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A Reinforcement Sensitivity Model of Affective and Behavioral Dysregulation in Marijuana Use and Associated Problems

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This study tested a model linking sensitivity to punishment (SP) and reward (SR) to marijuana use and problems via affect lability and poor control. A 6-month prospective design was used in a sample of 2,270 young-adults (64% female). The hypothesized SP × SR interaction did not predict affect lability or poor control, but did predict use likelihood at baseline. At low levels of SR, SP was associated with an increased likelihood of abstaining, which was attenuated as SR increased. SP and SR displayed positive main effects on both affect lability and poor control. Affect lability and poor control, in turn, mediated effects on the marijuana outcomes. Poor control predicted both increased marijuana use and, controlling for use level, greater intensity of problems. Affect lability predicted greater intensity of problems, but was not associated with use level. There were few prospective effects. SR consistently predicted greater marijuana use and problems. SP however, exhibited both risk and protective pathways. Results indicate that SP is associated with a decreased likelihood of marijuana use. However, once use is initiated SP is associated with increased risk of problems, in part, due to its effects on both affect and behavioral dysregulation.

Public Health Significance
The current study identifies important aspects of dysregulated affect and behavior that contribute to initiation and maintenance of marijuana use and that distinguish between problematic from non-problematic use. Identification of these factors is becoming increasingly important given the rapidly changing landscape surrounding marijuana use in the United States. Prevention and intervention programs that target regulatory deficits have the potential to minimize negative substance use outcomes as well as having broader impacts on individual well-being.

Keywords: reinforcement sensitivity theory, affect lability, impulsivity, marijuana use, marijuana problems

Marijuana is the most commonly used illicit substance in the United States (Substance Abuse and Mental Health Services Administration (SAMHSA), 2013; Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2015). It is especially prevalent among young adults, with approximately 20% of 18- to 25-year-olds reporting marijuana use in the past 30 days (SAMHSA, 2013; Johnston et al., 2015) and up to 10% of young adult college students exhibiting marijuana-related problems significant enough to warrant diagnosis of a marijuana use disorder (Caldeira, Arria, O'Grady, Vincent, & Wish, 2008; Substance Abuse and Mental Health Services Administration (SAMHSA), 2010). Given the number of negative consequences associated with marijuana use (e.g., Pearson, Liese, Dvorak, & Marijuana Outcomes Study Team, 2017), understanding individual difference factors that contribute to marijuana use and marijuana-related problems is an important area of research.

Substance use contains both strong approach (e.g., euphoric effects) and inhibitory cues (e.g., legal, health risks). As such, differential responsiveness to punishment versus reward may be associated with increases or decreases in use through reinforcement processes. It follows that underlying individual differences in sensitivity to punishment and reward are associated with variability in substance use (Colder et al., 2013; Franken & Muris, 2006), including marijuana use (Simons, Dvorak, & Lau-Barraco, 2009b). However, because sensitivity to punishment (SP) and sensitivity to reward (SR) are related to diverse personality/regulatory constructs of relevance to substance use (e.g., neuroticism, impulsivity; Braddock et al., 2011; Corr & McNaughton, 2008), the linkage between SR, SP, intermediate regulatory constructs, and risky substance use needs to be further examined. The goal of the current study is to delineate the independent and joint effects of SR and SP on more proximal affective and behavioral regulatory constructs and marijuana use and problems.

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Sensitivity to Punishment and Reward

There are several neuropsychological theories of personality (for review, see DeYoung & Gray, 2009). One prominent model is reinforcement sensitivity theory (RST; Corr, 2004; Corr & McNaughton, 2008; Gray & McNaughton, 2000) which posits that individual differences in personality, emotions, motivation, and behavior arise from three conceptual brain systems thought to differentially respond to reward and punishment. Consequently, they are believed to govern approach and avoidance behavior. One system, the behavioral approach system (BAS), is suggested to be sensitive to stimuli that signal reward and underlies approach motivation, positive affect, and reinforcement learning processes. Evidence suggests that individuals with a hypersensitive BAS tend to be impulsive, sensation-seekers who respond to reward-related cues with increases in positive affect and approach motivation (Carlson, Pritchard, & Dominelli, 2013; Corr, 2008; Gray, 1987). Hence, a hypersensitive BAS (i.e., sensitivity to reward) may be a risk factor for substance use motivated by positive reinforcement. Several studies have shown those with a high BAS display problematic substance use (Simons, Dvorak, & Batien, 2008; Wardell, O'Connor, Read, & Colder, 2011) and poor control over behavior motivated by reward (Braddock et al., 2011; Franken & Muris, 2006).

The two remaining systems, the behavioral inhibition system (BIS) and the fight-flight-freezing system (FFFS), are thought to govern responses to aversive stimuli and be engaged during avoidance behavior. The BIS inhibits behavior through increased arousal and risk assessment in response to stimuli signaling the loss of expected reward, uncertainty, and goal conflict. The BIS gives rise to emotional distress and is associated with trait negative affect, especially anxiety (Corr, 2008; Gray, 1982; Smillie, Pickering, & Jackson, 2006). The FFFS mediates responses to cues of punishment and nonreward, is responsible for escape behaviors, and gives rise to the emotions fear and panic. These two systems jointly control avoidance motivation and punishment sensitivity can be conceptualized as hypersensitivity of these two systems combined (Corr, 2004).

Given the primary combined function of these systems is to effectively inhibit behavioral approach to avoid punishment, some researchers have speculated that elevated SP may serve as a protective factor, deterring hazardous use to avoid negative consequences (Franken & Muris, 2006). In keeping with this view, several studies have found an inverse relationship between SP and substance use (Pardo, Aguilar, Molinuevo, & Torrubia, 2007; Simons & Arens, 2007; Wray, Simons, & Dvorak, 2011). Despite this support, SP is also heavily associated with trait negative affectivity (Erdle & Rushton, 2010; Smillie et al., 2006), which is linked to increased substance use and related problems (McCarthy, Curtin, Piper, & Baker, 2010; Simons, Gaher, Oliver, Bush, & Palmer, 2005b; Simons, Wills, & Neal, 2014). Hence, there may be an alternate pathway linking SP to increases in substance use and related problems. That is, because SP is associated with heightened negative affectivity it is possible that SP might lead to problematic substance use through negative reinforcement mechanisms. Indeed, some researchers have found positive relationships between SP and problematic substance use (Knyazev, 2004; Voigt et al., 2009). Taken together, it is clear that these relationships are complex and, as some researchers suggest (Wardell et al., 2011), it may be necessary to examine interactions between SP and SR to appropriately characterize the role of SP in the context of risky substance use.

Traditional views of RST (Gray, 1987) advocate that SR and SP stem from independent brain systems, while suggesting their influence on behavior is the result of a joint effort. This view is outlined by the joint subsystems hypothesis, which contends that these factors can interact to predict behavior and emotional experiences, with the effects being conditional upon each other (Corr, 2004, 2013). In line with this, research has examined the interaction between SR and SP as it relates to substance use. However, this has produced inconsistent findings, with some finding a positive relationship between SP and substance use, which is then amplified by high levels of SR (O'Connor, Stewart, & Marlatt, 2009; Wardell et al., 2011), while others have found a negative effect of SP on substance use, that was attenuated by high level of SR (Simons & Arens, 2007; Simons et al., 2008). As a result, the exact nature of these associations remains unclear. One way of clarifying this may lie in examining how SR and SP might be related to different dimensions of personality. In support of this, the joint subsystems hypothesis further advocates that greater SR and lower SP culminate in maximum poor behavioral control, whereas, the combined strength of SR and SP (with stronger input from SP than SR) gives rise to maximum affective instability (Corr, 2004, 2013). In that regard, SR and SP are associated with characteristic regulatory deficits in emotion and behavior (Braddock et al., 2011; Carlson et al., 2013; Markarian, Pickett, Deveson, & Kanona, 2013; Tull, Gratz, Latzman, Kimbrel, & Lejuez, 2010), which may mediate associations between SP and SR and substance use outcomes.

Affect Lability

Affect lability is a central feature of affect dysregulation (Jahng et al., 2011) and refers to the frequency, speed, and range of changes in affective states an individual experiences (Harvey, Greenberg, & Serper, 1989). This variation is characterized by large, erratic, shifts in feeling states in response to internal or external cues. Individuals high in affect lability are unable to dampen their emotional responses and therefore lack emotional stability. Affect lability is associated with substance use and associated problems (Simons & Carey, 2002, 2006; Wills, Walker, Mendoza, & Ainette, 2006) and often demonstrates unique associations with substance dependence symptoms, independent of use (Simons, Carey, & Wills, 2009a; Simons, Oliver, Gaher, Ebel, & Brummels, 2005c; Simons et al., 2014). Furthermore, affective instability has been shown to be a key risk factor, differentiating problematic use from nonproblem use (Kuvaas, Dvorak, Pearson, Lamis, & Sargent, 2014).

The relationship between affective dysregulation and substance use is complex, with emotional instability appearing to contribute to the development of problematic use in a number of ways. First, affective processing models of negative reinforcement implicate fluctuations in negative affect paired with repeated substance use as the core mechanism behind the development and maintenance of substance use disorders (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; McCarthy et al., 2010). The frequent and intense affective shifts experienced by those with high affect lability may strengthen the conditioned associations between emotional arousal and substance use (Simons et al., 2014). Sudden increases in

emotional arousal have also been shown to impair deliberative control processes and increase the effects of more automatic appetitive processes on behavior (Baker et al., 2004; Lieberman, 2007; Metcalfe & Mischel, 1999). Taken together, affect lability seems to be associated with problematic use patterns, but not more normative consumption (Simons & Carey, 2002; Simons et al., 2009a; Simons, Gaher, Correia, Hansen, & Christopher, 2005a; Simons et al., 2014).

In personality trait models such as the Five Factor Model (Costa & McCrae, 1996), emotional instability (i.e., lability) is subsumed by the trait neuroticism. In RST, neuroticism is posited to result from the joint effects of SP and SR, whereby those with high SP and high SR display the greatest emotional disturbance. That is, high SP is positively associated with emotional symptoms and the magnitude of that relationship (i.e., slope) is greatest in high SR individuals. Whereas, that relationship decreases in strength at lower levels of SR (Corr, 2004; Heym, Ferguson, & Lawrence, 2008). Hence, affect lability may be one mechanism linking SP and SR to substance use outcomes. However, research also identifies SP and SR as underlying characteristic differences in behavioral control or impulsivity (Carlson et al., 2013; Torrubia, Avila, Moltó, & Caseras, 2001), which are also closely tied to substance use behavior (Lejuez et al., 2010; Verdejo-García, Lawrence, & Clark, 2008).

Poor Control

Poor control of behavior (e.g., impulsivity) has been extensively linked to problematic substance use in both human and animal models (Dawe & Loxton, 2004; Lejuez et al., 2010) and is considered an important risk factor in the genesis and course of substance use behavior (Collado, Felton, MacPherson, & Lejuez, 2014; Simons et al., 2009a; Verdejo-García et al., 2008; Wills et al., 2013). Poor behavioral control is characterized by lack of planning and forethought, failure to consider risks, premature responding, inability to delay gratification, and difficulty restricting behavior to those required by situational demands (Daruna & Barnes, 1993; Plutchik & Van Praag, 1995; Wills et al., 2006). Poor control is thought to increase risk for substance use because an individual's behavior is governed by more immediate hot cognition and less by cooler, thoughtful processing of long-term consequences (Carver, 2005; Metcalfe & Mischel, 1999) and substance-related problems ensue as a result of deficits in the capacity to control behavior commensurate with situational demands (Tarter et al., 2003). Poor control is connected to use of a variety of licit and illicit substances (e.g., heroin, alcohol; Dissabandara et al., 2014; Simons & Carey, 2006; Simons et al., 2009a), including marijuana use (Day, Metrik, Spillane, & Kahler, 2013; Simons & Carey, 2002). Altogether, poor control appears to contribute significantly both directly to substance-related problems (Dvorak & Day, 2014; Simons et al., 2009a) and indirectly through its effects on use behavior (Simons et al., 2009a).

In the RST, impulsivity, or poor behavioral control, is posited to arise from the joint effects of SP and SR (Corr, 2004, 2013). Specifically, those with the poor behavioral control (i.e., inhibitory deficits) are characterized by high SR and low SP. This constellation minimizes the reciprocal inhibitory feedback loop between punishment and reward mechanisms and maximizes behavioral disinhibition. Subsequently, as SP raises it attenuates the positive relationship between SR and poor behavioral control, and vice versa (Avila & Parcet, 2001; Corr, 2004; Heym et al., 2008). Hence, poor behavioral control may mediate associations between SP, SR, and substance use behavior, given poor control's role as a propagator of substance use behavior.

The Current Study

In this study, we tested a reinforcement sensitivity model for marijuana use and problems using a 6-month prospective design. The hypothesized model is depicted in Figure 1. SP and SR were hypothesized to be indirectly associated with marijuana use and associated problems via affect lability and poor control. SP and SR were expected to interact in predicting the intermediary constructs of affect lability and poor control. Specifically, the positive association between SR and poor control is expected to be conditional upon levels of SP, such that the association is attenuated at higher levels of SP. Similarly, the positive association between SP and affect lability is expected to be conditional upon levels of SR, such that the association would be potentiated at higher levels of SR. Affect lability at baseline was expected to have direct associations with marijuana problems at Time 1 (T1) and Time 2 (T2). Poor

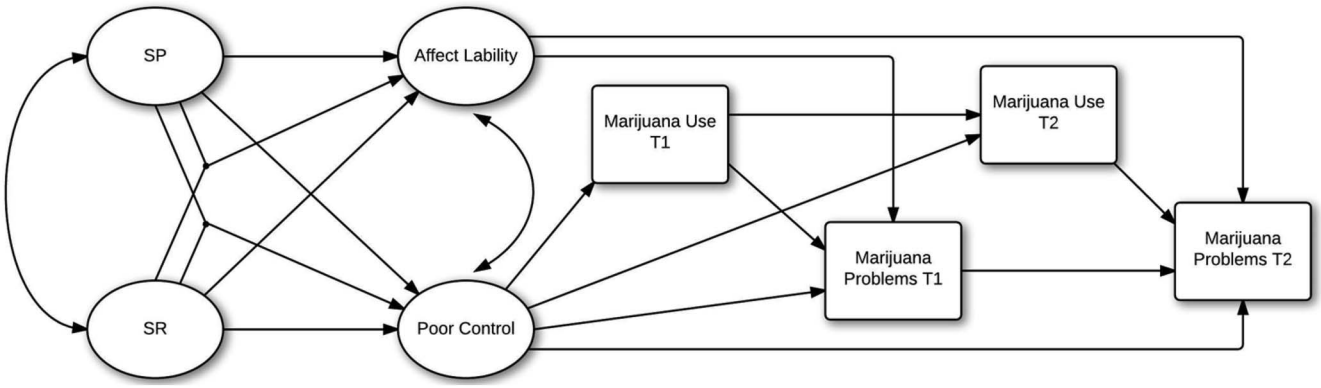

Figure 1. Conceptual model depicting hypothesized relationships. SP = sensitivity to punishment; SR = sensitivity to reward.

behavioral control was hypothesized to have direct effects on both marijuana use and problems at T1 and T2.

Method

Participants

Participant were 2,270 college students (64% female) that ranged in age from 18 to 25 years (M = 19.59, SD = 1.51). Ninety-four percent of the participants were White, 1% Asian, 1% African American, 1% Native American/Alaskan Native, and 3% other or did not wish to respond. Ninety-eight percent were non-Hispanic. Participants were recruited through e-mail and student newspaper advertisements. Of the participants who completed the baseline measures, 87% returned for a 6-month follow-up (T2), and of the returning participants, 94% were successfully matched to their baseline (T1) data. Four articles have been published from this dataset (Gaher, Hahn, Shishido, Simons, & Gaster, 2015; Simons et al., 2008, 2009a; Wray et al., 2011).

Procedure

Participants completed online questionnaires in a private space under the supervision of a research assistant and were provided informed consent. The study was approved by the institutional review board. To ensure participants' anonymity, each participant was assigned a unique code during their completion of the baseline questionnaires. Participants received $20 for completing the baseline assessment and $30 for completing the 6-month follow-up assessment. The average time interval between assessments was 203 days (SD = 50.35).

Measures

Sensitivity to punishment and reward. Sensitivity to punishment and sensitivity to reward were assessed by the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia et al., 2001). The SPSRQ is a 48-item scale with a dichotomous (yes/no) response format. For the current study, the factor solution obtained by O'Connor, Colder, and Hawk (2004) was used. Thus, the sensitivity to reward (SR) scale consisted of 17 items (α = .78; sample item: "Do you often do things to be praised?") and the sensitivity to punishment (SP) scale consisted of 18 items (α = .85; sample item: "Do you often refrain from doing something because of your fear of being embarrassed?"). Construct validity of these scales as indicators of behavioral inhibition and behavioral activation systems is supported by expected relationships with other individual difference measures (e.g., anxiety, impulsivity, neuroticism; Caseras, Àvila, & Torrubia, 2003; Torrubia et al., 2001). In the current model, SP and SR were latent variables with three indicators each, which were parcels formed by the item-to-construct method (Little, Cunningham, Shahar, & Widaman, 2002).

Affect lability. Affect lability was a latent variable measured by three indicators derived from the subscales of the Affective Lability Scale–Short Form (ALS; Oliver & Simons, 2004). Items are rated on a 4-point scale (1 = *very undescriptive* and 4 = *very descriptive*; sample item: "I switch back and forth between being extremely energetic and having so little energy that it's a huge effort just to get where I am going"). The three subscales assess affective lability in respect to depression or elation (eight items, α = .87), anxiety or depression (five items, α = .86), and anger (five items, α = .83).

Poor control. Poor control was a latent variable defined by three indicators. First, seven items from the Eysenck Impulsivity Scale (I7; Eysenck, Pearson, Easting, & Allsopp, 1985) assessed difficulty in controlling behavior (α = .75; sample item: "Do you generally do and say things without stopping to think?"). Second, we derived two scales from a measure of self-regulation (Kendall & Williams, 1982): a three-item scale representing impatience (α = .52; sample item: "I have to have everything right away") and a six-item scale representing distractibility (α = .81; sample item: "I like to switch from one thing to another"). These items were rated on a 5-point scale (1 = *not at all true of me* and 5 = *very true of me*). Previous research has demonstrated that these measures of impulsivity, impatience, and distractibility form a replicable subscale structure and load significantly on a factor of poor control (Wills et al., 2001; Wills, Sandy, & Yaeger, 2002).

Marijuana use. Marijuana use was an observed variable assessed by a 1-week grid with four 6-hr periods per day (Williams, Adams, Stephens, & Roffman, 2000). Participants indicated the number of time periods where they consumed marijuana in a typical week in the past 6 months. This measure of marijuana use has demonstrated good criterion validity in previous research (Simons & Carey, 2006; Simons et al., 2005a) and moderate relations with *DSM–IV* marijuana abuse and dependence (Williams et al., 2000).

Marijuana problems. Marijuana problems was an observed variable assessed by a substance use problems scale (Wills et al., 2002), reworded to focus on marijuana. This 16-item inventory measures marijuana-related problems in the past 6 months. Items were scored to indicate the presence or absence of each item with total scores ranging from 0 – 16. Sample items include: "You worried about your use," "A friend told you to cut down." Validity of the parent measure of substance-related problems is supported by moderate to strong expected associations with level of substance use, use motives, as well as peer and parental substance use levels across multiple samples (Wills, Pokhrel, Morehouse, & Fenster, 2011; Wills et al., 2002).

Analysis Plan

Structural equation models were estimated in Mplus 7 (Muthén & Muthén, 2013) using maximum likelihood robust estimation. We utilized a model building approach (i.e., increasing model complexity in steps) to sequentially examine the hypotheses and create a parsimonious model (see results). SR, SP, affect lability, and poor control were specified as continuous latent variables. Given that only 30% of participants reported marijuana use at T1 and just 23% at T2, use and problem variables were expected to have an "excess" of zeroes. Thus, marijuana use and problems were specified as observed variables with zero-inflated negative binomial (ZINB) distributions (Simons, Neal, & Gaher, 2006; Simons et al., 2014). The ZINB reference distribution is ideal for count outcomes that are positively skewed and include excessive zeros. The ZINB model includes two parts; the zero-inflated portion of the model, which determines the probability that an observation is "always" zero (i.e., in excess of what would be expected

from the negative binomial distribution), and a count portion, which models the count of the outcome conditional upon the likelihood of the observation being potentially nonzero (Cameron & Trivedi, 1998; Collins & Spelman, 2013). Gender was included as a covariate. As recommended by Kline (2011), a two-step approach was utilized, such that the measurement model was examined followed by the structural model. Guidelines for what constitutes a good fit vary, although a comparative fit index (CFI) $\geq$.95 is thought to represent very good fit (Hu & Bentler, 1999; Kline, 2011), and root mean square error of approximation (RMSEA) $<$ .06 as well as standardized root-mean-square residual (SRMR) values of .08 or lower are thought to indicate a close fit (Hu & Bentler, 1999). Chi-square values that are closer to zero and not significant are suggestive of good fit (Kline, 2011). However, in models that require numeric integration (such as ours), traditional model fit indices used to interpret the fit of structural equation models (e.g., CFI, RMSEA, SRMR, and χ^2) have not been developed. Instead, an alternative method for assessing the fit of each model needs to be employed (Klein & Moosbrugger, 2000; Maslowsky, Jager, & Hemken, 2015), whereby nested models are compared using a likelihood ratio test. This is operationalized as the relative fit of the base model and the expanded alternative model.

Results

Descriptive Statistics

One participant was excluded from the analysis because the majority of their data were missing. The analysis sample consisted of 2,269 individuals. Descriptive statistics for observed variables are in Table 1. At T1, participants reported using marijuana in an average of 1.68 (SD = 4.25) 6-hr time periods per week in the past 6 months, with approximately 30% of the total sample reported using marijuana at least once per week and approximately 23% reported at least one marijuana-related problem during that time. At T2, participants reported using marijuana an average of 1.23 (SD = 3.76) 6-hr time periods per week in the previous 6 months. At follow-up, approximately 23% endorsed using in at least once per week and approximately 16% reported having at least one marijuana-related problem in the 6 months since the baseline. Marijuana use and problems at T1 were highly correlated, r = .67, $p < .001$, as were use and problems at T2, r = .67, $p < .001$. Marijuana use and associated problems were moderately stable over time with correlations between the T1 and T2 assessments of use, r = .59, $p < .001$, and problems, r = .52, $p < .001$, respectively (see Table 2).

Measurement Model

An initial measurement model was estimated using maximum likelihood robust estimation that had latent factors for SP, SR, affect lability, and poor control. Gender was included as a covariate with paths to each factor. The initial measurement model was an adequate fit to the data χ^2 (56, N = 2,269) = 782.02, $p < .001$; RMSEA = .076 90% CI [.071, .080]; CFI = .93; standardized SRMR = .048. The modification indices were examined to evaluate whether to free additional constrained parameters in the model. Parameters were sequentially freed and the model reestimated. This process resulted in four modifications to the model: (a) measurement errors of the depression-elation indicator and the distractibility indicator were allowed to covary; (b) the measurement errors for SP-Parcel 3 and SR-Parcel 2 were allowed to covary; (c) a path from gender to the impatience indicator was freed; and (d) the measurement errors of SP-Parcel 3 and SR-Parcel 1 allowed to covary. Each of the freed parameters had a modification index greater than 29.00. Freeing these parameters resulted in an improved final model that was a good fit to the data χ^2 (52, N = 2,269) = 517.56, $p < .001$; RMSEA = .063 90% CI [.058, .068]; CFI = .95; SRMR = .042. Standardized factor loadings in the final model ranged from .62 to .87. Standardized correlations between latent variables were all significant ($p <$ .000) and positive, ranging from .18 to .60.

Table 1
Descriptive Statistics of Observed Variables

Variables	*M* (*SD*)	Median	Range	Skew	Kurtosis
ALS Anxiety-depression	8.73 (3.61)	8	5–20	.87	2.97
ALS Depression-elation	16.61 (5.30)	17	8–32	.28	2.50
ALS Anger	7.94 (3.22)	7	5–20	1.26	4.15
PC Impatience	5.96 (2.14)	6	3–15	.82	3.61
PC Distractibility	15.14 (4.85)	15	6–30	.35	2.65
PC Impulsivity	2.63 (2.12)	2	0–7	.44	2.06
SR Parcel 1	2.47 (1.39)	3	0–5	−.05	2.20
SR Parcel 2	3.40 (1.54)	3	0–6	−.18	2.24
SR Parcel 3	2.68 (1.54)	3	0–6	.09	2.27
SP Parcel 1	2.87 (1.71)	3	0–6	.09	2.02
SP Parcel 2	3.35 (1.72)	3	0–6	−.21	2.11
SP Parcel 3	2.52 (1.79)	2	0–6	.23	2.03
T1 Marijuana use	1.68 (4.25)	0	0–28	3.63	17.22
T1 Marijuana problems	.72 (1.75)	0	0–14	3.26	15.29
T2 Marijuana use	1.23 (3.76)	0	0–28	4.53	25.84
T2 Marijuana problems	.46 (1.43)	0	0–16	4.57	30.59

Note. N = 2,270. ALS = Affect Lability; PC = Poor Control; SR = Sensitivity to Reward; SP = Sensitivity to Punishment.

Table 2
Observed Variable Correlation Matrix

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Gender																	
2. SR Parcel 1	.20$^{\delta}$																
3. SR Parcel 2	.17$^{\delta}$	.55$^{\delta}$															
4. SR Parcel 3	.19$^{\delta}$	.57$^{\delta}$	.55$^{\delta}$														
5. SP Parcel 1	−.14$^{\delta}$	.09$^{\delta}$	.09$^{\delta}$	.16$^{\delta}$													
6. SP Parcel 2	−.17$^{\delta}$	−.01	.01	.07$^{\delta}$	.61$^{\delta}$												
7. SP Parcel 3	−.10$^{\delta}$	−.12$^{\delta}$	−.06†	−.02	.59$^{\delta}$	.65$^{\delta}$											
8. ALS Anxiety-depression	−.10$^{\delta}$	.07$^{\delta}$	.05*	.14$^{\delta}$	.43$^{\delta}$	.38$^{\delta}$	.32$^{\delta}$										
9. ALS Depression-elation	−.08$^{\delta}$	.15$^{\delta}$	.12$^{\delta}$	.22$^{\delta}$	.34$^{\delta}$	.31$^{\delta}$	.22$^{\delta}$	.63$^{\delta}$									
10. ALS Anger	−.01	.15$^{\delta}$	.18$^{\delta}$	.20$^{\delta}$	.26$^{\delta}$	.22$^{\delta}$	.19$^{\delta}$	.58$^{\delta}$	.52$^{\delta}$								
11. PC Impulsivity	.02	.30$^{\delta}$	.28$^{\delta}$	.34$^{\delta}$	.19$^{\delta}$	.12$^{\delta}$	.05†	.25$^{\delta}$	.33$^{\delta}$	.32$^{\delta}$							
12. PC Distractibility	.04	.19$^{\delta}$	.15$^{\delta}$	.27$^{\delta}$	.28$^{\delta}$	.28$^{\delta}$	.19$^{\delta}$	.32$^{\delta}$	.49$^{\delta}$	.30$^{\delta}$	.42$^{\delta}$						
13. PC Impatience	−.11$^{\delta}$	.23$^{\delta}$	.19$^{\delta}$	.25$^{\delta}$	.21$^{\delta}$	.15$^{\delta}$	.11$^{\delta}$	.29$^{\delta}$	.31$^{\delta}$	.29$^{\delta}$	.38$^{\delta}$	.47$^{\delta}$					
14. T1 Marijuana use	.15$^{\delta}$	.11$^{\delta}$	.08$^{\delta}$	.12$^{\delta}$	−.04*	−.04	−.05*	.07†	.08$^{\delta}$	.08$^{\delta}$	.14$^{\delta}$	.11$^{\delta}$	.05*				
15. T2 Marijuana use	.12$^{\delta}$	.07†	.06†	.07$^{\delta}$	−.02	−.01	−.03	.07†	.08$^{\delta}$	.08$^{\delta}$	.09$^{\delta}$	.08$^{\delta}$	.01	.59$^{\delta}$			
16. T1 Marijuana problems	.13$^{\delta}$	.16$^{\delta}$	.12$^{\delta}$	.16$^{\delta}$	.02	.01	−.02	.13$^{\delta}$	.16$^{\delta}$	.14$^{\delta}$	.19$^{\delta}$	.17$^{\delta}$	.13$^{\delta}$	.67$^{\delta}$	.45$^{\delta}$		
17. T2 Marijuana problems	.10$^{\delta}$	.10$^{\delta}$	.07$^{\delta}$	.09$^{\delta}$	−.01	.01	−.03	.11$^{\delta}$	.09$^{\delta}$	.09$^{\delta}$	.11$^{\delta}$	.11$^{\delta}$	.07$^{\delta}$	.45$^{\delta}$	.67$^{\delta}$	.52$^{\delta}$	

Note. N = 2,270. ALS = Affect Lability; PC = Poor Control; SR = Sensitivity to Reward; SP = Sensitivity to Punishment.
$^{\delta}p < .001$. $^{\dagger}p < .01$. $^{*}p < .05$.

Structural Model

Given of the complexity of the analysis, we used a model-building strategy designed to minimize Type I error and to develop a parsimonious model. At each step, joint tests of conceptually related effects (i.e., a test of all effects being zero) were conducted and likelihood ratio tests were examined to determine whether to add the hypothesized effects. Table 3 reports the result of the model building steps, linking each step to the study hypotheses. The final model is depicted in Figure 2 along with the resulting parameter estimates.

First, a latent variable structural model was estimated with gender as a covariate. This model included paths from SR and SP to affect lability and poor control, respectively, as well as a correlation between SR and SP. This model fit the data well, χ^2 (52, N = 2,269) = 517.56, $p < .001$; RMSEA = .063 90% CI [.058, .068]; CFI = .95; SRMR = .042. Next, a SP × SR latent variable interaction was introduced to the model with paths to both affect lability ($b = 0.03$, $p = .057$) and poor control ($b = 0.02$, $p = .468$). This addition did not significantly improve the model $\Delta\chi^2$ (2, N = 2,269) = 3.70, $p = .157$, and thus the interaction effects were removed.

Next, the observed marijuana outcomes (i.e., marijuana use and marijuana problems at both T1 and T2) were introduced to the previously fit latent variable model and a new structural model was

Table 3
Summary of Model Building Steps

Model	Model test	Comparison model	Parameters	Log-likelihood	AIC BIC	χ^2 Difference	p
Hypothesized model	Base model		78	−56191.39	112538.39 112985.69		
	Step 2. Test direct effects of PC to T2 use	Base	80	−56182.934	112525.94 112984.04	$\chi^2(2) = 15.59$	.000
	Step 3. Test direct effects of PC and ALS on T2 problems	Step 2	84	−56178.32	112524.65 113005.72	$\chi^2(4) = 6.64$	.156
Alternative model with direct effects	Step 4. Test direct effects of SR, SP, and SP × SR on T1 use	Step 2	86	−56154.41	112480.82 112973.35	$\chi^2(6) = 60.28$	.000
	Step 5. Test direct effects of SR, SP, and SP × SR on T1 problems	Step 4	92	−56146.26	112476.53 113003.42	$\chi^2(6) = 16.16$	.013
	Step 5.1. Test direct effects of SR and SP on T1 problems inflation	Step 4	88	−56147.85	112471.70 112975.68	$\chi^2(2) = 14.79$	.001
	Step 6. Test prospective direct effects of SR, SP, and SR × SP on T2 use	Step 5.1	94	−56143.56	112475.12 113013.47	$\chi^2(6) = 9.21$	.162
	Step 6.1. Test prospective direct effects of SR and SP on T2 use	Step 5.1	92	−56144.12	112472.24 112999.13	$\chi^2(4) = 7.289$	.121
	Step 7. Test prospective direct effects of SR, SP, and SR × SP on T2 problems	Step 5.1	94	−56138.16	112464.32 113002.67	$\chi^2(6) = 13.98$	.029
	Step 7.1. Test prospective direct effects of SR and SP on T2 problems	Step 5.1	92	−56139.17	112462.35 112989.24	$\chi^2(4) = 11.80$	.019

Note. SP = Sensitivity to Punishment; SR = Sensitivity to Reward; ALS = Affect Lability; PC = Poor Control.

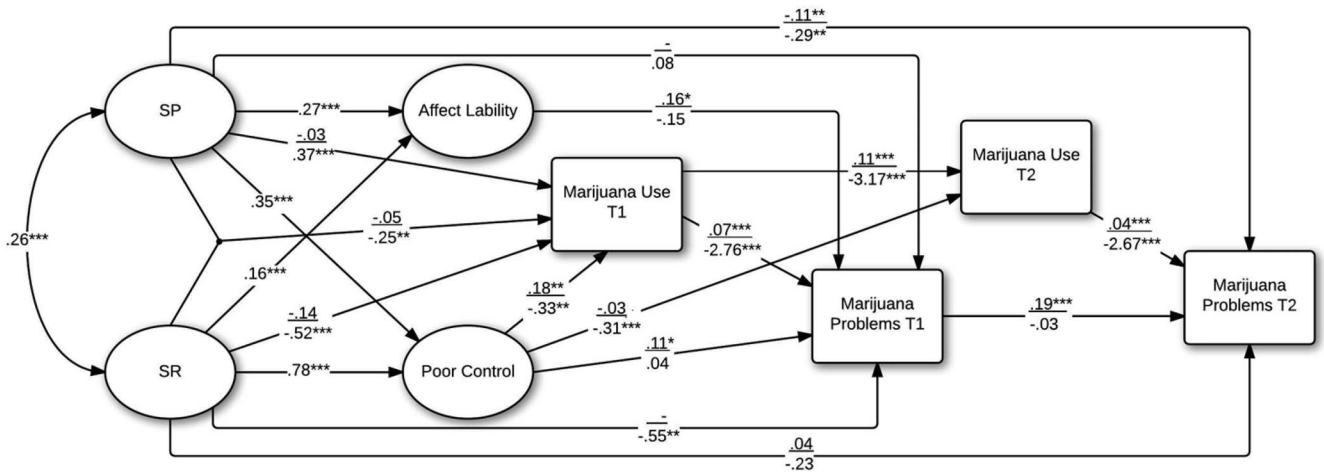

Figure 2. Final structural model. SP = sensitivity to punishment; SR = sensitivity to reward. All values are unstandardized coefficients. $N = 2{,}269$ persons. Marijuana use and problems coefficients above the horizontal line represent the count portion and below represent zero-inflation portion (e.g., abstaining). Gender was a covariate to SR, SP, Affect Lability, Poor Control, and Marijuana Use T1, but was omitted for clarity. $^{*}\ p < .05$. $^{**}\ p < .01$. $^{***}\ p < .001$.

estimated (Step 1). The observed marijuana outcomes were each specified as a count with a ZINB distribution. This new base model included cross-sectional direct paths from poor control to T1 use and problems and from affect lability to T1 problems as well as prospective direct effects of T1 marijuana use on T2 use and problems and prospective direct effect of T1 problems on T2 problems. Lastly, gender had direct paths to each of the substance use outcomes. Results from the base model indicated that gender was not associated with either substance use outcome at T2 nor was it significantly associated with T1 problems. These nonsignificant gender effects were subsequently dropped for a more parsimonious model. In Step 2, we tested the prospective effects of poor control on T2 marijuana use. This was a significant addition to the model, and thus, was retained (see Table 3). Step 3 tested the prospected effects of affect lability and poor control on T2 marijuana problems. This did not result in a significant addition and was dropped from the model.

Alternative model. The model estimation thus far tested the significance of expected effects and found partial support for hypothesized paths. In addition to the estimated paths, the hypothesized model (see Figure 1) was such that the effects of SR and SP were entirely indirect through affect lability and poor control. That is, the model specifies that direct effects of SP and SR on the marijuana outcomes are nonsignificant (i.e., zero). The following section tests whether this hypothesized structure is tenable by estimating a series of alternative models that test whether direct effects of SP and SR on the marijuana outcomes are nonsignificant.

In Step 4, we added directs effects of SR, SP, and SP × SR latent variable interaction on T1 use. This was a significant addition to the model and was retained (see Table 3). The SP × SR interaction was significant in predicting the inflation ($b = -0.25$, $p = .002$) but not count ($b = 0.05$, $p = .196$) outcome. The interaction is depicted in Figure 3. Consistent with research on this and other samples (Simons & Arens, 2007; Simons et al., 2008), SP had a conditional direct effect on the likelihood of being abstinent at T1. At low levels of SR (−1 *SD* below the mean) SP was associated with an increased likelihood of abstaining ($b = 0.89$, $p > .001$) this effect was attenuated as levels of SR increased (+1 *SD* above the mean; $b = 0.19$, $p = .198$). In Step 5, the effects of SP, SR, and SP × SR interaction on T1 problems were tested and represented a significant addition to the model. Yet, the SP × SR interaction effect was not significant for either the inflation ($b = -0.08$, $p = .406$) or count portions ($b = 0.04$, $p = .232$). Hence, the interaction was dropped and Step 5 reestimated. The joint test of the main effects on the outcomes was significant $^{\Delta}\chi^2$ $(4, N = 2{,}269) = 12.88$, $p = .012$. However, the inclusion of the effects of SR and SP resulted in all of the effects on the count portion (i.e., affect lability, poor control, SR, and SP) to be nonsignificant. Thus, to have a more interpretable and parsimonious model, the effects of SR and SP were retained for the inflation portion, but dropped from the count portion (see Step 5.1 in Table 3). Step 6 tested the prospective direct effects of SR, SP, and SR × SP interaction on T2 use. The step did not significantly improve the model, nor did addition of the main effects without the interaction terms (see Step 6.1 in Table 3). Hence, no additional paths to T2 use were retained. Lastly, Step 7 tested the prospective direct effects of SR, SP, and SR × SP interaction on T2 problems. This resulted in a significant improvement in the overall model, but the interaction effects were not significant: inflation ($b = 0.12$, $p = .373$); count ($b = 0.05$, $p = .354$). Thus, for parsimony, the interaction effects were dropped and the Step reestimated. This resulted in retaining direct paths from SP and SR to T2 problems (see Step 7.1 in Table 3). Finally, results of the log-likelihood ratio test for the final model (Step 7.1; AIC = 112538.39, BIC = 112985.69) versus the base model (AIC = 112462.32, BIC = 112989.24) favored the final model $^{\Delta}\chi^2$ $(14, N = 2{,}269) = 94.27$, $p > .000$.

Structural model summary. The final model estimates are presented in Figure 2. Importantly, gender was included as a

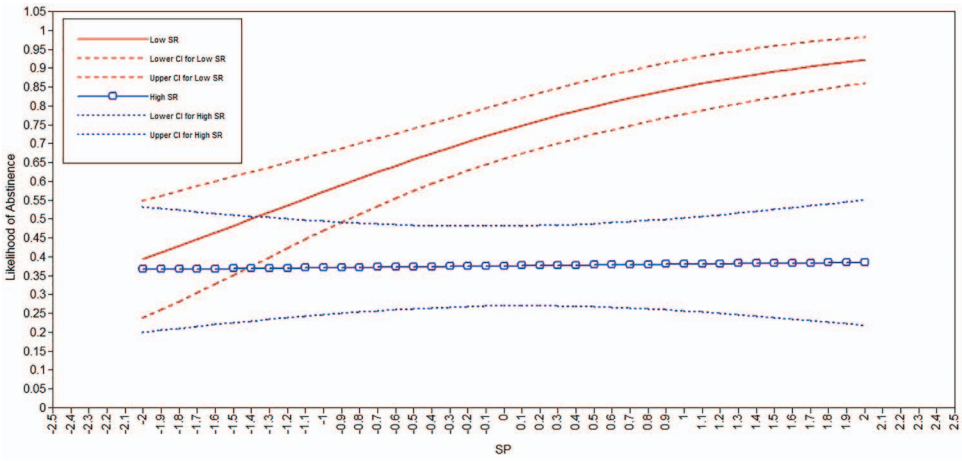

Figure 3. Direct effects of sensitivity to punishment on the likelihood of being abstinent at Time 1 as a function of sensitivity to reward. Low SR = sensitivity to reward −1 *SD*. High SR = sensitivity to reward +1 *SD*. Dashed lines represent 95% confidence intervals. See the online article for the color version of this figure.

covariate in the final model, however, it was omitted from the figure for clarity. Thus, those effects are listed here. Female gender was associated with SP ($b = -0.54$, $p > .001$), affect lability ($b = -0.10$, $p = .009$), and the likelihood of being a abstinent at T1 (i.e., T1 marijuana use inflation; $b = -0.47$, $p = .018$); whereas male gender was associated with SR ($b = 0.54$, $p > .001$) and the intensity of marijuana use at T1 (i.e., T1 marijuana use count; $b = 0.38$, $p = .001$). Gender was not associated with poor control ($b = -0.05$, $p = .544$). Also omitted for clarity from the figure of the final model was the correlation between the disturbance terms of affect lability and poor control. These were significantly related ($b = 0.40$, $p > .001$), indicating that these constructs have some shared sources of residual variance.

Indirect effects. The zero-inflated variables cannot be included as mediators in traditional tests of indirect effects. Hence, we provide tests of indirect effects that do not include marijuana use or problems as a mediator (see Table 4) using bias-corrected bootstrapped confidence intervals (MacKinnon, Lockwood, &

Table 4
Unstandardized Indirect Effects for Exogenous Variables Predicting Marijuana Outcomes

Variables/path	*b*	*SE*	95% CI
Sensitivity to punishment (SP)			
SP → ALS → T1 Problem Intensity	.06	.03	[.006, .129]
SP → PC → T1 Likelihood of Abstaining	−.17	.05	[−.288, −.072]
SP → PC → T1 Use Intensity	.09	.03	[.033, .151]
SP → PC → T1 Problem Intensity	.06	.02	[.015, .098]
SP → PC → T2 Likelihood of Abstaining	−.16	.05	[−.260, −.066]
SP → PC → T2 Use Intensity	−.01	.02	[−.053, .032]
Sensitivity to Reward (SR)			
SR → ALS → T1 Problem Intensity	.02	.01	[.003, .054]
SR → PC → T1 Likelihood of Abstaining	−.25	.07	[−.418, −.117]
SR → PC → T1 Use Intensity	.13	.04	[.048, .226]
SR → PC → T1 Problem Intensity	.08	.03	[.024, .148]
SR → PC → T2 Likelihood of Abstaining	−.23	.07	[−.369, −.081]
SR → PC → T2 Use Intensity	−.02	.03	[−.083, .043]
Total Effects			
SP → T1 Likelihood of Abstaining	.37	.10	[.190, .608]
SP → T1 Use Intensity	.06	.07	[−.113, .183]
SR → T1 Likelihood of Abstaining	−.76	.12	[−1.082, −.552]
SR → T1 Use Intensity	.00	.07	[−.153, .158]

Note. ALS = Affect Lability; PC = Poor Control; SR = Sensitivity to Reward; SP = Sensitivity to Punishment; Intensity = Count Portion of Zero-Inflated Negative Binomial (ZINB) Count Model; Likelihood = Inflation Portion of ZINB Count Model. The inflation portion of the ZINB count model reflects the likelihood of obtaining a zero (i.e., abstaining or having no problems, respectively). Therefore, a positive value represents an increased likelihood of obtaining a zero and a negative value indicates a decrease likelihood of obtaining a zero value (i.e. using). Indirect and total effects were calculated using bias corrected bootstrapped confidence intervals. Significance is represented by a confidence interval that does not contain zero. Total effect represent the effect of one exogenous variable at the mean level of the other.

Williams, 2004). As hypothesized, SR and SP demonstrated indirect effects on marijuana use and problems via affect lability and poor control. For example, SP was indirectly associated with a decreased likelihood of being abstinent (i.e., inflation) and increased use intensity (i.e., count) at T1 via poor control. However, the total effect of SP was significantly associated with an increased likelihood of being abstinent at T1. Similarly, SP was indirectly related with increased problem intensity at T1 through affect lability and poor control. SR exhibited indirect associations with a decreased likelihood of being abstinent, increased use intensity, and increased problem intensity at T1 via poor control. The indirect relationship between SR and T1 problem intensity via affect lability was also significant. As for T2 use, both SP and SR showed significant prospective indirect associations with a decreased likelihood of being abstinent at T2 through poor control. Whereas, the prospective indirect effects of SP and SR on use intensity at T2 via poor control were not significant.

Several total effects could not be estimated, given some paths had mediators that were zero-inflated. Therefore, in order to gain a sense of the overall pattern of the effects of SP and SR on the primary outcomes (i.e., marijuana use, marijuana problems) a series of *t* tests were conducted. We first compared levels of SP and SR between marijuana users (past 6 month use at baseline or follow-up, $n = 799$) and nonusers (no past 6-month use at baseline or follow-up, $n = 1471$). Those who used marijuana showed significantly lower levels of SP ($M = 8.20$, $SD = 4.53$) than those who did not ($M = 9.05$, $SD = 4.47$), $t(1618.84) = 4.31$, $p < .001$, $d = -0.19$. In contrast, SR was higher among marijuana users than nonusers ($M = 9.65$, $SD = 3.67$ vs. $M = 7.96$, $SD = 3.64$), $t(1629.73) = -10.53$, $p < .001$, $d = 0.46$. We next examined levels of SP and SR as a function of presence or absence of marijuana-related problems at T2. SP among those with problems ($n = 1917$, $M = 8.75$, $SD = 4.37$) did not significantly differ than those without problems ($n = 352$, $M = 8.75$, $SD = 4.53$), $t(499.50) = -0.002$, $p = .99$. However, those with problems had higher levels of SR ($M = 9.66$, $SD = 3.76$) than those who did not experience problems ($M = 8.35$, $SD = 3.69$), $t(481.72) = -6.00$, $p < .001$, $d = 0.35$. Finally, we compared levels of SP and SR among marijuana users who reported marijuana related problems at follow-up ($n = 323$), versus those who did not ($n = 475$). Those reporting marijuana problems ($M = 8.74$, $SD = 4.36$) displayed higher levels of SP than those without problems ($M = 7.83$, $SD = 4.60$), $t(714.58) = -2.84$, $p < .001$, $d = 0.20$; while those with problems ($M = 9.81$, $SD = 3.77$) did not significantly differ on levels of SR than those without problems ($M = 9.55$, $SD = 3.59$), $t(669.65) = -0.96$, $p = .34$.

Discussion

The purpose of this prospective study was to test a model of associations between individual differences in reinforcement sensitivity, affective and behavioral dysregulation, and marijuana outcomes at two time points in young adult college students. The effects of sensitivity to punishment and reward were expected to be indirect via affect lability and poor control. We also examined a series of theoretically based interaction effects to test conditional relationships. The analytic approach distinguished between variables that predicted the likelihood of using marijuana or having marijuana problems versus not and variables that predicted the amount (or intensity) of marijuana use or problems, respectively, when a person had experienced one of these outcomes. In general, there was support for the hypothesized indirect effects, but little evidence of interaction effects. In the following sections, we discuss the findings in greater detail.

Effects of Sensitivity to Punishment and Reward on Dysregulated Affect and Behavior

A major aim of this study was to examine if the effects of SP and SR on dysregulated affect and behavior were consistent with the joint subsystems hypothesis (Corr, 2004, 2013). In short, the joint subsystems hypothesis states the neuroticism and extraversion are produced by the joint effects of punishment and reward sensitivity. Accordingly, SP and SR should interact to produce the turbulent affective experience characterized by those high in neuroticism and the poor behavioral control seen in those high in extraversion. In the case of affect lability, it was hypothesized that the positive association between SP and affect lability would be potentiated at higher levels of SR. This prediction is grounded in evidence showing that high SR is associated with increased positive affect whereas high SP is related to increased negative affect (e.g., O'Connor et al., 2004). Thus, those individuals high in both SP and SR would likely experience competing valenced emotions, effectively promoting large and erratic vacillations from positive to negative feelings states. Yet, despite this strong rationale, the interaction between SP and SR failed to significantly predict affect lability. Instead, both SP and SR exhibited positive direct effects on affect lability. The pattern is consistent with an alternative formulation of the joint subsystems hypothesis that predicts two main effects, with SP having greater magnitude than SR (Corr, 2013). In these data, both SP and SR exhibited positive direct effects on affect lability in the appropriate magnitude cited by the joint subsystems hypothesis (i.e., SP > SR). Hence, SP and SR have additive rather than multiplicative effects on lability resulting in large, erratic, shifts in feeling states.

Similarly, the SP × SR interaction was not significantly associated with poor control. This finding was unexpected, given that the joint subsystems hypothesis clearly states that impulsivity should arise from an interaction between SP and SR, where the positive relationship between SR and poor control is maximized at low levels of SP (Corr, 2004; Simons et al., 2008). Despite this, we again found evidence of two main effects. SR exhibited a positive direct effect on poor control, as hypothesized. However, contrary to expectation, SP demonstrated a positive direct effect on poor control as opposed to the anticipated inverse relationship. This unexpected finding is inconsistent with the larger literature showing inverse associations between SP and disinhibition (e.g., Carlson et al., 2013; Torrubia et al., 2001) and it implies being sensitive to punishment in some way gives rise to poorly controlled behavior. There are several potential explanations for this finding. For example, this could reflect the positive relationship between neuroticism and impulsivity (e.g., Eysenck & Eysenck, 1977; Valero et al., 2014). Indeed, the construct *negative urgency* is characterized by high neuroticism and emotionally driven impulsive behavior, thus, integrating negative emotionality (i.e., neuroticism) and reckless action (i.e., impulsivity) into a single construct (Settles et al., 2012; Whiteside & Lynam, 2001). Therefore,

this finding may reflect negative emotionally driven impulsive behavior related to avoidance.

Sensitivity to Reward and Marijuana-Related Outcomes

The pattern of relationships found here consistently indicated that SR was associated with increased risk for marijuana use and problems. This is in line with mounting evidence suggesting that SR is a risk factor for substance use and associated problems (e.g., O'Connor & Colder, 2005; Pardo et al., 2007; Simons et al., 2009b). However, this study extends previous research by explicating the intermediate regulatory constructs by which being high in SR leads to increased marijuana use and problems. Importantly, this was the case for dysregulated affect and behavior as SR exhibited positive indirect effects on the marijuana-related outcomes through both affect lability and poor control.

The indirect effects of SR through poor control were consistent with the hypothesized model where poor control was expected to mediate associations between SR and substance use behavior, given poor control's role as a propagator of substance use behavior (Day et al., 2013; Simons & Carey, 2002). Specifically, SR exhibited an inverse indirect effect via poor control on the likelihood of being abstinent at T1 and T2. Similarly, SR demonstrated positive indirect effects through poor control on use and problem intensity at T1. These results are theoretically consistent with RST and makes sense in the context of a positive reinforcement pathway to increased marijuana use and problems. Individuals high in SR display a sensitivity to positive incentives that increase the propensity for approach behavior and decrease inhibitory control of behaviors motivated by reward (Corr, 2004; Franken & Muris, 2006) which, at least in part, places them at risk for learning the positive reinforcing effects of marijuana, effectively increasing the likelihood and intensity of use. This is consistent with the contemporary view of the central importance of reward neural circuitry in addictive behaviors (Koob & Volkow, 2010) and with studies suggesting that increased motivation for reward underlies impulsive behavior leading to increased risky health behavior (Braddock et al., 2011). In contrast to poor control, affect lability had significant direct paths to problem intensity, but not use. Interestingly, SR also exhibited direct effects on both use and problems, indicating that the sensitivity to positive incentives contributes to marijuana-related outcomes above and beyond its effects via dysregulated behavioral and affective control.

It is important to note that recent research suggests that the BAS construct is multidimensional with factors representing various processes involved in identifying and attaining reinforcers (Krupić, Corr, Ručević, Križanić, & Gračanin, 2016a; Krupić, Gračanin, & Corr, 2016b). These individual processes reflect differences in identification of reinforcers, planning behavior, execution of behaviors designed to obtain reinforcers, and the emotional reactions to receiving reward. SR, as measured here, has been found to load onto a factor related to execution of behavior to obtain reward (Krupić et al., 2016a). For these reasons, future research would be advanced by the inclusion of measures that allow for the BAS to be decomposed to facilitate more granular examination of the individual aspects of BAS functioning on substance use and problems (Corr & Cooper, 2016).

Sensitivity to Punishment and Marijuana-Related Outcomes

In contrast to SR, there have been a series of mixed findings in regard to the associations between SP and substance use, with some studies showing increased risk (O'Connor et al., 2009; Wardell et al., 2011) others showing decreased risk (Simons et al., 2008; Wray et al., 2011), and some finding no relationship (Colder et al., 2013). The results of this study clarify several of the existing discrepancies. The associations between SP and marijuana use and problems were complex. The relationship between SP and the likelihood using marijuana varied as a function of SR, such that the positive relationship between SP and the likelihood of abstaining was attenuated at high levels of SR. This finding is consistent with previous studies using these data (Simons et al., 2008) and others (Simons & Arens, 2007). This suggests that being sensitive to potential aversive consequences is a protective factor, buffering against use. However, as SR rises the protective effect of SP is diminished.

In addition, the effects of SP varied across outcomes within the model, exhibiting both risk protecting and risk promoting pathways. For example, SP was associated with an increased probability of abstaining from marijuana use at T1. In turn, this decreased probability of use at T1 is expected to have an inverse cascading effect on outcomes that come after it in the model contributing to lower overall risk. However, SP exhibited risk promoting effects as well. For example, SP was positively associated with T1 problem intensity via its effect on poor control and affect lability.

Unfortunately, we are unable to calculate the total effects due to distributions of the T1 marijuana use and problem variables. To remedy this, we conducted a series of *t* tests to facilitate interpreting the overall effects of SP. Here we found that the net effect of SP on problems in the full sample was zero, suggesting that the risk promoting and risk protective effects of SP cancel each other out. However, among marijuana users those high in SP were more likely to exhibit problems. Hence, the results of these analyses are consistent with the idea that SP decreases the likelihood initiating marijuana use, but once an individual has initiated use behavior, SP may result in increased risk for problems. This would explain the mixed findings seen across studies as the result would depend on different sample characteristics, the outcomes being modeled, and the interaction with SR. It is important to note that even though the net effect of SP was zero does not mean it is irrelevant. In fact, just the opposite. SP has different effects for different people in different contexts, making it an important factor to consider.

Clinical Implications

The pattern of results highlights that different intervention targets may be most appropriate at different stages of the marijuana use continuum. These findings implicate behavioral dysregulation as a core issue underlying the initiation of use. Preventative interventions, perhaps, may be best served focusing broadly on increased adaptive self-regulation skills, rather than emphasizing negative consequences resulting from use. Individuals most likely to respond to information about potential dangers (i.e., those high in SP) are already less likely to use. The results are consistent with conceptualizations of substance use disorder as manifestations of underlying behavioral and emotional dysregulation. Our findings indicate that affect dysregulation exhibits a direct effect on prob-

lems, over and above use level, suggesting that once use has been initiated those with emotion regulation deficits are more likely to have problems even at lower levels of use. Effective regulation of emotion and behavior is essential for adaptive functioning. Hence, prevention programs that target regulatory deficits have the potential to impact individual well-being in addition to minimizing negative substance use outcomes (Wills, Simons, Manayan, & Robinson, in press). In addition, targeted substance specific interventions may be useful in minimizing harm among those that use marijuana. For example, use of marijuana-related protective behavioral strategies is a robust predictor of lower marijuana use frequency and associated consequences (Bravo, Anthenien, Prince, Pearson, & Marijuana Outcomes Study Team, 2017; Pedersen, Hummer, Rinker, Traylor, & Neighbors, 2016). Interestingly, those who exhibit impulsivity-like traits have been shown to use these protective strategies less (Bravo et al., 2017). Hence, such interventions may benefit those that need it most.

Limitations

There are several limitations of the current study that should be noted. First, there were several potential indirect and total effects that could not be calculated because the mediator was modeled as two-parts (zeros and the count). This makes it difficult to fully characterize the effects of reinforcement sensitivity, affect lability, and poor control on problematic marijuana use. Second, though this is a complex model it is limited in that it does not include additional important familial and environmental factors that are relevant for understanding young adult substance use (cf. Simons et al., 2009a). Third, the current sample focused on college students and consisted largely of white participants. Results should be generalized to other populations with caution. Finally, this model was focused specifically on marijuana use and related problems, and as such, may be limited in its application to other substance use.

Summary

Overall, the current study identified a number of risk and protective pathways by which individual differences in reinforcement sensitivity are associated with corresponding increases or decreases in marijuana use and problems. Sensitivity to punishment and reward did not interact to predict affect lability or poor control as hypothesized, but displayed positive main effects on both affect lability and poor control. These constructs, in turn, exhibited independent positive effects on the intensity of marijuana problems, over and above the effects of use. The pattern of relationships consistently indicated that SR was associated with increased risk for marijuana use and problems: The effects were mediated, in part, by emotional and behavioral dysregulation, and moderated by SP.

In contrast to the consistent risk promoting effects of SR, associations between SP and marijuana use and problems were complex. The inverse association between SP and the likelihood of using marijuana varied as a function of SR, with the competing effects of SR attenuating the association. Once controlling for use level, SP was indirectly associated with increased intensity of problems due to its effects on emotional and behavioral dysregulation. Though speculative, the pattern of findings suggests that SP may reduce likelihood of marijuana involvement yet, due to its associations with dysregulation contribute to more problematic use patterns once use is initiated. These findings highlight the importance of decomposing outcomes into likelihood versus amount of use and problems as they had differential associations with predictors and underscores the importance of affect lability and poor control in predicting problematic marijuana use.

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