Variables Associated with Current Smoking Status in Chronic Pain Patients

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ABSTRACT

Objectives. Smokers may report more pain and may be at greater risk for psychiatric comorbidity. Smoking may be a major problem in chronic pain patients (CPPs). The goal of this study was to determine if pain and psychiatric comorbidity are associated with smoking status in CPPs.

Design. As part of a return-to-work grant study CPPs who could potentially return to work identified themselves as either current smokers (N = 81) or nonsmokers (N = 140). These two groups were compared on a large number of demographic, function, pain, disability, behavior, and psychiatric diagnoses variables gathered at admission into the grant study. The incidence of smoking was tested with either the student's t-test or chi-square to detect differences in continuous and categorical variables, respectively. Logistic regression was utilized to determine the predictive variables for smoking status by inputting significant independent variables (P < 0.01) from the prior analyses.

Setting. Pain facility.

Results. Five variables were found to explain 38.8% of the variance for smoking status. These were education; race (Caucasian); cups of coffee per day; a diagnosis of current alcohol abuse/dependence; and personality disorder.

Conclusions. Smoking status in CPPs is associated with some variables that are similar for smoking in the general and psychiatric populations (education, race, alcoholism). However, a number of variables expected to be relevant (e.g., mood disorders) were not associated with smoking status in CPPs. These results may not be generalizable to all CPPs as they are derived from CPPs who are return-to-work candidates.

Key Words. Smoking; Chronic Pain; Affective Disorders; Drug Use Disorders; Alcohol Use Disorders

Introduction

The Tobacco Supplement to the National Comorbidity Survey has determined that the lifetime prevalence for nicotine dependence in the United States is 24% [1]. Within chronic pain patients (CPPs), the prevalence of smoking may be higher. For example, Jamison et al. [2] have reported that 54% of their low back pain (LBP) patients smoked. This figure is important, not only because it represents a prevalence above that of the national survey or because it indicates that a large percentage of CPPs may have an addictive disorder (nicotine), but most important because smoking/nicotine may be associated with pain [3]. A large number of studies have addressed the issue of whether smoking is a risk factor for chronic LBP.
These studies have been reviewed by a number of researchers [4–7]. In the first review, Heliövaara reviewed 50 cross-sectional retrospective studies and concluded that smoking is associated with increased risk for LBP, but its causal role was questionable. Six years later this literature was again reviewed by Leboeuf-Yde [5]. She found consistent evidence against a causal association between smoking and sciatica/discal herniation. Four years later Leboeuf-Yde again reexamined this literature [6,8]. This time, she found some signs of causality consistently evident in large studies and concluded that smoking should be considered a weak risk factor for LBP. In the final recent review, Goldberg et al. [7] concluded that the data were consistent in indicating that smoking is associated with the incidence and prevalence of nonspecific LBP. This last conclusion is strengthened by a very sophisticated study. Here, Battie et al. [9] measured disk degeneration by magnetic resonance imaging (MRI) in twins highly discordant for smoking. They found that smoking twins had an 18% greater mean disk degeneration score across the entire lumbar spine. It is thought that the mechanism of premature disk degeneration in smoking relates to the action of nicotine [10]. This may occur by nicotine increasing serum proteolytic activity by releasing proteolytic enzymes from neutrophils [10]. This high serum proteolytic activity may speed up the disk degenerative process [10].

If smoking is associated with LBP, then one might expect smokers with LBP to have greater pain than nonsmokers with LBP. Some evidence in the literature indicates that this may be the case. Smokers appear to report more pain in general [11,12]. This relationship also appears to hold in case control studies where spinal patients are compared [13]. However, only one study [14] has specifically compared smokers and nonsmokers for pain severity. Here, smoking did not predict greater pain intensity. An association between smoking, mood disorders [15,16], dysthymia [17], and major depression [18] has been reported in the psychiatric literature. This relationship may arise from genetic factors that predispose both to smoking and major depression [19]. As affective disorders are common in CPPs [20], one could expect a potential association here between affective disorders and smoking status. Anxiety syndromes have also been reported in the psychiatric literature to be associated with smoking [18,21,22]. Risk for agoraphobia, generalized anxiety disorder, and panic disorder is increased in smokers [22]. These associations have not been explored in smoking CPPs.

Smoking may also be associated with drug use disorders. Nicotine dependence has been reported in the psychiatric literature to be associated with alcohol, cannabis, and cocaine dependence [18,23,24]. CPPs have also been noted to rely more on medications [2], such as narcotics [25,26]. However, these associations have also not been explored in CPPs. Finally, smoking may be associated with complaints of fatigue, increased coffee consumption, and insomnia [23]. These potential associations have also not been investigated in smoking CPPs. The goals of the present study were then the following: 1) to investigate if smoking CPPs have more pain than nonsmoking CPPs; 2) to determine if smoking CPPs have more psychiatric comorbidity vs nonsmoking CPPs, such as affective disorders; 3) to determine if smoking CPPs have more substance use disorder comorbidity; and 4) to determine if smoking CPPs are more prone to fatigue, coffee consumption, and insomnia vs nonsmoking CPPs. This study is described below.

Methods

Between March 1991 and March 1993, over 1000 consecutive CPP admissions to the University of Miami Comprehensive Pain Center were screened for possible selection for a National Institute on Disability and Rehabilitation Research Grant Study. Because this grant study dealt with prediction of return to employment postpain facility treatment, each selected CPP received a detailed assessment and follow-up post evaluation and/or post treatment. CPP inclusion criteria for the grant study were the following: 1) candidates for employment post treatment; 2) age range 19–62 years; 3) LBP as a presenting problem of greater than 6-month duration; 4) able to read English; 5) not retired; 6) not a housewife by profession; 7) not a student by profession; and 8) consenting to sign the informed consent to participate in the grant study. Exclusion criteria were the following: 1) noncandidate for employment post treatment (student by profession, housewife by profession, retired receiving social security, retired not receiving social security, or accepted for and/or receiving social security); 2) age less than 19 or over 62 years; 3) other forms of chronic pain other than LBP as the chief complaints, e.g., neck pain; 4) not able to read English; 5) any form of retirement; 6) requiring surgery for LBP at the time of...
admission to the Pain Center; and 7) refusal to sign the informed consent to enter the grant study. Those CPPs entering the grant study were followed up after evaluation and 1-month treatment at 1, 2, 3, 12, 18, 24, and 30 months.

After signing the consent form, the CPP completed a series of baseline demographic questionnaires and psychological inventories. These included the following: basic demographic information; fatigue question; pain score; visual analog scales (VASs) (various); pain disability questionnaire; sickness impact profile; functional assessment total score; Beck Depression Inventory; and the State-Trait Anxiety Inventory. In addition, each CPP received a psychiatric diagnostic interview by a senior psychiatrist utilizing Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (DSM-III-R) flow sheets and criteria. Only current DSM-III-R diagnoses were utilized in the analyses. In addition, for the purposes of the analyses abuse and dependence diagnoses for similar drugs, e.g., alcohol, cannabis, etc., were collapsed into one category, such as current alcohol abuse/dependence, current cannabinoid abuse/dependence, etc.

**Data Analysis**

Data were analyzed using the Statistical Package for the Social Sciences software. Frequency and descriptive statistics were calculated to check all relevant characteristics of the data. Patients classified themselves as either smokers (N = 81) or not (N = 140) according to the frequency breakdown of the number of packs of cigarettes smoked per day (Table 1). Smokers/nonsmokers classified themselves by answering a grant smoking question where smoking quantities were delineated as in Table 1. The classification (smoker/nonsmoker) was used as the dependent variable for analyses to determine differences on the instrument scores and scales and other variables of interest. The incidence of smoking was tested with either the Student’s t-test or chi-square to detect differences in continuous and categorical variables, respectively. Logistic regression was used to determine the predictors of whether or not a patient was a smoker by inputting significant independent variables (P < 0.01) from prior analyses. The alpha level used for all analyses was α = 0.01 in order to control for type I errors (chance) and spurious findings given the large number of variables that we tested.

Two additional logistic regression analyses were performed to determine if the amount of smoking was related to a different set of predictors. First, nonsmokers were compared with those patients who smoked up to one pack of cigarettes per day (N = 47). Next, nonsmokers were compared with those who smoked more than one pack of cigarettes per day (N = 34). Finally, the smoking variable was utilized as a semicontinuous dependent variable with prior significant variables in a linear regression. A power calculation was also performed and correlations were calculated between Beck Depression Inventory Scores and individual DSM Depression Diagnoses (Major Depression, Dysthymia, Adjustment Disorder with Depressed Mood).

**Results**

This sample consisted of 58% (N = 128) male and 42% (N = 93) female with a mean age of 41.1 (SD = 10.0, range = 19, 62). The ethnic distribution was 164 (74.2%) white, non-Hispanic; 18 (8.1%) black, non-Hispanic; 19 (8.6%) Hispanic; and 20 (9.1%) of unknown racial origin. The frequency distribution for number of packs of cigarettes smoked for the CPP sample is presented in Table 1. Approximately 37% of the CPPs entering the grant study classified themselves as smokers, while 15.4% were heavy smokers (greater than one pack per day).

**Table 1** Frequency distribution for the number of packs of cigarettes smoked per day for the CPP sample (N = 221)

<table>
<thead>
<tr>
<th>Category</th>
<th>Frequency</th>
<th>Percent</th>
<th>Valid Percent</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>None (N)</td>
<td>140</td>
<td>63.3</td>
<td>63.3</td>
<td>63.3</td>
</tr>
<tr>
<td>Less than 0.25 pack</td>
<td>9</td>
<td>4.1</td>
<td>4.1</td>
<td>67.4</td>
</tr>
<tr>
<td>0.25 to 0.5 pack</td>
<td>9</td>
<td>4.1</td>
<td>4.1</td>
<td>71.5</td>
</tr>
<tr>
<td>0.5 to 0.75 pack</td>
<td>11</td>
<td>5.0</td>
<td>5.0</td>
<td>76.5</td>
</tr>
<tr>
<td>0.75 to 1 pack</td>
<td>18</td>
<td>8.1</td>
<td>8.1</td>
<td>84.6</td>
</tr>
<tr>
<td>1 to 1.5 packs</td>
<td>25</td>
<td>11.3</td>
<td>11.3</td>
<td>95.9</td>
</tr>
<tr>
<td>1.5 to 2 packs</td>
<td>6</td>
<td>2.7</td>
<td>2.7</td>
<td>98.6</td>
</tr>
<tr>
<td>Greater than 2 packs</td>
<td>3</td>
<td>1.4</td>
<td>1.4</td>
<td>100.0</td>
</tr>
<tr>
<td>Total</td>
<td>221</td>
<td>100.0</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

CPP = chronic pain patient.
Comparisons Between Smokers and Nonsmokers for Demographic Variables at Pretreatment (Table 2)

Results are displayed as percentages within each category. No significant differences were found between smokers and nonsmokers for age, gender, marital status, fatigue status, Beck Depression Score, Functional Assessment Total Score, Sickness Impact Profile score, VAS average pain over the last 24 hours, VAS pain level considered intolerable, VAS pain level for which to take medications, VAS pain level considered disabling, Pain Disability Index Score, State Anxiety Total Score, and Trait Anxiety Total Score. The following DSM diagnoses were also nonsignificantly different between smokers and nonsmokers: Current Opioid Abuse/Dependence, Current Inhalant Abuse/Dependence, Current Psychoactive Abuse/Dependence, Current Cocaine Abuse/Dependence, Current Sedative Abuse/Dependence, Current Major Depressive Disorder, Dysthymic Disorder, Adjustment Disorder with Depressed Mood, Panic Disorder, Generalized Anxiety Disorder, Adjustment Disorder with Anxiety, and Conversion Disorder. Within each ethnic category, a higher percentage were nonsmokers compared with smokers ($\chi^2 = 13.3[3], P < 0.01$). For educational attainment, a higher proportion of smokers was found through the completion of high school, but a higher proportion of nonsmokers was found from the completion of vocational college through completion of a doctorate degree ($\chi^2 = 32.1[7], P = 0.001$). A higher percentage of smokers drank either none, up to three cups, or more than 10 cups of coffee per day, whereas a high rate of nonsmokers was found in the categories from three to nine daily cups of coffee ($\chi^2 = 22.3[6], P = 0.001$). Forty-eight percent of those subject involved in worker compensation cases were smokers ($\chi^2 = 18.1[1], P = 0.001$).

Our battery of study measures included assessments of psychopathology, pain, health, and functionality. While none of these measures was statistically different between smokers and nonsmokers (at the $P < 0.01$ level), several trends were found (see Table 3). Smokers tended to score higher on measures of depression, current level of
Variables Associated with Current Smoking Status

For current substance use diagnoses (Table 4), a higher proportion of those using alcohol were smokers (71%) compared with nonsmokers (29%; \( \chi^2 = 16.1, P < 0.001 \)). A greater percentage of those using cannabis were smokers (77%) compared with nonsmokers (23%; \( \chi^2 = 9.7, P < 0.01 \)). More of those using amphetamines were smokers (86%) compared with nonsmokers (14%; \( \chi^2 = 7.5, P < 0.01 \)). A trend was found for higher cocaine use among smokers (70%) compared with nonsmokers (30%; \( \chi^2 = 5.0, P < 0.03 \)). Differences for current opioid, sedative, inhalant, and psychoactive use were not significant between groups.

For psychiatric diagnoses (Table 4), a higher proportion of nonsmokers (77%) was diagnosed as obsessive compared with smokers (23%; \( \chi^2 = 15.4, P < 0.001 \)), but a higher percentage of...
smokers (62%) were histrionic relative to the non-smokers (38%; \( \chi^2 = 16.1[1], \ P < 0.001 \)).

**Logistic Regression Using Demographic and Clinical Characteristics as the Independent Variables and Smoking as the Dependent Variable (Table 5)**

A logistic regression was conducted with significant variables from prior analyses (Tables 2, 3, and 4), as the independents and smoking as the dependent variable. Subsequent sequential logistic regression analysis was performed to test the individual contribution of each predictor in the fit of the model with the omnibus step \( \chi^2 \) and Nagelkerke \( R^2 \). Table 5 includes the regression coefficient, step \( \chi^2 \) and significance, Nagelkerke \( R^2 \), Wald statistic, odds ratio, and 95% confidence intervals for the odds ratio. The overall chi-square for the analysis was significant (\( \chi^2 = 72.2[5], \ P < 0.001 \)), approximately 39% of the variance was explained by the model, and the model classified 75% of the subjects correctly. “Having less education,” “being Caucasian,” “drinking more daily cups of coffee,” “having a current alcohol use diagnosis,” and “being diagnosed as histrionic” were significant predictors of smoking. All five variables were highly significant according to the Wald test.

**Logistic Regression Using Demographic and Clinical Characteristics as the Independent Variables and Smoking (None vs Up to One Pack per Day) as the Dependent Variable (Table 6)**

The overall chi-square for the analysis was significant (\( \chi^2 = 43.3[3], \ P < 0.001 \)), approximately 31% of the variance was explained by the model, and the model classified 79% of the subjects correctly. “Having less education,” “having a current alcohol use diagnosis,” and “being diagnosed as histrionic” were significant predictors of smoking. All three variables were highly significant according to the Wald test.

**Logistic Regression Using Demographic and Clinical Characteristics as the Independent Variables and Smoking (Nonsmokers vs More Than One Pack per Day Smokers) as the Dependent Variable (Table 7)**

The overall chi-square for the analysis was significant (\( \chi^2 = 59.2[5], \ P < 0.001 \)), approximately 47% of the variance was explained by the model, and the model classified 87% of the subjects correctly. “Having less education,” “being involved in worker’s compensation,” “being white,” and “drinking coffee every day” were significant predictors of smoking. “Being married more often” was retained in the final equation, but was nonsignificant.

### Table 5

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>( \chi^2 ) (df), Sig.</th>
<th>( R^2 )</th>
<th>Wald, Sig.</th>
<th>Exp (B) (Odds Ratio)</th>
<th>95% CI for Exp (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>−0.523</td>
<td>32.9 (1), 0.001</td>
<td>0.193</td>
<td>21.3, 0.001</td>
<td>0.593</td>
<td>0.475 0.740</td>
</tr>
<tr>
<td>Race: Caucasian</td>
<td>1.268</td>
<td>17.2 (1), 0.001</td>
<td>0.283</td>
<td>8.7, 0.003</td>
<td>3.553</td>
<td>1.532 8.240</td>
</tr>
<tr>
<td>Cups of coffee per day</td>
<td>0.444</td>
<td>10.2 (1), 0.001</td>
<td>0.332</td>
<td>8.3, 0.004</td>
<td>1.559</td>
<td>1.152 2.109</td>
</tr>
<tr>
<td>Current alcohol abuse/dependence</td>
<td>1.021</td>
<td>7.7 (1), 0.005</td>
<td>0.369</td>
<td>4.0, 0.045</td>
<td>2.775</td>
<td>1.021 7.540</td>
</tr>
<tr>
<td>Histrionic</td>
<td>1.068</td>
<td>4.2 (1), 0.041</td>
<td>0.388</td>
<td>7.4, 0.007</td>
<td>2.910</td>
<td>1.345 0.740</td>
</tr>
</tbody>
</table>

Sig. = significance.

---

### Table 6

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>( \chi^2 ) (df), Sig.</th>
<th>( R^2 )</th>
<th>Wald, Sig.</th>
<th>Exp (B) (Odds Ratio)</th>
<th>95% CI for Exp (B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>−0.489</td>
<td>24.8 (1), &lt;0.001</td>
<td>0.189</td>
<td>13.4, &lt;0.001</td>
<td>0.613</td>
<td>0.472 0.796</td>
</tr>
<tr>
<td>Current alcohol abuse/dependence</td>
<td>1.394</td>
<td>11.5 (1), 0.001</td>
<td>0.268</td>
<td>6.8, &lt;0.01</td>
<td>4.031</td>
<td>1.410 11.52</td>
</tr>
<tr>
<td>Histrionic</td>
<td>1.477</td>
<td>6.9 (1), &lt;0.01</td>
<td>0.313</td>
<td>11.8, 0.001</td>
<td>4.382</td>
<td>1.887 10.18</td>
</tr>
</tbody>
</table>

Sig. = significance.
Variables Associated with Current Smoking Status

Linear Regression Using Demographic and Clinical Characteristics as the Independent Variables and Smoking (Nonsmokers vs More Than One Pack per Day Smokers) as the Dependent Variable (Table 8)

The F statistic for the linear regression was significant ($F(5,210) = 16.3, P < 0.001$) and the adjusted $R^2$, or variance explained in the model, was 26.2%. Smoking was predicted by “drinking coffee everyday,” “a lower level of education,” and “being white,” and trends were noted for “being diagnosed as histrionic” ($P < 0.05$), and using cannabis ($P < 0.05$).

Power Analysis for the Overall Study

At an alpha level of 0.01 with a sample size of 221, power was estimated to be 0.87 for the study. This level of power is considered adequate to detect our observed effects including a difference between smokers and nonsmokers for variables such as depression.

The correlation between the Beck Depression Inventory Scores and the various DSM depression diagnoses were as follows: for Major Depression $r = 0.44, P < 0.01$; for Dysthymia $r = 14, P < 0.05$, and for Adjustment Disorder with Depressed Mood $r = -0.03, P = 0.70$.

### Discussion

The hypotheses of this study were that CPPs who smoked should have more pain than CPPs who did not smoke and that the smokers would demonstrate affective psychiatric comorbidity, substance use disorder comorbidity, fatigue, coffee consumption, and insomnia. As pointed out in the Introduction, most of these hypotheses rested on previous literature.

In order to test these hypotheses, we performed three logistic regressions with variables that appeared to be highly associated with smoking (at a $P < 0.01$ level). Our first logistic regression (Table 5) for smokers and nonsmokers indicated that smoking in our CPPs was associated with five variables: education; race (Caucasian); cups of coffee per day; current alcohol consumption; and a diagnosis of histrionic personality disorder. The second logistic regression model (Table 6) comparing nonsmokers to those who smoked up to one pack/day essentially yielded similar variables: education, current alcohol consumption, and a diagnosis of histrionic personality disorder. The third logistic model (Table 7) using nonsmokers and those who smoked more than one pack per day yielded the following variables: cups of coffee per day, education, race (Caucasian), worker com-

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>Std. Error</th>
<th>$\chi^2$ (df), Sig.</th>
<th>$R^2$</th>
<th>Wald, Sig.</th>
<th>Exp (B) (Odds Ratio)</th>
<th>95% CI for Exp (B) Lower</th>
<th>95% CI for Exp (B) Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cups of coffee per day</td>
<td>0.496</td>
<td>0.201 (1), 0.001</td>
<td>0.176</td>
<td>7.0, &lt;0.01</td>
<td>1.642</td>
<td>1.138 2.371</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>0.419</td>
<td>0.117 (1), 0.001</td>
<td>0.269</td>
<td>7.0, &lt;0.01</td>
<td>0.658</td>
<td>0.482 0.897</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race: Caucasian</td>
<td>3.371</td>
<td>0.169 (1), 0.001</td>
<td>0.393</td>
<td>9.9, &lt;0.01</td>
<td>29.115 3.547 238.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worker's compensation</td>
<td>1.310</td>
<td>0.68 (1), 0.01</td>
<td>0.439</td>
<td>6.0, 0.014</td>
<td>3.708 1.299 10.6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of times married</td>
<td>0.564</td>
<td>0.40 (1), 0.046</td>
<td>0.466</td>
<td>3.8, 0.052</td>
<td>1.758 0.995 3.10</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sig. = significance.

### Table 7

Final model logistic regression results for smoking as the dependent variable with demographic and clinical characteristics as independent variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>Std. Error</th>
<th>$\chi^2$ (df), Sig.</th>
<th>$R^2$</th>
<th>Wald, Sig.</th>
<th>Exp (B) (Odds Ratio)</th>
<th>95% CI for Exp (B) Lower</th>
<th>95% CI for Exp (B) Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cups of coffee per day</td>
<td>0.419</td>
<td>0.107</td>
<td>0.236</td>
<td>3.922</td>
<td>0.000</td>
<td>0.209 0.630</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest level of schooling</td>
<td>-0.353</td>
<td>0.074</td>
<td>0.290</td>
<td>4.757</td>
<td>0.000</td>
<td>-0.499 -0.206</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race: White</td>
<td>1.068</td>
<td>0.294</td>
<td>0.221</td>
<td>3.631</td>
<td>0.000</td>
<td>0.488 1.648</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Histrionic</td>
<td>0.665</td>
<td>0.306</td>
<td>0.130</td>
<td>2.173</td>
<td>0.031</td>
<td>0.062 1.269</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current cannabis abuse/dependence</td>
<td>1.063</td>
<td>0.530</td>
<td>0.120</td>
<td>2.003</td>
<td>0.046</td>
<td>0.017 2.108</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Std. = standard; Sig. = significance.

### Table 8

Final model linear regression results for smoking status (number of packs smoked) as a continuous dependent variable with prior significant independent variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>Std. Error</th>
<th>Beta</th>
<th>Sig.</th>
<th>t</th>
<th>95% CI for B Lower</th>
<th>95% CI for B Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cups of coffee per day</td>
<td>0.419</td>
<td>0.107</td>
<td>0.236</td>
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<td>-0.499 -0.206</td>
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</tr>
<tr>
<td>Current cannabis abuse/dependence</td>
<td>1.063</td>
<td>0.530</td>
<td>0.120</td>
<td>2.003</td>
<td>0.046</td>
<td>0.017 2.108</td>
<td></td>
</tr>
</tbody>
</table>

Std. = standard; Sig. = significance.
penetration patient, and being married more often. The first model explained 39% of the variance, while the second and third models explained 31% and 47% of the variance, respectively. We also performed a linear regression analysis (Table 8) using the smoking variable as a continuous dependent variable with the prior significant independent variables. We were able to do these analyses as we had data on the actual numbers of packs of cigarettes smoked per day. Here again, the results were almost exactly the same as in the first logistic regression: smoking was predicted by drinking coffee daily, a lower level of education, being white, being diagnosed as histrionic, and using cannabis currently. The only difference was that alcohol was replaced by cannabis.

What can one observe from these results? First, it appears that a certain number of variables predict smoking status irrespective of number of cigarettes smoked. Second, smoking status at a high level (over pack/day) appears to be associated with slightly different variables (worker compensation status, married more often) and utilizing this group the model explained the largest percentage of the variance. Third, none of the derived models appeared to include all the variables hypothesized to be potentially predictive. Thus, this study's results supported only two of the above hypotheses (coffee and substance use disorders). These results will be discussed below.

The finding that mood disorder did not contribute to the final model (Table 5) was unexpected. This is because there is a high frequency of depression (major) in CPPs [20] and the psychiatric literature also indicates that there may be a shared genetic predisposition to both problems [17,18]. However, closer inspection of this literature revealed that the shared genetic predisposition finding for smoking depression may only relate to dysthymia, but not to major depression [17]. In addition, CPPs represent a potentially different genetic pool vs psychiatric patients or the general population in which these studies [15,18] were performed. There has only been one previous study [13] of smoking and mood disorders in CPPs (those with spinal disorders). Here a health status questionnaire was utilized to generate patients who had complaints of depression. These complaints were more frequent in smokers. Because neither a diagnostic method for depression nor a depression rating scale was utilized in this study, it is unclear what these results really mean. Finally, it is to be noted that our correlation results between the depression DSM diagnoses and Beck Depression Inventory Scores indicated that there was significant correlation between Major Depression and Dysthymia and Beck Depression Inventory Scores. Yet, the Beck Depression Inventory Scores were also not predictive of smoking status. Thus, our study then has two independent sources of data, which correlate with each other, but do not predict smoking status independently. This then also gives weight to our results indicating that perhaps smoking comorbidity for CPPs may differ from smoking comorbidity for psychiatric patient groups. Therefore, because of the above discussion, our results may not be in dramatic variance with the previous literature.

As noted in the Introduction, psychiatric studies indicate that smoking may be associated with anxiety syndromes. Our results did not reflect this finding. As noted for mood disorders, the discordance in the findings may reflect different population groups, as the psychiatric studies were performed on community samples.

In the general population, nicotine dependence has been found to be associated with illicit drug use (cannabinoids, cocaine) [18]. This finding has been further strengthened by a genetic study [24] of male twin pairs, which has demonstrated a shared genetic vulnerability to all forms of illicit drug use and alcohol, i.e., any form of addiction. Our results did demonstrate an association between illicit drugs (cannabis) and nicotine, but only in the linear regression analysis. This discordance in results could again be related to patient selection factors. There have been a number of psychiatric studies [18,23,27,28] demonstrating an association between alcohol abuse/dependence and nicotine dependence. Our result for alcohol and nicotine (Table 5) both support and are supported by these studies.

As noted in the Introduction, there may be an association between smoking and opioid use [29]. This association may also be present in female CPPs [25,26]. However, our results did not support these previous findings. This discordance may also relate to patient selection factors.

Coffee dependence may be another comorbidity associated with smoking [23]. It is interesting that in our group of CPPs, coffee drinking was shown to predict smoking, explaining 4.9% of the variance, which was greater than that of alcohol. Thus, our results support and are supported by this previous literature.

Recent studies in CPPs [30,31] have indicated that fatigue is a significant problem in CPPs. Fatigue has also been demonstrated in psychiatric...
patients to be associated with nicotine dependence [23]. As such, it was expected that an association between smoking and fatigue would be present in CPPs. No such association was found. This may be attributable to the fact that in the psychiatric study [23] the profile of mood states (POMS) was utilized to measure fatigue. The POMS is not a fatigue inventory. Our study utilized a five-point fatigue scale. To complicate this issue further, there has been one study in fibromyalgia patients [32], which has demonstrated no association between fatigue and smoking. As such, this issue requires further study.

As noted in the Introduction, there is some evidence that smokers may have greater pain [11–13]. Our study did not demonstrate this association. There is one study [14] in CPPs that supports our results. In addition, there is some evidence that nicotine may have analgesic properties in humans [33,34] and recently smokers have been demonstrated to have reduced pain perception vs nonsmokers [35]. As such, one could argue that smokers should then have less pain than nonsmokers. It appears then that this issue also requires further study.

An interesting finding in our study was that the presence of histrionic personality disorder predicted smoking. It is difficult to explain this finding, as there are no studies in the pain literature addressing personality disorders and smoking. However, it is well established in the psychiatric literature that personality disorders are associated with illicit drug use, alcohol use, and mood disorders, which in turn are associated with cigarette smoking [29]. This literature may then indirectly explain this last finding.

Finally, it is interesting to note that education (high school or less) and race (Caucasian) were the strongest predictors of smoking behavior in CPPs. This is not surprising, as similar findings are noted in the US general population [1]. Thus, our results are supported by this previous data.

There are a number of potential confounders to this study that will be discussed below. First, it is possible that some of the CPPs gave false information about their smoking status. This possibility has previously been reported in the literature by our group [36] in reference to other drugs, but not nicotine. There is a possibility that this previous finding translates to nicotine. The problem of false information could only have been controlled for by a biochemical analysis, which was not done. Thus, false information in reference to smoking status could have impacted on our findings. Second, as indicated in the methods and results, we did utilize a large number of variables in our analyses. However, we controlled for this by utilizing an alpha level of 0.01 instead of 0.05. This limited our type I error rate to a possible 1%, which is a very conservative method of not reporting a “chance” finding. Given how significant our findings were, it is unlikely that they were found by chance. Thus, we do not believe that this was a confounder in our results. Third, our data did not allow us to identify ex-smokers and/or ever smokers, but only current smokers. This issue could be a confounder to our results in not allowing us to identify two populations, those who never smoked and those who have and are now not smoking. At issue, however, is whether such a breakdown is relevant to the previous literature. To our knowledge, most previous smoking literature identifies patients who are current smokers and nonsmokers rather than identifying those who have never smoked and those who have smoked and quit and current smokers. As we wished to make comparisons between our results and this literature, the above problem does not confound our results. There is no doubt, however, that the identification of ex-smokers as a group can add to this area of scientific inquiry. Fourth, psychiatric diagnoses were assigned by one psychiatrist only without utilizing a standardized instrument such as the SCID. As such, no reliability data are available on the diagnoses assigned. However, these diagnoses were assigned by a senior psychiatrist utilizing DSM-III-R flow sheets and criteria. It is also to be noted that other sources of psychiatric pathology data, e.g., Beck Depression Inventory and State-Trait Anxiety Inventory, were utilized in the analyses. Similar to the DSM-III-R affective and anxiety diagnoses, these other sources of psychiatric pathology did not enter the models. As such, although the issue of reliability of DSM diagnoses could have been a confounder in the results of this study, it appears that the DSM data results are supported by other source results. Thus, it is unlikely that this issue confounded the results. Fifth, the data utilized in the study were gathered between 1991 and 1993 and we have published a number of studies utilizing this data set [37,38]. As such, it could be argued that these data are dated. However, there is no evidence in the pain literature that chronic pain populations have demographically changed. As such, the results of this study, if scientifically correct, would apply to current pain populations. Thus, we believe that the fact that these data are a decade old does not con-
found the application of these results (if scientifically correct) to current CPPs. Sixth, the range of variance explained by the various models was from 31% to 47%. One can therefore say that “only” 39% of the variance was explained by the derived variables. However, statisticians would say that this level of variance is not only statistically significant, but also substantively a high value, although 61% of the variance is unaccounted for. Thus, these results are important in spite of the variance values and are not a product of confounded data. The seventh and most important potential confounder is that of CPP selection and the impact of that on the results. It is to be noted that CPPs were selected to enter this grant study if they were candidates for employment after pain facility treatment. Thus, this group of CPPs does not represent the universe of CPPs at our pain center and because of the selection bias, more or fewer smokers could have been selected into the patient sample. However, this study is not about the percentage of smokers but about the prediction of smoking status in CPPs. As such, the results of this study are not confounded by this selection bias. However, because of the selection bias, the results only apply to the universe of CPPs who are candidates for employment. They do not necessarily translate to all CPPs. In addition, this last confounder may be the reason for the discrepancies between our results and the previous psychiatric literature.

Conclusions

Smoking in CPPs has been shown to be associated with some variables, which have been reported to be associated with smoking in the general and psychiatric population. However, a number of variables expected to be relevant, such as mood disorder, were not associated with smoking behavior. As smoking is a major problem in CPPs, further research on identifying variables associated with smoking is indicated. Because of the selection bias in the inclusion criteria of this study, the results only apply to CPPs who are candidates for employment.

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