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# Dysregulation as a correlate of cannabis use and problem use

**Hector I. Lopez-Vergara**[a], **Kristina M. Jackson**[a], **Lidia Z. Meshesha**[a], **Jane Metrik**[b,a]

[a]Center for Alcohol and Addiction Studies, Brown University School of Public Health, Providence, RI, 02903

[b]Providence VA Medical Center, Providence, RI, 02908

## Abstract

**Objective:** Cannabis users with a dysregulatory risk factor may be particularly vulnerable to engaging in more frequent and problematic cannabis use. Contemporary models of dysregulated behavior suggest that dysregulation emerges due to distinct mechanisms. The current study seeks to examine the dysregulatory correlates of cannabis involvement, including working memory capacity, delay discounting, impulsivity, and reward sensitivity.

**Method:** Participants were 104 non-treatment seeking frequent cannabis users (the average participant used cannabis 71% of the days/past 60 days [*SD*= 22%], with an average of two uses per day [*SD*= 1.2]). Mean age was 21.3 (*SD*= 4.3); 36.5% were female. Working memory was assessed via the Trail-Making Test-B and the Digit Span subtests of the WAIS-III, delay discounting was assessed via a computer-based task, trait impulsivity was self-reported via the Barratt Impulsiveness Scale, and reward sensitivity was self-reported via the Reward Dependence Scale and the Snaith-Hamilton Pleasure Scale.

**Results:** Structural equation modeling estimated the associations between different facets of dysregulation and cannabis involvement. Results suggest that poor working memory capacity and high trait impulsivity were associated with both use and problem use. Greater delay discounting was associated with problem use, but not with frequency of use. Low reward sensitivity was associated with more frequent cannabis use, but not with problem use.

**Conclusions:** Results confirm that the dysregulatory correlates of cannabis involvement consist of multiple dimensions of functioning. Prospective studies that assess the multidimensional structure of dysregulation and cannabis involvement are needed in order to disaggregate the dysregulatory antecedents and consequences of cannabis involvement.

### Keywords

marijuana; cannabis; working memory; delay discounting; impulsivity; reward sensitivity; dysregulation

Correspondence regarding this article should be directed to Jane Metrik, PhD, Center for Alcohol and Addiction Studies, Brown University, Box G-S121-4, Providence, RI 02912. Jane_Metrik@brown.edu.

## Introduction

Almost half of the U.S. population aged 12 or older reports lifetime use of cannabis (Center for Behavioral Health Statistics and Quality, 2016), and rates of use are likely to increase due to trends towards a more permissive legal environment (Hasin, 2018; Tarter, Kirisci, & Reynolds, 2014). However, only a fraction of cannabis users develop problematic forms of use (Zehra et al., 2018). The disproportion of people who experiment with cannabis versus those who develop problematic use indicates that there may be substantial individual differences in the etiology of cannabis-related problems and cannabis use disorder (Blanco, Rafful, Wall, Ridenour, Wang, & Kendler, 2014; Ystrom, Reichborn-Kjennerud, & Kendler, 2014). Examining individual differences in the disposition toward rapid, unplanned reactions to stimuli (i.e., dysregulation) may provide a way to elucidate who is likely to become involved in misuse of cannabis.

Increased levels of dysregulation have been hypothesized to be both an antecedent of cannabis involvement (Vanyukov et al., 2003) as well as a consequence of use (Solowij & Michie, 2006). Although there is no consensus on whether dysregulation is an antecedent or a consequence of use, when investigating the association between cannabis involvement and dysregulation it is important to consider that dysregulation is a highly heterogeneous, or multi-dimensional construct. Contemporary models of dysregulation converge in suggesting that behavior emerges due to distinct mechanisms, including constructs such as delay discounting, impulsivity, sensation seeking, or difficulty in behavior control (Beauchaine, 2015; De Wit, 2009; Nigg, 2017; Zucker, Heitzeg, & Nigg, 2011). Therefore, it is important to examine multiple processes that can explain dysregulation in cannabis users.

In response to the heterogeneous nature of dysregulation, a plethora of “dual-process” or “dual-pathway” models have been proposed (*for reviews see* Carver, Johnson, & Timpano, 2017; Evans & Stanovich, 2013; Shulman et al., 2016). Although there is not a single unitary dual-process model, Kahneman (2002) suggested that these types of models offer a useful heuristic to investigate two forms of information processing that may be relevant to explain human behavior: an executive (or deliberative) process and a reactive (or automatic) process.

### Proposed dysregulatory processes

Psychological processes that have been emphasized in dual-process models of dysregulated behavior (including substance use, psychopathology, and other health behaviors) include working memory capacity (Barrett et al., 2004; Evans, 2003), delay discounting (Schneider & Coulter, 2015; Sonuga-Barke, 2002), impulsivity (Dawe & Loxton, 2004; Steinberg et al., 2008), and sensitivity to reward (Lopez-Vergara & Colder, 2013; Steinberg, 2010).

Working memory reflects the ability to flexibly manipulate activated memory representations, particularly in light of interference or distraction (Baddeley, 2012). Low working memory capacity may influence drug involvement due to limited ability to exert behavioral control in the presence of drug-related cues (Hofmann et al., 2008). Poorer working memory has been shown to be associated with both cannabis use (Becker et

al., 2018; Schweinsburg et al., 2010; Crean, Crane, & Mason, 2011) and cannabis-related problems (Ma et al., 2018; Day et al., 2013).

Delay discounting reflects the tendency to choose immediate smaller rewards over larger delayed rewards (Bickel et al., 2014). Higher discounting of delayed rewards is associated with choice to use an immediately reinforcing drug over an alternative, socially-sanctioned behaviors with deferred rewards (i.e., studying to get a better grade on an exam or working extra hours for a promotion). Studies that have investigated delay discounting and cannabis suggest that greater discounting of delayed rewards is associated with increased cannabis use (Kollins, 2003) and problematic cannabis use (Aston, Metrik, Amlung, Kahler, MacKillop, 2016; Johnson et al., 2010; Strickland, Lile, & Stoops, 2017), but delay discounting does not seem to be acutely affected by delta(9)-tetrahydrocannabinol, the main psychoactive constituent of cannabis (McDonald, Schleifer, Richards, & de Wit, 2003; Metrik et al., 2012).

Trait impulsivity reflects individual differences in the personality disposition to act upon maladaptive behavioral options without considering potential consequences (Reynolds et al., 2006). Impulsivity is a robust correlate of a variety of problem behaviors (de Wit, 2009; Moeller et al., 2001), including both cannabis use (Gruber, et al., 2011; Vangsness et al., 2005) and problem cannabis use (Bidwell et al., 2013; Day et al., 2013; Gunn, Jackson, Borsari, & Metrik, 2018; Keough, Hendershot, Wardell, & Bagby, 2018; Pearson et al., 2018; Simons et al., 2005).

Finally, reward sensitivity reflects individual differences in the reactivity to appetitive stimuli (Corr, 2004). High levels of reward sensitivity have been posited to bias attention towards the potentially reinforcing properties of drug use, hence increasing probability for drug use (Lopez-Vergara et al., 2012). There is evidence that hypersensitivity to reward may be a risk factor for cannabis involvement. Increased cannabis involvement has been shown to be associated with hypersensitivity to reward (Emery & Simons, 2017; Papinczak, Connor, Harnett, & Gullo, 2018; Scalco & Colder, 2017; Simons & Arens, 2007), and Franken and Muris (2006) have shown that sensitivity to reward positively predicts a composite illicit drug use score that predominantly consisted of cannabis use.

There is theoretical clarity indicating that individual differences in working memory capacity represent a component of the “top-down,” deliberative system (Friedman & Miyake, 2017), whereas individual differences in reward sensitivity represents a phylogenetically ancient component of the “bottom-up,” reflexive system (Montag & Panksepp, 2017). However, delay discounting can be conceptualized as arising from both deliberative and/or reflexive systems (McClure & Bickel, 2014), as can impulsivity (Dalley & Robbins, 2017; Gullo, Loxton, & Dawe, 2014). Hence, it remains difficult to resolve whether delay discounting and impulsivity constitute deliberative and/or reactive processes. The goal of the current study is to investigate whether a relatively broad range of dysregulatory variables statistically overlap with cannabis involvement in one multivariate model that allows for comparison of effect sizes across dysregulatory variables.

### Current Study

This study draws from previous theoretical and empirical applications of dual-process models to investigate the association between cannabis use, problem cannabis use, and the following facets of liability for dysregulation: working memory capacity, delay discounting, impulsivity, and reward sensitivity. Previously, we have examined the associations of working memory and trait impulsivity with cannabis-related problems (Day et al., 2013), with problems more narrowly defined and not taking into consideration clinical symptoms of cannabis use disorder. This investigation builds on this prior work and expands it to other facets of dysregulation (i.e., delay discounting, reward sensitivity) and to the full spectrum of cannabis involvement from frequency of use, to cannabis-related problems, and to symptoms of cannabis use disorder. We hypothesize that lower working memory, higher delay discounting, higher impulsivity, and higher reward sensitivity will be associated with greater cannabis involvement. We do not have differential hypotheses for use versus problem use.

## Methods

### Participants

The present study is a secondary analysis of the data from a sample recruited for a parent cannabis administration study (Metrik et al., 2015). Participants ($n$= 104; $M_{age}$= 21.29, $SD$= 4.30; 36.54% female) were non-treatment seeking frequent cannabis users recruited from the community who met the following inclusion criteria: native English speakers, 18–44 years of age, non-Hispanic Caucasian (due to genetic aims of the parent study; Metrik et al., 2015), cannabis use at least 2 days per week in the past month and at least weekly in the past 6 months, and self-reported ability to abstain from cannabis for 24 hours without withdrawal. Exclusion criteria were: intent to quit or receive treatment for cannabis abuse, positive urine toxicology test result for drugs other than cannabis, pregnancy, nursing, past month DSM-IV Axis I affective or panic disorder, psychotic or suicidal state assessed by the Structured Clinical Interview for DSM-IV Non-Patient Edition (First et al., 2002), contraindicated medical issues by physical exam, and smoking more than 20 tobacco cigarettes per day (for a detailed description of the sample see Metrik et al., 2015). The study inclusion criteria were based on participant safety and scientific considerations in the parent laboratory study.

### Procedures

Participants completed a baseline battery of interview and self-report assessments, as well as two experimental double-blind sessions in which participants smoked active and placebo cannabis (Metrik et al., 2015). All data for the current study were drawn from the parent study's baseline session, with the exception of the delay discounting task, which was administered after participants smoked a marijuana placebo cigarette. As part of the experimental study, participants abstained from cannabis and tobacco smoking for 15 hours before each session. An alveolar carbon-monoxide (CO) of < 6 ppm was used to confirm no recent smoking (Cooper and Haney 2009; Metrik et al. 2015) with a Bedfont Scientific Smokelyzer®, and zero breath alcohol concentration was verified with an Alco-Sensor IV (Intoximeters, Inc., St Louis, MO., USA). Tobacco smokers were permitted to smoke a

tobacco cigarette following the CO test to prevent nicotine withdrawal. Participants were compensated upon study completion. Procedures were approved by the Institutional Review Board of Brown University.

## Measures

**Cannabis involvement ––—**The *Time-Line Follow-Back Interview* (TLFB; Dennis et al., 2004) assessed past 60-day number of marijuana days using a calendar assisted structured interview. The *Marijuana History questionnaire* assessed the number of times used cannabis per typical day (Metrik et al., 2009). Cannabis related problems were self-reported via a 22-item scale (Marijuana Problem Scale; MPS) (Stephens et al., 2000). A total count of problems was used; the MPS had a Cronbach's alpha of .76. Past-year symptoms of DSM-IV cannabis abuse and dependence were assessed via the Structured Clinical Interview for DSM-IV Non-Patient Edition (SCID; First et al. 2002).

Working memory capacity was assessed by the Trail-Making Test (Trails B) (Sanches-Cubillo et al., 2009) and the Digit Span subtests (forward and backward) of the WAIS-III (Wechsler, 1997). For Trail-Making, T-scores adjusted for age, gender, and education were used for all subsequent analyses. Hence higher scores are indicative of better cognitive capabilities. Although Trail-Making Test B may not be exclusively measuring working memory, there is evidence that working memory construct accounts for the most variance on this task (Sanchez-Cubillo et al., 2009) and we thus used the term working memory for the purposes of the current study. For Digit Span, a composite index score was used for all subsequent analyses. Hence higher scores are indicative of better cognitive capabilities.

Delay discounting was assessed using a computerized questionnaire with choice trials that vary in the amount of discounting delayed reinforcers (Richards et al., 1999). Participants had to choose between an immediate hypothetical monetary reinforcer or a larger amount after a delay (delay trials ranged between 0 and 365 days). The primary dependent variable was area under the curve connecting indifference points and the x-axis. The variable was scored such that higher levels were indicative of more discounting of delayed rewards, from 0.0 (no discounting) to 1.0 (steepest discounting) (Myerson et al. 2001).

Trait impulsivity was assessed via the Barratt Impulsiveness Scale (BIS; Patton & Stanford, 1995), a 30-item self-report measure scored on a Likert-scale from 1 (rarely/never) to 4 (almost always). Higher values are indicative of more impulsivity, the BIS had a Cronbach's alpha of .80.

Reward sensitivity was assessed by the Tridimensional Personality Questionnaire (TPQ-Short; Sher et al., 1995) and the Snaith Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995). For the TPQ, nine true-false items were used to assess reward dependence, with higher scores indicating hypersensitivity to reward. For the SHAPS, fourteen items assessed the ability to experience pleasure on a 4-point scale ranging from "strongly disagree" to "strongly agree," with higher scores representing an increased dispositional tendency to experience a hedonic tone characterized by pleasure. The reward dependence scale had a Cronbach's alpha of .74, and the SHAPS had a Cronbach's alpha of .91.

### Approach to analyses

Structural equation modeling was used to estimate the association between cannabis involvement and facets of dysregulation. A latent cannabis consumption factor was indicated by percent cannabis use days and the number of times cannabis was used in a typical day. A latent cannabis problems factor was indicated by self-reported number of problems from cannabis use in the past 90-days, number of cannabis abuse symptoms in the past year, and number of cannabis dependence symptoms in the past year. A latent working memory capacity factor was indicated by trail making and digit span tasks. A latent reward sensitivity factor was indicated by the reward dependence subscale of the TPQ and the Snaith-Hamilton Pleasure Scale. Delay discounting and impulsivity were modeled at the manifest level.

Analyses were conducted in Mplus version 7.11 (Muthen & Muthen, 2013). We used maximum likelihood estimation with robust standard errors (MLR); full information maximum likelihood (FIML) was used to estimate missing data. Due to the cross-sectional nature of the study, the structural associations between dysregulation and cannabis involvement were estimated without making assumptions about the causal directionality of effects.

## Results

Descriptive statistics and bivariate correlations are presented in Table 1. The sample was 36.5% female with a mean age of 21.29 (*SD*= 4.30, range= 18–42), with 64.3% of the sample reporting having completed at least one year of college, or being currently enrolled in college (*M*= 13.6 years of education, *SD*= 1.7). Participants reported cannabis use on an average of 71.35% (*SD*= 22.02%) of days in the 60-days prior to baseline (individuals ranged from using 18% of the days to 100% of the days), with a mean 2.03 (*SD*= 1.22) times a day. Additionally, there were 44 tobacco cigarette smokers (42.3%), who smoked 4.08 cigarettes (*SD*= 3.77) per day and 100 current drinkers (96.2%) who drank 8.5 (*SD*= 7.88) drinks per week.

Associations between the different facets of dysregulation and cannabis involvement are presented in Figure 1. The model had an excellent fit to the data, as indicated by *RMSEA*=.00 (*90% CI*= .00 – .07); *CFI*= 1.0; and *SRMR*= .04. Higher working memory capacity was associated with less cannabis consumption ($\beta$= −.48, *SE*= .16, *p*= .002) and less problems ($\beta$= −.31, *SE*= .12, *p*= .012); more discounting of delayed rewards was associated with problems ($\beta$= .25, *SE*= .13, *p*= .05) but not use frequency ($\beta$= .15, *SE*= .15 *p*= .32); higher levels of impulsivity was associated with more cannabis use ($\beta$= .63, *SE*= .14, *p*<.001) and more problems ($\beta$= .45, *SE*= .11, *p*<.001); and low reward sensitivity was associated with more cannabis use ($\beta$= −.46, *SE*= .24, *p*= .05) but not problems ($\beta$= −.17, *SE*= .15, *p*= .24). Associations with consumption tended to be larger than those with problems. The latent cannabis consumption and problematic use factors correlated at *r*= .58, *SE*= .15, *p*<.001.

## Discussion

We tested various dysregulatory correlates of cannabis involvement. Poor working memory and high trait impulsivity predicted both cannabis use and problems, suggesting that these two facets of dysregulation increase the likelihood of cannabis involvement in general; whereas steeper delay discounting predicted only cannabis problems. Our finding on delay discounting is consistent with meta-analytic evidence on other drugs, such as alcohol and tobacco involvement. Specifically, research on alcohol and tobacco use has indicated that delay discounting is more robustly associated with substance misuse relative to substance use (*for a quantitative review see* MacKillop et al., 2011). Similarly, our finding that delay discounting may be specifically related to cannabis problems is consistent with other studies examining delay discounting as a correlate of cannabis use disorder (Aston et al., 2016; Johnson et al., 2010).

Low levels of sensitivity to reward were associated with cannabis use, but not problems. The direction of this effect was in opposition to our hypotheses and to the few studies that have tested the association between reward sensitivity and cannabis use in college student and adolescent samples (e.g., Emery & Simons, 2017; Papinczak et al., 2018; Scalco & Colder, 2017). The direction of this effect is also inconsistent with previous findings testing the association between reward sensitivity and alcohol involvement (*for meta-analytic review see* Stautz & Cooper, 2013), but consistent with tobacco research implicating low reward sensitivity as a risk factor for smoking (Audrain-McGovern et al., 2012). The difference in composition of our sample (community vs. college/adolescent samples) may explain divergent findings. It is possible that college/adolescent samples represent earlier periods in the development of cannabis involvement, whereas our community sample may consist of more habitual users. It is possible that in more cannabis involved populations, low sensitivity to reward motivates cannabis use to cope with chronically low levels of reinforcement (Bowirrat & Oscar-Berman, 2005), and/or that use of cannabis leads to neurobiological changes that compromise the reward system (Koob, 2006). However, due to the unexpected nature of this association, replication is needed.

Future studies need to test the relationship between dysregulatory risk factors and involvement with different types of drugs in the same model (as opposed to collapsing across drugs, or solely focusing on a single type of drug). Multivariate models predicting variability in use of different drugs (amongst polysubstance users) may be more generalizable than contrasting sub-groups of “pure cannabis users” vs. “pure other drug users” because of the high epidemiological prevalence of polysubstance use (Connor, Gullo, White, & Kelly, 2014; Moss, Chen, & Yin, 2014), particularly marijuana and alcohol co-use (Metrik et al., 2018), and the high rates of alcohol use in our own cannabis-using sample. Similarly, future research may need to “unpack” cannabis use that occurs in isolation, vs. cannabis use that is simultaneous with other types of drug use. For example, emerging research on simultaneous alcohol and marijuana use demonstrates that the consequences and correlates of co-use differ relative to using either substance alone (Lee, Cadigan, & Patrick, 2017; Patrick, Fairlie, & Lee, 2018; Yurasek, Aston, & Metrik, 2017).

Finally, our findings suggest that cannabis use and problem use are substantially overlapping yet distinct factors. For example, the association between the cannabis use and problem use factors indicates that approximately a third of the variance between these factors is overlapping, yet approximately two thirds of their variance is unique. The degree of overlap between use and problem use in our sample is comparable to previous research on cannabis involvement (Godley et al., 2005; Zvolensky et al., 2006), as well as research on other drugs (Prince, Pearson, Bravo, & Montes, 2018). In other words, cannabis use itself may not be sufficient to explain the development of problem use, suggesting a need to examine predictors of cannabis related negative consequences beyond frequency of cannabis use. Our results, and previous findings (e.g., Dvorak & Day, 2014), suggest that individual differences in dysregulatory risk factors may be useful in quantifying variability in cannabis involvement.

## Conclusions, Limitations, & Future Directions

The present study provides support for a fundamental assumption of contemporaneous models of dysregulated behavior, and suggest that there are multiple facets of dysregulation that overlap with cannabis involvement. “Unpacking” the multi-dimensional nature of dysregulation has the potential to refine how we choose to “carve nature at its joints” (Duckworth & Steinberg, 2015), or in other words how we ask questions regarding the developmental antecedents and consequences of cannabis involvement.

The findings of this investigation should be considered in light of several limitations. Although our model conceptualizing cannabis involvement via frequency of use and use related problems fits the data well, there are likely individual differences in the quantity and potency of cannabis consumed that are not represented in our model (Prince, Conner, & Pearson, 2018). Furthermore, we utilized a uni-dimensional representation of trait impulsivity, which may be best conceptualized as a multidimensional construct (Carver & Johnson, 2018; Sharma, Markon, & Clark, 2014; Whiteside, Lynam, Miller, & Reynolds, 2005) with distinct facets shown to differ in their associations to cannabis (Gunn et al., 2018; Luba et al., 2018; VanderVeen, Hershberger, & Cyders, 2016; Wilson et al., 2018) and other drug use (Coskunpinar, Dir, & Cyders, 2013; Lopez-Vergara, Spillane, Merrill, & Jackson, 2016). Additionally, we were intentionally ambiguous in stipulating which dysregulatory mechanism belongs to the deliberative vs. reactive modes of information processing hypothesized in dual-process models because of unresolved theoretical issues in the field (Bellini-Leite, 2018; Glockner & Witteman, 2010). Future studies may benefit from using second-order latent variables to explicitly model the structure of associations between distinct dysregulatory risk factors (Krakauer, Ghazanfar, Gomez-Marin, Maclver, & Poeppel, 2017), similar to how the personality field has sought to quantify the structure of individual differences in character (Gullo et al., 2014).

Prospective studies such as the Adolescent Brain Cognitive Development (ABCD) Study (Jernigan, Brown, & Dowling, 2018) that investigate the dysregulatory precursors and consequences of cannabis involvement may help with causal attribution of the direction of these influences, particularly during periods of rapid transition into the initiation of cannabis use (early to middle or late adolescence) and the emergence of cannabis use disorders

(emerging adulthood). Clinical implications of our study emphasize that there may not be a single dysregulatory phenotype in cannabis users. Future research should test whether existing treatments are more likely to be efficacious depending on the phenotypic structure of the individual. For example, cannabis users whose phenotype involves alterations in reward sensitivity or delay discounting may be particularly responsive to prevention or intervention efforts that incorporate non-drug use sources of reinforcement; cognitive training paradigms may be particularly effective amongst individuals with pronounced working memory deficits; and/or users high in trait impulsivity may be particularly responsive to interventions that provide short term goal setting (or scaffolding).

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## Highlights

- Dysregulation is a correlate of cannabis involvement
- Dysregulation is a highly heterogeneous construct
- Cannabis use overlaps with working memory capacity, impulsivity, & reward sensitivity
- Problem cannabis use overlaps with working memory capacity, impulsivity, & delay discounting.

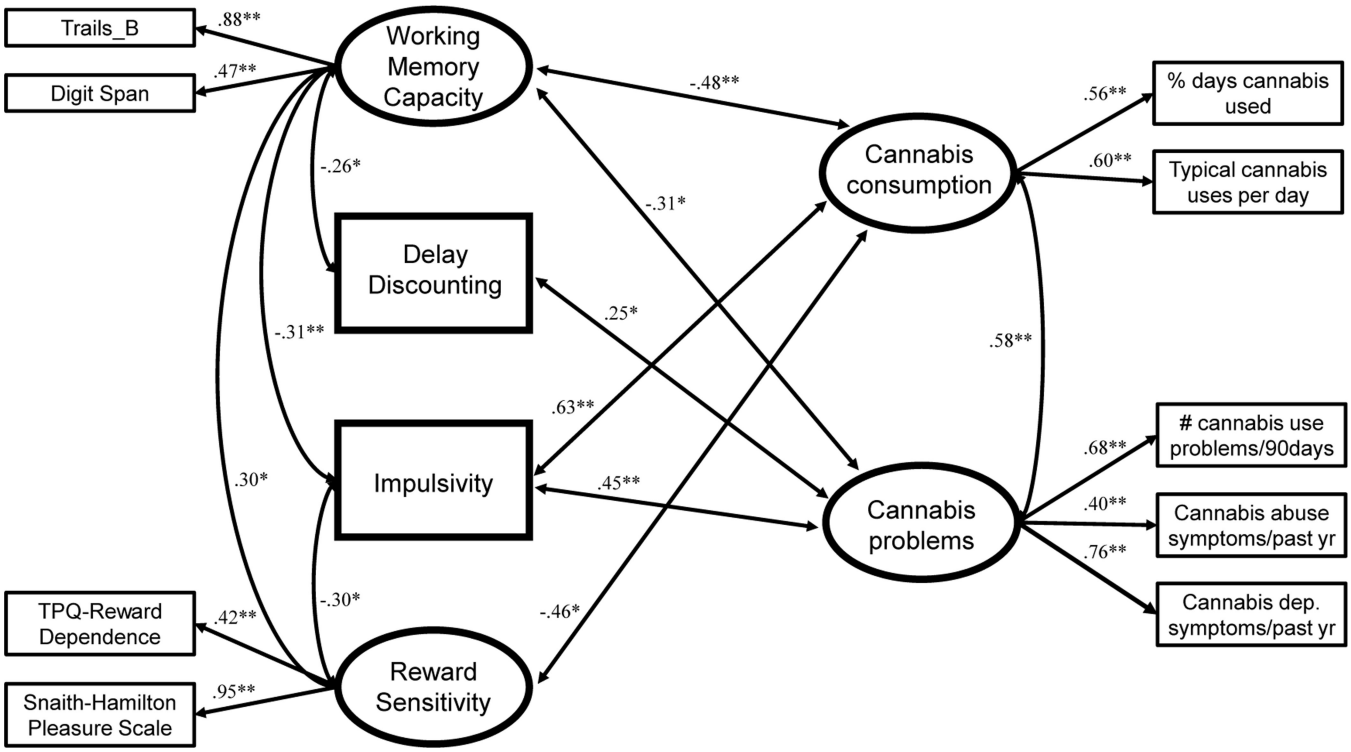


**Figure 1 –.**

Results of structural model of the association between facets of dysregulation and cannabis use and problem use

Note: All associations modeled, only significant associations depicted; for ease of interpretation all parameter estimates are standardized; * = p<.05; ** = p<.01

**Table 1 –**

Descriptive statistics and correlations

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Trail making B T-Score | --- | 41** | −.24* | −.27** | .01 | .25* | −.21 | −.26** | −.28** | −.07 | −.15 |
| 2. Digit span scaled score | | --- | −.11 | −.15 | .01 | .16 | −.10 | −.16 | −.02 | −.00 | −.11 |
| 3. Delay discounting | | | --- | .08 | −.10 | −.17 | .11 | .10 | .10 | .19 | .23* |
| 4. Impulsivity | | | | --- | −.12 | −.28 | .38** | .35** | 34** | .11 | .33** |
| 5. Reward dependence | | | | | --- | 0.4** | −.17 | −.12 | −.05 | .04 | .10 |
| 6. Pleasure scale | | | | | | --- | −.25* | −.25* | −.18 | −.10 | −.07 |
| 7. % cannabis use days | | | | | | | --- | .32** | .13 | .18 | .20* |
| 8. Times cannabis use/day | | | | | | | | --- | .20* | .14 | .35** |
| 9. Cannabis problems | | | | | | | | | --- | .32** | .51** |
| 10. Cannabis abuse symptoms | | | | | | | | | | --- | .28** |
| 11. Cannabis dependence symptoms | | | | | | | | | | | --- |
| *n* | 104 | 104 | 89 | 104 | 104 | 104 | 104 | 104 | 104 | 104 | 104 |
| *Mean* | 49.6 | 11.4 | .56 | 59.9 | 6.4 | 35.7 | 71.3 | 2.03 | 3.6 | .38 | 1.2 |
| *(SD)* | (10.2) | (2.6) | (.3) | (8.3) | (2.3) | (5.3) | (22) | (1.22) | (2.9) | (.61) | (1.2) |

Note:

* = p<.05

** = p<.01