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Acute Effects of Marijuana Smoking on Negative and Positive Affect

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Abstract

Human studies and animal experiments present a complex and often contradictory picture of the acute impact of marijuana on emotions. The few human studies specifically examining changes in negative affect find either increases or reductions following delta-9-tetrahydrocannabinol (THC) administration. In a 2 × 2, instructional set (told THC vs. told no THC) by drug administration (smoked marijuana with 2.8% THC vs. placebo) between-subjects design, we examined the pharmacologic effect of marijuana on physiological and subjective stimulation, subjective intoxication, and self-reported negative and positive affect with 114 weekly marijuana smokers. Individuals were first tested under a baseline/no smoking condition and again under experimental condition. Relative to placebo, THC significantly increased arousal and confusion/bewilderment. However, the direction of effect on anxiety varied depending on instructional set: Anxiety increased after THC for those told placebo but decreased among other participants. Furthermore, marijuana users who expected more impairment from marijuana displayed more anxiety after smoking active marijuana, whereas those who did not expect the impairment became less anxious after marijuana. Both pharmacologic and stimulus expectancy main effects significantly increased positive affect. Frequent marijuana users were less anxious after smoking as compared to less frequent smokers. These findings show that expectancy instructions and pharmacology play independent roles in effects of marijuana on negative affect. Further studies examining how other individual difference factors impact marijuana's effects on mood are needed.

Keywords

marijuana; anxiety; affect; expectancy; subjective effects

Long-term marijuana use is associated with mood and anxiety disorders (Agosti, Nunes, & Levin, 2002; Lynskey et al., 2002; Stinson, Ruan, Pickering, & Grant, 2006; Zvolensky et al., 2008). Although positive subjective effects (i.e., increased pleasure and reward enhancement) are most relevant in the initiation and progression to regular drug use, negative reinforcement (e.g., reduced negative affective states) becomes increasingly salient as dependence develops (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Robinson & Berridge, 2003). Marijuana use can both acutely reduce situational negative affect (e.g., tension; Haney, Ward, Comer, Foltin, & Fischman, 1999; McDonald, Schleifer, Richards, & de Wit, 2003; Phan et al., 2008) and allow chronic users to avoid unpleasant withdrawal symptoms (Budney, Moore, Vandrey, & Hughes, 2003). Not surprisingly then, disorders that have high levels of negative affect and arousal (e.g., posttraumatic stress disorder

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[PTSD]) lead to heightened drug motivation and are associated with protracted marijuana use (Bonn-Miller, Vujanovic, Feldner, Bernstein, & Zvolensky, 2007).

A proliferation of scientific evidence supports the major role of the endocannabinoid system in regulation of mood and anxiety states (Witkin, Tzavara, & Nomikos, 2005) and in the stimulating and intoxication effects of marijuana (Cooper & Haney, 2008; Huestis, 2007). Studies to date demonstrate a dose-dependent acute effect of marijuana on ratings of liking, good drug effect or euphoria, and satisfaction (Cooper & Haney, 2008; Hart, Haney, Vosburg, Comer, & Foltin, 2005; Metrik et al., 2009) with corresponding rapid transient increases in electroencephalographic alpha power, reflecting a neurophysiological correlate of the reinforcing effects (Lukas, Mendelson, & Benedikt, 1995). THC has positively reinforcing effects, with higher THC doses preferred over lower ones (Cooper & Haney, 2008).

In contrast to mostly homogeneous findings on positive affect following marijuana smoking, findings on the acute impact of cannabis on negative affect, and anxiety in particular, are often contradictory (Witkin et al., 2005). Despite marijuana's tension-reduction property, an often-cited motive for long-term use, studies also find acute increases in feelings of anxiety and panic following THC administration (D'souza et al., 2008; McDonald et al., 2003), consistent with its effect on increased physiologic arousal (Wachtel, ElSohly, Ross, Ambre, & de Wit, 2002). Thus, the aims of this study were to examine marijuana's subjective effects, broadly categorized as those related to drug “liking” (i.e., increased pleasure, reduced negative affective states; Robinson & Berridge, 2003) and potential moderators of these acute effects.

The reason for the mixed findings may be due to multiple factors including (a) *individual differences*: genetic background (Onaivi, Chakrabarti, Gwebu, & Chaudhuri, 1996; Schacht, Selling, & Hutchison, 2009), personality (Ashton, Golding, Marsh, Millman, & Thompson, 1981), history of use (Chait & Perry, 1992; Kirk & de Wit, 1999), and cognitive factors such as anxiety sensitivity (Bonn-Miller, Zvolensky, & Bernstein, 2007; Bonn-Miller, Zvolensky, Marshall, & Bernstein, 2007; Buckner & Schmidt, 2009) or marijuana expectancies (Schafer & Brown, 1991); (b) *drug-related differences*: the dose-dependent biphasic effects of marijuana (i.e., anxiolytic effects from low doses and anxiogenic effects from higher doses; Sulcova, Mechoulam, & Fride, 1998; Witkin et al., 2005) and cannabinoid composition (i.e., THC's anxiogenic and cannabidiol's (CBD) anxiolytic properties; Zuardi, 2008), and (c) *differences in experimental methodology*: assessments employed (e.g., neuroimaging vs. subjective; Phan et al., 2008) and timing of assessment. Increases in subjective anxiety immediately after smoking may be in part due to the initial stimulation and physiological effects of marijuana, including strong cardiovascular effects (Curran, Brignell, Fletcher, Middleton, & Henry, 2002; Wachtel et al., 2002). THC reliably increases heart rate with peak elevations produced by smoked marijuana occurring within 10 to 15 minutes of administration and returning to baseline levels after 90 minutes (Hart, van Gorp, Haney, Foltin, & Fischman, 2001). Interestingly, peak physiological arousal may coincide with positive affective reactions (i.e., euphoria) and negative reactions (i.e., anxiety). It is possible that people are interpreting their elevated heart rate as a sign of anxiety.

In addition to the pharmacological action, differences in acute drug response may also be caused by drug expectancies (Marlatt & Rohsenow, 1980). A drug-taking situation involves expectations about the administration of a drug (i.e., stimulus expectancy, such as “I am smoking marijuana”) and expectancies about the effects associated with the drug (i.e., outcome expectancies, such as “Marijuana makes me relaxed”). Marijuana expectancies have been shown to be related to marijuana use (Aarons, Brown, Stice, & Coe, 2001; Galen & Henderson, 1999; Schafer & Brown, 1991) and to explain the relationship between social

anxiety and marijuana use and problems (Buckner & Schmidt, 2008, 2009). Such outcome expectancies may differentially impact behavioral responses to THC or marijuana placebo. In a recent balanced-placebo design (BPD) study, Metrik and colleagues found independent effects of a marijuana stimulus expectancy manipulation (told THC vs. told no THC) in addition to the pharmacological effects (smoked marijuana with 2.8% THC vs. placebo) on subjective measures, marijuana ratings, and marijuana smoking behavior (Metrik et al., 2009). Although THC and stimulus expectancy of THC both increased ratings of satisfaction in comparison to placebo, only the pharmacological effect was significant for affective rating of “It made me feel better.” Whether marijuana outcome expectancies moderate effects of stimulus expectancies or THC has not been tested. However, outcome expectancies have been shown to moderate alcohol's effects on behavioral tasks (Vogel-Sprott & Fillmore, 1999) and nicotine's effects on negative affect (Juliano & Brandon, 2002), suggesting that such moderation is possible.

The aims for this study were to use the BPD to examine marijuana's pharmacologic and stimulus expectancy effects on (a) positive and negative affect, (b) physiological and subjective stimulation, and (c) subjective intoxication. Effects were expected to be in the same direction for each independent variable. We hypothesized that THC would significantly increase physiological response (heart rate), subjective reports of stimulation and intoxication, and that both THC and instructions that THC was smoked would increase positive affective reaction. In light of the mixed findings on marijuana's effects on anxiety, we did not hypothesize a specific direction of the effect on measures of negative affect. Based on the extant literature, we hypothesized that individuals with more salient outcome expectations of cognitive and behavioral impairment from marijuana would report increased tension and anxiety after marijuana. In contrast, more positive outcome expectations of relaxation and tension-reduction from marijuana would lead to a greater decrease in anxiety and a greater increase in positive affect after smoking. Finally, because frequent users should show tolerance (Chait & Perry, 1992; D'souza et al., 2008; Kirk & de Wit, 1999; Lichtman & Martin, 2006), the influence of stimulus expectancy and THC on affect was hypothesized to be greater among infrequent users as compared to more frequent users.

Method

Participants

The study was approved by the Institutional Review Board of Brown University. Marijuana smokers (N = 114) were recruited from the community through newspaper advertisements, flyers, and social media websites (Facebook, Craigslist). To be included, participants had to meet the following inclusion criteria: native English speakers, 18 to 30 years of age (to generalize to most frequent users), marijuana use at least once a week in the past month and at least 10 times in the past 6 months but not more than 6 days per week, ability to abstain from marijuana for 24 hours without withdrawal, no history of substance abuse treatment and no intent to quit or receive treatment for cannabis abuse, not using other illicit drugs, not pregnant by urine screen at each visit, not nursing, no past month affective disorder or history of panic attacks, not psychotic or suicidal, not meeting criteria for alcohol dependence in the past 12 months, no contraindicated medical issues by physical exam by a study physician, not smoking more than 20 tobacco cigarettes a day, and no prior knowledge about the study procedures or contact with study participants (e.g, significant other).

Potential participants were screened by telephone (N = 1139) before completing an intake interview, at which they signed informed consent. Of the 172 eligible screens who signed consent, 43 were deemed ineligible at baseline (mostly because of medical, psychiatric, and marijuana use criteria), 12 additional individuals dropped out prior to the second session, and 3 participants experienced adverse events and thus did not complete the postsmoking

assessments. Results are based on 114 participants, except the Profile of Mood States (POMS), which was only administered to 97 participants.

Participants were told to refrain from all marijuana and tobacco smoking for 12 hours, with this time frame selected to avoid the onset of cannabis withdrawal (Budney et al., 2003) and to not drink alcohol for 24 hours and caffeine for 1 hour before both sessions. Because a negative urine THC screen could require several weeks, an alveolar carbon monoxide (CO) test was used instead to confirm no recent smoking (Cooper & Haney, 2009). Tobacco smokers were given an opportunity to smoke a tobacco cigarette following the CO test to prevent nicotine withdrawal at the second session. Self-reported smoking abstinence was verified by CO reading ≤ 6 ppm using a Bedfont Scientific Smokelyzer breath CO monitor. Zero breath alcohol concentration was verified with an Alco-Sensor IV (Intoximeters, Inc., St. Louis, MO). Positive THC urine screens were obtained from 58% of participants at baseline and from 64% of participants at the beginning of the smoking session.

Design and Randomization

The study involved a 2 × 2 randomized factorial design crossing drug administration (2.8% THC or 0% THC) with instructional set (Told THC or Told Placebo) (Metrik et al., 2009). Urn randomization (Wei, 1978) balanced conditions on gender, college status, and tobacco smoking: (a) Told THC/Received THC (n = 29), (b) Told THC/Received Placebo (n = 28), (c) Told Placebo/Received THC (n = 28), and (d) Told Placebo/Received Placebo (n = 29).

Procedure

Participants completed a baseline nonsmoking and an experimental smoking sessions on average 14.3 (SD = 8.3) days apart. Participants were informed that the study evaluated the effects of marijuana on mood and behavior, and that they would be randomly assigned to smoke one marijuana cigarette that contained THC or one marijuana placebo cigarette with THC removed. Experimental sessions occurred in a 75 ft^2 ventilated smoking room with a one-way mirror wall through which research staff observed participants at all times and communicated by intercom. At baseline, participants completed a battery of assessment questionnaires including demographic and substance use questions for descriptive purposes. Prior to smoking during the second session, participants completed subjective-effect questionnaires, baseline heart rate was measured, and then they were instructed about which cigarette they were assigned to smoke (see Metrik et al., 2009, for details of the instructional set manipulation procedures). Postsmoking assessment (heart rate, cigarette ratings, questions pertaining to the credibility of the instructional set manipulation, and subjective effects) was designed to capture intoxication effects at their peak at 16 minutes after the start of the smoking, T1 (e.g., Lukas et al., 1995). Cigarette ratings were completed again at an average of 108 minutes from the start of smoking, T2. Participants in Told THC or Received THC conditions remained in the laboratory for 4 hours after smoking, passed a field sobriety test, were paid $145 for completing the sessions, and were transported home in a taxi. Participants in the deception conditions were fully debriefed regarding the deception following the completion of the study.

Marijuana Administration

Marijuana cigarettes (0% or 2.8% THC) were provided by the National Institute of Drug Abuse, rolled at both ends, humidified before use, and smoked according to the standardized paced puffing procedure (Foltin, Fischman, Pedroso, & Pearlson, 1987; Metrik et al., 2009).

Descriptive Measures

The *Time-Line Follow-Back* (TLFB; Dennis, Funk, Godley, Godley, & Waldron, 2004) was used to establish a 60-day retrospective marijuana (number of days smoked), alcohol use, and tobacco cigarettes baseline. *Marijuana History and Smoking Questionnaire* includes questions about age of onset, number of hours spent smoking per day, amount of money spent monthly on marijuana, and other questions (Metrik et al., 2009). *Marijuana Withdrawal Checklist* has 15 items ranging from 0 (not at all) to 3 (severe) with 10 symptoms comprising a withdrawal discomfort score (Budney, Novy, & Hughes, 1999); not clinically meaningful in this sample: 2.7 (*SD* = 2.8). *Alcohol use disorder diagnoses* were based on the Structured Clinical Interview for DSM-IV Nonpatient Edition (SCID; First, Spitzer, Gibbon, & Williams, 2002).

Assessment of Credibility of the Instructional Set Manipulation

Participants completed cigarette ratings (from 0 “no effect at all” to 4 “a very strong effect”), estimated cigarette potency (from 0 “not at all” to 4 “extremely”) and THC content (0 = none 0%, 1 = low dose < 2%, 2 = moderate dose 2%–3%, 3 = high dose 3%–4%), and indicated whether they felt deceived about the content of the drug they received (Metrik et al., 2009).

Physiological Effects

Because heart rate is a sensitive physiological indicator of THC absorption (Chait & Perri, 1992), heart beats per minute were recorded (Datascope Accutorr Plus NIBP automated vital sign monitor; Datascope Corp., Paramus, NJ) via a blood pressure cuff attached to a participant's nondominant arm.

Subjective Effects of Marijuana

Subjective effects of marijuana were determined using the 12-item ARCI-Marijuana scale (ARCI; Chait, Fischman, & Schuster, 1985; Martin, Sloan, Sapira, & Jasinski, 1971). Affective reactions to marijuana were assessed with several cigarette-rating items such as cigarette satisfaction: “It was satisfying,” liking: “I liked it,” and “makes me feel better” (Metrik et al., 2009), scored on 5-point Likert scales ranging from 0 (“not at all”) to 4 (“extremely”). *Self-Assessment Manikin* (SAM; Bradley & Lang, 1994) is a brief, momentary affect measure that includes two pictographic scales: (a) valence (pleasant vs. unpleasant), and (b) arousal; each scored on 5-point Likert scales. POMS; McNair, Lorr, & Droppleman, 1971) is a 30-item measure of state affect that includes adjectives along six dimensions including tension anxiety on a 5-point Likert scale (0 = not at all, 4 = extremely). ARCI-M, SAM, and POMS were administered prior to smoking and at T1.

Marijuana Outcome Expectancies—Cognitive-Behavioral Impairment (10 items) and Relaxation-Tension Reduction (8 items) scales of the *Marijuana Effect Expectancy Questionnaire* (MEEQ; Schafer & Brown, 1991) were administered to assess beliefs about possible consequences of smoking marijuana. The MEEQ is scored on 5-point Likert scales and has good psychometric properties (Aarons et al., 2001).

Data Analysis Plan

Differences between experimental conditions were tested with analyses of variance (ANOVAs) and chi-square tests. To examine the credibility of the instructional set manipulation, we examined the proportion of participants whose answers regarding the drug they received were congruent with their instructional set (Metrik et al., 2009). We then used regression models to analyze pharmacologic and stimulus expectancy effects of marijuana on measures of affect and arousal while covarying presmoking scores. Repeated measures

ANOVAs, 2 (T1 and T2) × 2 (Drug) × 2 (Expectancy) were used for the postsmoking marijuana ratings and for the ARCI-M scale (ANCOVA: covarying baseline ARCI-M scores). Correlations among the POMS Anxiety with other affective and arousal variables determined the overlap of anxiety and arousal measures. Regression analyses were conducted to determine the role of physiological arousal (by self-report and heart rate) in anxiety response to marijuana. Separate multiple regression analyses were conducted to test the hypotheses that (a) marijuana outcome expectancies for cognitive-behavioral impairment, (b) outcome expectancies for relaxation and tension reduction, and (c) frequency of marijuana use moderated marijuana's acute effect on the three subjective measures of affect (the POMS Tension-Anxiety subscale, the SAM valence item, and the marijuana rating of “feeling better”). These hypotheses were tested in separate regression models with presmoking values of the dependent variables (for the POMS and the SAM) entered on the first step as covariates, two main effects of stimulus expectancy and drug manipulations entered on the second step, outcome expectancy subscale or frequency of marijuana use variable entered on the third step, and two interactions of each respective moderator with stimulus expectancy and drug manipulation, respectively, entered on the fourth step to predict the affective reaction postsmoking. All tests of statistical significance were conducted with an alpha level set at .05.

Results

Table 1 presents descriptive statistics for the sample. The four experimental conditions did not differ significantly on any of these descriptive variables, and effect sizes were of relatively small magnitude (η^{2}'s: .002–.099, Cramer's ϕs: .041–.225, all *p*s > .17).

Credibility of the Instructional Set Manipulation

In the Told THC/Received Placebo condition, two participants (7%) indicated that they had suspicions about “THC content.” In the Told Placebo/Received THC condition, seven (25%) reported that they suspected their cigarettes had THC in them. No one in other conditions endorsed any deception.

Marijuana Cigarette Ratings—All cigarette ratings were significantly higher in the THC than Placebo conditions across time (see Table 2). There were no significant drug × stimulus expectancy interaction effects for any of the cigarette rating scales.

Valence and Affect—Significant main effects of the drug but not of stimulus expectancy were observed on the POMS Tension-Anxiety (*B* = .57, *SE* = .15, sr^2 = .10, *p* < .001) and Confusion-Bewilderment (*B* = .16, *SE* = .08, sr^2 = .04, *p* < .05) subscales. These two subscales were square root transformed to correct positive skewness, but raw means (*SD*) are shown for ease of interpretation. Post hoc tests revealed that compared to baseline, tension was nonsignificantly increased in the Told Placebo/Received THC condition (*paired t*[*df* = 24] = .35, *p* = .72), slightly decreased in the Told THC/Received THC condition (*paired t*[*df* = 24] = .92, *p* = .37), and significantly decreased in the two Received Placebo conditions after the smoking (*paired t*'s[*df*'*s* = 22 and 23] = 3.72 and 2.73, *p*'s < .01; see Figure 1). Comparison of the two instructional set conditions revealed significant differences at baseline with increased tension in the Told THC conditions, *M*(*SD*) = 2.06 (2.46), as compared to the Told Placebo conditions, *M*(*SD*) = 1.18(1.49), *p* < .05, revealing a randomization failure on this variable. There were no baseline differences on other subjective measures. Direction of effect was consistent within drug and instructional set conditions across other variables. Significant main effect of stimulus expectancy but not of the drug was observed on the Vigor-Activity subscale (*B* = 1.08, *SE* = .52, sr^2 = .03, *p* < .

05), with decreased vigor in the Told Placebo relative to Told THC conditions. No interaction effects approached significance for any of the POMS scales.

On the SAM valence scale, both stimulus expectancy main effect (B = .43, SE = .15, sr^2 = .05, p < .01) and drug main effect (B = .48, SE = .15, sr^2 = .06, p < .01) were significant. The drug by stimulus expectancy manipulation interaction was just outside of traditional significance level (B = –.41, SE = .21, sr^2 = .02, p = .055) and indicated that valence was more negative in Told Placebo vs. THC conditions when only placebo was smoked (B = .40, SE = .15, sr^2 = .09, p < .01). However, there was no significant difference between the Told THC and Told Placebo groups (B = .04, SE = .14, sr^2 = .001, p = .81) among those who received THC. On the marijuana rating of “feel better,” valence was more positive in the THC conditions as compared to Placebo conditions, as depicted in Table 2 and Figure 2.

Heart Rate, Arousal, and Intoxication Effects—As expected, THC significantly increased heart rate relative to placebo at T1(B = 38.20, SE = 3.08, sr^2 = .48, p < .001). There were no significant main or interaction effects with stimulus expectancy. For the ARCI-M summary scores, there were significant main effects of drug, $F(1, 109) = 62.12$, p < .001, and stimulus expectancy, $F(1, 109) = 4.68$, p < .05, manipulations indicating significantly higher ARCI scores of marijuana's effects in the THC conditions, relative to Placebo conditions, over time. There was a significant main effect of time, $F(1, 109) = 8.56$, p < .01, and a significant drug by time interaction, $F(1, 109) = 6.82$, $p \leq .01$, such that THC significantly increased subjective ARCI scores of marijuana's effects, relative to placebo, with a higher increase immediately postsmoking than at the end of the postsmoking testing period. The stimulus expectancy manipulation by time interaction was nonsignificant, suggesting no significant differences in stimulus expectancy effect averaged across time. Significant main effect of the drug was also observed on the SAM arousal ratings, with THC significantly increasing self-reported levels of arousal at T1 postsmoking (B = .61, SE = .18, sr^2 = .09, p < .001). The main and interaction effects of stimulus expectancy were not significant.

Analyses of Associations between Affective and Arousal Variables

Correlations among the T1 postsmoking affective, arousal, and heart rate variables by drug condition along with each measure's mean and standard deviation are presented in Table 3. In the whole sample, POMS tension-anxiety scale (square root transformed) did not correlate significantly with positive affective measures (SAM valence and “feel better” cigarette rating: rs –.06 and .10, respectively), but was significantly correlated with changes in heart rate from presmoking to postsmoking, subjective arousal, and subjective intoxication on the ARCI-M (rs .22–.28). Correlations between subjective arousal on the SAM with heart rate change and the ARCI-M scores was higher (rs .31 and .43, respectively), with the latter two measures moderately correlated (r = .51). Regression models controlling for presmoking POMS anxiety indicated that changes in heart rate did not explain additional variance (sr^2 < .01) in the postsmoking POMS Anxiety over and above the drug (sr^2 = .10) and expectancy (sr^2 < .01) terms. However, postsmoking arousal was a significant predictor of the postsmoking POMS Anxiety over and above drug, expectancy, and heart rate change terms with presmoking anxiety and arousal levels controlled (B = –.21, SE = .08, sr^2 = .04, p = .01).

Marijuana Outcome Expectancies and Frequency of Use in Prediction of Postsmoking Affective Reactions—In the POMS anxiety moderational model, a drug by impairment outcome expectancy interaction was significant (B = .42, SE = .20, sr^2 = .03, p < .05). In those given THC, higher outcome expectancies were associated with greater anxiety after smoking, controlling for baseline anxiety (B = .47, SE = .15, sr^2 = .14, p < .01).

In the absence of THC, there was no significant association between outcome expectancies and anxiety (B = .18, SE = .14, sr^2 = .03, p = .19). There were no significant main or moderation effects of tension reduction expectancies on anxiety, $p > .05$. Both main effects of drug manipulation (B = .56, SE = .15, sr^2 = .10, $p < .001$) and frequency of marijuana use (B = –.01, SE = .003, sr^2 = .05, $p < .01$) were observed on anxiety, with frequent users reporting less anxiety after smoking as compared to less frequent users. When predicting "it made me feel better," there were significant main effects of drug (B = .85, SE = .20, sr^2 = .12, $p < .05$), stimulus expectancy (B = .71, SE = .20, sr^2 = .08, $p \leq .001$), and outcome expectancies for relaxation and tension reduction (B = .58, SE = .18, sr^2 = .07, $p \leq .001$), with no moderation effects. There were no significant effects of the moderators for SAM valence.

Discussion

Acute influences of marijuana and expectancies were found on positive and negative affect. Although valence became more positive after smoking active marijuana as compared to placebo, the direction of effect on anxiety varied depending on the individuals' stimulus expectancies. Anxiety decreased after smoking in all but one of the experimental conditions, the one in which participants were told Placebo but received THC. Furthermore, marijuana users who expected marijuana to cause more impairment on their thoughts and behavior displayed more anxiety after smoking active marijuana.

Although baseline differences in anxiety between the two instructional set conditions call for caution in the interpretation of the significant drug effect, results may reflect that anxiety postsmoking in the Told Placebo/Received THC condition resulted from inherent conflict between the interoceptive cues from significant drug-induced physiological arousal and the instructional set, which explicitly stated that placebo would have no effect on mood. Deceptive instructions leave participants wondering what they are ingesting in an experiment, which leads subjects to search for internal cues (Rohsenow & Marlatt, 1981). In contrast, consistency between instructional set and drug effect in the Told THC/Received THC did not increase anxiety in that group despite increases in heart rate and arousal. This finding, coupled with the fact that anxiety decreased in both of the Received Placebo conditions, illustrates the importance of considering expectancies in the context of drug administration studies. Although physiological reaction to a drug that contradicts preexisting beliefs about the ingested substance may increase anxiety, pharmacological effect that is consistent with the expected effect (i.e., tension reduction) or expectation of no effect on mood (under placebo) may decrease anxiety after smoking. Correspondingly, marijuana's divergent effect on anxiety was also dependent on the preexisting expectation of impairment from marijuana. Interestingly, unlike marijuana impairment expectancies, outcome expectancies for relaxation and tension reduction were not associated with changes in anxiety but were with marijuana rating of "it made me feel better." This suggests that tension-reduction outcome expectancies may play a more prominent role in positive affect, whereas impairment expectancies are more closely related to negative affect.

These findings are consistent with previous studies showing that the effects that people expect from a drug can significantly influence their behavior in accordance with those beliefs. For example, outcome expectancies can reduce or intensify the degree of alcohol-induced behavioral impairment (Vogel-Sprott & Fillmore, 1999) and also moderate the effect of tobacco stimulus expectancy on anxiety (Juliano & Brandon, 2002). Previous marijuana studies have also found that marijuana expectancy influences acute response to marijuana and to placebo (Chait & Perry, 1992; Jones, 1971; Kirk, Doty, & de Wit, 1998; Pihl, Segal, & Shea, 1978; Stark-Adamec, Adamec, & Pihl, 1981). However, the current study has extended these findings by (a) delineating the degree to which both

pharmacological and stimulus expectancy effects were responsible for the acute effects of marijuana on anxiety and positive affect, and (b) demonstrating the contribution of individual differences, namely, outcome expectancies, to marijuana's effects on anxiety. Results from this study also support the role of drug tolerance (Babor, 2006) in that frequent marijuana users exhibited less anxiety after the smoking as compared to less frequent smokers. Several marijuana studies have reported that participants with less marijuana experience reported stronger subjective effects (Chait & Perry, 1992; D'souza et al 2008; Kirk & de Wit, 1999).

Limitations

Discussion of current findings is necessarily limited to the cannabinoid composition of the marijuana material used in the experiment (i.e., THC's effects).Several recent studies have clearly identified distinct pharmacological actions of THC and CBD, another nonpsychoactive cannabinoid in marijuana with pharmacological properties that are widely explored for therapeutic (Pertwee, 2009; Rahn & Hohmann, 2009; Russo, 2008) and possibly psychiatric indications (Fride & Russo, 2006; Marsicano et al., 2002; Musty, 2002; Parolaro, Realini, Vigano, Guidali, & Rubino, 2010). Whereas THC was shown to induce anxiety, CBD has ameliorated anxiety (Fusar-Poli et al., 2009; Zuardi, 2008) and also modulated THC's psychoactive effects (Morgan, Freeman, Schafer, & Curran, 2010). In these studies, the ratio of CBD to THC was equal or greater than one, whereas marijuana available for research in the United States has inconsequential levels of CBD (Ilan, Gevins, Coleman, ElSohly, & de Wit, 2005; Russo & McPartland, 2002; Wachtel et al., 2002). Lack of support for the tension-reduction effects of marijuana may simply be a matter of the different chemical composition of the drug that we used. Additional limitations that must be noted include the single dose, the laboratory setting, the timing of measurement, and controlled smoking method. Assessment of negative affect was conducted during the peak heart rate and physiological arousal but not during the descending limb of THC's effect. Finally, in our effort to standardize smoking administration, participants were asked to smoke the whole cigarette rather than smoking ad lib as is typical, and this may actually induce anxiety in some individuals.

In light of the diverse experimental and individual difference factors that impact marijuana's effects on mood, further investigations are critical. Future studies should consider examining other individual differences that characterize persons most sensitive to such effects and that may differentially impact subjective responses. For example, recent advances in cannabis genetics suggest that genetic variability influences sensitivity to its effects (Haughey, Marshall, Schacht, Louis, & Hutchison, 2008; Schacht et al., 2009) and may be implicated in psychiatric conditions related to mood deregulation (Barrero et al., 2005; Ujike & Morita, 2004). Therefore, future laboratory studies might examine candidate genes for marijuana in the context of the BPD, as it allows for narrow phenotypes (e.g., pharmacologic effect of a drug independent of the expectancy effects; Metrik et al., 2009). Further in-depth investigations with a greater variety of affective measures and continuous assessment of affect during the entire intoxication period (1–2 hours postsmoking) are needed to replicate and extend our findings on mood. Next steps might involve the examination of different doses of THC and other smoking methods (*ad libitum*). Ideally, future laboratory studies should attempt to examine marijuana's effects on mood in clinical populations of marijuana users with comorbid affective disorders. However, implementing such a study would have obvious ethical implications. However, greater knowledge from experimental studies with nontreatment-seeking clinical populations that use various subjective, biological, and behavioral measures of emotional regulation will advance our understanding of marijuana's effect on emotions. Furthermore, the intriguing findings on CBD's effects on emotions demand more research specifically focused on the pharmacologic action of this cannabinoid

in relation to THC. In sum, experimental studies are important steps in advancing our understanding of the complex relationship between affective disorders and marijuana use.

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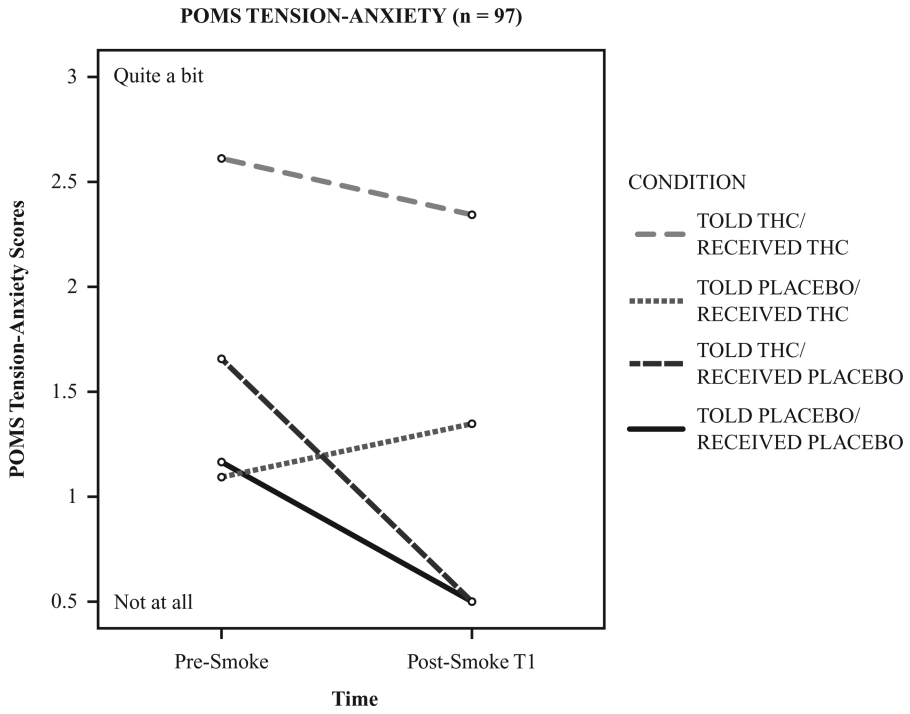

Figure 1.

Raw (untransformed) POMS Tension-Anxiety Scores prior to smoking and at 16 minutes (T1) postsmoking by BPD condition. Presmoking to postsmoking changes in anxiety in both Received THC conditions are not statistically significant; decreases in anxiety in both Received Placebo conditions are significant.

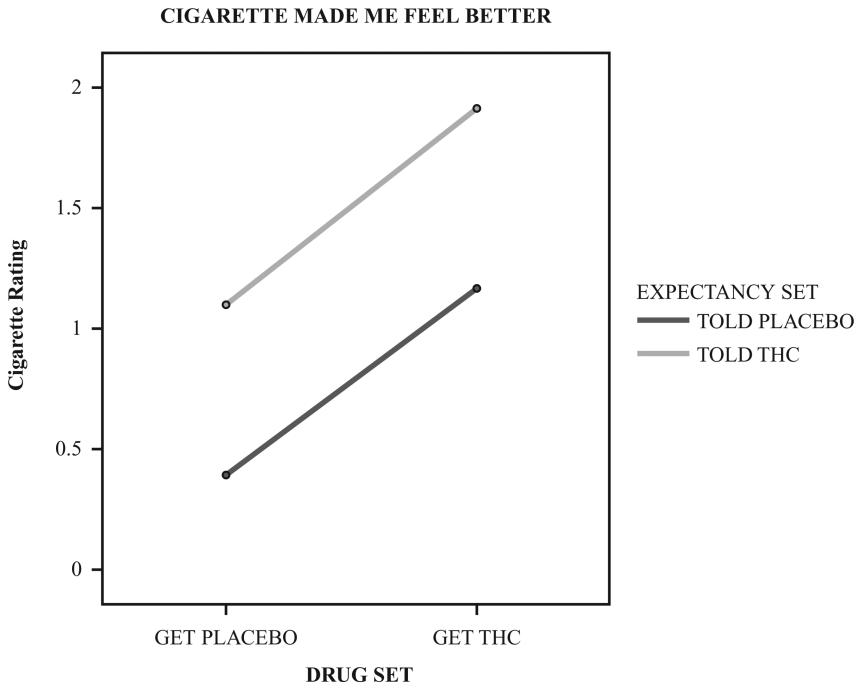

Figure 2.
Mean marijuana ratings for “Cigarette Made Me Feel Better” at 16 minutes (T1) postsmoking by drug and expectancy manipulation conditions.

TABLE 1

Demographics and Substance Use Sample Characteristics (N = 114)

Variable	*M*	*SD*
Age	21.5	3.3
Age of marijuana initiation	15.4	1.5
Age of regular use of marijuana	16.5	0.9
Percent of marijuana use days	37.7	22.8
Times used marijuana on an average day	1.75	1.0
Money spent on marijuana in the past 6 months ($)	332.38	398.37
Marijuana relaxation and tension-reduction expectancy	3.64	0.57
Marijuana impairment expectancy	2.75	0.72
Number of alcohol drinks per week	8.30	9.43
Percent of heavy drinking days	9.33	12.39
Percent of smoking tobacco days	53.3	41.7
Number of tobacco cigarettes per day	5.29	5.12

	n	%
Men	75	66
White[a]	76	67
African American	7	6
American Indian/Alaskan Native	1	1
Asian American	6	5
Mixed ethnic origin/other	18	16
Hispanic ethnicity	12	11
Marital status		
Never married	101	89
Married	4	3
Cohabitating	8	7
Divorced	1	1
In college	75	66
Education		
High school or general educational development (GED) diploma	17	15
Less than high school	3	3
Some college	79	69
Bachelor's degree or higher	15	13
Marijuana ounces used per week		
Less than 1/16th	29	25
1/16th	25	22
1/8th	20	17
More than 1/8th	40	36
DSM-IV alcohol abuse	24	21
Tobacco users	53	46

Note.

[a] Refers to non-Hispanic White. Percentages are based on available data per group.

TABLE 2

Means and Effect Sizes for Main Effects of Expectancy and Drug Manipulations for Cigarette Ratings

	Instructed					Received				
	THC		Placebo			THC		Placebo		
	M	(*SD*)	*M*	(*SD*)	Effect Size	*M*	(*SD*)	*M*	(*SD*)	Effect Size
Taste	1.46	1.39	1.02	1.28	.02	1.62	1.42	0.86	1.16	.09**
Smell	1.61	1.17	1.09	1.07	.05*	1.55	1.23	1.14	1.02	.04*
Similarity	1.05	1.21	0.56	0.95	.06**	1.25	1.31	0.37	0.62	.20***
Potency	2.07	0.92	0.96	1.21	.31***	2.21	1.06	0.82	0.91	.41***
THC content	1.55	0.69	0.51	0.78	.38***	1.35	0.97	0.70	0.68	.19***
Satisfaction	1.93	1.29	0.96	1.24	.13***	1.91	1.34	0.98	1.20	.18***
Liking	1.93	1.33	1.07	1.28	.11***	1.95	1.37	1.05	1.23	.17***
Feel better	1.54	1.23	0.82	1.15	.12***	1.60	1.27	0.77	1.07	.14**

Note. Means (*SD*) are from Tl postsmoking assessment for all variables except for Potency and THC content ratings completed at T2. Effect sizes for expectancy and drug between-subject effects are indicated as partial η^2 from 2 (Tl and T2) × 2 (Drug) × 2 (Expectancy) repeated measures analyses of variance. There were no significant interaction effects with time or drug × expectancy, except for taste: expectancy by time, $F(1, 110) = 4.34$, $p < .05$, with higher T2 than Tl ratings in the Told THC vs. Placebo.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

TABLE 3

Correlations Among T1 Postsmoking Affect–related Variables by Drug Manipulation Condition

Variable	*M*	*SD*	1.	2.	3.	4.	5.	6.	7.
Received Placebo (*n* = 57)									
1. ΔHR	73.35	11.55	—						
2. SAM-A	1.69	0.80	.03	—					
3. ARCI-M	1.53	1.79	–.06	.19	—				
4. ΔPOMS[a]	0.53	1.47	.21	–.16	–.08	—			
5. Feel better	0.77	1.07	–.02	.13	.35**	–.23	—		
6. SAM-V	3.85	0.65	–.12	.21	.20	–.10	.11	—	
7. Expectancy	—	—	.002	.02	.20	–.13	.38**	.29*	—
Received THC (*n* = 57)									
1. ΔHR	113.75	23.90	—						
2. SAM-A	2.33	1.09	.14	—					
3. ARCI-M	5.13	2.65	.09	.38**	—				
4. ΔPOMS[a]	1.86	2.46	.09	.34*	.30*	—			
5. Feel better	1.60	1.27	–.01	–.20	.17	–.16	—		
6. SAM-V	4.04	0.74	.002	–.02	.25	–.05	.39**	—	
7. Expectancy	—	—	.01	.07	.11	–.09	–.29*	–.04	—

Note. Spearman's rank correlation coefficients reported for correlations for the Expectancy manipulation variable; all other correlation statistics are based on Pearson's Product-Moment correlation coefficients. SAM-V = Self-Assessment Manikin Valence (possible range: 1–5); SAM-A = Self-Assessment Manikin Arousal (possible range: 1–5); ΔPOMS = POMS Tension-Anxiety Scale Presmoke to Postsmoke Change Score, correlations based on square-root transformed values but means (*SD*) based on raw values for interpretation (possible range: 0–4); ΔHR = Heart Rate Presmoke to Postsmoke Change Score but means (*SD*) based on responses at T1; ARCI-M = ARCI-Marijuana Scale Summary Score (possible range: 0–12); Feel Better = Cigarette Rating "Made Me Feel Better" (possible range: 0–4); Expectancy = Expectancy Manipulation (0 = Told Placebo, 1 = Told THC).

[a]POMS variables: $n = 47$ in placebo conditions and $n = 50$ in THC conditions.

*$p \leq .05$.

**$p \leq .01$