub>A</sub>	19.38 (3.46)	18.41 (2.94)	0.23
SSEIT <sub>U</sub>	14.97 (2.18)	13.77 (2.69)	<b>0.03</b>
SSEIT <sub>S</sub>	18.33 (2.91)	16.91 (2.91)	0.05

**Notes:** The categorical values are presented as numbers. Parametric variables are presented as mean and standard deviation. P-values <0.05 are shown in bold.

**Abbreviations:** BDI, Beck Depression Inventory; SSEIT<sub>A</sub>, Schutte Self-Report Emotional Intelligence Test – appraisal of emotions; SSEIT<sub>R</sub>, Schutte Self-Report Emotional Intelligence Test – mood regulation; SSEIT<sub>S</sub>, Schutte Self-Report Emotional Intelligence Test – social skills; SSEIT<sub>U</sub>, Schutte Self-Report Emotional Intelligence Test – utilization of emotions.

higher severity of depressive ( $F=5.35$ ;  $p=0.02$ ) and anxiety symptoms ( $F=10.93$ ;  $p=0.001$ ). Higher pain levels were also associated with lower education ( $F=6.38$ ;  $p=0.01$ ). By contrast, subjects who reported no/mild physical pain had higher scores in two of the four EI factors: self-reported mood regulation ( $F=9.60$ ;  $p=0.003$ ) and utilization of emotions ( $F=4.63$ ;  $p=0.03$ ). The detailed comparisons of individuals with moderate/severe physical pain and those with mild or no pain are presented in Table 1.

All variables that were significantly associated with pain levels were entered into a logistic regression analysis. After controlling for age and gender, mood regulation (OR=0.83; 95% CI: 0.70–0.99;  $p=0.04$ ), anxiety severity (OR=2.91; 95% CI: 1.11–7.62;  $p=0.03$ ), and education (OR=0.76; 95% CI: 0.59–0.99;  $p=0.04$ ) remained significant predictors of pain severity. The overall model was significant (chi-square=20.48,  $df=8$ ,  $p=0.009$ ) and explained 30% of the variance in pain severity (Table 2).

## Mediation analyses

It was hypothesized that mood regulation would mediate the effect of both depression and anxiety on pain, thus two sets of mediation analyses were conducted. The first analysis revealed that the indirect effect of depression on pain through mood regulation was significant, with an unstandardized point estimate of 0.03 and a 95% CI of 0.002–0.08. Furthermore, the direct effect of depression on pain (0.05; SE=0.02;  $p<0.03$ ) became nonsignificant when mood regulation was included as a mediator of the direct effect in the model (0.02; SE=0.03;  $p=0.52$ ). Thus, mood regulation fully mediated the relationship between depression and pain (Figure 1).

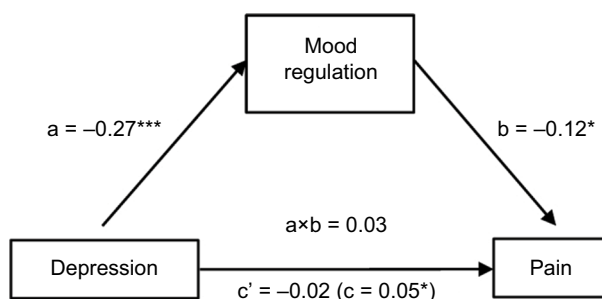
The second analysis yielded a significant indirect effect of anxiety on pain through mood regulation (0.25) with a 95% -confidence interval from 0.04 to 0.66. Mood regulation only

**Table 2** Multivariate model of logistic regression analysis for the prediction of moderate/severe physical pain in alcohol-dependent patients

	OR (95% CI)	p-value
SSEIT <sub>R</sub>	0.83 (0.70–0.99)	0.04
SSEIT <sub>U</sub>	1.05 (0.78–1.42)	0.75
Age	1.02 (0.96–1.08)	0.56
Gender	1.78 (0.28–4.90)	0.82
Education	0.76 (0.59–0.99)	0.04
Neuroticism	0.99 (0.88–1.12)	0.95
Severity of anxiety symptoms	2.91 (1.11–7.62)	0.03
Severity of depressive symptoms	0.95 (0.88–1.03)	0.21

**Notes:** Model:  $R^2$  (Nagelkerke)=0.31. Chi-square=20.48;  $df=8$ ;  $p=0.009$ .

**Abbreviations:** SSEIT<sub>R</sub>, Schutte Self-Report Emotional Intelligence Test – mood regulation; SSEIT<sub>U</sub>, Schutte Self-Report Emotional Intelligence Test – utilization of emotions.

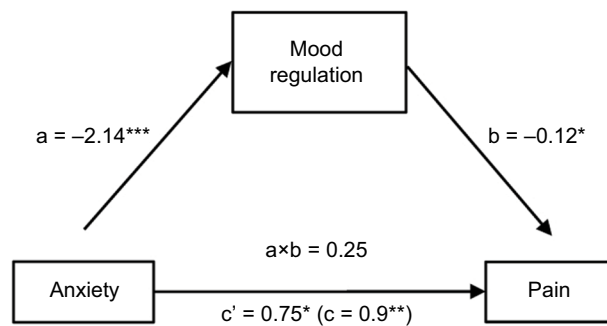
**Figure 1** Mood regulation as a mediator of the relationship between depression and pain.

**Notes:** The values in the figure are unstandardized path coefficients. \* $p<0.05$ , and \*\*\* $p<0.001$ .

partially mediated the anxiety–pain relationship, as anxiety still had a significant direct effect on pain (0.75; SE=0.32;  $p<0.02$ ), albeit lower than without mood regulation being controlled for (0.89; SE=0.31;  $p<0.005$ ) (Figure 2).

## Discussion

The purpose of our study was to examine how various components of self-reported EI relate to self-reported pain severity in AD individuals. To our knowledge, this is the first study to



**Figure 2** Mood regulation as a mediator of the relationship between anxiety and pain.

**Notes:** The values in the figure are unstandardized path coefficients. \* $p < 0.05$ , \*\* $p < 0.01$ , and \*\*\* $p < 0.001$ .

examine the association between self-reported EI and pain in this clinical population. The study results show that while controlling for negative affect and sociodemographic factors in AD patients, deficits in emotional regulation – a component of EI measured by the SSEIT scale – were a significant correlate of self-reported pain severity during the previous 4 weeks. Utilization of emotions was associated with pain severity before controlling for other variables, but the other two components of EI were not associated with pain. Anxiety and lower education – albeit not depression – were also associated with pain in the multivariate analysis. Education has consistently been shown to be a significant correlate of physical pain severity.<sup>52–55</sup>

The mood regulation factor in the SSEIT scale reflects a person's ability to understand, recognize, and manage his/her feelings. It measures the capacity to maintain positive feelings on the basis of both previous experiences of doing so and the anticipation and involvement in activities that enhance positive feelings. The better their ability at mood regulation, the easier for individuals to acquire, maintain, and benefit from positive affect.

The study results also revealed that mood regulation fully mediated the relationship between depression and pain, and partially the relationship between anxiety and pain. In other words, greater levels of negative affect (depression or anxiety) were associated with worse emotional regulation skills and more pain. Our results suggest that the self-perceived ability to regulate emotions is a major contributor to the relationship between negative affect and pain. Negative affect may increase the perception of pain, and pain may increase negative affect, but their bidirectional effects depend on emotional regulation competencies in AD patients. These results are consistent with studies in healthy controls and chronic pain populations, focusing on the influence of the negative/positive emotions utilization skills in the pain–affect relationship.<sup>35,56–60</sup>

The results of our study are in line with observations that emotional traits and abilities are associated with self-reported pain severity in chronic pain patients as well as pain-free participants.<sup>34,36,37,61</sup> In addition, the results of the previous studies reveal that with the changing emotional state (negative or positive affect), pain sensitivity, and perception changes accordingly, depending on the valence of the emotion induced.<sup>62,63,21</sup> Studies on the effects of mood induction (positive, negative, or neutral) in pain-free participants show that the induction of negative mood results in a significant decrease in CPT pain tolerance, whereas the effect is reversed for the positive mood induction condition.<sup>64</sup> Importantly, increased positive affect weakens the association between negative affect and pain.<sup>57–60</sup> This finding has been corroborated by longitudinal observations showing that pain reduction is predicted by increases in positive and decreases in negative affect.<sup>35,56</sup> Conversely, pain can lead to long-lasting negative emotions, with the modulating effect of individual personality characteristics or previous reflection related to pain experience referred to as “secondary pain affect.”<sup>65</sup> Chronic “unspecific” pain may itself be considered a form of masked depression.<sup>66</sup> Through its relationship to aggravated depressive symptoms, pain increased the probability of depression recurrence.<sup>67</sup> Across studies, patients with chronic conditions (back pain, arthritis, fibromyalgia, and chronic widespread pain) generally have a two- to eight-fold greater likelihood of being diagnosed with a depressive disorder.<sup>68</sup> Meta-analytic data confirmed the pain-reducing properties of antidepressants (vs placebo) in chronic back pain patients.<sup>69</sup> Importantly, once a cycle of chronic or recurrent pain and negative affect (as in AD subjects) has developed, it might be difficult and clinically irrelevant to determine the current causal relationship between the two conditions.<sup>33</sup>

Among the various emotional correlates of pain, significant attention is now being directed to the variably defined theoretical concept of emotion regulation. In a longitudinal study, Paquet et al<sup>56</sup> found that increased affect regulation was related to lower levels of pain intensity. Ruiz-Aranda et al<sup>36</sup> showed that pain-free women scoring high in the self-perceived ability to use positive thinking to repair negative mood, reported less sensory and affective pain during the CPT procedure. Importantly, once the experimental task was over, the negative impact of pain induction on mood was less severe in those scoring high in emotional repair. These results are consistent with the other studies showing that higher emotional repair is associated with lower perception of pain.<sup>35,56</sup>

Our results have clinical implications. Emotional dysregulation is a significant correlate of alcohol use disorders,



even in the absence of chronic pain, and drinking alcohol is commonly used by patients as an ineffective self-medication strategy for both managing pain<sup>70</sup> and coping with negative affect.<sup>71</sup> Growing research data suggest common underlying neurobiological mechanisms for chronic pain and addiction, including reward deficiency and anti-reward processes, incentive sensitization, and aberrant learning.<sup>15</sup> As noted by Navratilova and Porreca<sup>16</sup> chronic pain is a constant challenge for relief and can suppress or surpass other emotions, including natural rewards, leading to negative affect and anhedonia (as a reflection of reward deficiency). These negative affective states supported with the pain-induced dysregulation of reward/reinforcement circuitry may lead to excessive substance use and possibly, for some vulnerable individuals, contribute to the development of addiction.<sup>38</sup>

Recent preliminary findings revealed that deficits in utilizing and regulating emotions were an independent predictor of relapse in AD patients,<sup>7</sup> whereas chronic pain was shown to be associated with worse pain-related and substance-related outcomes among adults treated for substance use disorders.<sup>13,14,72</sup> These observations underlie the clinical significance of further studies on emotional competencies and pain in AD individuals. Given that 1) pain is a highly prevalent and problematic comorbidity in addiction treatment settings and 2) emotional regulation deficits are a significant correlate of both pain and AD, it is reasonable to develop and test therapeutic strategies to improve emotional skills in patients with this comorbidity. The efficacy of psychological interventions targeted at reducing pain and improving functioning in persons with a broad spectrum of pain-related conditions has been demonstrated.<sup>73,74</sup> However, these treatments have not been well tested in those with AUDs. Mindfulness has been suggested as a potentially beneficial therapy for chronic pain,<sup>74</sup> and has also been used for relapse prevention in AD patients.<sup>75</sup> Our data suggest that affect regulation skills training might reduce negative affect and its effect on pain intensity.

The results of our study need to be interpreted with caution because of several limitations. First, the cross-sectional design of the study precludes any conclusions regarding causation and/or directionality of the revealed associations, specifically, whether higher depressive or anxiety severity leads to greater self-reported pain or the painful experience generates higher negative affect, with the mediating effect of emotion regulation. Our findings should be confirmed within the experimental design of longitudinal observations. Moreover, our single-item, self-report measure of pain intensity in the previous 4 weeks is unidimensional, albeit utilized in

other studies.<sup>12,76,77</sup> It does not assess for the perceptual threshold, cause, location, or type of pain as well the specific context for its chronicity, origin, or continuation into the present. Except for the exclusionary criterion of opioid use, the data on the utilization of other pain management strategies such as nonsteroidal anti-inflammatory drugs or physical therapy, in the group of AD patients with moderate or greater pain, were not available. Finally, our study group was confined only to patients who entered inpatient treatment for AD, which may not be representative of the broader population of individuals with AD and comorbid pain syndromes.

## Conclusion

The results of the current study extend previous findings and suggest that emotion regulation deficits are related to severity of self-reported pain in AD subjects. Comprehensive psychotherapeutic interventions focusing on the improvement of mood regulation skills might improve the effectiveness of the treatment of AD patients with the comorbid pain symptoms.

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## Disclosure

The authors report no conflicts of interest in this work.

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