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Personality and Affect When the Central Nervous System is Sensitized: An Analysis of Central Sensitization Syndromes in a Substance Use Disorder Population

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Abstract

Functional somatic syndromes, or more recently termed central sensitivity syndromes (CSS), comprise a significant portion of the chronic pain population. Although it is evident that personality is intricately related to the pain experience, it has not been widely studied. This article examines the impact of CSS on the clinical presentation of individuals presenting to treatment for a substance use disorder (SUD), with an emphasis on personality and emotional functioning. We examined personality profiles of individuals presenting to treatment with SUD between three groups: those with a CSS ($n = 30$), non-CSS chronic pain ($n = 79$), and no pain ($n = 232$). Based on previous research and a psychodynamic conceptualization of CSS, we hypothesized that predictors of the presence of a CSS in this sample would be higher rates of overall anxiety, traumatic stress, perfectionistic traits, and a need for interpersonal closeness. Logistic regression analyses did not support our hypothesis. Exploratory analyses indicated which personality traits most strongly predicted the presence of CSS. We discuss these findings using descriptive psychopathology literature, with recommendations for future research.

Keywords

central sensitivity; substance use; affect regulation; personality; MMPI-II

Chronic, persistent pain is highly prevalent in substance abuse treatment settings, with prevalence rates ranging from 24–61% (Jamison, Kauffman, & Katz, 2000; Rosenblum et al., 2003). The impact of chronic pain on addiction complexity and prognosis is significant. Individuals with chronic pain in treatment for substance use disorder (SUD) present with more severe SUD symptoms than those without pain (Potter, Prather, & Weiss, 2008). Chronic pain is also associated with characteristics that predict poor SUD treatment

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outcomes, including extensive treatment histories, earlier age of first use, functional impairment, health problems, and high psychiatric comorbidity (Jamison et al., 2000; Potter, Hennessy, Borrow, Greenfield, & Weiss, 2004; Potter et al., 2008).

Functional somatic syndromes, or more recently termed “central sensitivity syndromes” (CSS; Yunus, 2005), comprise a significant proportion of the chronic pain population—accounting for up to 30% of outpatient visits in primary care settings (Kirmayer, Groleau, Looper, & Dao, 2004). Central sensitivity syndromes are a similar and overlapping group of syndromes that are bound by a unifying pathophysiological mechanism of pain and/or sensory amplification, which is commonly referred to as the process of central sensitization (CS; Ablin & Clauw, 2009; Yunus, 2005). Individuals with CSS display diffuse pain sensitivity (hyperalgesia) and reduced pain thresholds (allodynia), which can occur without the presence of painful stimuli (Woolf, 2011). This suggests that CSS conditions are disorders of *pain processing* in the central nervous system, and explains why conditions often overlap, are not confined to one specific region of the body, and patients can be diagnosed with several CSS conditions (Ablin & Clauw, 2009; Woolf, 2011; Yunus, 2005). This process can be observed in patients presenting with pain in multiple locations, functional disturbance in different organ systems, and complaints surrounding fatigue and exhaustion (Henningsen, Zipfel, & Herzog, 2007). Examples of CSS conditions include fibromyalgia, irritable bowel syndrome, chronic fatigue syndrome, migraine, and temporomandibular joint disorder (Yunus, 2007a, 2007b). These conditions are common, costly, persistent, and can be disabling (Wessely & White, 2004). Not surprisingly, patients with high levels of these symptoms engage in healthcare use at rates that far surpass use by peers, costing the U.S. healthcare system 100 billion dollars annually (Barsky, Orav, & Bates, 2005).

The impact of CSS on those presenting to treatment for SUD is largely unknown. Although it is evident that personality is intricately related to the pain experience, it has not been widely studied (Malin & Littlejohn, 2012). To date, a review examining the relationships between chronic pain and SUD concluded there were no patterns of demographic, pain-related, or psychiatric variables consistently distinguishing pain patients with or without a history of SUD (Morasco et al., 2011). The existing literature is extremely limited and has yet to examine emotional or personality functioning in depth. Furthermore, to the best of our knowledge, there has yet to be an investigation of CSS conditions separately. Acknowledging a dearth in literature, experts have called for further examination of SUD in pain patients in order to provide clinical guidance to manage this comorbidity (Adams & Turk, 2015). The purpose of this article is to examine the impact of CSS on the clinical presentation of individuals presenting to treatment for an SUD, with an emphasis on personality and emotional functioning.

ALLOSTASIS: STRESS AND CENTRAL SENSITIZATION

Research strongly indicates that childhood adversity, severe life-threatening stress, and chronic stress can result in permanent, irreversible enhancement of the responsiveness of the central stress circuitry and vulnerability to development of CSS later in life (Henningsen et al., 2007; Mayer, Naliboff, Chang, & Coutinho, 2001; McKernan, Carr, & Nash, 2016;

Roelofs & Spinhoven, 2007). Other factors identified in case-control studies that increase vulnerability to CSS include accidents, physical injury, and infection (Ablin & Clauw, 2009). Woolf (2011) relates these events by discussing "stress," whether physical or psychological, as a system in the body that is activated by real or perceived threat to homeostasis. When signaled, the body internally activates programs of neural, hormonal, and behavioral activity in response, which involves the autonomic nervous system, HPA-axis, metabolic, and immune systems that lead to *protection* and *adaptation* in the face of threat (Sterling & Eyer, 1988; Woolf, 2011).

There is longstanding interest in the effects of stress on health, due to the strain that it places on the adaptive capacity of individuals over time, which increases risk of disease (McFarlane, 2010). The key to understanding the link between chronic stress and disease involves the principle of allostatic load (McEwen, 1998; Sterling & Eyer, 1988). Allostatic load results when the stress-regulation systems of the body are overworked, fail to shut off after the stressful event is over, or when systems fail to react adequately to initial stress—leading other systems to over-react. Allostatic load is the wear and tear that results on the body and brain as a result, for example, in atrophy of muscle tissue, impaired tissue repair, immune system suppression, and morphological alterations of brain structures (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Interestingly, other shared underlying mechanisms have been identified in CSS that may be partly responsible for symptom expression involve these systems and include neurogenic inflammation, dysfunction of the autonomic nervous system, and hypothalamic pituitary dysfunction (Ablin & Clauw, 2009; McBeth et al., 2005). More specifically, there is thought to be an adaptation of the neuroendocrine system through "down-regulating" the HPA-axis after excessive, chronic stress overload (Mayer et al., 2001).

APPROACHING PERSONALITY CORRELATES OF CENTRAL SENSITIZATION

Different components of personality may modulate the process underlying central sensitization through influence on these pain-modulating mechanisms (Malin & Littlejohn, 2012). Chronic stress response dysregulation involves changes to a wide variety of cognitive, interpersonal, and affective dimensions of the person (Gross, 2002; McEwen, 2004). From a psychodynamic perspective, these various styles can be understood as inter- and intrapersonal adaptations to stress, particularly around managing anxiety and affect be it through implicit emotion regulation mechanisms, dissociation of awareness, or styles of relating to oneself and others. Such adaptations in personality and affective functioning may have long-term negative consequences as they can precipitate further psychopathology and can come at significant physiological cost.

Given the pervasiveness of pain responses to stimuli that are normally not painful (allodynia) in central sensitization disorders and the impaired affect regulation among these patients (Finan, Zautra, & Davis, 2009; Nicol et al., 2016; Okifuji & Turk, 2002), we anticipated that individuals with this disorder would have higher need for interpersonal closeness when compared to others with SUDs. The presence of functional somatic complaints has been

connected to insecure attachment in some patients, where individuals rely on secondary attachment strategies in response to stress (Luyten, Van Houdenhove, Lemma, Target, & Fonagy, 2013; Waller & Scheidt, 2006). These secondary attachment strategies can manifest in hypersensitive and/or deactivated attachment (Luyten et al., 2013; Shaver & Mikulincer, 2007). When patients utilize deactivating strategies, there is a striving for autonomy and resilience, with significant underlying vulnerability present (Van Houdenhove & Luyten, 2008). Alternatively, when using hyperactivating strategies, patients seek excessive comfort and relief from others—often leading to further disappointment and frustration (Waller & Scheidt, 2006). Having high pain sensitivity to touch could also disturb ordinary security and comfort, resulting in an anxious seeking for further regulation from others. Both are strategies related to unmet attachment needs and ultimately contribute to further increase in allostatic load. Thus, this dimension could be understood from an attachment perspective (Fonagy, Gergely, & Jurist, 2004), which highlights the ongoing importance of close relationships in regulating affect throughout the lifespan.

One current finding from a psychodynamic perspective on the relevance of personality processes for the persistence of CSS disorders comes from Luyten et al.'s (2011) ecologically valid daily experience sampling of chronic fatigue syndrome (CFS) patients. In this work, self-critical perfectionism was shown to impact experiences of depression through increasing stress sensitivity. The authors also demonstrate that high levels of self-critical perfectionism can lead to the generation of more person-dependent daily hassles, for example, "A conflict with your partner" (Luyten et al., 2011). Self-critical perfectionistic patients often deal with these daily conflicts through a vicious, maladaptive cycle of withdrawal/isolation, depression, and suppression of distress (Luyten, Blatt, & Corveleyn, 2005; Luyten et al., 2013). It appears that self-critical perfectionism may be implicated in the development of chronic fatigue syndrome, and possibly CSS, through long-term down-regulation of the stress system (Van Houdenhove, Eede, & Luyten, 2009). In addition, self-critical or maladaptive perfectionism has been indicated as a contributing personality dimension that can worsen symptoms of CSS such as pain and fatigue (Kempke et al., 2013; Luyten et al., 2011).

COMORBIDITY

Among treatment-seeking individuals with a drug use disorder, 60% have at least one comorbid mood disorder and 43% at least one comorbid anxiety disorder (Grant, Stinson, Dawson et al., 2004). Upwards of 80% of women with an SUD report lifetime histories of physical or sexual assault, and among SUD treatment seekers, posttraumatic stress disorder rates range from 25–55% (Coffey, Dansky, Falsetti, Saladin, & Brady, 1998; Dansky, Saladin, Brady, Kilpatrick, & Resnick, 1995; Hien, Cohen, Miele, Litt, & Capstick, 2004). In addition, the rates of personality disorder pathology among substance abusers are roughly four times higher than the general population (Verheul, 2001). In this study, we are testing for personality differences in CSS patients within a sample of substance dependent patients. Given the extraordinarily high rates of comorbidity (Grant et al., 2004), lifetime stressors (Fetzner, McMillan, Sareen, & Asmundson, 2011), and general interpersonal difficulties known to the SUD population (Trull, Jahng, Tomko, Wood, & Sher, 2010), comparing these

two groups can help demonstrate the unique effects of CSS among a sample with otherwise high levels of psychiatric distress.

PRESENT STUDY

Given these theoretical considerations and previous research, we hypothesized a constellation of personality traits that would distinguish, in our sample of patients with SUDs, those with comorbid CSS disorders from all others. After controlling for somatic symptoms, we hypothesized that anxiety, need for interpersonal closeness, symptoms of traumatic distress, and perfectionistic traits would predict the presence of a CSS condition.

In addition to testing the above hypotheses we sought to conduct an exploratory analysis on the MMPI-II scales that most differentiated patients with a CSS from all others. Further, we describe personality trait differences on the MMPI-II clinical scales among those with CSS conditions, non-CSS chronic pain conditions, and those without a chronic pain disorder presenting to treatment for SUD.

METHOD

Procedures

Records were evaluated from a residential substance abuse and dependence treatment program in the southeastern United States. Archival medical records data (2007–2009) were collected and analyzed for this current study. Upon entering treatment, patients undergo a series of medical and psychological assessments, including full medical intakes by a licensed M.D., urinary drug screening, semistructured interviews by mental health counselors, and a battery of psychological assessments. These documents are reviewed collaboratively in an interdisciplinary treatment team setting, where diagnoses are made based on *DSM-IV-TR* (American Psychiatric Association, 2000) criteria. All patients accepted to treatment have a primary diagnosis of an SUD. This process is overseen by a licensed clinical psychologist, medical director, and psychiatrist.

Classification of CSS and Chronic Pain Conditions—To determine patients' chronic pain status and the potential of a CSS diagnosis, we reviewed medical intake records and diagnosis at discharge. Upon intake, patients are asked if they experience chronic pain and are evaluated for current and pre-existing health complaints by a medical professional, and given an ICD-9-CM (World Health Organization & Practice Management Information, 2012) medical diagnosis. Patients' records were evaluated and classified into one of three categories based on their medical diagnoses and self-reported pain: CSS, non-CSS chronic pain, and no chronic pain. Following Yunus's criteria for CSS (Yunus, 2007b), those with a CSS diagnosis given by a medical provider were classified as having a CSS. Classification was primarily based on medical diagnoses. In rare instances ($N = 16$, 4.6% of cases), when it was unclear as to whether or not a medical diagnosis was "historical" or indicative of an active chronic pain condition, we used self-reported chronic pain to a medical professional as a confirmatory factor.

Drug of Choice Classification—In order to assess patients' substance misuse, we evaluated their drug of choice (DOC). To do this, multiple points of information pertaining to substance use were separated and independently analyzed by two raters. The details of this process are outlined in McKernan et al. (2015), including the stepwise decision-making model guiding this process. In brief, information was gathered from self-reported drug use, current use patterns, past history, treatment history, urinalysis on intake, discharge diagnosis, and substance use scales from structured psychological assessments. To determine DOC, two independent raters compared self-reported information to medical records data, utilizing additional information to corroborate self-reported statements and/or determine a DOC when inconsistencies arose. Using Cohen's Kappa, inter-rater agreement was 0.91, $p < .001$.

Using this algorithm, individuals were categorized into one of six groups: (1) Depressants (alcohol, benzodiazepines, barbiturates), (2) Opiates (narcotics, analgesics), (3) Stimulants (cocaine, amphetamines), (4) Cannabis, (5) Polysubstance Dependence, and (6) Indeterminate (insufficient information). We used this classification system to assess whether group differences exist based on drug of choice, which has evidence of impacting personality characteristics and affective functioning (Khantzian, 1997; McKernan et al., 2015; Suh, Ruffins, Robins, Albenese, & Khantzian, 2008).

Participants

There were 341 total patients in the sample. Of this total, 30 (8.79%) were categorized as having a CSS condition. There were 79 patients (23.17%) diagnosed with a non-CSS chronic pain condition and 232 patients (68.04%) without any comorbid chronic pain condition. On average, patients were 36.96 years old ($SD = 12.41$) and 66.57% ($N = 227$) of the sample was male.

Of the CSS patients, 17 (56.67%) were diagnosed with a migraine condition, 5 (16.67%) were diagnosed with fibromyalgia, 2 (6.67%) were diagnosed with restless leg syndrome, 2 (6.67%) were diagnosed with chronic fatigue syndrome, 2 (6.67%) were diagnosed with irritable bowel syndrome, 1 (3.33%) was diagnosed with myofascial pain syndrome, and 1 (3.33%) was diagnosed with dysmenorrhea.

Measures

Demographics—Patients were asked standard demographics questions when entering the treatment facility, including age, gender, race/ethnicity, employment status, and marital status.

Medical Records Data—During intake procedures, all patients entering treatment underwent a history and physical exam (H&P) with a medical provider. During this assessment patients indicated their current symptoms, including pain levels (both acute and chronic), and their medical history was reviewed. We utilized pain-related diagnoses resulting from these assessments and interdisciplinary treatment team *DSM-IV-TR* diagnoses (Axis III) to classify patients into CSS, non-CSS chronic pain, and non-pain groups. This information was reviewed and discussed extensively by the primary (LCM) and secondary (MF) author. Any diagnosis that was unclear was excluded from analysis.

Minnesota Multiphasic Personality Inventory–2nd Edition (MMPI-2)—The MMPI-2 is a 567-item self-administered questionnaire in true/false format that assesses the existence of various forms of psychopathology and personality (Butcher, Graham, Tellegen, & Kaemmer, 1989). The MMPI-2 is frequently used to assess psychopathology in clinical and research settings because of its high reliability and validity (Butcher & Williams, 2000).

Several scales from the MMPI-2 were used to operationalize traits found in our hypotheses. *Anxiety* was operationalized by the Anxiety supplementary scale (A), *traumatic distress* was operationalized by the Posttraumatic Stress Disorder–Keane (PK) scale, and *need for interpersonal closeness* was operationalized by the Need For Affection subscale of the Hysteria clinical scale (Harris-Lingos Hysteria Subscale 2). In addition, *somatic symptoms* was operationalized by the Somatic Complaints subscale of the Hysteria clinical scale (Harris-Lingos Hysteria Subscale 4).

Unrelenting Standards Schema of the Young Schema Questionnaire–3rd Edition (YSQ-L3)—The YSQ-L3 is a 232-item self-administered questionnaire that assesses for the presence of early maladaptive schemas (Young & Brown, 2003; Young, Klosko, & Weishaar, 2003). The Unrelenting Standards schema was selected out of the YSQ-L3 in order to operationalize *perfectionism* for our study. We do not consider the many other YSQ-L3 scales at any stage of this report. The items are answered on a 6-point scale (1 = completely untrue of me, 6 = describes me perfectly), where respondents answer in accordance with their view of themselves. Items rated “4” or higher contribute to an individual’s’ overall schema score, indicating that a particular schema may be relevant to the respondent. An example item of this schema is as follows: “When I make a mistake, I deserve strong criticism.” We contend that someone rating this and the other items of the schema highly would be indicating a high degree of self-critical perfectionism. Evidence supports the factor structure, reliability, and validity of this measure (Cockram, Drummond, & Lee, 2010; Saariaho, Saariaho, Karila, & Joukamaa, 2009).

RESULTS

Demographic and Treatment Characteristics

Before conducting our hypothesis testing, we tested a series of demographic and treatment characteristics for any differences that might distinguish the comorbid CSS patients from all other patients. Table 1 provides a description of these tests. We found no significant differences between these two groups across a number of demographic and clinical variables. The following demographic variables were described and tested for differences either by independent samples *t*-test of chi-square exact test: age, gender, occupational status, marital status, and number of children. Likewise, the following clinical variables were examined: number of Axis I diagnoses (using *DSM-IV-TR* nosology), presence of an Axis II diagnosis (using *DSM-IV-TR* nosology), and length of stay in the treatment program (in days).

We then investigated potential for any trends in drug of choice among individuals with comorbid CSS conditions. In a chi-square analysis, we found no differences in proportions of drug of choice membership between those with CSS and those without, $\chi^2(5) = 1.51$, p

= .92. The proportions of members to each of the six drug of choice categories are remarkably similar for between patients with CSS and those without. As shown in Table 1, the distribution of drug of choice categories was essentially mirrored among patients with and without a CSS condition.

Hypothesis Testing: Distinguishing Differences of CSS Patients

In order to examine the primary hypotheses, we conducted a logistic regression predicting the presence of a CSS condition across two blocks of predictors. The first block served as control variables for the primary hypotheses. For this block, we added the MMPI-2-derived measure of somatic complaints (Harris-Lingos Hysteria Subscale 4). As expected, somatic complaints independently predicted the presence of a CSS condition, OR [95% CI] = .97 [.942–1.00], Wald = 3.92, $p = .048$. While it is somewhat interesting that the presence of a CSS condition predicts more general somatic complaints, this measure serves primarily as a control variable in the hypothesized model as we are intending to assess personality and affect above and beyond variance explained by general somatic complaints.

For the second block, we added the four hypothesized variables: anxiety, need for interpersonal closeness, traumatic distress, and perfectionism. None of these variables independently predicted the presence of a CSS, ps > .05, nor did this block explain incrementally more variance as a whole, $p = .98$. Therefore, our hypotheses were not supported. Our data seem valid for logistic regression analysis as the presence of CSS is not sufficiently rare to require a more specialized methodology (see King & Zeng, 2001, on this topic).

Exploring Personality Differences

A Search for the Strongest MMPI-2 Predictors of a CSS Diagnosis—Given that we were unable to establish evidence in support of a constellation of personality traits distinguishing those with CSS, we explored our personality data for the strongest predictors of a comorbid CSS condition in a stepwise logistic regression. Entry and removal of predictors in each iteration of the model were determined by level of significance (p value). At each step, the following algorithm was employed. P values from predictors below 0.05 were entered into the model and any predictors already in the model with subsequent p values above 0.10 were removed.

This model went through two steps, with one predictor entered at each step. In the second step, both predictors were retained in the model. The first predictor of CSS diagnoses entered into the model was the Fake Bad Scale (FBS), where those with a CSS diagnosis scored *lower* on FBS, OR [95% CI] = .97 [.946–.996], Wald = 5.25, $p = .022$. This suggests that patients with comorbid CSS conditions may be more forthcoming and genuine regarding the problems they are encountering (versus malingering) than other patients with SUDs.

In the second step, the Antisocial Practices (ASP) content scale was entered into the model with no removal of the FBS predictor, OR [95% CI] = 1.04 [1.00–1.08], Wald = 3.95, $p = .047$. Those who scored higher on the ASP scale had a slightly higher likelihood of having a

comorbid CSS. As a whole, the model was statistically significant, but explained a small amount of variance, $\chi^2(2) = 10.86$, $p = .004$, Nagelkerke $R^2 = .07$. This suggests that patients with a comorbid CSS may be more prone to everyday antisocial acts than other patients with SUDs.

The FBS independently predicted presence of a CSS (a lower FBS score being more likely to have a CSS diagnosis), but the ASP scale did not independently predict the presence of a CSS apart from the effect of the FBS, OR [95% CI] = 1.004 [.97–1.04], Wald = .06, $p = .81$. The predictive power of antisocial practices appeared dependent on patients being forthcoming in their responding.

Characteristics of Presence of CSS on MMPI-2 Clinical Scales

In order to explore the personality characteristics of individuals with CSS conditions more broadly, we compared the MMPI-II clinical scales across the CSS patients with all other patients.

First, a univariate ANOVA with all MMPI-2 clinical scales as dependent variables and CSS diagnosis as the only factor, we found no evidence for overall differences between having a comorbid CSS or not in overall responding across the MMPI-II clinical scales, $F(10, 330) = .93$, $p = .51$. There were no clinical scales that significantly differentiated individuals with CSS conditions, even without correcting for multiple comparisons.

Comparing Individuals with CSS, Non-CSS Chronic Pain, and All Others—

Following our initial analysis, we decided to break down the non-CSS sample into two different comparison groups, those with chronic pain and those without. We then compared the MMPI-II clinical scales across the CSS patients, chronic pain patients (not including CSS patients), and all remaining patients with neither condition. Grouping into these three categories, we were able to see more clearly any potential personality differences in the CSS patients beyond the experience of chronic pain alone.

For this stage, we first constructed a MANOVA with all MMPI-2 clinical scales as dependent variables and CSS diagnosis as the only factor, we found no evidence for overall differences between having a comorbid CSS or not in overall responding across the MMPI-II clinical scales, $F(20, 660) = 1.05$, $p = .40$. Exploring a series of univariate ANOVAs for each clinical scale, we found that overall group differences were greatest for Depression scale, $F(2, 338) = 2.79$, $p = .06$.

Pairwise comparisons on this scale revealed some significant differences of the CSS patient group from the others. On average, CSS patients had lower Depression scale scores ($M = 58.42$, $SD = 12.46$) than both the chronic pain patients ($M = 65.14$, $SD = 13.67$) and the non-pain reference group ($M = 64.84$, $SD = 15.22$), ps $< .05$. Table 2 provides a companion table to this figure, describing average T-scores of the MMPI-2 clinical scales by group along with the results of univariate ANOVA tests. These findings were somewhat counterintuitive and we attempt to understand them in the context of all of our findings in the section to follow.

DISCUSSION

We sought to examine the differences in personality and emotional functioning among individuals with centrally sensitive chronic pain conditions in a residential treatment setting for SUDs. To our knowledge, this is the first study to examine the characteristics of CSS in the SUD population. As this is a new area of study, we drew from previous research and a psychodynamic framework to inform a conceptualization of individuals with CSS via a constellation of personality traits.

From this model, we hypothesized that predictors of the presence of a CSS in this sample would be higher rates of overall anxiety, traumatic stress, perfectionistic traits, and a need for interpersonal closeness. In order to most effectively assess this model, we controlled for age, gender, and the self-reported severity of somatic symptoms. While somatic complaints (MMPI-II Harris Lingoes scale Hy4) independently predicted the presence of a CSS, none of the hypothesized personality differences independently predicted the presence of a CSS over and beyond somatic complaints.

We then sought to explore which MMPI-II scales may differentiate the CSS group from others. We employed a stepwise logistic regression algorithm predicting the presence of a CSS using MMPI-II clinical and content scales in order to determine the strongest predictors of CSS in our sample. We found that a model including the Fake Bad Scale (FBS) and the Antisocial Practices (ASP) content scales best predicted a CSS. FBS negatively predicted a CSS, while ASP positively predicted it. That is, those who had a comorbid CSS were more forthcoming about themselves (versus malingering) and were also more likely to endorse engaging in everyday aberrant behaviors.

We also explored which MMPI-II clinical scales might differentiate groups. We began with a comparison between CSS patients and all others, which yielded no differences. Following this, we sought to explore further by breaking up the non-CSS comparison group into those with chronic pain (but no CSS) and all others without either. In this analysis, patients with comorbid CSS exhibited significantly *lower* ratings on the Depression scale when compared to other groups (indicating less symptoms of depression). We were initially surprised by these findings, which at face value appear contradictory to what has been published about affectivity and CSS. Previous literature has found that distinctive traits in patients with CSS are negative affectivity and poor affect regulation (White, Nielson, Harth, Ostbye, & Speechley, 2002; Zautra et al., 2005). In addition, it is consistently reported through meta-analyses that individuals with a CSS have experienced much higher rates of trauma exposure than the general population (Afari et al., 2014; Paras et al., 2009). Furthermore, previous research using the MMPI-II to examine profiles of individuals with fibromyalgia and chronic headache found elevations in Hypochondriasis (Hs), Depression (D), Hysteria (Hy), and in some cases Schizophrenia (Sc) scales at levels that are significantly different from profiles of other health conditions (Ahles, Khan, Yunus, Spiegel, & Masi, 1991; Bigal et al., 2003; Johnson et al., 2010; Paolucci et al., 2015). Accordingly, the “contemporary norms” for fibromyalgia syndrome established by Yunus (Yunus, Ahles, Aldag, & Masi, 1991) also indicate scale elevations on Hs (67.12 +/− 8.8) and Hy (67.7 +/− 9.6) that were significantly higher than those reported in our sample.

Noting the high proportion of tension-type headache and migraine patients in our CSS sample (17 of 30), we examined existing literature to assess whether there are characteristics specific to this group which could provide insight into our findings. Large population-based studies conclude that those with tension and migraine headaches are more depressed, anxious, and phobic—with psychiatric comorbidities increasing with chronicity and substance use (Antonaci et al., 2011; Jette, Patten, Williams, Becker, & Wiebe, 2008). Furthermore, perfectionism and neuroticism are consistently cited as personality traits associated with these conditions (Antonaci et al., 2011). Although there is no relationship between migraine and increased risk for substance dependence, major depressive disorder is twice as more common among migraine sufferers who abuse opioids (Hung, Liu, Cheng, & Wang, 2009; Jette et al., 2008). Therefore, literature regarding comorbidity supports our initial hypotheses, making these findings regarding lower rates of depression even more interesting.

Why would a group of patients with central sensitivity pain conditions, in a residential substance use setting, who have more extensive treatment histories for substance use display “normal” or “non-pathological” personality profiles on the MMPI-II when compared to those without chronic pain? We pose this question with the warning that these relationships were discovered in an exploratory mode and need to be followed up with in further research. That said, we have subsequently discovered that there is some correspondence of these findings with previously published research.

Chronic Alienation from Affect

We turned to the descriptive psychopathology literature in order to help make sense of these exploratory findings for future research. There is some precedent to the connection between a tendency toward everyday social deviance (“psychopathic practices”) and somatization, which Lilienfeld and Hess (2001) state as “one of the most puzzling comorbidities in descriptive psychopathology” (p. 20). In a sample of healthy undergraduates, these authors provide evidence for the hypothesis that the link between proneness to antisocial behaviors (“secondary psychopathy”) and somatization is mediated by anxiety. Rather than a deficiency in behavioral inhibition, the stress brought on by social deviance or risk-taking behavior might add to the individual’s allostatic load which impacts their degree of somatization. This direct sequence of events that the authors propose may not apply in psychiatric populations, where an allostasis to chronic traumatic experiences is more likely among individuals.

Considering a sample of patients with comorbid CSS, however, we may find that the role of negative affectivity is less pronounced in mediating this relationship between somatic symptoms and antisocial practices. This might suggest a kind of extensive dissociation of autonomic nervous system function in this population, perhaps marking a significant change in the relationship between their own somatic experience and behavior. In this way, there may be paths toward and/or from the development of a CSS which lead to a diminished emotional awareness or alexithymia rather than anxiety. This emotional distancing may also lead to a decreased capacity to experience depression, for example, which is otherwise a

normal or even adaptive response to a difficult life situation. One might think of this like being “checked out” of your body—disconnected or even alienated in some chronic ways.

There may be multiple routes to such a state, which may share in common much with high rates of trauma. Since the connection with trauma and CSS has been shown (Afari et al., 2014), potentially these interesting findings may elucidate or speak to the impact of trauma and how that shows up in the pain condition. There are indications of the lasting effects of disruption in stress regulation systems in previous research examining personality and emotional factors associated with functional illnesses. Research denotes that individuals with CSS experience higher levels of negative affectivity and general psychological distress (Aaseth et al., 2011; Arnold et al., 2006; Johnson et al., 2010; White et al., 2002). Furthermore, individuals are found to be highly sensitive to anxiety, struggle to differentiate affect (Porcelli, Taylor, Bagby, & De Carne, 1999), and to cope with or regulate emotion (Lumley et al., 2011). Notably, CSS have been associated with high rates of alexithymia and catastrophizing (Drossman et al., 2000; Edwards, Bingham, Bathon, & Haythornthwaite, 2006; Porcelli et al., 1999). These emotional awareness deficits are linked to somatosensory amplification, where individuals experience a hyper-vigilance to bodily sensations, in turn increasing them (Edwards et al., 2006). Subsequently, these factors are often cited as not only precipitating, but perpetuating factors of CSS.

Interaction of Substance Use and Central Sensitization

A second possible explanation for these findings can be found amidst previous investigations of MMPI-II profiles of individuals with tension-type headache and fibromyalgia. It may be that our sample of individuals with CSS fall into a particular cluster of patients within the chronic pain population, and this depends on the intensity and chronicity of pain as opposed to having a CSS *per se*. Conducting a cluster analyses of the MMPI in fibromyalgia and rheumatoid arthritis patients, Ahles and colleagues (1991) found three distinct subgroups or “profiles” to classify respondents into: one considered a “normal” profile (all T scores < 70), another the “chronic pain profile” (T > 70 on Hs, Hy, and/or D only), and lastly a “psychological disturbance profile” (T > 70 on Hs, D, Hy, Psychasthenia (Pt), Sc or 4+ scale elevations). These profile characteristics were replicated by Rappaport (Rappaport, McAnulty, Waggoner, & Brantley, 1987) in a sample of chronic headache sufferers, who also found a fourth “sub-clinical” cluster with elevated response patterns on Hs, Hy, and D that were not clinically significant. The authors concluded that diagnostic category was unrelated to cluster membership—instead chronicity and intensity of pain was most influential.

The extent to which an SUD, of which all of the individuals in our sample were diagnosed, modifies the degree to which these traits are manifest remains an open question. Our sample of CSS patients would, on average, fall within the “normal” profile of Ahles et al. (1991) and Rappaport, McAnulty, Waggoner, and Brantley (1987). One wonders how, according to a self-medication hypothesis (Khantzian, 1997; McKernan et al., 2015), the substance use might mute or silence general affectivity in these patients.

Limitations

There are limitations within this study that may have impacted our results and conclusions. First, this study involved a retrospective review of medical records. Although our classification of chronic pain conditions was based on a diagnosis given by a medical provider, having the opportunity to interview patients directly would have confirmed diagnoses and provided more information regarding pain severity and the impact of symptoms on daily life. It is possible that some diagnoses were missed or not made by the medical provider. In terms of emotionality, we were also unable to assess patients after a long period of abstinence or observe pre-addiction personality/emotional functioning. Therefore, it is possible that extended substance use or withdrawal influenced patient responses to assessments. On site, individuals do not complete assessments if identified as experiencing withdrawal symptoms until adequately stabilized, helping to temper this possibility; however, it remains a study limitation. In addition, we were unable to assess the temporal association of mood and substance use. Although longitudinal studies denote that depression tends to arise after the development of chronic pain (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997), having this information (particularly age of first use) could have provided further insight into the interplay of emotionality and sensitization in this sample. Last, limitations include the homogeneity of the sample, therefore, conducting a similar study among a more diverse sample would elucidate the greater potential for generalizability of findings.

Future Research

Further research into the interrelationship of somatic complaints, emotional alienation, and antisocial practices might prove a useful way forward toward understanding how personality and affect are operating among these patients. While our initial hypotheses were not supported, it remains possible that there may be an issue of awareness of affect which comorbid CSS and SUDs may seriously limit. Investigating the role of alexithymia or poor emotion regulation and level of trauma and how that interacts with our findings would be a potential next step for understanding just how such pervasive disorders can disrupt personality and emotionality. Given the exploratory nature of our positive results, it still remains that there may not be a relationship between any personality variables and the presence of CSS. Research on this topic is rather new and should continue to progress with some measure of doubt.

Additionally, given what is known about allostatic load and the neurobiology of how trauma changes the nervous system (e.g., how complex trauma develops), future researchers could use brain imaging to try and understand the presence/impact of trauma and brain atrophy/dysfunction and its connection with these CSS disorders and SUD. Putting these findings and research in conversation with advances in our understanding of pathologies of embodiment (Ataria, 2016) might help to sort through thorny theoretical and methodological issues around the prolonged altered experience of the body and emotion.

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Table 1

Demographic and Clinical Characteristics of Substance Use Disorder Patients With and Without a Comordbid CSS

	Without Comorbid CSS (*N* = 311)	With Comorbid CSS (*N* = 30)				
Variable	**Mean (*SD*) /Count (%)**	**Mean (*SD*) /Count (%)**	***t* /χ^2**	***df***	***p* (two-sided)**	**Missing cases**
Demographic						
Age	37.13 (12.48)	35.23 (11.71)	0.80	339	0.43	0
Gender			0.17		0.84	0
Male	206 (66.24%)	21 (70%)				
Female	105 (33.76%)	9 (30%)				
Occupational Status			0.02	1	0.88	35
Employed	112 (40%)	10 (38.46%)				
Unemployed	168 (60%)	16 (61.54%)				
Marital Status			0.82	2	0.66	16
Single	100 (33.78%)	12 (41.38%)				
Married	117 (39.53%)	11 (37.93%)				
Divorced/Separated/Widowed	79 (26.68%)	6 (20.69%)				
Number of Children	1.21 (1.16)	.93 (1.08)	1.26	336	0.21	3
Clinical						
Number of Axis I Diagnoses (DSM-IV TR)	2.45 (1.46)	2.26 (1.26)	0.70	328	0.49	11
Presence of Axis II Diagnosis (DSM-IV TR)		0.173	1	0.68	2	
Yes	39 (12.62%)	3 (10%)				
No	270 (87.38%)	27 (90%)				
Length of Stay (Days)	56.31 (45.33)	58.07 (67.82)	−0.192	336	0.85	3
Drug of Choice						
Depressant	157 (50.5%)	17 (56.7%)	1.51	5	0.92	0
Opiate	94 (30.2%)	8 (26.7%)				
Stimulant	29 (9.3%)	3 (10%)				
Cannabis	19 (6.1%)	2 (6.7%)				
Polysubstance	9 (2.9%)	0 (0%)				
Indeterminate	3 (1.0%)	0 (0%)				

Note. The percentage given next to counts of the categorical variables is the percentage of available cases from that CSS category (i.e., not including missing cases in the total number).

Table 2

Average MMPI-2 Clinical Scales by Group

MMPI-2 Scale	Without Comorbid Pain Condition (*N* = 232)	With Comorbid Chronic Pain (non-CSS; *N* = 78)	With Comorbid CSS (*N* = 31)		
	Mean (*SD*)	Mean (*SD*)	Mean (*SD*)	*F*	*p* (two-sided)
Hypochondriasis (Hs)	60.07 (13.67)	60.21 (14.03)	56.32 (13.55)	1.07	0.34
Depression (D)	64.84 (15.22)	65.14 (13.67)	58.42 (12.46)*	2.79	0.06
Hysteria (Hy)	60.72 (15.36)	59.85 (12.17)	56.84 (14.45)	1.00	0.37
Psychopathic Deviate (Pd)	66.61 (11.84)	65.40 (11.46)	64.10 (11.75)	0.81	0.45
Masculinity/Femininity (Mf)	47.92 (9.93)	50.21 (10.27)	50.71 (14.52)	2.00	0.14
Paranoia (Pa)	61.48 (14.49)	61.95 (14.24)	56.58 (13.49)	1.75	0.18
Psychasthenia (Pt)	64.47 (15.46)	63.28 (13.63)	59.87 (12.63)	1.37	0.26
Schizophrenia (Sc)	62.06 (15.96)	61.08 (15.60)	58.23 (14.38)	0.84	0.43
Hypomania (Ma)	56.18 (12.85)	54.10 (11.42)	57.71 (16.32)	1.12	0.33
Social Introversion (Si)	52.50 (11.16)	53.94 (11.52)	50.06 (12.06)	1.33	0.27

Note.

*Indicates a pairwise *p*-value from other groups of less than .05. There was only one such difference. Degrees of freedom for each analysis were constant: between groups *df* = 2; within groups *df* = 338.