

Testing the Biosocial Cognitive Model of Substance Use in Cannabis Users Referred to Treatment

Zoë E. Papinczak^{1,5}, Jason P. Connor^{1,2,3}, Gerald F. X. Feeney^{1,3}, Paul Harnett⁵, Ross McD. Young^{3,4} & Matthew J. Gullo^{1,3}

¹Centre for Youth Substance Abuse Research, Faculty of Health Sciences, The University of Queensland, Brisbane, QLD 4072, Australia

²Discipline of Psychiatry, School of Medicine, The University of Queensland, Brisbane, QLD 4029, Australia

³Alcohol and Drug Assessment Unit, Princess Alexandra Hospital, Brisbane, QLD 4102, Australia

⁴Faculty of Health, Queensland University of Technology, Brisbane, QLD 4059, Australia

⁵School of Psychology, The University of Queensland, Brisbane, QLD 4072, Australia

*Correspondence to:

Matthew J. Gullo, Centre for Youth Substance Abuse Research, The University of Queensland, Brisbane, QLD 4072, Australia

Email: m.gullo@uq.edu.au

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ABSTRACT

Background: The Biosocial Cognitive Theory (bSCT) hypothesizes two pathways linking dimensions of impulsivity to substance use. The first predicts that the association between reward sensitivity and substance use is mediated by positive outcome expectancies. The second predicts that the relationship between rash impulsiveness and substance use is mediated by refusal self-efficacy. This model has received empirical support in studies of alcohol use. The present research provides the first application of bSCT to a cannabis treatment population and aims to extend its utility to understanding cannabis use and severity of dependence.

Design: 273 patients referred for cannabis treatment completed a clinical assessment that contained measures of interest.

Setting: A public hospital alcohol and drug clinic.

Measurements: The Sensitivity to Reward Scale, Dysfunctional Impulsivity Scale, Cannabis Expectancy Questionnaire, Cannabis Refusal Self-Efficacy Questionnaire and Severity of Dependence Scale–Cannabis were completed, along with measures of cannabis consumption.

Findings: The bSCT model provided a good fit to the data for cannabis use and severity of dependence outcomes. The association between reward sensitivity and each cannabis outcome was fully mediated by positive cannabis expectancies and cannabis refusal self-efficacy. The relationship between rash impulsiveness and each cannabis outcome was fully mediated by cannabis refusal self-efficacy.

Conclusions: Findings support the application of the bSCT model to cannabis use and dependence severity and highlight the important role of social cognitive mechanisms in understanding the association between impulsivity traits and these outcomes. The differential association of impulsivity traits to social cognition may assist targeted treatment efforts.

Keywords: cannabis, dependence, expectancies, impulsivity, marijuana, self-efficacy

1. INTRODUCTION

Cannabis is the most commonly used illicit drug worldwide (Hall, 2015). The global number of cannabis users is estimated at approximately 125 – 203 million people, or 2.8% - 4.5% of the global population (Degenhardt and Hall, 2012). Of those who ever try cannabis, it is estimated that 10% will become daily users and 20 – 30% will become weekly users (Hall and Pacula, 2003). Engaging in frequent and persistent cannabis use is associated with increased risk for adverse outcomes including cannabis dependence (Hall and Degenhardt, 2009), long-term cognitive impairments (Solowij et al., 2002) and psychosis (van Os et al., 2002). The effectiveness of secondary prevention and treatment interventions will be enhanced by an understanding of the risk factors and psychological processes that influence and maintain cannabis use and dependence.

Impulsivity and cannabis-related cognition are two sets of psychological risk factors associated with cannabis consumption (Hayaki et al., 2010; Lyvers et al., 2013; Stautz et al., 2017), cannabis dependence (Blanco et al., 2014; Connor et al., 2013; Hayaki et al., 2011) and treatment outcomes (Bentzley et al., 2016; Gullo et al., 2017b; Litt et al., 2008). The Biosocial Cognitive Model (bSCT model) integrates biologically-based personality traits and social cognitive factors to provide a mechanistic account of substance use. In summary, this model predicts that impulsivity conveys risk for cannabis use through its influence on cannabis related-learning and cannabis-related cognitions. The present study will evaluate this model in a sample of cannabis users in treatment.

1.1 Impulsivity

Impulsivity plays a key role in substance use disorders. Current evidence indicates that impulsivity is comprised of two biologically-based dimensions which independently contribute to the development of drug abuse and dependence (Dawe et al., 2004; DeWit and Richards, 2004; Potenza and Taylor, 2009; Woicik et al., 2009). These dimensions are

reward sensitivity (reward drive) and *rash impulsiveness* (disinhibition). At the trait level, reward sensitivity reflects individual differences in the inherent salience of rewarding stimuli. Reward sensitive individuals are more likely to notice, desire and actively pursue rewards, including drugs of abuse. Conceptually, it is similar to *Sensation Seeking* (Steinberg, 2010; Woicik et al., 2009), *Choice Impulsivity* (Potenza and Taylor, 2009), and particularly Gray's (1970) *Behavioural Approach System*. The other trait, rash impulsiveness, refers to individual differences in the capacity to modulate and inhibit prepotent approach behaviours, regardless of potential negative consequences. Rash impulsive individuals have a tendency to persist in previously reinforced behaviour, despite that behaviour no longer resulting in reward (Gullo and Dawe, 2008). Conceptually, it is similar to *Impulsivity* (Steinberg, 2010; Woicik et al., 2009), *Response Impulsivity* (Potenza and Taylor, 2009), and Barratt's (1972) *Impulsiveness* dimension.

While each trait bears similarities to other constructs, there are important differences (e.g., Carlson et al., 2013; Gullo et al., 2014a). For example, it is high reward sensitivity in particular that is argued to drive the learning bias that produces stronger associations between cannabis cues and anticipated reward (i.e., positive cannabis expectancies; Gullo et al., 2010). While Sensation Seeking and Choice Impulsivity may also be related to positive expectancies, theoretically, it should only be those components related to reward sensitivity that are behind this association (Gray, 1975; Gullo et al., 2010). Similarly, while there is less variance among rash impulsiveness-like traits, it has been argued that it is those components related to disinhibition and non-planning, as conceptualized in rash impulsiveness, that affect beliefs about control over cannabis (i.e., cannabis refusal self-efficacy; Gullo et al., 2010). Both impulsivity traits are associated with greater cannabis use (Griffith-Lendering et al., 2012; Lyvers et al., 2013; Prince van Leeuwen et al., 2011; Stautz et al., 2017).

1.2 Biosocial Cognitive Theory

Biologically-based personality traits, like reward sensitivity and rash impulsiveness, are distal risk factors for drug abuse. One mechanism through which these traits are predicted to influence drug use is via their influence on social learning (Bandura, 1986), and the subsequent acquisition and maintenance of drug-related cognitions (Gullo et al., 2010; Smith and Anderson, 2001). These proximal, cognitive risk factors directly influence drug use. The bSCT model (see Figure 1) proposes that reward sensitivity and rash impulsiveness convey risk for substance use via two distinct, cognitively-mediated pathways.

The first pathway involves reward sensitivity influencing cannabis use via its effect on *positive cannabis expectancies* (i.e., the set of expectations that consuming cannabis will result in positive outcomes; Gullo et al., 2010; Papinczak et al., 2018). Highly reward sensitive individuals are predicted to experience a positive learning bias, in which they are more likely to attend to, encode, and recall the positive effects of using cannabis (Gullo et al., 2010; Smith and Anderson, 2001). They may also experience greater euphoria and reinforcement from cannabis use. Therefore, these individuals are prone to acquire and maintain strong positive cannabis expectancies. Positive cannabis expectancies are robustly associated with greater cannabis consumption and this effect has been demonstrated among adolescents (Alfonso and Dunn, 2007; Skenderian et al., 2008), adults (Brackenbury et al., 2016; Galen and Henderson, 1999) and cannabis dependent individuals (Boden et al., 2013; Connor et al., 2013, 2011).

The second pathway involves rash impulsiveness influencing cannabis use through its effect on *cannabis refusal self-efficacy* (i.e., self-confidence in one's ability to refuse cannabis; Gullo et al., 2010; Papinczak et al., 2018). Rash impulsive individuals have inherent difficulties in inhibitory control, particularly during situations involving the refusal of immediate rewards. Reduced cannabis refusal self-efficacy is considered to result, in part,

from a generalized belief of poor impulse control due to past failures to refuse rewards (Gullo et al., 2010). Therefore, these individuals are predicted to have greater self-perceived difficulty resisting cannabis, which is then reinforced by subsequent disinhibited use. Cannabis refusal self-efficacy is a robust predictor of cannabis consumption, and is negatively associated with cannabis use among frequent users (Connor et al., 2013; Hayaki et al., 2011; Young et al., 2012). Cannabis refusal self-efficacy is also an important predictor of abstinence during and after the treatment of cannabis dependence (Kadden et al., 2007; Litt et al., 2008; Litt and Kadden, 2015) and is negatively associated with cannabis dependence severity (Connor et al., 2013; Young et al., 2012).

In addition to directly impacting cannabis use, positive cannabis expectancies indirectly influence use via their effect on cannabis refusal self-efficacy. Theoretically, higher perceived reinforcement from drug use will undermine self-efficacy for drug refusal (Bandura, 1986; Gullo et al., 2010). Cannabis refusal self-efficacy has been found to fully mediate the association between positive cannabis expectancies and cannabis use in studies of university students (Papinczak et al., 2018) and cannabis dependent outpatients (Gullo et al., 2017b), and partially mediated this association in a study of cannabis users referred for treatment (Connor et al., 2013).

The majority of the empirical evidence in support of the bSCT model has been obtained from studies of alcohol use. These studies have examined a range of different samples including university students (Gullo et al., 2010; Harnett et al., 2013; Leamy et al., 2016), adults recruited from the community (Kabbani and Kambouropoulos, 2013) and alcohol dependent inpatients (Gullo et al., 2014b, 2010). Only one study has examined the bSCT model in the context of cannabis use. Papinczak and colleagues (2018) recently evaluated the model in a study of youth cannabis use and obtained cross-sectional data from a sample of 252 university students. This study found that the bSCT model provided a good fit

to the data. Positive cannabis expectancies and cannabis refusal self-efficacy partially mediated the relationship between reward sensitivity and cannabis use. Cannabis refusal self-efficacy on the other hand, fully mediated the association between rash impulsiveness and cannabis use. While this initial evidence is promising, the results of Papinczak et al. (2018) may be limited by the infrequent use of cannabis reported by the sample.

1.3 Present Study

It is not known whether social cognition would serve to mediate the association of impulsivity with cannabis use in a treatment population. Furthermore, there may be differences in the strength of the model pathways when comparing a clinical sample to a student sample, as has been previously found in alcohol studies (Gullo et al., 2014b, 2010; Leamy et al., 2016). If found, such differences could have important implications for targeted approaches to cannabis prevention and treatment, through highlighting distinct pathways of risk.

The present study provides the first test of a new Biosocial Cognitive Theory in cannabis users referred for treatment. This research will test the bSCT model across two outcomes: cannabis use and cannabis dependence severity. To summarise, the bSCT model hypothesizes that (see Figure 1):

1. Cannabis refusal self-efficacy will mediate the association between rash impulsiveness and cannabis outcomes.
2. Positive cannabis expectancies will mediate the association between reward sensitivity and cannabis outcomes.
3. Cannabis refusal self-efficacy will mediate the association between positive cannabis expectancies and cannabis outcomes.

2. METHOD

2.1 Participants and Procedure

Data were obtained from all 346 cannabis users who attended a cannabis assessment between 1 February 2016 and 30 September 2017 as part of the Queensland Illicit Drug Diversion Initiative. All patients attended this diversion program voluntarily and under police direction as an alternative to criminal prosecution for a minor cannabis-related offence (i.e., possession of <50g of cannabis or paraphernalia).¹ The diversion program consists of a two-hour assessment of substance use and psychosocial functioning and incorporates motivational interviewing. As part of the assessment protocol, all patients who attend the diversion program complete an assessment battery that includes the measures listed in Section 2.2. These completed assessment batteries were analysed in the present study. The present study analysed cases from consecutive patients who presented at a public hospital alcohol and drug clinic in Brisbane, Australia during the study period. Ethics approval was obtained from the hospital human ethics committee to access de-identified, retrospective data for patients in the diversion program. The response rate was 100% of individuals assessed at the site.

2.2 Measures

Reward sensitivity was measured using the 10-item version of the Sensitivity to Reward Scale (Cooper and Gomez, 2008). Each item describes a behaviour in which a yes (1) or no (0) response is made. The convergent, concurrent and discriminant validity of the scale has been established (Cooper and Gomez, 2008). Factor analytic studies have shown it loads on a factor with measures of the same construct (e.g., BAS-Drive; Franken and Muris, 2006).

Rash impulsiveness was measured using the 12-item Dysfunctional Impulsivity Scale (Dickman, 1990). Each item describes a behaviour in which a true (1) or false (0) response is

¹ No other types of crimes are offered the option to attend the diversion program. There is currently no data available on the number of individuals who decline drug diversion, but anecdotal reports by police suggest that “everyone” chooses it over prosecution.

made. The criterion validity of the scale has been confirmed (e.g., delay discounting; Mobini et al., 2007) and it loads on a factor with measures of the same construct (e.g., Novelty Seeking; Miller et al., 2004).

Positive cannabis expectancies were assessed using the positive expectancies scale (18 items) from the Cannabis Expectancy Questionnaire (CEQ-P) (Connor et al., 2011). Responses were rated on a 5-point Likert scale (1 = *Strongly disagree* to 5 = *Strongly agree*). The criterion validity and factor structure of the scale have been confirmed (Connor et al., 2011).

Cannabis refusal self-efficacy was measured using the 14-item Cannabis Refusal Self-Efficacy Questionnaire (CRSEQ) (Young et al., 2012). It comprises three subscales that can be summed to provide a total score: *emotional relief* (six items) *opportunistic* (five items) and *social facilitation* (three items). Responses were made on a 6-point Likert scale (1 = *I am very sure I could NOT resist smoking cannabis* to 6 = *I am very sure I could resist smoking cannabis*). The factor structure and criterion validity of the CRSEQ has been previously established in clinical samples (Young et al., 2012).

Severity of cannabis dependence was determined using the five-item Severity of Dependence Scale – Cannabis (SDS-C) (Gossop et al., 1995). This measure assesses the degree of psychological dependence experienced by users (Swift et al., 2000). The SDS-C cut-off for DSM-IV cannabis dependence is a score greater than 2, which corresponds with 64% sensitivity and 82% specificity (Swift et al., 1998). The construct and criterion validity of the SDS-C has been confirmed in adult and adolescent samples of cannabis users (Ferri et al., 2000; Martin et al., 2006). The SDS-C was used as a continuous indicator of severity of cannabis dependence in the present study.

Cannabis use was assessed using a single item in which patients indicated the typical “quantity of cannabis used per week” in the past month and responded using an open-

response format to indicate grams used per week . That is, “_____grams/week”. The convergent validity of this item was evidenced by a small significant correlation with SDS-C scores ($r = .32$) in the present sample which is consistent with correlations reported in past research (Ferri et al., 2000; Steiner et al., 2008).

2.3 Data Analyses

Structural equation modelling with robust maximum likelihood estimation was conducted in *R* using the *lavaan* package. The comparative fit index (CFI), Tucker Lewis index (TLI), goodness-of-fit index (GFI), root mean square error of approximation (RMSEA) and standardized root mean square residual (SRMR) were used to evaluate model fit. The following cut-offs were used to indicate “good” fit: CFI ≥ 0.95 ; TLI ≥ 0.95 ; GFI ≥ 0.90 ; RMSEA ≤ 0.06 ; SRMR ≤ 0.08 (Hooper et al., 2008; Hu and Bentler, 1999). The χ^2 test of model fit ($\alpha = 0.05$) is also reported, although this typically overestimates poor fit in large samples (Bentler, 2007). The final useable sample was $n = 269$; further details concerning data analyses, data screening and assumption checks are reported in the Supplementary Material.

3. RESULTS

Sample characteristics are presented in Table 1. Descriptive statistics and Cronbach’s alphas for the model variables are reported in Table 2. The inter-correlations between model variables are reported in Table 3. The fit indices for the structural models are presented in Table 4. Results of the χ^2 difference tests ($\Delta\chi^2$) indicated no difference between partial and full mediation models, which supports the latter, more parsimonious model (see Table 4). This is consistent with the direct pathways which were non-significant. Reward sensitivity was not directly associated with cannabis use ($\beta = .14, p=.090$) or severity of cannabis dependence ($\beta = .11, p=.097$). Rash impulsiveness was not directly associated with cannabis use ($\beta = .03, p=.701$) or dependence severity ($\beta = .05, p=.415$). In the following sections, the

full mediation model pathways are reported in the text and figures. The direct effects of the structural models are reported in Table 5.

3.1 Cannabis Use

The bSCT model provided a good fit to the data (see Table 4, Models 1 and 2). The model pathways are reported in Figure 2. Overall, the model explained 18.9% of the variance in cannabis use, which is a moderate-to-large effect size (Cohen, 1988). Hypothesis 1 was supported as a significant mediation effect was found for the rash impulsiveness pathway (unstandardized indirect effect = 0.347, 97.5% CI = 0.191 – 0.533). Rash impulsiveness was directly associated with lower levels of cannabis refusal self-efficacy ($p < .001$) and this association had a moderate effect size ($\beta = -0.36$). Cannabis refusal self-efficacy was directly associated with lower levels of cannabis use ($p < .001$) and this was a moderate effect size ($\beta = -0.38$).

Hypothesis 2 was supported as a significant mediation effect was found for the reward sensitivity pathway (unstandardized indirect effect = 0.060, 97.5% CI = 0.006 – 0.139). Reward sensitivity was directly associated with stronger positive cannabis expectancies ($p = .006$). Stronger positive cannabis expectancies were associated with greater cannabis use – both directly ($p = .017$) and indirectly via reduced levels of refusal self-efficacy ($p < .001$; unstandardized indirect effect = 0.246, 97.5% CI = 0.135 – 0.379). Therefore, Hypothesis 3 was supported.

3.2 Cannabis Dependence Severity

The bSCT model provided a good fit to the data (see Table 4, Models 3 and 4). The model pathways are shown in Figure 3. Overall, the model explained 30.6% of the variance in severity of cannabis dependence, which is a large effect size (Cohen, 1988). Hypothesis 1 was supported as a significant mediation effect was found for the rash impulsiveness pathway (unstandardized indirect effect = 0.179, 97.5% CI = 0.111 – 0.256). Rash impulsiveness was

directly associated with lower levels of cannabis refusal self-efficacy ($p < .001$) and this effect was moderate ($\beta = -0.37$). Cannabis refusal self-efficacy was directly associated with severity of cannabis dependence ($p < .001$) and this association had a large effect size ($\beta = -0.55$).

Hypothesis 2 was supported as a significant mediation effect was found for the reward sensitivity pathway. Reward sensitivity was directly associated with greater positive cannabis expectancies ($p = .006$), which were associated with severity of cannabis dependence indirectly via reduced refusal self-efficacy (unstandardized indirect effect = 0.123, 97.5% CI = 0.075 – 0.178). This supported Hypothesis 3. Positive cannabis expectancies were not directly associated with severity of cannabis dependence ($p = .905$).

4. DISCUSSION

This study provides primary support for the generalizability of the Biosocial Cognitive Theory (bSCT) to cannabis use and severity of cannabis dependence. The bSCT model was able to predict a moderate-to-large amount of variance in typical weekly cannabis use and severity of cannabis dependence within a group of cannabis users referred for treatment. Overall, the results of the two models were consistent. The social cognitive mechanisms of positive expectancies and refusal self-efficacy fully mediated the effects of reward sensitivity and rash impulsiveness, respectively. Therefore, these results provide a theoretical explanation for how these biologically-based impulse personality traits convey risk for cannabis use and dependence.

The two mediational pathways proposed by bSCT were supported in this study. Our results suggest that cannabis refusal self-efficacy may be an important mechanism through which rash impulsiveness indirectly influences cannabis use and dependence. This is in line with the prediction that rash impulsive individuals have greater perceived difficulty refusing substances, possibly due to past failed attempts to refuse rewards generally and awareness of

difficulties with inhibitory control (Gullo et al., 2010). This weakened sense of refusal self-efficacy may place rash impulsive individuals at greater risk for problematic cannabis use.

The reward sensitivity pathway was more complex. Our findings suggest that reward sensitivity is associated with cannabis use and dependence indirectly through the mechanism of positive cannabis expectancies and its subsequent negative association with cannabis refusal self-efficacy. Reward sensitivity was associated with stronger positive cannabis expectancies, which is consistent with the notion of a positive learning bias in the context of substance use (Gullo et al., 2010). These heightened positive expectancies among reward sensitive individuals, may place them at greater risk for cannabis use and dependence, theoretically through undermining their refusal self-efficacy.

The results of this study are consistent with Social Cognitive Theory's prediction that self-efficacy is the key, proximal predictor of behaviour (Bandura, 1986). As has previously been found with alcohol, cannabis refusal self-efficacy had the strongest, direct association with cannabis use (moderate effect size) and severity of dependence (large effect size), and it provided the mechanism through which positive expectancies conveyed the majority of their association with these outcomes. Together, these results add to the body of literature highlighting the important protective role that refusal self-efficacy plays against problematic cannabis use (Hayaki et al., 2011; Young et al., 2012); and indicate that refusal self-efficacy is an important target for cannabis interventions (Kadden and Litt, 2011).

Based upon the findings of the present study, the bSCT may reveal new avenues for targeted treatments. Of proximal importance is refusal self-efficacy, consistent with previous studies, which can be increased directly and indirectly through several different approaches. Teaching and practicing coping skills to better manage high risk situations, and the sense of mastery that comes with it, is one way to increase self-efficacy (Bandura, 1986; Litt et al., 2008; Marlatt and Gordon, 1985; Stephens et al., 1995). According to bSCT, the

prioritization of coping skills to develop could be informed by consideration of patient characteristics. For example, given the similar magnitude of the association with self-efficacy between rash impulsiveness and positive expectancies, a patient with higher expectancies may benefit more from behavioural experiments and cognitive restructuring skills that target their exaggerated beliefs (Beck et al., 1993; Brown, 1993). Our findings also suggest that individuals high in rash impulsiveness may be at greater risk of large reductions in cannabis refusal self-efficacy after a lapse, increasing the likelihood of relapse (Marlatt and Gordon, 1985). Treatment targeted at individuals high in rash impulsiveness may involve a greater degree of relapse prevention support. If a causal connection is found to exist between rash impulsiveness and cannabis refusal self-efficacy, rash impulsiveness could be targeted directly through process-oriented techniques included in cognitive and dialectical behavioural therapies (Butz and Austin, 1993; Coates et al., 2018; Lineham, 1993).

The results of the present study are largely consistent with past research which has evaluated the bSCT in the context of alcohol abuse and dependence (Gullo et al., 2014b, 2010; Harnett et al., 2013; Kabbani and Kambouropoulos, 2013) and cannabis use (Papinczak et al., 2018). This consistency across studies suggests that the same social cognitive mechanisms are operating across substances of abuse and levels of use, and are consistently and uniquely, associated with different dimensions of impulsivity. Therefore, the bSCT model is likely to apply to substance use, broadly. Further research is required to confirm this for other substances, although, it raises the possibility of common pathways of risk that could be targeted to reduce or prevent problematic substance use.

Despite some similarities across studies, there were important differences between the results of the present research and Papinczak et al. (2018), who evaluated the bSCT model of cannabis use in a sample of university students. First, unlike Papinczak et al. (2018), there was no direct association between reward sensitivity and the cannabis outcomes in the

present clinical sample as effects were fully mediated by positive expectancies. Second, both cannabis use and dependence severity were more strongly associated with the rash impulsiveness pathway in the clinical sample, compared to university students. These findings suggest that the reward sensitivity pathway may be more influential during the initiation and experimental phases of cannabis use, while the rash impulsiveness pathway may play a stronger role in abuse and dependence. This suggests that there may be benefits to targeting different bSCT pathways and mechanisms when approaching cannabis use prevention versus treatment for dependence.

Some limitations in the present study are acknowledged. Firstly, the study was cross-sectional in its design and therefore the directions of the observed effects and causality cannot be determined. Secondly, there may be limitations to the generalisability of the findings as the sample was a group of cannabis users who were offered treatment as part of a police-mandated program and attended voluntarily as an alternative to criminal prosecution. Given this treatment context, there may be an increased likelihood of participants under reporting their cannabis use, however, at the commencement of the session, participants were assured of the confidentiality of their responses. Thirdly, the self-report SDS-C was used to assess dependence severity, rather than clinical interviews. Furthermore, although the SDS-C has been validated on DSM-IV criteria, it has not yet been validated for DSM-5 cannabis use disorder. Fourthly, cannabis use was assessed with a single-item in which participants indicated the typical quantity of cannabis they consumed per week during the past month. Although typical use measures are commonly used, they provide a less precise indication of actual use in comparison to the timeline followback method (Sobell & Sobell, 1992). Based upon these limitations, future research could utilise a community sample of cannabis users and would benefit from employing a longitudinal design, clinical interviews to diagnose

DSM-5 cannabis use disorder and the timeline followback to measure cannabis consumption with corroboration from biochemical measures (Smith et al., 2018).

To conclude, this research supported bSCT when applied to the understanding of cannabis use and severity of cannabis dependence in a clinical population. The findings offer an explanation for how biologically-based impulsive personality traits convey risk for problematic cannabis use at the cognitive level of analysis. Future treatments for problematic cannabis use could be enhanced by focusing on the complex interplay of bSCT factors.

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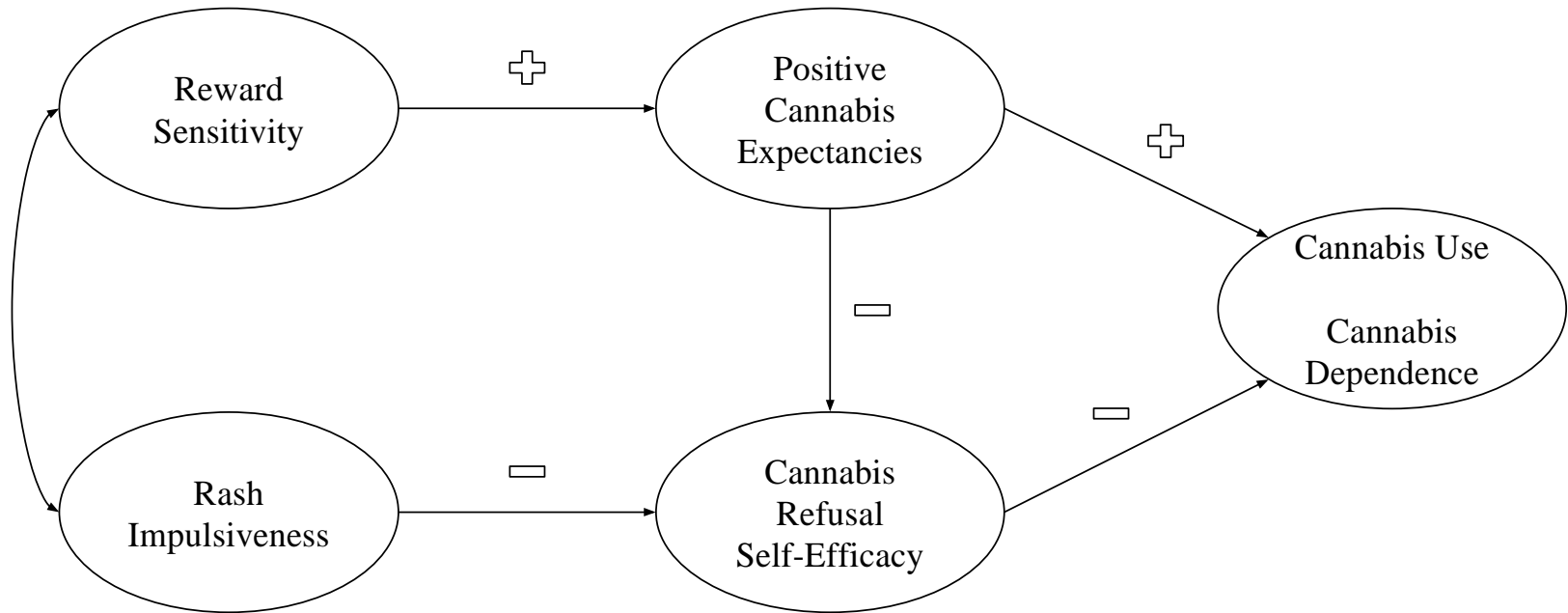


Figure 1.

Table 1Sample characteristics ($N = 273$).

	<i>M</i>	<i>SD</i>	Range
Age (years)	26.31	9.39	14 - 61
Cannabis use – typical weekly consumption (grams)	4.06	10.09	0 - 70
Cannabis use – number of days in the last 28 (days)	9.58	10.09	0 - 28
Cannabis use – age of first use (years)	17.17	4.80	7 - 54
Hazardous drinking (AUDIT score)	7.12	6.18	0 - 34
	<i>N</i>	<i>n</i>	%
Gender			
Female	273	60	22
Male		213	78
Country of Birth			
Australia	273	206	75.5
New Zealand		14	5.1
England		10	3.7
Other (not specified)		43	15.8
Finished High School			
Yes	270	206	76.3
No		64	23.7
Aboriginal or Torres Strait Islander			
Neither	271	264	97.4
Aboriginal		6	2.2
Torres Strait Islander		1	0.4
Currently Employed			
Yes	272	222	81.6
No		50	18.4
Receiving Government Benefits			
Yes	272	61	22.7
No		211	77.3
SDS Cannabis Dependent			

Yes	270	104	38.5
No		166	61.6
Cannabis Primary Drug of Concern			
Yes	267	246	92.1
No		21	7.9
Cannabis Used in Past 28 Days			
Yes	250	217	86.8
No		33	13.2
Usual Frequency of Cannabis Use			
0 days per week	253	2	0.8
1-3 days per week		82	32.4
4-6 days per week		17	6.7
Daily		73	28.9
Fortnightly		17	6.7
Opportunistically		55	22.7
Monthly		7	2.8
Tobacco Smoker			
Yes	273	62	22.7
No		211	77.3

Note. SDS = Severity of Dependence Scale; AUDIT = Alcohol Use Disorders Identification Test.

Table 2Means, standard deviations and Cronbach's alphas of the measured variables ($N = 273$).

Scale	<i>M</i>	<i>SD</i>	α
Sensitivity to Reward Scale ^a	4.05	2.52	.72 (.83)
Dysfunctional Impulsivity Scale ^b	3.04	2.94	.64 (.90)
Cannabis Expectancy Questionnaire			
Positive ^c	51.69	12.48	.92
Cannabis Refusal Self-Efficacy			
Questionnaire			
Emotional Relief ^d	27.76	8.81	.97
Opportunistic ^d	21.24	7.08	.92
Social Facilitation ^d	15.32	3.50	.87
Severity of Cannabis Dependence Scale ^e	2.26	2.48	.77

Note. All sum scores. The poly-choric Cronbach's alphas of the dichotomous scales are presented in brackets.

^aHigher scores reflect greater reward sensitivity.

^bHigher scores reflect greater rash impulsiveness.

^cHigher scores reflect higher positive cannabis outcome expectancies.

^dHigher scores reflect greater cannabis refusal self-efficacy.

^eHigher scores reflect greater cannabis dependence severity.

Table 3Pearson's correlations between the measured variables ($N = 273$).

Variable	1.	2.	3.	4.	5.	6.	7.	8.
1. Reward sensitivity	-							
2. Rash impulsiveness	.23 ^{***}	-						
3. Positive cannabis expectancies	.16 [*]	.03	-					
4. Emotional relief refusal self-efficacy	-.15 [*]	-.38 ^{***}	-.30 ^{***}	-				
5. Opportunistic refusal self-efficacy	-.18 ^{**}	-.27 ^{***}	-.28 ^{***}	.76 ^{***}	-			
6. Social facilitation refusal self-efficacy	-.03	-.24 ^{***}	-.31 ^{***}	.74 ^{***}	.65 ^{***}	-		
7. Cannabis use	.22 ^{**}	.21 ^{**}	.25 ^{***}	-.37 ^{***}	-.36 ^{***}	-.32 ^{***}	-	
8. Severity of cannabis dependence	.20 ^{**}	.30 ^{***}	.22 ^{***}	-.54 ^{***}	-.42 ^{***}	-.35 ^{***}	.32 ^{***}	-

Note. * $p < .050$; ** $p < .010$; *** $p < .001$

Table 4
Fit Indices for the Hypothesized Structural Models ($n = 269$).

Model	$\chi^2(df)$	CFI	TLI	GFI	NFI	RMSEA (CI90%)	SRMR	$\Delta\chi^2(df)$
1. Full Mediation Model – cannabis use	54.92* (24)	.97	.96	.997	.95	.07 (.05 - .10)	.04	
2. Partial Mediation Model – cannabis use	48.08* (22)	.98	.96	.997	.96	.07 (.04 - .10)	.03	
3. Full Mediation Model – severity of dependence	54.04* (24)	.97	.96	.997	.95	.07 (.05 - .10)	.04	
4. Partial Mediation Model – severity of dependence	48.70* (22)	.98	.96	.997	.98	.07 (.04 - .10)	.03	
Difference between Model 1 and 2								5.94 [#] (2)
Difference between Model 3 and 4								5.34 [^] (2)

Note. CFI = comparative fit index; TLI = Tucker Lewis index; GFI = goodness-of-fit index; NFI = normed fit index; RMSEA = root mean-square error of approximation; SRMR = standardized root mean square residual.

* $p < .001$; # $p = .050$; ^ $p = .070$

Table 5
Summary of Direct Effects from the Biosocial Cognitive Models ($n = 269$).

Pathway	Standardized Coefficients (β)	Unstandardized Coefficients	Standard Error (SE)	p -value
<i>Cannabis Use</i>				
RS \rightarrow PCE	.18	.30	.11	.006
PCE \rightarrow CRSE	-.37	-.75	.13	<.001
PCE \rightarrow Can Use	.11	.20	.08	.017
CRSE \rightarrow Can Use	-.38	-.33	.06	<.001
RI \rightarrow CRSE	-.36	-1.06	.18	<.001
<i>Severity of Dependence</i>				
RS \rightarrow PCE	.18	.30	.11	.006
PCE \rightarrow CRSE	-.37	-.75	.13	<.001
PCE \rightarrow Can Dep	.01	.01	.05	.905
CRSE \rightarrow Can Dep	-.55	-.16	.02	<.001
RI \rightarrow CRSE	-.37	-1.09	.18	<.001

Note. RS = Reward Sensitivity; RI = Rash Impulsiveness; PCE = Positive Cannabis Expectancies; CRSE = Cannabis Refusal Self-Efficacy; Can Use = Cannabis Use; Can Dep = Severity of Cannabis Dependence.

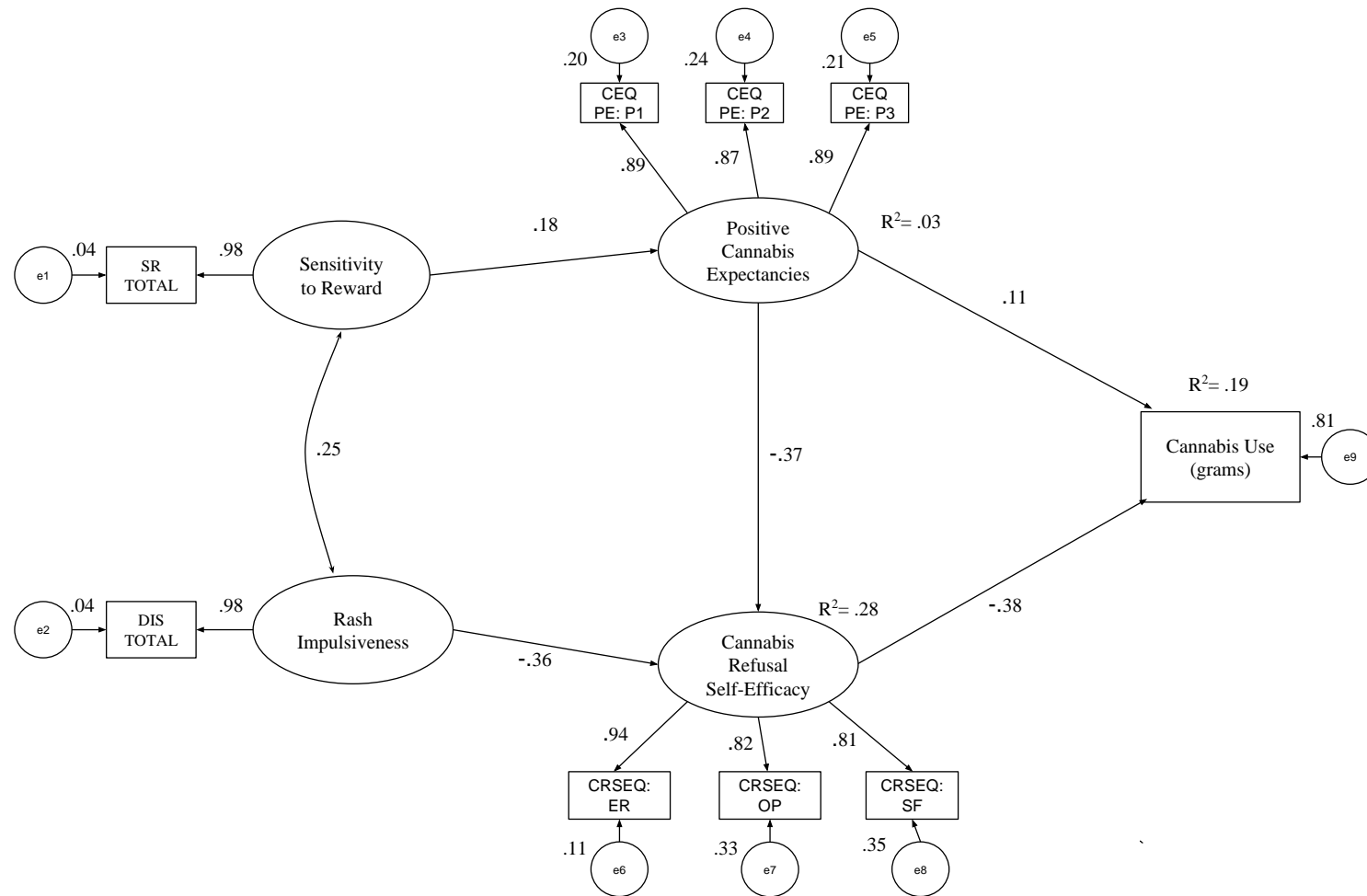


Figure 2.

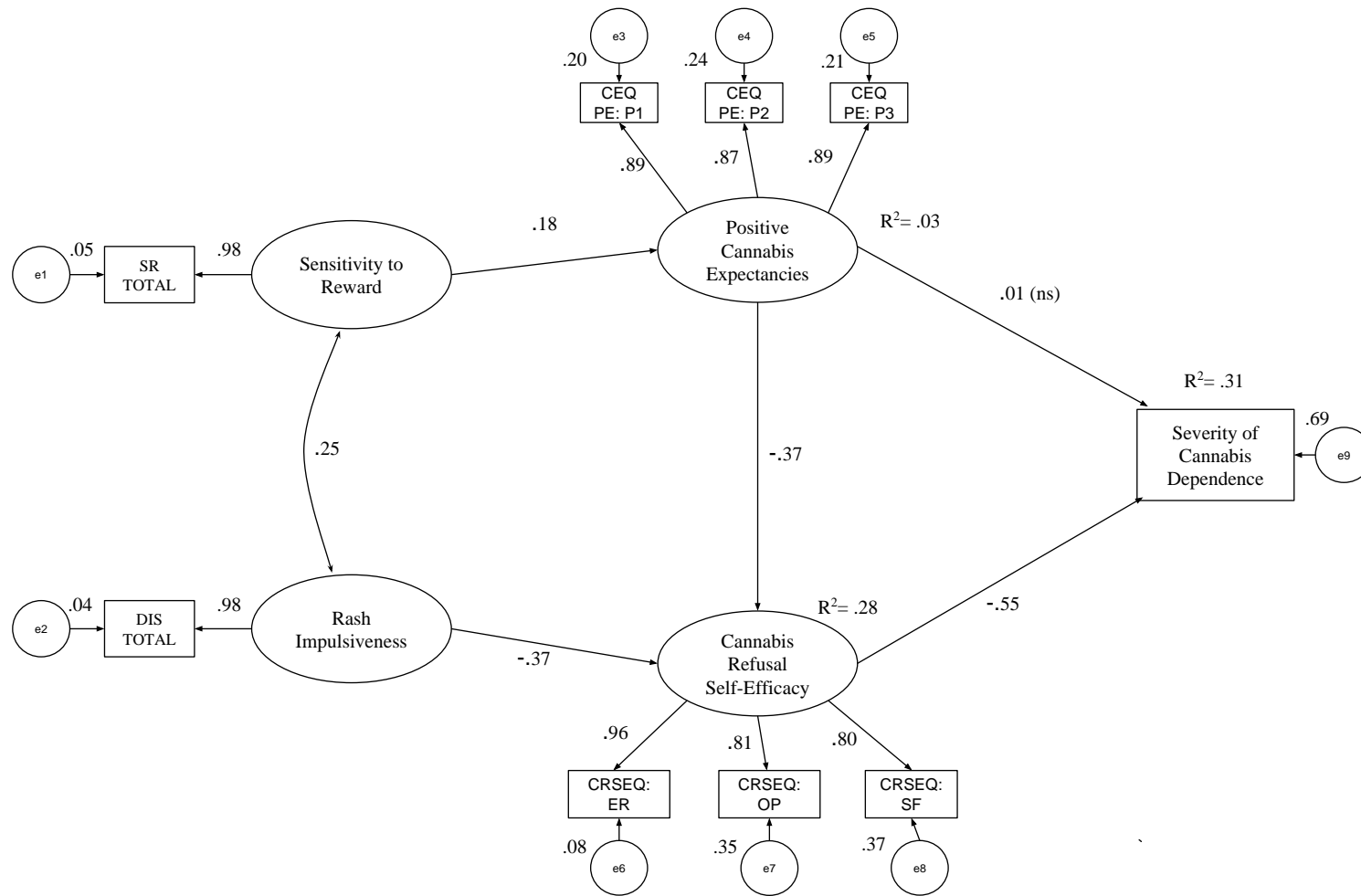


Figure 3.

Figure Captions

Figure 1. Biosocial Cognitive Model of Cannabis Use and Cannabis Dependence Severity.

Figure 2. The Biosocial Cognitive Model of Cannabis Use. Standardized parameter estimates are presented. All estimates are statistically significant at $p < .05$.

Figure 3. The Biosocial Cognitive Model of Severity of Cannabis Dependence. Standardized parameter estimates are presented. All estimates are statistically significant at $p < .05$, except where indicated (ns).

Author Contributions

Zoë Papinczak and Dr Matthew Gullo designed the study. Zoë Papinczak conducted the literature review, undertook the statistical analyses and wrote the first draft of the manuscript. All authors contributed to and approved the final manuscript.

AUTHOR DISCLOSURES

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Author Contributions

Zoë Papinczak and Dr Matthew Gullo designed the study. Zoë Papinczak conducted the literature review, undertook the statistical analyses and wrote the first draft of the manuscript. All authors contributed to and approved the final manuscript.

Conflict of Interest

None declared.

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Conflict of Interest

None declared.

HIGHLIGHTS

- Impulsivity associated with cannabis use and dependence via social cognition.
- Reward sensitivity associated with cannabis outcomes via positive expectancies.
- Rash impulsiveness associated with cannabis outcomes via refusal self-efficacy.
- Targeting social cognition may reduce risk conveyed by an impulsive personality.

Supplementary Material

Data Analyses

Total scores on the three subscales of the CRSEQ served as indicators for the latent cannabis refusal self-efficacy factor (Young et al., 2012). For the positive cannabis expectancies latent factor, random parcels of items from the CEQ-P were formed to create the indicators as the CEQ-P is a unidimensional scale (Little et al., 2002; Matsunaga, 2008). As the measures of reward sensitivity and rash impulsiveness contained a smaller number of dichotomously-scored items (10-12 items), these personality constructs were modelled as single-indicator latent variables, each with their measurement error set as 1 – Cronbach's alpha (Bollen, 1989; Sass and Smith, 2006). Cannabis use (grams) and severity of dependence (SDS-C total score) were modeled as non-latent measured variables, as in Connor et al. (Connor et al., 2013).

Mediation was tested in two ways. First, the fit of the hypothesized bSCT model (specifying full mediation) was compared to that of a partial mediation bSCT model that specified direct associations between impulsivity traits and cannabis outcomes. Models were compared using the χ^2 difference test ($\Delta\chi^2$) (Holmbeck, 1997). Second, the indirect mediation effect itself was estimated using *RMediation* package, which utilises the distribution-of-the-product method (Tofighi and MacKinnon, 2011). Robust standard errors were used when calculating the indirect effects (Preacher, 2015).

Data Screening and Assumption Checks

The original data set contained the responses of 346 previously unanalysed cases. Overall, there was 14.1% missing data. 52 participants were missing more than 50% of their data and thus were excluded from further analyses. A further 21 participants were excluded as they had not completed any items on at least one of the model measures. A sample size of 273 remained, in which there was 0.82% missing data. Data were missing completely at random

(MCAR) as Little's (Little, 1988) MCAR test was non-significant, $\chi^2(181) = 180.41, p = 0.498$. Missing data were estimated using the Full Information Maximum Likelihood method (Graham, 2009).

The data were screened for outliers and this revealed that 12 participants were univariate outliers and 4 participants were multivariate outliers. The multivariate outliers significantly reduced multivariate normality and inflated model fit and therefore were excluded from further analyses, leaving 269 cases. While this improved multivariate normality, Mardia's test revealed that this assumption was still violated in both the cannabis use model (normalized coefficient = 29.90, $p < .001$) and severity of dependence model (normalized coefficient = 7.27, $p < .001$). Therefore, Yuan-Bentler corrected model fit statistics and Huber-White robust standard errors were used across the analyses to control for any potential biases (Rosseel, 2012; Tong et al., 2014; Yuan and Bentler, 2000).

The models were re-run controlling for the effects of sex, age, hazardous alcohol use (as indicated by total scores on the Alcohol Use Disorders Identification Test [AUDIT; (Saunders et al., 1993)] and psychological functioning (as indicated by total scores on the General Health Questionnaire [GHQ-28, (Goldberg and Williams, 1998)]). Results were unaffected. Neither sex nor age were significantly associated with the cannabis outcomes, consistent with Connor et al. (2013). Similarly, hazardous alcohol use and psychological functioning were not associated with cannabis use or severity of cannabis dependence. Therefore, these were not retained in the final models.

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