THE ASSOCIATIONS BETWEEN MARIJUANA USE, EMOTION DYSREGULATION, AND HEART RATE VARIABILITY AMONG HEALTHY YOUNG ADULTS

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Abstract

Recent epidemiological data have revealed that, in past years, there has been an increase in daily marijuana consumption by young adults. To gain better insight into factors that may contribute to problem use, the present study sought to examine associations between marijuana use and emotion regulation in a sample of 210 generally healthy young adults between ages 18 and 30 (mean age = 20.5, *SD* = 2.2; 52% women) using both self-report and experimental data. Using self-report data, this study found that those who frequently used marijuana reported significantly higher depression scores, compared with those who abstained. Furthermore, though no differences in self-reported anxiety were noted, those who regularly used marijuana employed more emotion regulation strategies to cope with affects than abstainers, especially "acceptance" and "rumination." In addition to self-report measures, the current study examined heart rate variability (HRV), a widely accepted indicator of emotion regulation capacities, in three groups of young adult subjects (abstinent, moderate, and frequent users). Despite prior literature reporting differences in HRV between those who use marijuana and those who abstain from use, this study did not find baseline differences in its sample. However, subjects who reported frequent marijuana use demonstrated greater physiological reactivity in response to testing, compared with the moderate and abstinent groups. Taken together, the self-report and physiological data may suggest that those who regularly use marijuana may have more difficulty regulating emotions which, in turn, may motivate them toward higher rates of use. Notably, these findings endured after controlling for co-occurring alcohol and cigarette use, as well as depression, each of which have been reported to affect HRV. Clinically, the reported findings may impart clinicians with increased insight into factors

which may contribute, in part, to the emotion regulation strategies and behavioral patterns of those who present seeking treatment for marijuana use. Results may also suggest that people entering treatment for marijuana use may benefit from therapies that emphasize emotion regulation skills.

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Introduction

Until the 1990's, marijuana was widely regarded as a non-addictive substance that caused few long-term health consequences (Jager, 2012). However, research has since demonstrated the addictive potential of marijuana, as well as the existence of a clinically distinct withdrawal syndrome (Budney & Hughes, 2006; Copersino et al., 2006; Levin et al., 2010). In fact, after alcohol use disorders, cannabis use disorder is the second leading cause for seeking addiction treatment. A 2013 National Survey on Drug Use and Health reported that an estimated 5.7 million persons aged 12 or older used marijuana on a daily or almost daily basis and, furthermore, 845,000 people received treatment for marijuana use at drug treatment centers during 2013 (Substance Abuse and Mental Health Services Administration [SAMHSA], 2013). Notably, the number of daily or almost daily marijuana users reported by SAMHSA reflects an increase of 2.6 million users compared to 2006 data. Considering that, as of March 6, 2017 (States decriminalized, n.d.), 20 states in the United States and the District of Columbia have decriminalized or legalized recreational marijuana use for those aged 21 and older, the number of users seeking treatment for marijuana may climb higher.

Although an estimated 9.1% of people reporting lifetime marijuana use become dependent[1] (National Institute on Drug Abuse [NIDA], 2005), it is important to note that

[1] According to the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; *DSM-5*; American Psychiatric Association, 2013), Cannabis Use Disorder may be diagnosed on the basis of a problematic pattern of use occurring within a twelve-month period which causes clinically significant impairment or distress along with at least two delineated symptoms (e.g., unsuccessful efforts to quit, craving, tolerance).

the addiction risk for marijuana may be somewhat lower than for other frequently used substances. For instance, it is reported that clinical dependence syndromes may be developed at rates of 15-16% and 12-13%, respectively, for cocaine and alcohol (Wagner & Anthony, 2002). Nevertheless, given the high prevalence of marijuana abuse, especially among youth, it is crucial to understand the mechanisms by which people who casually use marijuana may become heavy users. One important pathway to heavy use may be users' need to better regulate emotions (i.e., "self-medication";Simons & Carey, 2000).

To gain insight into the role of emotion regulation and marijuana use, this study examined the association between marijuana use and emotion dysregulation among young adults using self-report measures, as well as using heart rate variability (HRV) as a physiological indicator of one's emotion regulatory capacities. In addition, HRV was assessed for its associations with self-report measures of emotion dysregulation, depression, and anxiety. Finally, utilizing regression analysis, this study examined whether there was a unique association between marijuana use and HRV among young adults, controlling for emotional dysregulation and symptoms of depression and anxiety, as well as alcohol and tobacco use. In this dissertation, the available literature regarding the relationships between marijuana use and mood disorders (i.e., depression and anxiety) will also be briefly reviewed, followed by a review on the topics of emotion regulation and HRV.

Literature Review

Importantly, in the *DSM-5* (APA, 2013), the previous diagnoses of Cannabis Abuse and Cannabis Dependence were combined into a singular Cannabis Use Disorder diagnosis.

Marijuana and Mood Disorders

Though the literature has not yet demonstrated a causal relationship between marijuana use and mood disorders, there is mounting evidence that they are associated. Recently, two meta-analysis reviews of marijuana studies were published. In the first, Kedzior and Laeber (2014) examined 31 studies[2] on marijuana and anxiety, resulting in a combined sample of approximately 112,000 subjects from non-institutionalized populations in 10 countries. Notably, they found subjects meeting the criteria for anxiety disorders using scores on standardized scales or the DSM /ICD criteria for anxiety disorders[3] were more likely to use marijuana or have cannabis use disorder, reporting statistically significant odds ratios (ORs) of 1.24 (95% confidence interval [CI]: 1.06-1.45) and 1.68 (95% CI: 1.23-2.31), respectively. Moreover, subjects meeting the criteria for concurrent anxiety and depression were more likely to use cannabis, having an OR of 1.68 (95% CI: 1.17-2.40). Importantly, the authors suggested that, although these are significant associations, the correlations may be greater in samples seeking professional treatment for either marijuana use or anxiety. They also suggested that the relatively

[2] Studies were included in the meta-analysis if they reported: (1) data from a non-institutionalized general population; (2) anxiety diagnosis based on the *DSM-V*/International Classification of Diseases (ICD; World Health Organization, 1992) diagnostic criteria or anxiety severity score; (3) cannabis use/no use in cases with anxiety/no anxiety; (4) appropriate odds ratios and 95% confidence intervals; and (5) data sufficient to compute odds ratios. Studies were excluded if they: (1) did not report data from healthy non-users; (2) reported data from those seeking treatment for cannabis use disorder and/or presented with high psychiatric comorbidity; or (3) reported inadequate data to compute any effect size.

[3] Kedzior and Laeber (2014) provide a table of the assorted anxiety assessments and the DSM/ICD editions used in the 31 studies included in their meta-analysis review.

smaller effect sizes were likely due to the heterogeneous definition of anxiety disorders and use duration across studies.

In a second meta-analysis article, Lev-Ran et al. (2014) reported combined results from 14 studies examining marijuana use and depression in a sample of 76,058 subjects. Similar to Kedzior and Laeber (2014), the researchers found a greater prevalence rate of depression among marijuana users (OR = 1.17; 95% CI: 1.05-1.3), with the OR for heavy users increasing to 1.62 (95% CI: 1.21-2.16). Notably, Lev-Ran et al. included only the studies that controlled for baseline rates of depression. They also evaluated studies using the Newcastle-Ottawa Scale (Wells et al., 2005) to ensure that the studies included in the meta-analysis had high methodological quality. Therefore, the evidence on the associations between marijuana and depression and anxiety appears strong. In the forthcoming "Emotion Regulation" section, the role of emotion regulation is highlighted in the development of mood disorders. Prior to that discussion, however, it is important to contextualize the relationship between marijuana and mood disorders by examining associations between other psychogenic substances (e.g., nicotine, alcohol, cocaine) and depression and anxiety.

Other addiction problems and mood disorders. In a meta-analysis of 47 studies on smoking, nicotine dependence, and anxiety disorders, Moylan, Jacka, Pasco, and Berk (2012) found that nicotine dependence predicted the onset of agoraphobia, social phobia, specific phobia, and PTSD, with ORs ranging between 2.4 and 5.1. Similarly, in a sample of 1007 young adults, Breslau, Kilbey, and Andreski (1991) determined that the OR for moderately dependent nicotine users to develop an anxiety disorder was 4.2, compared to an OR of 1.5 for those reporting mild nicotine use.

Using data from the 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions, Hasin, Stinson, Ogburn, and Grant (2007) found that the OR for alcohol use disorder co-occurring with an anxiety disorder was 1.9 for 12-month use and 10.4 for lifetime alcohol dependence. Furthermore, in a meta-analysis of 15 studies that investigated associations between alcohol use disorders and major depression, Boden and Fergusson (2011) determined that the adjusted OR between drinking and clinical levels of depression ranged from 1.03 to 4.21, with a pooled OR ranging from 2.00 to 2.09. In another compelling epidemiologic meta-analysis of a non-substance based addictive behavior and depression, Ho et al. (2014) reported that the OR between internet addiction (e.g., internet gaming, addictive downloading and online shopping, excessive social networking cite use) determined from eight studies was 2.77 (95% CI: 2.04-3.75) for major depression and 2.7 (95% CI: 1.46-4.97) for anxiety disorders.

Finally, and perhaps most pertinent to the present study, Hadland et al. (2011) analyzed data from 447 street youths to determine the associations between marijuana, heroin, methamphetamine, and crack/cocaine use and depression. The researchers found that, compared to daily marijuana users, the adjusted OR for subjects reporting major depression and weekly heroin use was 2.64 (95% CI: 1.39-4.99); for weekly methamphetamine use, 1.88 (95% CI: 1.88-3.42); and for weekly crack/cocaine use, a statistically non-significant OR of 1.41 (95% CI: .79-2.52). Despite the relative elevated rates of depression found among heroin and methamphetamine users compared to the rates of depression among those who use marijuana, the associations between marijuana and mood disorders remains a troubling health concern.

Emotion Regulation

Prior to discussing emotion regulation, it is important to briefly examine how human emotion is conceptualized in the recent literature on emotion regulation. Citing the classic work by William James (1884), Gross (1998) noted that emotions constitute adaptive behavioral and physiological "response tendencies." Pursuant to this view, emotions are understood as flexible reaction sequences arising when one perceives a situation as conferring distinct opportunities or challenges. Emotional responses may thus entail activation of behavioral, experiential, autonomic, and neuroendocrine systems. Most pertinent to emotion regulation researchers, an individual may modulate (i.e., regulate) the response tendencies that constitute the emotion response.

Recently, Sheppes, Suri, and Gross (2015) highlighted several common features that unify the discrete emotions. Namely, they proposed that emotions: (1) generate when situational stimuli are interpreted as being central to personal, social, and cultural goals;[4](2) implicate changes across subjective, behavioral, and physiological domains; and (3) can be adjusted to suit the needs of a particular situation. They also indicated that the multisystemic changes accompanying an emotion may be further exposed by describing the intensity, duration, frequency, and category of the emotional response.

Emotion regulation has been defined as "the processes by which individuals influence which emotions they have, when they have them, and how they experience and

[4] Notably, though Gross's seminal 1998 article explicitly endorsed James's "response-tendency" perspective of emotions, his recent formulation seems to be equally accommodating of Schachter and Singer's (1962) competing theory of emotion, which emphasizes the role of cognitive interpretation in emotional responding.

express these emotions" (Gross, 1998, p. 275). Emotion regulation investigators have generally focused on two dimensions of emotion regulatory processes: the (1) *function* of emotion regulation in organizing internal processes such as attention, memory, and action readiness, as well as the emotion's role in informing social communication; and the (2) *ways* in which emotions are regulated (e.g., cognitive control) to afford the individual a chance to adapt reactions to situational demands and expectancies (Cole, Michel, & Teti, 1994).

Integrating these divergent foci, Gratz and Romer (2004) proposed that emotion regulation is a comprehensive, multidimensional construct involving: (1) acceptance of emotional responses; (2) ability to control impulsive behaviors; (3) ability to engage in goal-directed behaviors when experiencing negative emotions; (4) emotional awareness; (5) access to emotion regulation strategies; and (6) experience of emotional clarity. These discrete factors were incorporated into a 36-item scale called Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Conversely, impairments in any one of these capacities may be understood to indicate emotion dysregulation (Weiss, Sullivan, & Tull, 2015).

Responding to the need for greater specificity in defining emotion regulatory processes, Sheppes, Gaurav, and Gross (2015) recently proposed an extended process model of emotion regulation. This new model posits that emotion regulation difficulties may arise in the (1) identification of the need to regulate emotions; (2) selection among available regulatory options; (3) implementation of a regulatory tactic; and (4) monitoring of regulatory strategy across time. Moreover, they highlight the elements of one's unique perceptions, values, and actions in determining emotion regulatory

processes. Notably, in their expanded model, the researchers attempted to link various psychopathologies to maladaptation of the regulatory processes at discrete stages. For instance, panic attacks may be viewed as resulting from the overrepresentation of subtle signs (i.e., perception element) of current emotional states during the identification stage of the regulation sequence. Though the importance of this new model to the field is not yet fully known, its proposal highlights the need for better clarity regarding the constituent parts of the emotion regulatory process.

Emotion regulation and marijuana use. There is increasing consensus in the literature that marijuana use is associated with maladaptive attempts to regulate emotional states. In a frequently cited study, Bonn-Miller, Vujanovic, and Zvolensky (2008) recruited a community sample of 136 young adults to examine the relationship between emotion dysregulation and motives for marijuana use. Although several prior studies reported that marijuana use was related to certain emotion-related symptoms, the study by Bonn-Miller et al. was the first to use the empirically constructed DERS and the Marijuana Motives Measure (MMM; Simons, Correia, Carey, & Borsari, 1998) to explore the possible role of emotion dysregulation in motivating marijuana use. The MMM is a 25-item self-report measure to assess factors that may motivate marijuana use, consisting of the following five subscales: (1) Coping, (2) Expansion, (3) Conformity, (4) Enhancement, and (5) Social. Interestingly, Bonn-Miller et al. found that emotion dysregulation was correlated only with coping motives. Moreover, they found that the "Nonacceptance of Emotional Response" dimension[5] of the DERS accounted for the

[5] A representative question from the scale is: "When I'm upset, I feel guilty for feeling that way."

bulk of the relationship between emotion dysregulation and coping motives. This finding suggests the potential importance of emotion dysregulation and coping motives in predicting higher levels of marijuana use, as one's inability to tolerate distressing affect may motivate greater marijuana consumption.

A sampling of other notable findings in the literature concerning emotion dysregulation and marijuana use reveals the following: dysregulation predicts marijuana-related problems independent of gender and use frequency (Simons & Carey, 2002); difficulties in emotion regulation fully mediate the association between posttraumatic stress symptoms severity and marijuana use coping motives (Bonn-Miller, Vujanovic, Boden, & Gross, 2011); low levels of emotional clarity predict higher levels of problem marijuana use and an interaction exists between high cognitive reappraisal and low emotional clarity (Boden, Gross, Babson, & Bonn-Miller, 2013); and coping motives mediate the relationship between social anxiety and problem marijuana use, though social anxiety is unrelated to use frequency alone (Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007).

Although the literature suggests that marijuana use may be motivated by an attempt to regulate negative affective states, ultimately leading to maladaptive symptoms, there is a dearth of research showing this link in experimental studies. By observing HRV in an experimental study for those who use vs. do not use marijuana, we can actually compare their responses at baseline and when stimulated. Thus, the remainder of this proposal will examine the associations between HRV, emotion regulation, and marijuana use.

Emotion regulation and HRV. An individual's ability to regulate emotion is dependent on biological and environmental (i.e., learning) influences (Gross & Muñoz, 1995). Physiologically, the central autonomic network is comprised of cortical (prefrontal and anterior cingulate cortices) and subcortical (e.g., hippocampus, amygdala) structures that control emotional responding via feedback and feedforward loops (Thayer & Lane, 2000). A cardiac response to emotionally evocative environmental stimuli is indicative of emotional responding, which can be assessed. Various objective measures have been developed to track this critical physiological activity. One such index is HRV (see Appelhans & Luecken, 2006 for a review).

HRV refers to dynamic changes in inter-beat heart rate intervals. The measure tracks the dynamic range of adjustments between the sympathetic (excitatory) and parasympathetic (inhibitory) branches of the autonomic nervous system (ANS), as well as central nervous system (CNS) regulation of the ANS. HRV reflects both the magnitude and timing of emotional responses and has emerged as a widely accepted gauge of an individual's capacity to regulate emotion (Mun, von Eye, Bates, & Vaschillo, 2008). High HRV is reflective of greater capacity for regulated emotional responses, whereas low HRV has been associated with high mortality, psychological distress, and poor health outcomes (Appelhans & Luecken, 2006).

Due to the importance of HRV as a physiological indicator of health and wellbeing, substantial research has been devoted to examining how certain substances effect HRV. It is well established in the literature that acute alcohol intoxication decreases HRV (Koskinen, Virolainen, & Kupari, 1994; Vaschillo et al., 2008). Researchers have, however, also found that non-dependent subjects who reported

moderate alcohol consumption may have higher HRV than both non-drinkers and heavy drinkers. After analyzing 24 articles investigating associations between alcohol and HRV, Karpyak, Romanowicz, Schmidt, Lewis, and Bostwick (2014) determined that, in non-dependent adult drinkers, daily consumption of one alcoholic beverage drink for females and up to two drinks for males, was correlated with higher levels of HRV compared to abstainers and light drinkers. However, the researchers also discovered that subjects who consumed more than one and two daily alcoholic drinks, for women and men, respectively, had lower HRV than both abstainers and moderate drinkers. These findings suggest that drinking in moderation may lead to improved psychophysiological functioning, however, salutary effects are negated and HRV decreases once consumption exceeds moderate levels.

Research on other psychoactive substances has routinely found that drug use is associated with lower HRV. For instance, studies on nicotine and HRV have found that HRV is lower in nicotine users, and that HRV increases following smoking cessation (Harte & Meston, 2014; Hayano et al., 1990; Minami, Ishimitsu, & Matsuoka, 1999). Similarly, studies on cocaine have found that HRV decreases in response to cocaine administration (Vongpatanasin, Taylor, & Victor, 2004). Finally, in a sample of 80 treatment seeking heroin users, Chang et al. (2012) reported lower HRV among patients entering treatment, and also noted increased HRV in patients who complied with a methadone treatment regimen.

Surprisingly, there is dearth of epidemiological literature examining HRV in relation to marijuana use. Schmid, Scönlebe, Drexel, and Mueck-Weymann (2010) found that young men who reported marijuana use had higher HRV than non-marijuana users.

Paradoxically, the investigators also noted that marijuana users scored lower on the WHO-5 (World Health Organization, 1998), a five-item measure of psychological wellness. These puzzling findings led Schmid et al. to conclude that the relationship between psychological wellness and marijuana use may be complex and that marijuana may have a salutary effect on the cardiovascular system of marijuana users.

The Schmid et al. (2010) study was limited in several respects. Foremost, the researchers relied on a convenience sample of men and consequently had a sample selection bias. For example, the researchers did not collect data from women; thus, the effect of marijuana use on female HRV remains unknown. Moreover, the sample came from men eligible for military service and residing in Leipzig, Germany, leaving open questions about the association between marijuana use and HRV for other populations. Finally, the criterion Schmid et al. established for inclusion in the marijuana group may have been problematic. The researchers classified participants to the marijuana use group based on the presence of tetrahydrocannabinol (a chemical compound found in marijuana) in the blood stream alone, without any measures of frequency or quantity of use.

Though not an exploration of HRV, Somaini et al. (2012) also recently examined psychobiological responses to unpleasant emotions in marijuana users. In their study, the researchers examined hypothalamus-pituitary-adrenal (HPA) functioning as measured by adrenocorticotropic hormone (ACTH) and cortisol levels in response to neutral and unpleasant stimuli. They also examined participants' subjective responses to such stimuli using the State-Trait Inventory Y-1 (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) and the Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1994).

Somaini et al. (2012) found that, compared to healthy controls and those who regularly used marijuana in the past but had abstained for the past 6 months, marijuana dependent participants who continued to use marijuana reported the lowest level of emotional reactivity to negative stimuli as measured by the STAI. However, the researchers also found that current marijuana users reported the highest levels of trait anxiety, indicating a state of persistent arousal which may make them less reactive to additional stressors. Biologically, hyperactive HPA activity was found to be highest in active marijuana users, and only partially recovered after 6-month abstinence, compared to controls who had never used. Dampened ACTH and cortisol responses to negative stimuli were also reported in regular marijuana users, indicating lower arousal to aversive stimuli. It is important to note that the study could not establish if HPA dysregulation is a cause or result of marijuana dependence because of the cross-sectional design of the study.

In conclusion, the limited available evidence in the literature is inconsistent about the nature of the associations between marijuana use and emotion regulation especially in connection with adaptability. According to Schmid et al. (2010), the higher HRV levels found in marijuana users appears to indicate better ability to regulate emotion; however, the HPA dysfunction detected by Somaini et al. (2012) indicates otherwise. In addition, the important questions raised by Schmid et al. (2010) and Somaini et al. (2012) indicate that there is a knowledge gap in this area. This inconsistency may be attributed to the different samples studied in different studies. In addition, the available evidence in the literature, including the findings from meta-analysis reviews, are correlational data suggesting associations without a clear direction as to the direction of this association.

Therefore, it is important to examine these associations in healthy young adults in an experimental study before their ability to respond to adverse stimuli has been compromised.

The Current Study

The present study is aimed at addressing the following aims:

AIM 1: Examining the associations between marijuana use and self-reported measures of anxiety, depression, and emotion dysregulation in a sample of young adults.

AIM 2: Replicate and extend Schmid et al. (2010) in a broader sample of young adults to determine if marijuana use is positively correlated with higher HRV at baseline.

AIM 3: Determine whether participants endorsing marijuana use have higher HRV reactivity as the environmental demands increase.

AIM 4: Determine whether there is a unique effect of marijuana use on HRV, both at baseline and under testing conditions, after statistically controlling for other covariates, including typical alcohol and cigarette use.

Method

Participants

Participants were 210 healthy individuals between ages 18 and 30 (mean age = 20.5, *SD* = 2.2; 52% women) who participated in an existing experimental study designed to assess emotional responses to sensory stimulation (Mun, 2012). The majority of participants were Asian 46.2%; 35.2% were Caucasian, 9% were African American, and 9% identified as other. The parent study was conducted in three sequential phases, with each informing successive phase experiments. Since the aim of the parent study was to examine responses to sensory stimulation in the form of sensates (i.e., active ingredients

used for consumer products such as lotion) applied to the skin, those with skin problems and allergies were excluded from the study. In addition, individuals with physical conditions such as diabetes, respiratory problems, kidney or liver disease, or cardiovascular problems, or those who were pregnant or planning to become pregnant were excluded from participation. Finally, those who had high blood pressure (over 140 systolic and/or over 90 diastolic) or who were over- or under-weight (20% above or below from ideal weight) were excluded. The overall designs of the experiments were very similar and thus the data sets from all three phases were combined.

Recruitment and Procedures

Participants were recruited for the study during spring and summer of 2011 through word of mouth, university bulletin board fliers, website advertisement, and advertisement in the university newspaper. Those who expressed interest were contacted via phone to complete a brief screening interview in which they were assessed on the exclusion criteria described above, with the exception of blood pressure, height, and weight. Eligible participants were then scheduled for an individual experimental session between 10 am and 4 pm to control for circadian rhythms. At this time, they were instructed to abstain from any alcohol or drugs for 24 hours prior to testing, and asked to fast for 1 hour prior to testing. Upon arrival, informed consent was obtained, and blood pressure, height, and weight were assessed. Individuals who did not meet inclusion criteria based on these measures were informed they were not eligible to continue, thanked for their time, and compensated with $10. The remaining participants continued with the experimental session that consisted of both self-report and physiological assessments. Each session lasted approximately 1.75 hours, and participants were paid

$20 at the end of testing (this was increased to $25 midway through the summer in an effort to boost participation). All procedures were reviewed and approved by the university Institutional Review Board (Protocol 11-278M). The IRB granted exemption status for the present study (IRB Protocol # E16-238).

Participants were first directed to a computer and asked to complete a self-report form containing both demographic and health-related questions using Microsoft InfoPath 2007. A respiration strain gauge belt (Piezo respiratory belt transducer) was attached to the participant's chest, and electrodermal activity was assessed by attaching electrodes on volar surfaces of either medial or distal phalanges on the third and fourth fingers. One transducer was attached to the index finger for finger pulse, and one sensor was attached to the arm to measure skin temperature. ECG data were collected from electrodes on the right and left arms and the left leg.[6]

Participants were then seated in a comfortable chair positioned approximately 1-2 meters in front of a computer screen in a dimly lit, sound-attenuated and well-ventilated room (70-75°F). Participants then performed a standardized, cognitively low-demand task in which they were asked to view a sequence of colored objects (rectangles, circles, squares, or triangles) presented to them on the computer screen and silently count the number of blue objects. The objects appeared at the rate of one object per 10 s for a five-min period (Task 1). This is known as a "plain vanilla task" (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992) and was used to equate the influence of cognitive load on HRV across participants (Jorna, 1992; Sloan, et al., 1994). SuperLab 4.5 (Cedrus Corporation, San Pedro, CA) was used for the stimulus presentation. Physiological reactivity was

[6] EEG data were also collected from a small subset of participants, but not investigated in the present study.

measured during this task and served as baseline data. All physiological records were collected at a rate of 2,000 samples per s using the ADInstruments PowerLab data acquisition and analysis systems (ADInstruments, Colorado Springs, CO).

After the baseline procedure, participants were tested for two sensory stimulations as a part of the parent study, which included providing self-reported sensory experiences, saliva samples, as well as recording physiological data during repetition of the plain vanilla task (respectively, Task 2 and Task 3). For the purpose of the present study, the application of sensates and the increasing testing demands during Task 2 and Task 3 are posited to have induced mild distress in participants. Due to physiological recording, participants were told to restrain from movement during each task session and the entire experimental session took 1.5 to 2 hours. Therefore, we reasoned that physical discomfort and fatigue would be more likely as the experiment went on and, consequently, used task time periods (Tasks 2 and 3) as a proxy stress variable. Participants also completed self-report questionnaires on alcohol use and related problems, emotional regulation strategies, depressive symptoms, and anxiety using the InfoPath form described above.

Measures

Heart rate variability. To maintain consistency with Schmid et al. (2010), Root Mean Square of Successive Differences (RMSSD) in normal-to-normal beat intervals was used to assess HRV. RMSSD is a time domain measure of heart period variability and is sensitive to high-frequency heart period fluctuations in the respiratory frequency range and has been used as an index of vagal cardiac control. RMSSD was assessed during the initial 5 min baseline period (M=50.5, SD=31.6) prior to sensory stimulation, at Task 2 (M=54.8, SD=35.7), and at Task 3 (M=55.8, SD=35.5) (see Procedures above).

RMSSD indices were log transformed. Basal HRV refers to RMSSD assessed during the initial baseline period, whereas HRV reactivity refers to RMSSD indices measured during the latter task phases.

Marijuana use. Marijuana use was self-reported on a single question. Subjects were asked to give the frequency of marijuana use during the past month. Use was assessed on a rating scale between 0 – 6. Specifically, participants were asked to answer "How often have you used Marijuana or Hashish in the PAST MONTH" by selecting among the following options: 0 = *Never used marijuana or hashish* (*n*= 123); 1 = *not used in the past month* (*n*=46); 2 = *once a month* (*n*=8); 3 = *two or three times a month* (*n*=11); 4 = *once or twice a week* (*n*=10); 5 = *three or four times a week* (*n*=7); 6 = *every day or nearly every day* (*n*=5). Due to low response rates for higher frequency use, marijuana users were grouped into the following three use categories: 0 = *less than once per month* (*n*=169) ("Abstinent"); 1 = *once to three times per month* (*n*=19) ("Moderate"); 2 = *weekly or greater* (*n*=22) ("Frequent"). More detailed frequency statistics for marijuana use with this sample are reported in Table A1.

Alcohol use. Alcohol use was assessed using self-report measures of quantity, heavy episodic drinking, and alcohol-related problems. Alcohol use quantity in a typical week in the past month was assessed using the Daily Drinking Questionnaire (DDQ; Collins, Parks, & Marlatt, 1985). Heavy episodic drinking was assessed by asking participants the number of times they consumed five drinks or more (four or more for women) within two hours over the past month. Alcohol-related problems for the past three months were assessed using the 18-item version of the Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989, 2000). The RAPI assesses the extent to which

participants' daily functions and social relationships were affected by drinking and whether they experienced a higher alcohol tolerance or a blackout. For the 18 items presented, participants were asked to respond how often an outcome had occurred, in the past three months, using the following options: 0 = *none*; 1 = *1-2 times*; 2 = *3-5 times*; 3 = *more than five times*. The RAPI has demonstrated reliability and discriminant construct validity in both general population and clinical samples of adolescents and young adults (White & Labouvie, 1989, 2000) and the 18-item version correlates above 0.9 with the 23-item version (White & Labouvie, 2000). In the present study, Cronbach's alpha for the RAPI was .92. Means and standard deviations for these measures are reported in Table A2.

Tobacco use. Tobacco use was self-reported on a single question. Subjects were asked to give the frequency of tobacco use during the past month. Use was assessed on a rating scale between 0 – 6. Specifically, participants were asked to answer "How often have you used Cigarettes in the PAST MONTH" by selecting among the following options: 0 = *Never used cigarettes* (*n*= 149); 1 = *not used in the past month* (*n*=23); 2 = *once a month* (*n*=14); 3 = *two or three times a month* (*n*=10); 4 = *once or twice a week* (*n*=10); 5 = *three or four times a week* (*n*=1); 6 = *every day or nearly every day* (*n*=9).

Emotion regulation. Emotion regulation was assessed using the Cognitive Emotion Regulation Questionnaire short version (CERQ; Garnefski, Kraaij, & Spinhoven, 2002) that was designed to assess one's ability to cope with negative events. This measure includes 18 items which represent different thoughts one might hold in unpleasant situations (e.g., I think that I have to accept that this has happened) across the following nine subscales: self-blame, acceptance, rumination, positive refocusing, refocus

on planning, positive reappraisal, putting into perspective, catastrophizing, and other-blame. Participants were asked to indicate the frequency of each thought when experiencing a negative event on a 5-point scale ranging from 1 = *almost never* to 5 = *almost always*. Items within each subscale were prorated and summed to create scores reflecting different coping styles. Means, standard deviations, and Cronbach's alphas for CERQ subscales are reported in Table A3.

Depressive symptoms. Depressive symptoms were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D consists of 20 self-report items and provides a unitary measure of current depressive symptoms, with an emphasis on the affective component, depressed mood. Participants were asked to indicate how many days during the past week they had experienced the emotions or behaviors indicated in each of the items. The response options for these items ranged from 0 = *rarely or none of the time* to 3 = *most or all of the time*. Items were prorated for missing response and summed to create a total CES-D scale score (M=12.6, SD=8.1; a score of 16 or greater in the CES-D is used as a cut-off score for clinical depression). Cronbach's alpha for the CES-D with this sample was .87.

Anxiety symptoms. The State-Trait Anxiety Inventory for Adults (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was used to assess anxiety. This measure includes two forms, Y-1 (M=36.9, SD=9.2) and Y-2 (M=40.2, SD=8.9), each with 20 items. Form Y-1 is used to assess feelings of anxiety in the moment, or state anxiety. Participants were asked to indicate their feelings on a 4-point scale ranging from *not at all* (1) to *very much so* (4). Form Y-2 is used to measure general feelings of anxiety, or trait anxiety. Similar to form Y-1, participants were asked to read the items

and indicate their feelings on a 4-point scale ranging from *almost never* (1) to *almost always* (4). Items specific to each form were prorated summed to create total state and trait anxiety scores. Researchers and clinicians using the STAI suggest a clinical anxiety cutoff score of 39-40 (Knight, Waal-Manning, & Spears, 1983). Cronbach's alphas for forms Y-1 and Y-2 were .90 and .89, respectively, with this sample.

Results

Associations between Marijuana Use and Self-Reported Measures of Anxiety, Depression, and Emotion Dysregulation

Emotion regulation. Analysis of Variance (ANOVA) was conducted to determine if Abstinent, Moderate, and Frequent marijuana use groups differed in emotion regulation. Though there were no statistically significant differences in emotion regulation strategies detected across marijuana use groups, descriptive statistics demonstrated that Moderate and Frequent marijuana users reported greater use of each emotion regulation strategy. Moreover, differences between marijuana users vs. non-users approached statistical significance on the CERQ's "acceptance" (F=2.7, p= .07) and "rumination" (F=2.5, p=.09) subscales. Means, standard deviations and results of ANOVA F-tests are reported in Table 1.

Table 1

CERQ Descriptive Statistics and ANOVA F-tests between Marijuana Groups

Subscale	Group	*M* (*SD*)	*F*	*p*
Self-blame	Abstinent	4.9 (1.7)	1.7	.19
	Moderate	5.5 (1.8)		
	Frequent	5.4 (2.1)		
	Total	5.0 (1.8)		
Acceptance	Abstinent	6.5 (2.0)	2.74	.07

Table 1 – Continued

	Moderate	6.6 (1.7)		
	Frequent	7.5 (2.2)		
	Total	6.6 (2.0)		
Rumination	Abstinent	6.0 (1.8)	2.48	.09
	Moderate	6.8 (2.0)		
	Frequent	6.5 (2.1)		
	Total	6.1 (1.8)		
Positive refocusing	Abstinent	4.5 (1.9)	2.13	.12
	Moderate	3.8 (1.6)		
	Frequent	5.0 (1.7)		
	Total	4.5 (1.9)		
Refocus on planning	Abstinent	6.8 (2.0)	.50	.61
	Moderate	7.0 (1.6)		
	Frequent	7.2 (1.9)		
	Total	6.9 (1.9)		
Positive reappraisal	Abstinent	7.1 (2.1)	.93	.40
	Moderate	7.1 (2.1)		
	Frequent	7.7 (2.0)		
	Total	7.1 (2.1)		
Putting into perspective	Abstinent	6.3 (2.1)	1.3	.28
	Moderate	5.7 (1.7)		
	Frequent	6.8 (2.1)		
	Total	6.3 (2.1)		
Catastrophizing	Abstinent	4.3 (1.7)	2.19	.12
	Moderate	4.9 (2.5)		
	Frequent	5.0 (2.4)		
	Total	4.4 (1.9)		
Other blame	Abstinent	3.9 (1.2)	.59	.56
	Moderate	3.7 (1.3)		

Table 1 – Continued

	Frequent	4.1 (1.1)
	Total	3.9 (1.2)

Notes. With the exception of "other blame," *F*-test for each subscale had 173 Abstinent, 19 Moderate, and 23 Frequent subjects, respectively, and *dfs* for the *F*-test were 2 and 212. For "other blame," there were 172 Abstinent subjects, and *dfs* = 2, 211.
None of the ANOVA results were statistically significant at $p < 0.05$.

Depressive symptoms. ANOVA was conducted to determine if Abstinent, Moderate, and Frequent marijuana users differed in depressive symptoms. A statistically significant difference in depressive symptoms was found between Abstinent, Moderate, and Frequent marijuana users, $F\ (2, 212) = 4.44$, $p < .01$, $\eta^2 = .04$. Means and standard deviations are reported in Table 2, and Figure 1 illustrates the relationship between marijuana use and depressive symptoms.

Table 2

CES-D Descriptive Statistics and ANOVA F-tests between Marijuana Groups

Group	*N*	*M* (*SD*)	*F*	*p*
Abstinent	173	11.9 (7.5)	4.44	.01
Moderate	19	14.9 (9.9)		
Frequent	23	16.7 (10)		

Note. *dfs* = 2, 212

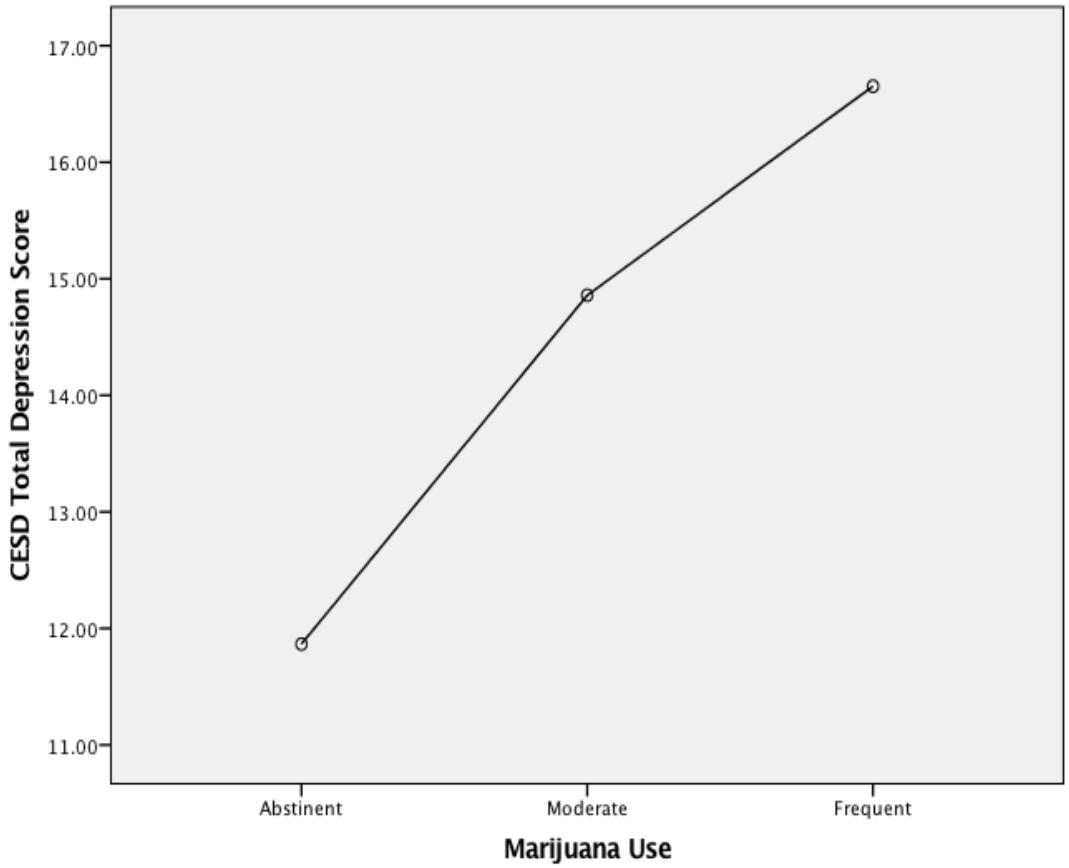

Figure 1. *Marijuana use and depression symptom (CES-D) scores*

Furthermore, results of Tukey post hoc comparisons indicated that Frequent marijuana users (M = 16.65, SD = 9.95) reported significantly higher depressive symptoms than Abstinent (M = 11.87, SD = 7.47) users, but not Moderate (M = 14.86, SD = 9.88) users. Tukey results are reported in Table A4.

Trait and state anxiety. ANOVA was conducted to determine if Abstinent, Moderate, and Frequent marijuana users differed in trait anxiety. No significant difference was found between Abstinent, Moderate, and Frequent marijuana users and trait anxiety, F (2, 212) = 1, p = .37. Descriptive statistics are reported in Table 3.

Table 3

Descriptive Statistics and ANOVA F-tests of Trait Anxiety, and State Anxiety at Baseline, Task 2, and Task 3 by Marijuana Group

	Group	*M* (*SD*)	*F*	*p*
Trait Anxiety	Abstinent	39.7 (8.5)	1.00	0.37
	Moderate	42.0 (8.7)		
	Frequent	41.7 (11.1)		
State Anxiety Baseline	Abstinent	36.8 (9.0)	0.08	0.92
	Moderate	37.3 (11.1)		
	Frequent	37.5 (9.9)		
State Anxiety Task 2	Abstinent	36.9 (9.3)	0.19	0.82
	Moderate	36.9 (10.6)		
	Frequent	38.2 (11.7)		
State Anxiety Task 3	Abstinent	38.3 (9.7)	0.08	0.90
	Moderate	38.5 (10.6)		
	Frequent	39.2 (12.1)		

Notes. With the exception of Task 3 "State Anxiety", each scale had 169 Abstinent, 19 Moderate, and 22 Frequent subjects, respectively, and *dfs* =2, 210. At Task 3, there were N=168 Abstinent subjects, and *dfs*=2, 208.
None of the ANOVA results were statistically significant at $p < 0.05$.

In addition to determining whether there was an association between marijuana use and trait anxiety, ANOVA was conducted to determine whether Abstinent, Moderate, and Frequent marijuana users differed in state anxiety at baseline, Task 2, and Task 3 (see Table 3). No statistically significant differences were found between Abstinent, Moderate, and Frequent marijuana users at baseline, F (2, 212) = .08, p = .92; Task 2, F (2, 212) = .19, p = .82; and Task 3, F (2, 212) = .08, p = .93.

Marijuana Use and HRV at Baseline

ANOVA was conducted to determine if Abstinent, Moderate, and Frequent marijuana users differed in RMSSD at baseline. No significant difference was found between Abstinent, Moderate, and Frequent marijuana users at baseline, F (2, 212) = 1.39, p = .25. Descriptive statistics are reported in Table 4.

Marijuana Use and HRV at Task 2 and Task 3

ANOVA was conducted to determine if Abstinent, Moderate, and Frequent use groups differed in HRV reactivity (when stimulated). Unlike the self-report results, this analysis examined a physiologically based indicator of subjects' ability to regulate emotion under increasingly time-consuming and stressful testing conditions. At Task 2, RMSSD did not statistically differ among those reporting Abstinent, Moderate, and Frequent levels of marijuana use, F (2, 207) = 2.57, p = .08. However, at Task 3, ANOVA results indicated that Frequent marijuana use was associated with higher levels of RMSSD, F (2, 206) = 4.52, p <.01, η^2 =.04. Descriptive statistics and f-test results are reported in Table 4 below.

Table 4

Descriptive Statistics and ANOVA F-test of RMSSD at Baseline, Task 2, and Task 3

	Group	*M* (*SD*)	*F*	*p*
Baseline	Abstinent	48.7 (30.0)	1.4	.25
	Moderate	59.7 (35.9)		
	Frequent	55.9 (39.1)		
Task 2	Abstinent	52.1 (33.2)	2.6	.08
	Moderate	67.3 (38.5)		
	Frequent	64.9 (48.0)		
Task 3	Abstinent	52.3 (30.7)	4.2	.01*
	Moderate	68.0 (41.5)		
	Frequent	72.4 (54.6)		

Table 4 – Continued

Notes. * p <.01. With the exception of RMSSD at Task 3, there were 172 Abstinent, 19 Moderate, and 22 Frequent subjects, respectively, and *dfs* = 2, 207. At Task 3, there were 170 Abstinent subjects (*dfs* = 2, 206).

Furthermore, Tukey post hoc test results indicated that subjects in the Abstinent group had significantly lower RMSSD than those reporting weekly use or greater; *M*=52.27, *SD*=30.67 vs *M*=72.42, *SD*=54.57 (see Table A5). Figure 2 below illustrates the change in RMSSD for Abstinent, Moderate, and Frequent groups at baseline, Task 2, and Task 3.

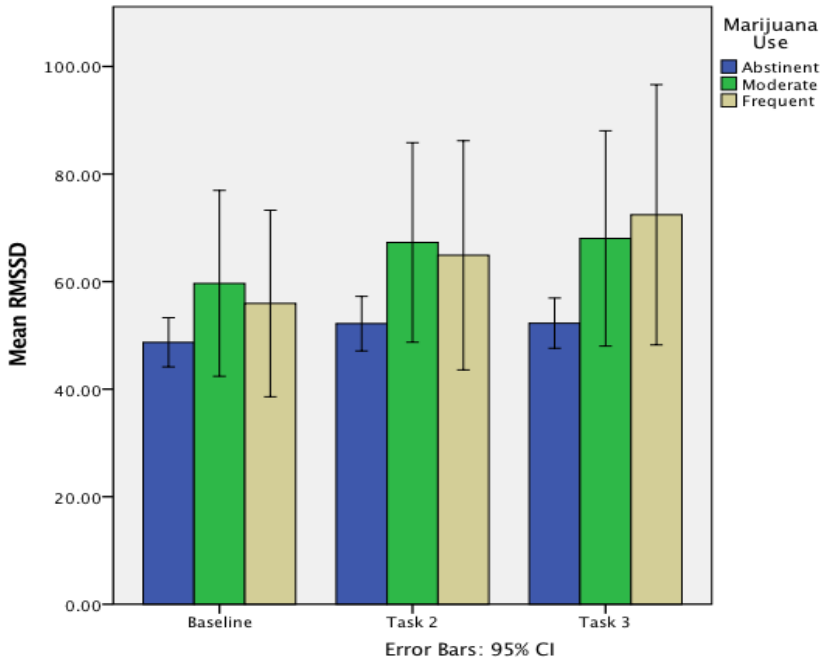

Figure 2. *RMSDD for abstinent, moderate, and frequent groups at baseline, Task 2, and Task 3*

At baseline, there was no statistical difference between subjects at either of the three use levels. However, at Task 3, Frequent users of marijuana had significantly greater RMSSD measures compared to Abstinent subjects; p <.01.

Marijuana Use and HRV after Controlling for Covariates

To determine if there was a unique association of marijuana with RMSSD at Task 3, cigarette and alcohol use were entered as covariates in a standard multiple regression analysis. Table 5 below shows the results of regression analysis (see Table A6 for correlations among alcohol use, cigarette use, and marijuana use). Even after controlling for typical alcohol and cigarette use, multiple regression analysis revealed that those who used marijuana exhibited greater RMSSD, $R^2 = .04$, F (3, 205) = 2.92, $p < .05$, with marijuana use uniquely accounting for 3.4% of the variance of RMSSD.

Table 5

Raw and Standardized Coefficients of Regression Analysis of RMSSD at Task 3

	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	*t*	*p*
(Constant)	53.01	3.804		13.933	0
Alcohol Use	-.09	1.943	-.004	-.046	.963
Cigarette Use	-.71	1.882	-.031	-.377	.707
Marijuana Use	11.8	4.354	.216	2.71	.007

Although not hypothesized, the present study additionally used depression symptom scores as a between-subject covariate in a within subjects repeated measures ANOVA. After applying a Huynh-Feld correction, depressive symptoms were unrelated to RMSSD measures, F (1.61, 329.5) = 1.56, $p = .21$. However, marijuana use was significantly associated with RMSSD measures over time, F (3.22, 329.5) =3.14, $p = .02$. Figure 3 below illustrates the relationship between marijuana use and RMSSD after controlling for depression scores.

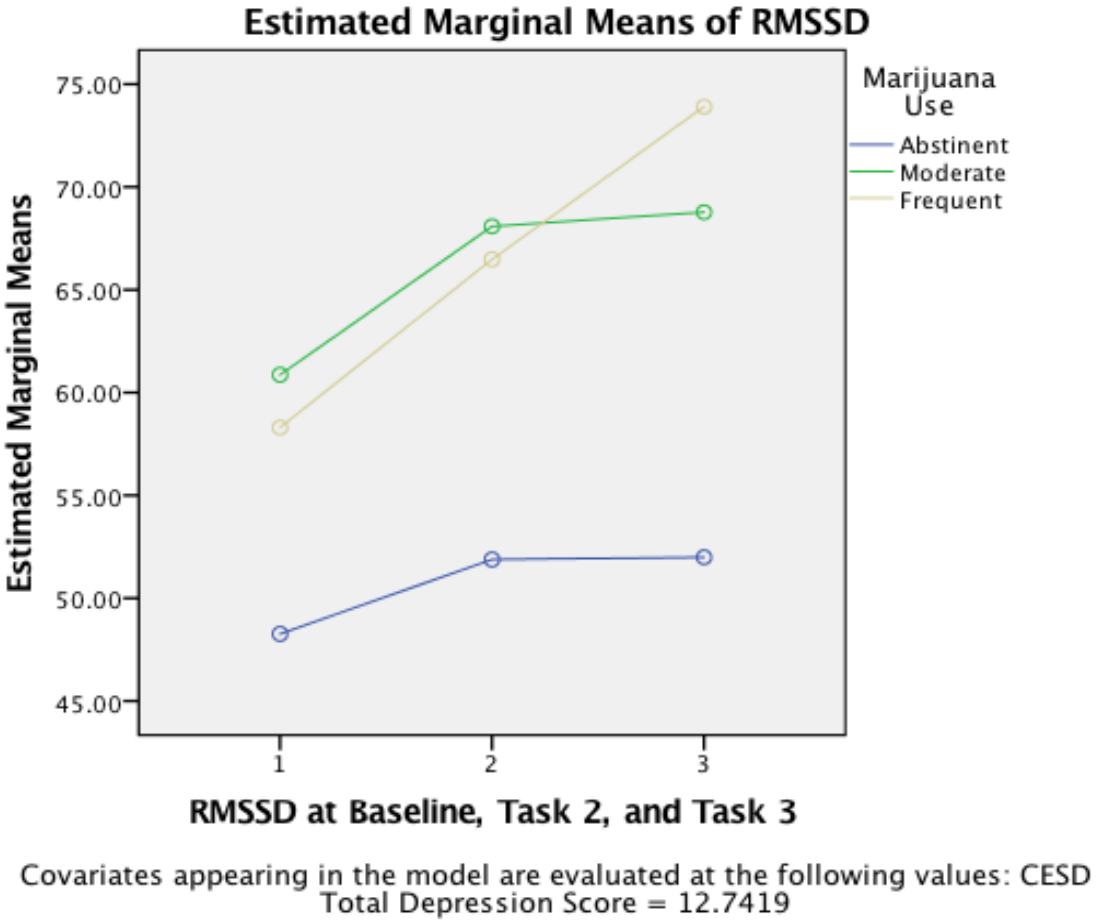

Figure 3. *Relationship between RMSSD and marijuana with depression included as a covariate.*

Discussion

Using self-report and psychophysiological measures, this study set out to explore the role of emotion dysregulation in motivating marijuana use in a sample of healthy young adults. Consistent with the literature reporting positive associations between marijuana use and mood disorders (Kedzior & Laeber, 2014; Lev-Ran et al., 2014), Frequent marijuana users were expected to report higher rates of anxiety, depression, and emotion dysregulation when compared to Moderate and Abstinent use groups. In addition to self-report measures, this study also examined associations between marijuana use and HRV, a physiological measure of adaptive emotional responding. Schmid et al. (2010) had previously reported higher basal HRV in a limited male sample of marijuana users, and this study sought to replicate those findings in a broader sample of young adults. Moreover, this was the first study to take repeated HRV measurements to explore changes in marijuana users' HRV under increasingly stressful testing conditions, as well as to control for alcohol and smoking, each of which have been associated with reduced HRV (Hayano et al., 1990; Vaschillo et al., 2008).

Anxiety, Depression, and Marijuana Use

Contrary to expectations (Kedzior & Laeber, 2014), this study did not detect associations between subjects' marijuana use and measures of state and trait anxiety. There are several possible explanations for these null findings. First, it is important to note that our sample was generally small and, therefore, we may have lacked statistical power to detect small group differences in state and trait anxiety. Furthermore, many studies reported in the literature utilized clinical samples in which users may have had more demonstrable differences in anxiety levels when compared to controls. In our

sample of healthy college students, however, it is possible that subjects experienced similar academic stressors and heightened levels of anxiety. This possibility of the restricted range affecting negatively on the inference may be demonstrated by examination of mean STAI scores (see Table 3) within the sample, which approach the suggested clinical anxiety cutoff score of 39-40 (Knight, Waal-Manning, & Spears, 1983).

Frequent marijuana users reported experiencing significantly higher levels of depression compared to Abstinent participants. This finding is consistent with the literature (Lev-Ran et al., 2014) and suggests that, within our sample, Frequent marijuana users experienced significantly greater depressive symptoms than Abstinent subjects. Moreover, Frequent users, on average, experienced clinical levels of depression, exceeding the CES-D's clinical cutoff score of 16 (M=16.7). This finding is notable because, unlike the ubiquity of anxiety in college students (Sax, 1997), clinical level depression is not typically considered to be a part of the normative collegiate experience. Clinically, this finding is of heightened importance because, though occasional marijuana use may be dismissed by some as a benign way of relieving stress, for those experiencing depressive symptoms, the drug may have increased potential for misuse.

Emotion Regulation and Marijuana Use

Difficulties regulating emotions may lead to depressive symptomology (Sheppes, Gaurav, & Gross, 2015), which, in turn, may be partially relieved by marijuana use. Although no statistically significant differences were found, marijuana users appear to generally employ more emotion regulation strategies on each of the CERQ's subscales. This may cautiously suggest that young adults who reported using marijuana exerted

more efforts to regulate emotions and were more reactive to their affects. Of the nine subscales of the CERQ, marijuana groups appear to differently use "acceptance" (F=2.7, p = .07) and "rumination" (F=2.5, p =. 09) strategies. This finding may tentatively suggest that marijuana use may be motivated as emotion regulation strategies. For instance, though "acceptance" may be adaptive if it implies an ability to move past an immediate stressor, when paired with a high degree of "rumination," it may cause one to perseverate on negative expectancies or stimuli without feeling personal agency to induce a positive change. For a person caught in what can be a paralyzing emotional bind, marijuana may decrease interfering ruminative thinking patterns.

Marijuana Use and HRV

This work reported several important findings regarding associations between marijuana use and HRV. First, in this sample, no baseline differences in HRV were detected among young adults who were Frequent, Moderate, and Abstinent in their marijuana use. This finding is particularly notable because prior research (Schmid et al., 2010) reported higher baseline HRV in marijuana users when compared to non-users. One possible explanation for the discrepancy in Schmid et al.'s findings and the findings reported herein may be that differences in the samples and methodologies used between the two studies. For instance, in the work by Schmid et al., men were recruited from a sample of potential military cadets presenting for a physical examination attendant to enlistment. Thus, the sample was fairly unique, which may make the findings from the study less generalizable to the broader populations. Conversely, our sample was recruited from a diverse pool of healthy young adults, most of whom were attending a public research university.

The second notable finding from this study was that Frequent marijuana users were found to have significantly higher HRV than Abstinent subjects near the end of the experiment. Though higher HRV is generally linked to better health outcomes (Appelhans & Luecken, 2006), when assessed in response to an immediate stressor, significant changes in HRV may signify that an individual is exerting substantial efforts to cope; similar difficulties in autonomic self-regulation have been reported in high-risk drinkers (Mun et al., 2008). Notably, this result remained significant after controlling for alcohol and cigarette use, as well as depression. Therefore, the data, on the whole, cautiously suggest that the associations between marijuana use and mood dysregulation symptoms may be driven by the motivation to "self-medicate" via up-regulating positive emotions and down-regulating negative emotions. While it would be more adaptive to utilize cognitive emotion regulation strategies and to have greater momentary HRV reactivity to a stressor, this study suggests that over time, this effortful and autonomic responses, when repeated, may not be adaptive for some individuals, increasing their allostatic load (McEwen, 1998).

Limitations and Future Directions

There are several important limitations to this study. First, the study used a sample of young adults recruited on a college campus and, thus, results may not be generalizable to other populations. Furthermore, marijuana use, a key variable, was assessed solely by self-report, and without indication of the method of ingestion, dosages typically consumed, or the psychotropic properties of the marijuana used. Since marijuana may vary greatly in potency and chemical structure, gaining experimental control of the marijuana condition may be especially important for future studies.

Despite its limitations, this study highlights the need for further resources to be allocated to studying the physiological effects of marijuana, the reasons why some people use the drug more than others, and possible differences in the substance's effects at various use levels and within different populations. This is an exciting field of study which, due to the spreading legalization and decriminalization of marijuana, is imperative for drug and alcohol researchers to undertake. Further knowledge in this area may help prevent addictions, guide public policy with respect to establishing norms for responsible marijuana use, and suggest new therapeutic strategies for those seeking to reduce consumption. This field promises to continue its rapid evolution in coming years, and this study may help provide anchoring and direction for future researchers and clinicians.

Clinical Implications

Treatments for marijuana dependence have been derived from behavioral approaches to the treatment of other substance use disorders (e.g., alcohol, cocaine). Approaches such as Cognitive Behavioral Therapy (CBT), Motivational Enhancement (MET), and Contingency Management (CM), and combinations of these methods, have received the greatest empirical support and are widely practiced in outpatient treatment centers (Budney, Roffman, Stephens, & Walker, 2007). CBT for marijuana use is typically administered over the course of 6 to 14, 45 to 60-minute sessions, and focuses on the following areas: functional analysis of marijuana use; identifying triggers to use; planning to cope with cravings; learning drug refusal and problem-solving skills (Steinberg et al., 2005; Budney et al., 2007). MET, conducted in 1-4, 60-90 minute sessions (Budney, Higgins, Radonovich, & Novy, 2000; Steinberg et al., 2005), addresses ambivalence about quitting marijuana. Therapists using MET help guide the patient

toward committing to change using certain therapeutic skills (e.g., exploring pros and cons of use, encouraging self-efficacy, rolling with resistance). Finally, CM encourages abstinence by providing varying types of reinforcement for maintaining abstinence (Budney et al., 2000; Budney, Moore, Rocha, & Higgins, 2006). In clinical trials, CM participants have been required to undergo urine screens twice per week to confirm marijuana abstinence. Participants who provided drug-negative samples for the complete duration of the 14-week monitoring period were eligible to receive vouchers worth $570 (Budney et al., 2000; Budney et al., 2006).

Although leading investigators often tout the efficacy of behavioral treatments for marijuana dependence (Budney et al., 2007), further analysis gives cause to temper such claims. For instance, in two randomized trials including CBT, MET, Multidimensional Family Therapy, and Adolescent Community Reinforcement conditions, involving 600 adolescent participants, Dennis et al. (2004) reported a small Cohen's effect size of $f = .1$; they also found that two thirds of the participants reported continued substance use at 12-month follow-up. Moreover, in a recent meta-analysis of randomized controlled trials of marijuana treatments, Davis et al. (2014) found that, when behavioral therapies were compared with active control groups (e.g., treatment as usual) instead of waitlist groups, there were no statistically significant group differences in treatment outcomes. Finally, it is important to note that youth entering marijuana treatment programs may present with lower motivation than other groups, as treatment seekers may be court mandated and more likely to be in a pre-contemplative stage of treatment (Sinha, Easton, & Kemp, 2003).

The limitations to the prevailing approaches of treating marijuana dependence reviewed above may suggest the potential clinical importance of the results from the current study. First, the findings may provide insight into why a subset of casual marijuana users become heavy users and misuse marijuana. Clearly, individuals who experience greater difficulty coping with stressors may gain more relief from the psychogenic effects of marijuana. Moreover, this study may provide specificity into the combination of regulation strategies (i.e., acceptance, rumination) that may make marijuana an appealing substance of choice for some users. However, it is also possible that marijuana users may engage more strategies regardless of whether they are positive or negative. Other strategies, such as catastrophizing and positive refocusing, for example, also tended to be endorsed by frequent marijuana users in this sample. If replicated and extended by future research, these findings would provide a basis for increased incorporation of more effective emotion regulation strategies (e.g., mindful meditation) into treatment for marijuana misuse. Notably, certain contemporary behavioral treatments which emphasize tolerating distressing affects, reducing reactivity to negative thoughts, and increasing behaviors linked to personally identified reinforcers have shown limited promise in treating marijuana dependence (Twohig, Shoenberger, & Hayes, 2007). Finally, this study may also indicate that HRV biofeedback, as well as other forms of therapeutics which improve autonomic regulation, such as exercise, may confer a particularly important benefit to those struggling to quit marijuana use.

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Appendix

Table A1. Percentage of Participants Reporting Past Month Marijuana Use

Table A2. Alcohol Use Descriptive Statistics

Table A3. Descriptive Statistics and Cronbach's Alpha for CERQ Subscales

Table A4. Tukey Comparison of Abstinent, Moderate, and Frequent Groups and CES-D Scores

Table A5. Tukey Comparison of RMSSD of Abstinent, Moderate, and Frequent Groups at Baseline, Task 2, Task 3

Table A6. Pearson Correlations between HRV and Alcohol, Cigarettes, and Marijuana at Baseline, Task 2, and Task 3

Table A1.

Percentage of Participants Reporting Past Month Marijuana Use

Response	*N*	*%*
0	125	58.1
1	48	22.3
2	8	3.7
3	11	5.1
4	11	5.1
5	7	3.3
6	5	2.3
Combined Responses	*N*	*%*
Abstinent (0-2)	173	80.5
Moderate (3-4)	19	8.8
Frequent (5-6)	23	10.7

Table A2.

Alcohol Use Descriptive Statistics

Measures	*M*	*SD*
DDQ	6.9	12
Heavy drinking	1.1	2.2
RAPI	3.1	5.8

Subjects completing each measure were DDQ = 202; Heavy drinking = 214; RAPI = 215.

Table A3.

Descriptive Statistics and Cronbach's Alpha for CERQ Subscales

Subscale	*M*	*SD*	α
Self blame	5.0	1.8	.70
Acceptance	6.6	2.0	.78
Rumination	6.1	1.8	.55
Positive refocusing	4.5	1.9	.71
Refocus on planning	6.7	1.9	.59
Positive reappraisal	7.1	2.1	.72
Putting into perspective	6.3	2.1	.72
Catastrophizing	4.4	1.9	.77
Other blame	3.9	1.2	.70

Table A4.

Tukey Comparison of Abstinent, Moderate, and Frequent Groups and CES-D Scores

(I) Marijuana Use	(J) Marijuana Use	Mean Difference (I-J)	Std. Error	*p*
Abstinent vs.	Moderate	-3	1.9	0.27
	Frequent	-4.8*	1.8	0.02
Moderate vs.	Abstinent	3	1.9	0.27
	Frequent	-1.8	2.5	0.75
Frequent vs.	Abstinent	4.8*	1.8	0.02
	Moderate	1.8	2.5	0.75

Note. * $p < 0.05$.

Table A5.

Tukey Comparison of RMSSD of Abstinent, Moderate, and Frequent Groups at Baseline, Task 2, Task 3

	(I) Marijuana Use	(J) Marijuana Use	Mean Difference (I-J)	Std. Error	*p*
RMSSD					
Baseline	Abstinent	Moderate	-10.9	7.6	.32
		Frequent	-7.2	7.2	.57
	Moderate	Abstinent	10.9	7.6	.33
		Frequent	3.7	9.9	.98
	Frequent	Abstinent	7.2	7.2	.57
		Moderate	-3.7	9.9	.92
RMSSD					
Task 2	Abstinent	Moderate	-15.2	8.6	.18
		Frequent	-12.8	8	.25
	Moderate	Abstinent	15.2	8.6	.18
		Frequent	2.4	11.1	.98
	Frequent	Abstinent	12.8	8	.25
		Moderate	-2.4	11.1	.98
RMSSD					
Task 3	Abstinent	Moderate	-15.8	8.4	.15
		Frequent	-20.1*	7.9	.03
	Moderate	Abstinent	15.8	8.4	.15
		Frequent	-4.4	10.9	.92
	Frequent	Abstinent	20.1*	7.9	.03
		Moderate	4.4	10.9	.92

Note. * $p < .05$.

Table A6.

Pearson Correlations between HRV and Alcohol, Cigarettes, and Marijuana at Baseline, Task 2, and Task 3

	Baseline	Alcohol Use	Cigarette Use	Marijuana Use
Baseline HRV	1	.003	.025	.076
Alcohol Use	--	1	.462*	.464*
Cigarette Use	--	--	1	.526*
Marijuana Use	--	--	--	1
	Task 2	Alcohol Use	Cigarette Use	Marijuana Use
Task 2 HRV	1	.058	.109	.141**
Alcohol Use	--	1	.462*	.410*
Cigarette Use	--	--	1	.468*
Marijuana Use	--	--	--	1
	Task 3	Alcohol Use	Cigarette Use	Marijuana Use
Task 3 HRV	1	.071	.069	.200***
Alcohol Use	--	1	.461*	.409*
Cigarette Use	--	--	1	.467*
Marijuana Use	--	--	--	1

Notes. *Correlations between alcohol use, cigarette use, and marijuana use were significant ($p < .001$).

** Marijuana use was correlated with RMSSD at Task 2 ($p < .05$).

*** Marijuana use was correlated with RMSSD at Task 3 ($p < .01$).