Abstract
My thesis explored the relation between cannabis use (CU) and two distinct subtypes of aggression. Substantial prior research has examined the association between CU and aggression; however, empirical evidence has not yet provided a clear or complete picture of this relationship. This may be due to the fact that to date no studies have considered the important distinction between instrumental aggression (IA) and reactive aggression (RA). The aim of this study was to differentiate these subtypes of aggression while controlling for covariates such as psychopathy and trait aggression which has the potential to reconcile the apparently contradictory findings in the literature. Also of interest was the role that gender plays in the relationship between cannabis and IA/RA. Samples from three different populations, who all completed a series of self-report questionnaires, were examined for this study. The sample from the university population consisted of 427 participants, the sample from the online population consisted of 434 participants, and the sample from the treatment population consisted of 68 participants. The data were analysed using independent samples t tests, hierarchical multiple regressions, and two-way ANOVAs. The results from all three samples indicated that there is little to no relation between CU and IA/RA. In cases where a relation was found, it disappeared when accounting for psychopathy and trait aggression. These findings were consistent across all frequencies of CU. Furthermore, the results showed that there was no interaction between gender and any frequency of CU when looking at the relation with IA/RA. Collectively, these findings indicate that CU is not associated with aggressive behaviour and propose answers to the question of why there is such differing findings in the existing literature. The public health impact of CU remains controversial and these findings have important theoretical, methodological, and clinical implications.
Preface

Ethics approval for my research was approved by the University of British Columbia’s Research Ethics Board. The ethics approval certificate number for my research project is H14-00296.

There have been no publications using the data from this study to date.
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Dedication

I would like to dedicate this thesis to my father, Robin Carroll, who passed away during the early stages of this project. You are the one who inspired me to be the man I am today and I am forever grateful for all of your leadership and guidance throughout my life. I wish I had the opportunity to show you the end result of the hard work and dedication I put into this project but I know that I made you proud. Gone but not forgotten.
CHAPTER 1 Introduction

Clinical and cultural lore has specifically proposed a positive relationship between cannabis use (CU) and the propensity to engage in aggressive behaviour; however, no conclusive evidence has been found to determine whether such an association exists. The most notable example of the sensationalizing of aggression associated with CU is in the 1936 ‘educational’ film *Reefer Madness*, where the foreword warns viewers that CU leads to “acts of shocking violence...ending in incurable insanity” (Boyd, 2010). Produced with the support of the Federal Bureau of Narcotics, *Reefer Madness* depicts white middle-class youth as vulnerable to the negative effects of CU including violent outbursts and psychosis (Starks, 1983). Shortly after the film's release, the 1937 *Marijuana Tax Act* in the US was enacted and cannabis has since been criminalized in federal law. Further, the putative association between CU and aggression has played an important role in the establishment of prohibitionist policies, and the war on drugs has been fueled by unsupported claims of the negative effects of CU.

Regardless of the unsubstantiated negative effects and legal restrictions that have been placed on cannabis, CU has flourished and it currently stands as the most used illicit substance in Canada and throughout the world. It is estimated that 180 million people worldwide use cannabis, of which Canadians account for over 3.5 million users (Statistics Canada, 2012). The start of this trend was during the 1960s when the number of cannabis users in Canada grew exponentially (Alexander, 2003). This was also the time when research efforts began to focus on the medical and psychological effects of cannabis. Today, a substantial amount of information pertaining to the psychological implications of cannabis is founded on empirical data, leaving little doubt about the reliability and validity of the findings.
On the other hand, empirical findings related to the association between aggression and cannabis are equivocal; some research has suggested that CU is positively associated with aggressive behaviour, due to alterations of higher cognitive functioning (Moore & Stewart, 2005), negative consequences of a cannabis withdrawal syndrome (Milin, Manion, Dare, & Walker, 2008), or tolerance to deviance, risk-taking behaviour, and rebelliousness (Hall & Degenhardt, 2007). In contrast, other research has concluded that CU is not associated with aggression because of its sedative and quieting nature (Pujazon-Zazik & Park, 2009), increased positive mood states (Plancherel et al., 2005), and the preconceived expectancies of CU (Alfonso & Dunn, 2007). It has been suggested that a possible reason for this lack of resolution is that past research has focused on a variety of aggressive behaviours and has not fully differentiated the subtypes of aggression when looking at the effect of CU (Ostrowsky, 2011).

The distinction between instrumental aggression (IA) and reactive aggression (RA) has the potential to elucidate this apparent discrepancy. IA is characterised by planned and unprovoked behaviour that is a means to an end, such as money or power, whereas RA is characterised by impulsive and defensive responses to provocation or frustration, or an end in itself (Tapscott, Hancock, & Hoaken, 2012). Examining the unique dimensions that encompass both subtypes of aggression will help answer the question of whether or not CU is associated with aggression.

1.1 Theories regarding the relationship between cannabis and aggression

A tripartite conceptual framework has been proposed to explain the relationship between substance use and aggression (Goldstein, 1985). The first factor of this model is the economic compulsive model and suggests that some people may act in an aggressive manner for economic gain in order to support costly drug use (Goldstein, 1985; Oser, Mooney, Staton-Tindall, &
Leukefeld, 2009). However, the aggression associated with acts such as robbery and theft perpetuated by the motivation for money to purchase drugs has been proposed to be largely related to contextual factors and not actual drug use (Weiner, Sussman, Sun, & Dent, 2005). Since the purpose of this study was to examine the relation between CU and aggression, the economic compulsive model does not consider the main variables of interest for the current study.

The second factor of the model is systemic aggression, which is intrinsic to criminal behaviour surrounding the drug trade, and refers to the traditionally aggressive patterns of interaction within illicit drug distribution and use (Goldstein, 1985). Trafficking and obtaining of an illegal substance lead to violent and aggressive behaviour for the purpose of economic gain, to support a drug habit, or to maintain the rules and regulations of the drug trade (White & Hansell, 1998). For instance, disputes over territory between rival dealers, to chastise or eliminate informants, and punishment for failing to pay an owed debt are examples of violent behaviour considered to be systemic aggression (Boles & Miotto, 2003). However, the emphasis of systemic aggression is on actions within the drug trade and not on substance use and therefore is also not of interest to this study.

The third and final factor of the model is psychopharmacological aggression (Goldstein, 1985) and, of the three, is most pertinent to this study. Psychopharmacological aggression has since been expanded to be the psychopharmacological model (Kuhns & Clodfelter, 2009; Pihl & Hoaken, 1997; White & Hansell, 1998) and postulates that the negative effects of substances increase irrational behaviour and arousal leading to aggressive or violent behaviour. CU is associated with many psychopharmacological effects including cognitive impairments. Even though long-term CU does not appear to produce any severe impairment in cognitive
functioning, chronic CU may be related to minor cognitive impairments including complex executive functions and problems with attention (Gray, 1999). For example, cannabis users have shown a greater deterioration in tests of reaction time, speed, and accuracy when compared to nonusers (Mendhiratta et al., 1988; Thames, Arbid, & Sayegh, 2014). In addition, heavy users evidenced significantly greater impairments in attention and executive functioning compared to light users (Pope & Yurgelun-Todd, 1996; Battisti et al., 2010).

It also has been shown that acute cannabis intoxication can alter different aspects of memory functioning, including short-term memory, recognition memory, and free recall, as well as other higher cognitive functions (Hart, van Gorp, Haney, Foltin, & Fischman, 2001; Maykut, 1985). What is of specific interest is the consistent effect of acute cannabis intoxication on users’ ability to split their attention between tasks and perform complex reaction time tasks. Several studies have demonstrated that CU lowers one’s ability to attend to multiple stimuli simultaneously across the field of vision, increases errors in identifying peripheral stimuli, and increases reaction time to peripheral stimuli (Barnett, Licko, & Thompson, 1985; Harbey, Sellman, Porter, & Frampton, 2007; Marks & MacAvoy, 1989). Also, CU has been linked to increases in the number of premature responses on reaction time tasks and the amount of time needed to read stories and complete mental calculations (Hart, van Gorp, Haney, Foltin, & Fischman, 2001). Hart et al. (2001) concluded that acute CU is also related to difficulties inhibiting inappropriate responding and is indicative of executive cognitive impairments.

These cognitive impairments may have important implications for explaining the relationship between CU and aggression. For example, acute and chronic CU may decrease the ability to attend and process complex stimuli that are likely present in situations of interpersonal conflict, decrease the ability to inhibit inappropriate responding, and increase premature
responding in conflict situations, all of which may increase the likelihood of an aggressive response to conflict (Howard & Menkes, 2007). In other words, both simple and complex cognitive functions negatively affected by CU in laboratory tests may be similar to those required to effectively cope with interpersonal conflict, thereby increasing risk for reactive aggression.

Related to the cognitive effects of acute CU are the cognitive effects of alcohol use. Alcohol myopia (Steele & Josephs, 1990) is a model that provides the framework that can help explain alcohol-related aggression. According to Steele and Josephs (1990), alcohol has a ‘myopic’ effect on attentional capacity by restricting the range of internal and external cues that can be perceived and processed resulting in the allocation of attentional resources to the most salient cues. In hostile situations, this promotes aggression through focusing attention on provocative cues due to their more threatening and salient nature (Giancola, Duke, & Ritz, 2011). Although not researched with CU, these principles may also apply to the relation with aggression based on the cognitive effects mentioned above.

Another reason to propose an association between aggression and the psychopharmacological effects of CU is the withdrawal symptoms from cannabis dependence. A review on cannabis withdrawal demonstrated that aggression was consistently observed across studies (Budney, Moore, Vandrey, & Hughes, 2003). Based on the results, it is suggested that the relation between CU and aggression may be the strongest during periods of withdrawal, and that withdrawal periods may be just as predictive, if not more predictive, than acute intoxication (Budney et al., 2003). However, another review of the published literature notes that even though individuals with withdrawal symptoms may experience heightened agitation and possible
aggression, these symptoms are not specific to periods of cannabis withdrawal and may be due to personality factors (Smith, 2002).

Other acute and chronic psychopharmacological effects of cannabis related to aggression are panic reactions or panic disorders and paranoia (Sussman, Stacy, Dent, Simon, & Johnson, 1996). George et al. (2000) suggested that symptoms of autonomic arousal and a sense of fear or loss of control are associated with aggression and panic and that both conditions may involve exaggerated fear-related responses. CU is known to be associated with an increased likelihood for developing panic attacks and panic disorders (Zvolensky et al. 2008).

1.2 Contradictory findings

Despite the theory that the psychopharmacological model illustrates a positive connection between CU and aggression, the empirical evidence surrounding this connection remains inconclusive. Several prominent studies attempting to examine the relation between CU and aggression have come to differing conclusions regarding the connection. Positive, negative, and mixed relations have been reported in cross-sectional and prospective studies using questionnaire and survey data (Ardent et al., 2007; Denson & Earleywine, 2008; White & Hansell, 1998).

Regarding cross-sectional research, an analysis of 119 cannabis dependent patients from 19 substance dependence treatment centers found that subjects who reported problems controlling their violent behaviour attempted to use cannabis more frequently to reduce their aggression, but more often reacted with aggression while under the influence (Arendt et al., 2007). In contrast, Macdonald, Erickson, Wells, Hathaway, and Pakula (2008) found differing results while recruiting participants from various treatment agencies in Ontario. While looking at the 1019 patients enrolled in treatment programs for cocaine, cannabis (128 of whom were in treatment for a primary problem with cannabis), alcohol, tobacco, other drugs, and gambling, the
researchers found that the frequency of CU was not significantly related to aggression when controlling for other factors. However, because these studies used selective samples of those who sought treatment for substance abuse, the generalizability of these findings is questionable.

Further, personal in-home interviews on a representative U.S. sample of 18,352 participants were used to examine the relation of substance abuse and aggression/suicide. It was found that men who reported frequent CU were over twice as likely to report fighting while under the influence of cannabis alone than when under the influence of both cannabis and alcohol (Dawson, 1997). What is of specific interest is that this association was only seen in those who reported frequent CU and not in the entire population of the study indicating that the association may only be with a higher level of CU. Conversely, one of the largest studies to date examining the relationship between CU and aggression was facilitated by The Marijuana Policy Project, The National Organization for the Reform of Marijuana Laws, and The Drug Policy Alliance who sent a query to their mailing lists for participation (Denson & Earleywine, 2008). Data were collected from 6910 participants and it was concluded that there was no relation between CU and aggression once other factors were taken into account (such as gender, age, and other drug use), even among frequent, long-time users (Denson & Earleywine, 2008).

Longitudinal studies have reported equally inconsistent findings. For instance, Arseneault, Moffitt, Caspi, Taylor, and Silva (2000) conducted a study that examined the risk of aggression with alcohol dependence, cannabis dependence, and schizophrenic disorders. Utilizing a sample of 961 people (consisting of 91% of consecutive births in Dunedin, New Zealand for one year) followed from the age of 3-21 years, the results indicated that after controlling for sex, socioeconomic status, and other psychiatric disorders (including alcohol dependence), those qualified as being cannabis dependent were 3.8 times more likely to report
aggression over controls, and 28% of the risk for becoming violent was uniquely attributable to cannabis dependence. However, the Young in Norway Longitudinal Study found no evidence that CU is associated with an increased risk of subsequent non-drug-specific criminal charges, including violent crimes, when controlling for other factors (Pedersen & Skardhamar, 2010). The data were collected from a population-based sample of 1,353 Norwegian adolescents who were followed-up with four survey based data collections over a 13-year span from 1992-2005. While it is impressive that these studies were able to examine an entire birth cohort over several years, they failed to take into account the selective nature of their population, and thus their results may be due to cultural or cohort effects such as the differing drug use attitudes, cannabis expectancies, and the legal status of cannabis.

Although the majority of prospective studies conclude either a positive or no association between CU and aggression, a few studies reveal mixed or contradictory findings. For instance, in a longitudinal study that followed adolescents for 15 years to examine the effects of drug use on aggression, it was found that CU had a negative effect on aggression from early (ages 12-18) to late adolescence (ages 15-21), but had a significant positive effect from late adolescence (ages 18-21) to early adulthood (ages 25-28) (White & Hansell, 1998). White and Hansell (1998) recruited 1,201 participants from across the United States who were tested four times between 1979 and 1994. Moreover, using data from a sample of 437 high school or adjudicated delinquent males, Watts and Wright (1990) found that CU did not predict aggression among adolescent Mexican-Americans, but did significantly predict aggression among Caucasians and African Americans. Lastly, using self-report survey data from 4,390 high school seniors and dropouts from California and Oregon from a 5-year follow-up study, it was found that the frequency of CU measured at grade 7 was a significant predictor of the amount of aggression.
engaged in at grade 12, but did not significantly predict the occurrence of grade 12 aggression (Ellickson & McGuigan 2000).

Given the mixed conclusions of previous research, it is clear that we do not have a definitive answer regarding the association between CU and aggression. Because of this, further exploration on the issue at hand is needed. However, in order to conduct the necessary research, the possible reasons for the contradictory findings must first be identified.

1.3 Reasons for contradictory findings

1.3.1 Subtypes of aggression

One possible reason for the contradictory findings is that previous studies have not differentiated the aforementioned subtypes of aggression. More specifically, research has shown that there is a two-factor model for aggressive behaviour (Crick & Dodge, 1996; Dodge & Coie, 1987; Poulin & Boivin, 2000). First characterized by competing theories of aggression (Bandura, 1983; Berkowitz, 1983), instrumental aggression (IA) has been described as "non-provoked aversive acts aimed at influencing others in an attempt to gain resources or for the purposes of domination or intimidation" (Dodge & Coie, 1987, p. 1147). Reactive aggression (RA) can be described as "a hostile act displayed in response to provocation or a perceived threat and is generally impulsive and occurs with strong negative affect" (Dodge & Coie, 1987, p. 1147). Poulin and Boivin (2000) provided empirical support for the construct validity of the two subtypes of aggression presenting distinct patterns of relations that are congruous with the theoretical definitions. However, the study also demonstrated a high correlation between the subtypes of aggression suggesting that some aggressive behaviour may contain elements of both reactivity and instrumentality.
Even though the two subtypes of aggression are highly correlated, they consistently produce unique dimensions (Boivin, Dodge, & Coie, 1995; Coie, Dodge, Terry, & Wright, 1991; Dodge, Price, Coie, & Christopoulous, 1990). For example, RA in adolescents is uniquely associated with negative emotionality, specifically anxiety, in adulthood (Fite, Raine, Stouthamer-Loeber, Loeber, & Pardini, 2009). In contrast, IA in adolescents is uniquely associated with measures of adult psychopathic features and antisocial behaviour in adulthood (Fite et al., 2009). Furthermore, IA children are more likely to engage in online acts of bullying when compared to RA children, who were more likely to engage in face-to-face bullying (Burton, Florell, & Gore, 2013).

The clear distinction between the subtypes of aggression is important due to the reactive nature of the aggression that is primarily associated with CU. Based on the psychopharmacological effects mentioned above, the aggression that would be related to CU is arguably spontaneous, provoked, or a reaction to external influences. According to social information processing models, RA is a result of a deficit in the interpretation stages of a situation where the aggressor exhibits hostile attributional biases in response to ambiguous provocation situations (Crick & Dodge, 1996). With this information in mind, such deficits could result from the acute cognitive impairments that CU can cause. Moreover, the aggression associated with the effects of withdrawal and panic reactions would likely be considered RA.

1.3.2 Limitations of the populations

The most common limitation seen in prior research is inherent in the populations of the studies and the measures used. Previous research has recruited participants from a selective or biased population making their results less generalizable. Drawing samples from a selective population, such as the single cohort samples from small cities, may not give an accurate representation of
the general public and the results may be due to the location of data collection. Factors such as
the legal restrictions placed on drugs (van Ours, 2007), expectancies of CU (Kristjansson,
Agrawal, Lynskey, & Chassin, 2012), and overall attitudes regarding cannabis (Simons & Carey,
2000) result in different negative effects of CU, including aggression.

Analysis of samples taken from treatment centres were focused on cannabis dependent
individuals who also are not an accurate representation of the general public. And lastly,
populations that were drawn from advocates of the pro-cannabis movement may have inherent
biases revolving around cannabis and aggression resulting in skewed results that would favour a
negative association. Further, all of the aforementioned studies used different measures of
aggression and CU making it difficult to interpret discrepant results between the studies. As a
whole, the samples would produce an incomplete representation of RA and IA or of CU in
society. Obtaining information from multiple samples while consistently measuring the
association of these two main types of aggression and CU is imperative to clarifying the
controversy in the research.

1.3.3 Covariates

One final reason for the contradictory findings in the literature may be due to a lack of
controlling for some of the most pertinent covariates. Previous research has failed to take into
account the effect of covariates such as trait aggressive behaviour and psychopathy when looking
at the influence of CU on aggressive behaviour. Trait aggressive behaviour can be defined as a
proneness to act in an aggressive manner to certain situations due to a comparatively stable
personality predisposition (Coccaro, Berman, & Kavoussi, 1997). Whereas state aggression
involves immediate aggressive actions towards provocation or anger-related cues and are not due
to a personality predisposition (Krcmar & Farrar, 2006). Failure to control for aggression due to
a personality predisposition may have impacted the state aggression attributed to CU in previous studies.

The other covariate of importance is psychopathy which is known to be strongly associated with aggressive behaviour (Hart & Hare, 1997; Porter & Woodworth, 2006). For example, when comparing inmates who had high levels of psychopathy with those who had low levels of psychopathy, it was found that high psychopathy scores were related to an increase of post-release violence four times greater than low psychopathy scores (Hart, Kropp, & Hare, 1988). Psychopathy has also been shown to be associated with both IA (Walsh, Swogger, & Kosson, 2009) and RA (Blair, 2010). In fact, recent evidence has shown that these associations are distinct for each subtype of violence (Hecht, Berg, Lilienfeld, & Latzman, 2015). Similar to trait aggression, psychopathy poses a predisposition to aggressive behaviour and may have influenced the findings in past literature.

1.4 Aims and hypotheses

The specific aims of my thesis were twofold. First, I examined the relation between CU and IA/RA while controlling for trait aggression and personality factors (i.e., psychopathy). This included an exploration of the difference in patterns and prevalence of IA/RA between cannabis users and non-users. Second, I provide a more refined examination of the confounding factors that are problematic in previous research to clarify the contradictory findings. The specific confounding factors of interest included the effect of frequency of CU, gender differences, and the populations under examination.

My analyses focused on the limitations in the populations of previous research to make my results more consistent regarding the measures used and to increase the generalizability. Previous research has primarily focused on one type of population for their analysis, the most
common being university students (Watts & Write, 1990; White & Hansell, 1998), patients in addictions treatment centers (Arendt et al., 2007; Macdonald, Erickson, Wells, Hathaway, & Pakula, 2008), or single cohort samples from a distinct cultural background (Arseneault, Moffitt, Caspi, Taylor, & Silva, 2000; Pedersen & Skardhamar, 2010). The current study utilized all three of these populations (university students at UBCO, treatment patients at The Bridge, and Amazon’s mechanical Turk [MTurk] for a community sample) to provide a more detailed look at the association of CU and aggression, and to examine the extent to which previous contradictory findings may be due to population biases.

In addition, frequency of CU is a factor that warranted further examination. Empirical evidence indicates a link between anxiety and frequency of CU, specifically problematic CU (Agosti, Nunes, & Levin, 2002; Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007; Oyefeso, 1991). This association is shown to be strongest among anxiety conditions where panic is common (Buckner et al., 2008), and panic may be a catalyst in the association between CU and aggression. In turn, I will be using the Cannabis Use Disorder Identification Test (CUDIT) (Adamson & Sellman, 2003) to examine frequency of CU and problematic CU. Frequency of CU, as determined by the CUDIT, may account for some of the variance in the relation under examination, and controlling for two other covariates will give us a more complete picture of the relationship.

Gender also plays a key role in substance use and aggression and may be a contributing factor in the differences between IA/RA as they relate to CU. For example, social stigma helps to differentiate substance use patterns among males and females (Lancaster, 1994). Females experience a greater amount of the social stigma and social disapproval regarding substance use resulting in more prevalent diagnosable substance use disorders in males but greater social
problems associated with substance use in females (Brady & Randall, 1999). Aggression also has a stronger association with males as they perpetrate the majority of all violent crimes, and their aggression is often more severe than that perpetrated by females (Gammelgard et al., 2012). For these reasons it was important to examine the differences in gender when completing the analyses for this study.

Lastly, I also controlled for covariance due to two other related factors. First, I used the Life History of Aggression Questionnaire (LHA) (Coccaro, Berman, & Kavoussi, 1997) to control for trait aggressive behaviours. Observed associations between CU and aggression may reflect a third factor such as trait aggressive behaviour and should be controlled for to ensure that the relationship found is related to CU and not a third factor. Second, I will use the Self-Report Psychopathy scale (SRP-III) to control for psychopathy in the populations. Psychopathy is related to the prediction of aggression and measures such as the SRP-III are recognized to be reliable predictors of aggression in numerous contexts across diverse samples (Coid et al., 2009; Gordts et al., 2015; Hare, 2003; Hemphill, Hare & Wong, 1998; Skeem & Mulvey, 2001; Walsh & Kosson, 2008).

Regarding the effect of CU on the subtypes of aggression, my first hypothesis is that we will find a relation between CU and RA due to the psychopharmacological effects of cannabis discussed earlier. My second hypothesis is that a higher frequency of CU will result in higher levels of RA because of the increased problems associated with higher CU frequency and the aforementioned hypothesised association between CU and RA. Lastly, it is hypothesized that, among the cannabis users, we will find a distinct relationship between gender and subtypes of aggression. More specifically, it is predicted that females will exhibit higher levels of IA associated with their CU due to the social implications of premeditated aggression and males will
display higher levels of RA associated with their cannabis use due to their higher levels of aggression in general and the impulsive nature of RA.

The public health impact of CU remains controversial, and the elucidation of the association between cannabis and aggression has the potential to inform this important ongoing debate. The proposed study addressed the controversy that surrounds the relation between CU and aggressive behaviour. A better understanding of the role of substance use within the context of aggression will be valuable in designing prevention and treatment programs that can be used in the community to mitigate some of the negative effects of both aggression and CU on society. For example, cannabis cessation could be incorporated in aggression management programs if found to be related to subtypes of aggression.
CHAPTER 2 Method

2.1 Participants

2.1.1 University population

The first group of participants was drawn from a sample of undergraduate students from the University of British Columbia Okanagan campus. Previous research suggests that the frequency of CU is a significant predictor of predatory aggression in late adolescence and early adulthood (Ellickson & McGuigan, 2000). Moreover, university students also exhibit high rates of substance use, with approximately one third reporting hazardous drinking and CU, either independently or in combination (Centre for Addiction and Mental Health, 2005), which makes an undergraduate sample appropriate for this particular project. Eligibility requirements included being at least 17 years old, fluent in English, and enrolled in a course offering credit for participating in research projects.

A total of 427 university students (age range: 17-35 years) were recruited to participate in an online survey. There was a range of ethnic backgrounds (283 Caucasian [66.28%], 45 Chinese [10.54%], 20 South East Asian [4.68%], 19 South Asian [4.45%], 19 Arab/West Asian [4.45%], 11 Latin American [2.58%], 10 Korean [2.34%], 8 Aboriginal [1.87%], 5 African American [1.17%], 5 Japanese [1.17%], and 2 Filipino [0.47%]), as well as a range of religious affiliation (316 not affiliated [74.00%], 36 Catholic [8.43%], 32 Protestant [7.49%], 31 Other [7.26%], 7 Muslim [1.64%], 4 Jewish [0.94%], and 1 Orthodox [0.23%]). See Table 1 for demographic information and CU history.
2.1.2 Online population

The second sample was drawn from a larger online population through Amazon's Mechanical Turk (MTurk). It has been shown that larger online pools of participants, namely MTurk samples, are significantly more diverse when compared to common university samples and are more demographically diverse than typical internet samples (Buhrmester, Kwang, & Gosling, 2011). Participants can be recruited rapidly and inexpensively, realistic compensation rates do not affect data quality, and the results acquired are as reliable as those obtained through traditional methods (Buhrmester, Kwang, & Gosling, 2011). Eligibility criteria for this sample included being at least 17 years old, fluent in English, and residents of North America.

A total of 434 participants (age range: 18-71 years) were recruited to participate in an online survey through MTURK. There was a range of ethnic backgrounds (342 Caucasian [78.80%], 30 African American [6.91%], 23 Latin American [5.30%], 9 Chinese [2.07%], 8 Filipino [1.84%], 5 South East Asian [1.15%], 5 South Asian [1.15%], 4 Aboriginal [0.92%], 4 Japanese [0.92%], 3 Arab/West Asian [0.69%], and 1 Korean [0.2%]), as well as a range of religious affiliation (304 not affiliated [70.0%], 68 Protestant [15.7%], 30 Catholic [6.9%], 21 Other [4.8%], 9 Jewish [2.1%], 1 Muslim [0.2%], and 1 Orthodox [0.23%]). See Table 1 for demographic information and CU history.

2.1.3 Treatment population

The third group of participants was drawn from individuals enrolled in a residential treatment program within the community. As mentioned above, prior research has shown that cannabis dependent patients in treatment programs who reported problems controlling their aggressive behaviour attempted to use cannabis more frequently to reduce their aggression, but more often
reacted with aggression while under the influence (Arendt et al., 2007). Eligibility requirements included being at least 17 years old, fluent in English, and enrolled in the 6-week treatment program for drug/alcohol addiction.

A total of 68 participants (age range: 20-71 years) were recruited to participate in a semi-structured interview and survey. There was a range of ethnic backgrounds (55 Caucasian [80.88%], 12 Aboriginal [17.65%], and 1 South Asian [1.47%]). See Table 1 for demographic information and CU history.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>University</th>
<th>Online</th>
<th>Treatment</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>M age (SD)</strong></td>
<td>20.10 (2.16)</td>
<td>35.59 (10.64)</td>
<td>40.34 (12.12)</td>
<td>467.15*</td>
</tr>
<tr>
<td><strong>% Female (N)</strong></td>
<td>70.26 (300)</td>
<td>51.38 (223)</td>
<td>52.94 (36)</td>
<td>17.37*</td>
</tr>
<tr>
<td><strong>% Any CU (N)</strong></td>
<td>53.40 (228)</td>
<td>60.83 (264)</td>
<td>82.35 (56)</td>
<td>13.24*</td>
</tr>
<tr>
<td><strong>% Current CU (N)</strong></td>
<td>35.83 (153)</td>
<td>21.43 (93)</td>
<td>57.35 (39)</td>
<td>13.24*</td>
</tr>
<tr>
<td><strong>% Infrequent CU (N)</strong></td>
<td>28.57 (122)</td>
<td>8.29 (36)</td>
<td>23.53(16)</td>
<td>28.97*</td>
</tr>
<tr>
<td><strong>% Frequent CU (N)</strong></td>
<td>7.26 (31)</td>
<td>13.13 (57)</td>
<td>33.82(23)</td>
<td>28.97*</td>
</tr>
</tbody>
</table>

Note. *p < .001

### 2.2 Procedure

#### 2.2.1 University sample

All university participants were recruited through the SONA Research Participation System at the University of British Columbia – Okanagan. SONA is an online recruitment system that
allows students at the university to participate in research for course credit. A link to the survey, created through Qualtrics, was posted on SONA. Qualtrics is a private research software, for which UBC has a licence, and enables researchers to build a survey and distribute it to potential participants using the website link. Participants were informed that the survey would take approximately 60 min to complete and were compensated one course credit for participating.

2.2.2 Online sample

Participants in the online group were recruited through Amazon’s Mechanical Turk (MTurk), an online crowdsourcing marketplace hosted by Amazon where researchers can recruit qualified individuals (also known as ‘workers’) worldwide to participate in research projects. A link to my survey through Qualtrics was posted on the MTurk website and was available to any worker within North America. Participants were told that the survey would take approximately 60 min to complete and were compensated $3.00 CAD for participating in the survey. To ensure the validity of the responses, we also incorporated a Chapta verification code and an attention filter question within the survey as attentional checks. Lastly, we included a survey end code to establish which participants fully completed the survey to be compensated appropriately.

2.2.3 Treatment sample

All treatment participants were recruited at The Bridge Residential Treatment Facility in Kelowna, BC, Canada. The Bridge is an intensive residential treatment facility that offers a treatment program from which the third group of participants were recruited. The Bridge provides treatment for substance use disorders in 20 residential beds that alternates between a 6-week program for males and a 6-week program for females. The measures in my study were embedded in a larger project examining aggression and addiction. Pre-consent was gained by the
staff at The Bridge in order for the research assistant to approach the participant. Full consent was then obtained by the research assistant. Individuals recruited participated in a semi-structured interview and then were asked to fill out self-report measures. Total participation time was approximately 1.5 hr and participants were compensated $20.00 CAD for their participation.

2.3 Materials

2.3.1 IA and RA

The Impulsive/Premeditated Aggression Scale (IPAS) was designed to characterise aggressive behaviour as predominantly premeditated (IA) or predominantly impulsive (RA) in nature (Stanford et al., 2003). Participants were asked to consider their aggressive acts during the past year and complete the IPAS in relation to those acts. Fifteen of the items focus on impulsive aggressive (IA) characteristics and 15 items focus on premeditated aggressive (PM) characteristics. The items are scored on a 5-point numeric scale that ranges from 1 (Strongly disagree) to 5 (Strongly agree). This measure has been shown to have high overall internal consistency and high reliability within the two subscales (Kuyck, de Beurs, Barendregt, & van den Brink, 2013).

2.3.2 CU frequency

The Cannabis Use Disorder Identification Test (CUDIT) (Adamson & Sellman, 2003) was used to assess frequency of CU and to screen participants for the presence of a CU disorder. Adapted from the Alcohol Use Disorders Identification Test (AUDIT) (Saunders, Assland, Babor, De La Fuente, & Grant, 1993), the CUDIT first asks participants if they have used cannabis over the past 6 months. The participants are then asked to answer 10 items, in which the first 8 require an indication of how often the question applies over five time points, ranging from never to 4 or
more times a week, and the last two items asking only “yes” or “no” over the past 6 months. This measure has shown high sensitivity and positive predictive values in large samples of adolescents and adults (Adamson & Sellman, 2003; Annaheim, Rehm, & Gmel, 2008). For the purpose of this study, CU frequency was dichotomized into infrequent CU (monthly or less, and 3-4 times a month) and frequent CU (3-4 times a week, and 4 or more times a week).

### 2.3.3 Trait aggressive behaviour

The Life History of Aggression Questionnaire (LHA) is a 5-item questionnaire (Coccaro, Berman, & Kavoussi, 1997) that was developed to estimate the magnitude of trait aggressive behaviour. Participants are asked to rate on a 6-point scale the number of times their actions reflected a certain behaviour ranging from 0 (Never Happened) to 5 (Happened so many times that I couldn't give a number). This test has been shown to have high test-retest stability, interrater agreement, and internal consistency reliability (Coccaro et al., 1997).

### 2.3.4 Psychopathy

The Self-Report Psychopathy scale (SRP-III) is a 40-item questionnaire (Paulhus, Neumann, & Hare, in press) that was created to measure psychopathy in community samples. Along with the total SRP-III score, four subscale scores are also calculated (callous affect, interpersonal manipulation, erratic lifestyle, and criminal tendencies). Participants are asked to rate on a 5-point scale the extent to which each of the items applies to them ranging from 1 (Not at all) to 5 (Very much). The scale has shown good convergent and discriminate validity (Mahmut, Menictas, Stevenson, & Homewood, 2011).
2.4 Analytic procedures

There are three different levels of analyses for this study based on my three main hypotheses. First, independent-samples t tests were conducted on the cannabis users versus the non-users to determine whether there were mean differences between the groups regarding both IA and RA. Significant results were then followed up with a hierarchical regression to include both trait aggression and psychopathy to determine whether CU can predict the level of IA or RA after the covariates have been accounted for. This analytic procedure was conducted on all three of the samples recruited for this study.

For my second hypothesis, independent-samples t tests were conducted on the infrequent cannabis users versus the frequent cannabis users to determine whether there were mean differences between the groups regarding both IA and RA. Significant results were also then followed up with a hierarchical regression to include both trait aggression and psychopathy to determine whether frequency of CU can predict the level of IA or RA within the cannabis users after the covariates have been accounted for. This analytic procedure was conducted on all three of the samples recruited for this study.

For my third hypothesis, two-way ANOVAs were conducted within each of the three samples to examine the interaction of gender and both CU and frequency of CU to determine whether there is an interaction effect of gender. Significant results were then followed up with an analysis of the simple main effects to determine the strength and the nature of the interaction. Non-significant results were followed up with analyses of the main effects of gender and CU or CU frequency independently to establish in an effect is evident without the interaction term due to the non-significance of the interaction.
2.5 Supplementary Analyses

Due to the possible confounding nature of the participants who have identified themselves as previous (but not current) or infrequent cannabis users, I decided to include additional supplementary analyses. The first set of analyses in this project examined any previous cannabis use compared to no cannabis use at all which may not give an accurate picture of the influence of cannabis use on aggressive behaviour. Similarly, those who identified themselves as infrequent cannabis users (3 to 4 times a month or less) may not be using cannabis enough to determine whether a relation would exist between their CU and aggressive behaviour. For these reasons, it is important to explore the relationship of CU and aggressive behaviour between the non-users and the frequent cannabis users. The same procedures described above (independent-samples \( t \) tests and follow-up hierarchical regressions, and two-way ANOVAs and follow-up tests) were conducted on the non-users versus the frequent cannabis users in all three of the samples recruited for this study.
CHAPTER 3 Results

3.1 Descriptive statistics

3.1.1 University sample

The prevalence of CU in this sample was consistent with the expected frequencies established from previous research (Centre for Addiction and Mental Health, 2005) (See Figure 1). The frequency of LHA scores showed low levels of trait aggression ($M = 10.99$, $SD = 4.61$, range: 5-27). The LHA demonstrated high reliability within this sample ($\alpha = .78$) (See Table 2). The data showed moderate levels of psychopathy given the SRP scores ($M = 2.17$, $SD = 0.42$, range: 1.20-3.89). The SRP-III demonstrated high reliability within this sample ($\alpha = .78$) (See Table 2). IA was also low within this sample ($M = 22.53$, $SD = 6.34$, range: 10-37). Lastly, the data showed moderately higher levels of RA ($M = 25.04$, $SD = 6.66$, range: 10-43). The IPAS also demonstrated high reliability within this sample ($\alpha = .89$) (See Table 2). Intercorrelations for LHA scores, SRP-III scores, and IA/RA scores can be found in Table 3.

3.1.2 Online sample

The frequency of CU in this sample was consistent with the expected frequencies established from previous research (Denson & Earleywine, 2008) (See Figure 1). The frequency of LHA scores showed low levels of trait aggression ($M = 12.82$, $SD = 5.29$, range: 5-30). The LHA demonstrated high reliability within this sample ($\alpha = .83$) (See Table 2). The data showed moderate levels of psychopathy given the SRP scores ($M = 2.13$, $SD = 0.50$, range: 1.14-3.86). The SRP-III demonstrated high reliability within this sample ($\alpha = .73$) (See Table 2). IA was also low within this sample ($M = 22.38$, $SD = 6.43$, range: 10-42). Lastly, the data showed moderately higher levels RA ($M = 27.37$, $SD = 7.30$, range: 10-45). The IPAS also demonstrated
high reliability within this sample \((\alpha = .86)\) (See Table 2). Intercorrelations for LHA scores, SRP-III scores, and IA/RA scores can be found in Table 4.

### 3.1.3 Treatment sample

The frequency of CU in this sample is consistent with the expected frequencies established from previous research (Macdonald et al., 2008) (See Figure 1). The frequency of LHA scores showed low levels of trait aggression \((M = 19.22, SD = 6.25, \text{range: 7-30})\). The LHA demonstrated high reliability within this sample \((\alpha = .86)\) (See Table 2). The data showed moderate levels of psychopathy given the SRP scores \((M = 2.63, SD = 0.48, \text{range: 1.58-3.95})\). The SRP-III demonstrated high reliability within this sample \((\alpha = .62)\) (See Table 2). IA was also low within this sample \((M = 26.30, SD = 6.43, \text{range: 10-40})\). Lastly, the data showed moderately higher levels RA \((M = 33.14, SD = 7.64, \text{range: 18-49})\). The IPAS also demonstrated high reliability within this sample \((\alpha = .81)\) (See Table 2). Intercorrelations for LHA scores, SRP-III scores, and IA/RA scores can be found in Table 5.

#### Figure 1

- **CU Prevalence (%)**
- **No CU**
- **Previous CU**
- **Current CU**

<table>
<thead>
<tr>
<th>Sample</th>
<th>No CU</th>
<th>Previous CU</th>
<th>Current CU</th>
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<tbody>
<tr>
<td>University</td>
<td>46.60</td>
<td>35.83</td>
<td>17.56</td>
</tr>
<tr>
<td>Online</td>
<td>39.40</td>
<td>39.40</td>
<td>21.43</td>
</tr>
<tr>
<td>Treatment</td>
<td>57.35</td>
<td>25.00</td>
<td>17.65</td>
</tr>
</tbody>
</table>

*Figure 1. CU prevalence for the university, online, and treatment samples.*
Table 2

*Reliability Coefficients*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Alpha</th>
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<tbody>
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<td><strong>University Sample</strong></td>
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<td>LHA</td>
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<tr>
<td>SRP-III</td>
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</tr>
<tr>
<td>IPAS</td>
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<tr>
<td>CUDIT</td>
<td>.78</td>
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<tr>
<td><strong>Online Sample</strong></td>
<td></td>
</tr>
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<td>LHA</td>
<td>.83</td>
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<tr>
<td>SRP-III</td>
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<tr>
<td>IPAS</td>
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<tr>
<td>CUDIT</td>
<td>.82</td>
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<tr>
<td><strong>Treatment Sample</strong></td>
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<tr>
<td>LHA</td>
<td>.86</td>
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<tr>
<td>SRP-III</td>
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</tr>
<tr>
<td>IPAS</td>
<td>.81</td>
</tr>
<tr>
<td>CUDIT</td>
<td>.77</td>
</tr>
</tbody>
</table>

Note. LHA = Lifetime History of Aggression Scale, SRP-III = Self-Report Psychopathy Scale, IPAS = Impulsive and Premeditated Aggression Scale, CUDIT = Cannabis Use Disorder Identification Test

Table 3

*Intercorrelations for University Sample*

<table>
<thead>
<tr>
<th></th>
<th>LHA</th>
<th>SRP-III</th>
<th>Instrumental</th>
<th>Reactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>LHA</td>
<td>.31*</td>
<td>.31*</td>
<td>.45*</td>
<td></td>
</tr>
<tr>
<td>SRP-III</td>
<td>.31*</td>
<td>.38*</td>
<td>.20*</td>
<td></td>
</tr>
<tr>
<td>Instrumental</td>
<td>.31*</td>
<td>.38*</td>
<td>.23*</td>
<td></td>
</tr>
<tr>
<td>Reactive</td>
<td>.45*</td>
<td>.20*</td>
<td>.23*</td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .001
Table 4

*Intercorrelations for Online Sample*

<table>
<thead>
<tr>
<th></th>
<th>LHA</th>
<th>SRP-III</th>
<th>Instrumental</th>
<th>Reactive</th>
</tr>
</thead>
<tbody>
<tr>
<td>LHA</td>
<td>.34**</td>
<td>.23**</td>
<td>.45**</td>
<td></td>
</tr>
<tr>
<td>SRP-III</td>
<td>.34**</td>
<td>.53**</td>
<td>.27**</td>
<td></td>
</tr>
<tr>
<td>Instrumental</td>
<td>.23**</td>
<td>.53**</td>
<td>.11*</td>
<td></td>
</tr>
<tr>
<td>Reactive</td>
<td>.45**</td>
<td>.27**</td>
<td>.11*</td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .05  **p < .001

Table 5

*Intercorrelations for Treatment Sample*

<table>
<thead>
<tr>
<th></th>
<th>LHA</th>
<th>SRP-III</th>
<th>Instrumental</th>
<th>Reactive</th>
</tr>
</thead>
<tbody>
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<td>.30*</td>
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</tr>
<tr>
<td>SRP-III</td>
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<td>.58**</td>
<td>-.18</td>
<td></td>
</tr>
<tr>
<td>Instrumental</td>
<td>.59**</td>
<td>.58**</td>
<td>.09</td>
<td></td>
</tr>
<tr>
<td>Reactive</td>
<td>.30*</td>
<td>-.18</td>
<td>.09</td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .05  **p < .001

3.2 Hypotheses

3.2.1 CU and subtypes of aggression

3.2.1.1 University sample
An independent-samples *t* test was run within the university sample to determine whether there was a significant difference in IA between cannabis users and those who have never used cannabis. There were no outliers in the data, as assessed by inspection of a boxplot. IA scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test (*p* > .05), and there was homogeneity of variances as assessed by Levene’s test for equality of variances (*p* = .48). There was no statistically significant difference in IA among cannabis users (22.70, *SD* = 6.29) and non-users (22.33, *SD* = 6.29), 95% CI [-1.60 to 0.86], *t*(414) = -.59, *p* = .55 (See Figure 2). Due to the non-significant finding, the follow-up hierarchical regression was not run.

\[
\begin{array}{c|c|c}
\text{Aggression Type} & \text{Non-User} & \text{Cannabis User} \\
\hline
\text{Instrumental} & 20 & 18 \\
\text{Reactive} & 25 & 24 \\
\end{array}
\]

*Figure 2*. Mean IA and RA scores for non-users vs. cannabis users within the university sample.

An independent-samples *t*-test was also run within the university sample to determine whether there was a significant difference in RA between cannabis users and those who have never used cannabis. There were no outliers in the data, as assessed by inspection of a boxplot. RA scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test (*p* > .05), and there was homogeneity of variances as assessed by Levene’s test for
equality of variances ($p = .14$). There was no statistically significant difference in RA among cannabis users ($25.26, SD = 6.99$) and non-users ($24.78, SD = 6.27$), $95\%$ CI [-1.77 to 0.81], $t(416) = -.74, p = .46$ (See Figure 2). Due to the non-significant finding, the follow-up hierarchical regression was not run.

3.2.1.2 Online sample

An independent-samples $t$ test was run within the online sample to determine whether there was a significant difference in IA between cannabis users and those who have never used cannabis. Two participants in the online sample for instrumental aggression were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. IA scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .37$). There was no statistically significant difference in IA among cannabis users ($22.70, SD = 6.28$) and non-users ($21.89, SD = 6.64$), $95\%$ CI [-2.05 to 0.44], $t(432) = -1.27, p = .20$ (See Figure 3). Due to the non-significant finding, the follow-up hierarchical regression was not run.

An independent-samples $t$ test was also run within the online sample to determine whether there was a significant difference in RA between cannabis users and those who have never used cannabis. There were no outliers in the data, as assessed by inspection of a boxplot. RA scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .47$). RA was higher among cannabis users ($28.30, SD = 7.21$) than among the non-users ($25.93, SD = 7.23$), with a statistically significant increase of 2.37, $95\%$ CI [-3.77 to -0.98], $t(432) = -3.35, p < .01$ (See Figure 3).
Cannabis users had a significantly higher level of RA compared to non-users.

Due to the significant findings, a hierarchical multiple regression was run to determine whether CU was a significant predictor of RA while including the covariates of trait aggression and psychopathy. See Table 6 for full details on each regression model. The full model of trait aggression, psychopathy, and CU to predict RA (Model 2) was significant, $R^2 = .21$, $F(3, 430) = 39.10, p < .001$; adjusted $R^2 = .21$. However, the addition of CU to the prediction of RA (Model 2) led to a non-significant change in $R^2$ of <.01, $F(1, 430) = .24, p = .62$. 

Figure 3. Mean IA and RA scores for non-users vs. cannabis users within the online sample.
Table 6

CU Predicting RA in Online Sample

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>t</th>
<th>F</th>
<th>Adjusted $R^2$</th>
<th>$R^2\Delta$</th>
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<td>.21**</td>
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<tr>
<td>LHA</td>
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<td>8.84**</td>
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<td>.21**</td>
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<tr>
<td>SRP-III</td>
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<td>2.91*</td>
<td>.21</td>
<td>.00</td>
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<tr>
<td>Model 2</td>
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<td>.21</td>
<td>.00</td>
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<td>8.57**</td>
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<td>SRP-III</td>
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<td>.33</td>
<td></td>
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<td>.21</td>
<td>.00</td>
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</tbody>
</table>

Note. *$p < .01$  **$p < .001$

3.2.1.3 Treatment sample

An independent-samples $t$ test was run within the treatment sample to determine whether there was a significant difference in IA between cannabis users and those who have never used cannabis. There were no outliers in the data, as assessed by inspection of a boxplot. IA scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .87$). There was no statistically significant difference in IA among cannabis users ($26.38, SD = 6.45$) and non-users ($25.92, SD = 6.67$), 95% CI [-5.03 to 4.10], $t(52) = -.21, p =$
.84 (See Figure 4). Due to the non-significant finding, the follow-up hierarchical regression was not run.

An independent-samples t test was also run within the online sample to determine whether there was a significant difference in RA between cannabis users and those who have never used cannabis. There were no outliers in the data, as assessed by inspection of a boxplot. Reactive aggression scores for cannabis users and non-users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .80$). There was no statistically significant difference in RA among cannabis users ($32.47, SD = 7.62$) and non-users ($36.00, SD = 6.27$), 95% CI [-1.82 to 8.88], $t(51) = 1.33$, $p = .19$ (See Figure 4). Due to the non-significant finding, the follow-up hierarchical regression was not run.

Figure 4. Mean IA and RA scores for non-users vs cannabis users within the treatment sample.
3.2.2 CU frequency and subtypes of aggression

3.2.2.1 University sample

An independent-samples $t$ test was run within the university sample to determine whether there was a significant difference in IA between infrequent cannabis users and frequent cannabis users. Four participants in the university sample for IA were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. IA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .23$). There was no statistically significant difference in IA among infrequent cannabis users (23.61, $SD = 5.43$) and frequent cannabis users (25.17, $SD = 6.69$), 95% CI [-3.90 to 0.78], $t(144) = -1.32, p = .19$ (See Figure 5). Due to the non-significant finding, the follow-up hierarchical regression was not run.

![Figure 5](image-url)

*Figure 5.* Mean IA and RA scores for infrequent vs. frequent cannabis users within the university sample.
An independent-samples $t$ test was also run within the university sample to determine whether there was a significant difference in RA between infrequent cannabis users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. RA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .68$). There was no statistically significant difference in RA among casual cannabis users ($25.08, SD = 6.83$) and heavy cannabis users ($26.71, SD = 7.13$), 95% CI $[-4.41$ to $1.16], t(146) = -1.15, p = .25$ (See Figure 5). Due to the non-significant finding, the follow-up hierarchical regression was not run.

3.2.2.2 Online sample

An independent-samples $t$ test was run within the online sample to determine whether there was a significant difference in IA between infrequent cannabis users and frequent cannabis users. One participant in the online sample for IA was identified as an outlier, as they had a score greater than 3 standard deviations above the group mean, and was removed from the analysis. IA scores for non-users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .51$). There was no statistically significant difference in IA among infrequent cannabis users ($23.31, SD = 6.72$) and frequent cannabis users ($22.53, SD = 6.32$), 95% CI $[-1.97$ to $3.53], t(90) = .56, p = .57$ (See Figure 6). Due to the non-significant finding, the follow-up hierarchical regression was not run.

An independent-samples $t$ test was also run within the online sample to determine whether there was a significant difference in RA between infrequent cannabis users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. RA
scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .38$). There was no statistically significant difference in RA among infrequent cannabis users (27.99, $SD = 8.39$) and frequent cannabis users (29.04, $SD = 7.29$), 95% CI [-4.32 to 2.22], $t(91) = -.64$, $p = .53$ (See Figure 6). Due to the non-significant finding, the follow-up hierarchical regression was not run.

![Mean Aggression Scores](image.png)

*Figure 6. Mean IA and RA scores for infrequent vs. frequent cannabis users within the online sample.*

### 3.2.2.3 Treatment sample

An independent-samples $t$ test was run within the treatment sample to determine whether there was a significant difference in IA between infrequent cannabis users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. IA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s
test for equality of variances ($p = .57$). There was no statistically significant difference in IA among infrequent cannabis users (26.50, $SD = 6.37$) and frequent cannabis users (28.13, $SD = 6.64$), 95% CI [-6.83 to 3.58], $t(28) = -.64$, $p = .53$ (See Figure 7). Due to the non-significant finding, the follow-up hierarchical regression was not run.

![Figure 7](image_url)

**Figure 7.** Mean IA and RA scores for infrequent vs. frequent cannabis users within the treatment sample.

An independent-samples $t$ test was also run within the treatment sample to determine whether there was a significant difference in RA between infrequent cannabis users and frequent cannabis users. One participant in the treatment sample for RA was identified as an outlier, as they had a score greater than 3 standard deviations above the group mean, and was removed from the analysis. RA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of
variances as assessed by Levene’s test for equality of variances \((p = .20)\). There was no statistically significant difference in RA among infrequent cannabis users \((30.97, SD = 5.11)\) and frequent cannabis users \((32.70, SD = 7.50)\), 95% CI \([-7.43 to 3.98]\), \(t(26) = -.62, p = .54\) (See Figure 7). Due to the non-significant finding, the follow-up hierarchical regression was not run.

### 3.2.3 Gender and subtypes of aggression

#### 3.2.3.1 Cannabis users versus non-users

##### 3.2.3.1.1 University sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and CU on the expression of IA within the university sample. Two participants in the university sample for IA were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. IA scores for males and females were not normally distributed, as assessed by Shapiro-Wilk’s test \((p < .05)\); however, as the sample sizes are large and AVOVA’s are considered fairly robust to deviations of normality (Maxwell & Delaney, 2004), the analysis was continued. The homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances \((p > .05)\).

The interaction effect between gender and CU for IA was not statistically significant, \(F(1, 410) = .24, p = .62\), partial \(\eta^2 < .01\) (See Figure 8). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, \(F(1, 410) = 23.37, p < .01\), partial \(\eta^2 = .05\). The unweighted marginal means of IA scores for females was 21.62 \((SE = .36)\) and 24.81 \((SE = .55)\) for males. Mean IA scores for males were statistically significantly higher than for females, 3.19, 95% CI \([1.89 to 4.49]\), \(p < .01\).
An analysis of the main effect of CU was also performed which was not significant, $F(1, 410) = .08$, $p = .77$, partial $\eta^2 < .01$. The unweighted marginal means of IA scores for cannabis users was 23.31 ($SE = .46$) and for non-users was 23.12 ($SD = .46$). Mean IA scores for cannabis user was not statistically significantly different than for non-users, $0.19$, 95% CI [-1.11 to 1.49], $p = .77$.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and CU on the expression of RA within the university sample. There were no outliers in the data as assessed by a boxplot, and RA scores for males and females were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$). The homogeneity of variances assumption was violated as assessed by Levene’s test for equality of variances ($p = .04$); however, as group sizes are approximately equal and large, the scores are normally distributed, and the ratio of the largest group variance to the smallest group variance is less than 3, the two-way ANOVA was still run as this procedure is robust to heterogeneity of variance under these conditions (Jaccard, 1998).

![Figure 8](image.png)

**Figure 8.** Mean IA and RA scores for male and female non-users vs. cannabis users within the university sample.
The interaction effect between gender and CU for RA was not statistically significant, $F(1, 412) = 3.42, p = .07$, partial $\eta^2 < .01$ (See Figure 8). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 412) = 1.32, p = .25$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for females was 24.76 ($SE = .39$) and 25.58 ($SE = .60$) for males. Mean RA scores for males were not statistically significantly different than for females, 0.82, 95% CI [-0.58 to 2.22], $p = .25$.

An analysis of the main effect of CU was also performed which was also not statistically significant, $F(1, 412) = .01, p = .97$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for cannabis users was 25.15 ($SE = .49$) and for non-users was 25.18 ($SE = .52$). Mean RA scores for cannabis users was not statistically significantly higher than for non-users, 0.03, 95% CI [-1.37 to 1.43], $p = .97$.

### 3.2.3.1.2 Online sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and CU on the expression of IA within the online sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and CU for IA was not statistically significant, $F(1, 430) = .56, p = .45$, partial $\eta^2 < .01$ (See Figure 9). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, $F(1, 430) = 13.09, p < .01$, partial $\eta^2 = .03$. The unweighted marginal means of IA scores for females
was 21.16 ($SE = .44$) and 23.46 ($SE = .45$) for males. Mean IA scores for males were statistically significantly higher than for females, $2.59$, 95% CI [1.03 to 3.48], $p < .01$.

An analysis of the main effect of CU was also performed which was not significant, $F(1, 430) = 1.55$, $p = .21$, partial $\eta^2 < .01$. The unweighted marginal means of IA scores for cannabis users was 22.70 ($SE = .39$) and for non-users was 21.94 ($SE = .49$). Mean IA scores for cannabis user was not statistically significantly different than for non-users, $.78$, 95% CI [-0.45 to 2.00], $p = .21$.

![Figure 9](image_url)

*Figure 9.* Mean IA and RA scores for male and female non-users vs. cannabis users within the online sample.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and CU on the expression of RA within the online sample. Two participants in the online sample for RA were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. RA scores for males and females
were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and CU for RA was not statistically significant, $F(1, 428) = .96, p = .33$, partial $\eta^2 < .01$ (See Figure 9). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 428) = .08, p = .77$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for females was 27.09 ($SE = .49$) and 27.29 ($SE = .50$) for males. Mean RA scores for males were not statistically significantly different than for females, .20, 95% CI [-1.18 to 1.58], $p = .77$.

An analysis of the main effect of CU was also performed which was statistically significant, $F(1, 428) = 12.65, p < .01$, partial $\eta^2 = .03$. The unweighted marginal means of RA scores for CU was 28.44 ($SE = .44$) and for non-users was 25.94 ($SE = .55$). Mean RA scores for cannabis users was statistically significantly higher than for non-users, 2.50, 95% CI [1.12 to 3.88], $p < .01$.

### 3.2.3.1.3 Treatment sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and CU on the expression of IA within the treatment sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and CU for IA was not statistically significant, $F(1, 50) = 1.29, p = .67$, partial $\eta^2 = .02$ (See Figure 10). Therefore, an analysis of the main
effect for gender was performed which indicated that the main effect was also not statistically significant, \( F(1, 50) = 1.36, p = .25 \), partial \( \eta^2 = .03 \). The unweighted marginal means of IA scores for females was 27.23 (\( SE = 1.47 \)) and 24.52 (\( SE = 1.81 \)) for males. Mean IA scores for males were not statistically significantly different than for females, -2.72, 95% CI [-7.40 to 1.96], \( p = .25 \).

An analysis of the main effect of CU was also performed which was not statistically significant, \( F(1, 50) = .18, p = .67 \), partial \( \eta^2 < .01 \). The unweighted marginal means of IA scores for cannabis users was 26.37 (\( SE = 2.10 \)) and for non-users was 25.38 (\( SE = 1.01 \)). Mean IA scores for CU were not statistically significantly different than for non-users, .99, 95% CI [-3.69 to 5.67], \( p = .67 \).

*Figure 10.* Mean IA and RA scores for male and female non-users vs. cannabis users within the treatment sample.
A two-way ANOVA was conducted to determine whether there was an effect of gender and CU on the expression of RA within the treatment sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p < .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and CU for RA was not statistically significant, $F(1, 49) = .79, p = .38$, partial $\eta^2 = .02$ (See Figure 10). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, $F(1, 49) = 20.33, p < .01$, partial $\eta^2 = .29$. The unweighted marginal means of RA scores for females was 38.25 ($SE = 1.41$) and 28.16 ($SE = 1.74$) for males. Mean RA scores for males were statistically significantly lower than for females, -10.09, 95% CI [-14.59 to -5.59], $p < .01$.

An analysis of the main effect of CU was also performed which was not statistically significant, $F(1, 49) = 2.00, p = .16$, partial $\eta^2 = .04$. The unweighted marginal means of RA scores for cannabis users was 31.62 ($SE = .97$) and for non-users was 34.79 ($SE = 2.02$). Mean RA scores for cannabis users were not statistically significantly different than for non-users, -3.17, 95% CI [-7.67 to 1.33], $p = .16$.

### 3.2.3.2 CU frequency

#### 3.2.3.2.1 University sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and frequency of CU on the expression of IA within the university sample. Seven participants in the university sample for IA were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. IA scores for males and
females were normally distributed, as assessed by Shapiro-Wilk’s test \((p > .05)\), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances \((p > .05)\).

The interaction effect between gender and frequency of CU for IA was not statistically significant, \(F(1, 139) = .42, p = .52\), partial \(\eta^2 < .01\) (See Figure 11). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, \(F(1, 139) = .93, p = .34\), partial \(\eta^2 = .01\). The unweighted marginal means of IA scores for females was 24.29 \((SE = .79)\) and 25.44 \((SE = .90)\) for males. Mean IA scores for males were not statistically significantly different than for females, 0.20, 95% CI [-1.18 to 1.58], \(p = .77\).

![Figure 11](image.png)

*Figure 11*. Mean IA and RA scores for male and female infrequent vs. frequent cannabis users within the university sample.
An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 139) = 2.35, p = .13$, partial $\eta^2 = .02$. The unweighted marginal means of IA scores for infrequent cannabis users was 23.95 ($SE = .54$) and for frequent cannabis users was 25.78 ($SE = 1.07$). Mean IA scores for infrequent cannabis users was not statistically significantly different than for frequent cannabis users, $-1.84$, 95% CI [-4.20 to 0.53], $p = .13$.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and frequency of CU on the expression of RA within the university sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and frequency of CU for RA was not statistically significant, $F(1, 141) = 2.32, p = .13$, partial $\eta^2 = .02$ (See Figure 11). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 141) = 1.52, p = .22$, partial $\eta^2 = .01$. The unweighted marginal means of RA scores for females was 26.74 ($SE = .94$) and 24.88 ($SE = 1.18$) for males. Mean RA scores for males were not statistically significantly different than for females, $-1.86$, 95% CI [-4.85 to 1.12], $p = .22$.

An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 141) = .86, p = .36$, partial $\eta^2 = .01$. The unweighted marginal means of RA scores for infrequent cannabis users was 25.11 ($SE = .68$) and for frequent cannabis users was 26.51 ($SE = 1.35$). Mean RA scores for infrequent cannabis users were not statistically significantly different than for frequent cannabis users, $-1.40$, 95% CI [-4.39 to 1.58], $p = .36$. 
3.2.3.2.2 Online sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and frequency of CU on the expression of IA within the online sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and frequency of CU for IA was not statistically significant, $F(1, 89) = .16, p = .69$, partial $\eta^2 < .01$ (See Figure 12). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 89) = .48, p = .49$, partial $\eta^2 = .01$. The unweighted marginal means of IA scores for females was $22.52$ ($SE = 1.02$) and $23.51$ ($SE = 1.00$) for males. Mean IA scores for males were not statistically significantly different than for females, $0.99$, 95% CI [-1.86 to 3.83], $p = .49$.

![Figure 12](image_url)

**Figure 12.** Mean IA and RA scores for male and female infrequent vs. frequent cannabis users within the online sample.
An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 89) = .18, p = .67$, partial $\eta^2 < .01$. The unweighted marginal means of IA scores for infrequent cannabis users was 23.32 ($SE = 1.12$) and for frequent cannabis users was 22.71 ($SE = .89$). Mean IA scores for infrequent cannabis users were not statistically significantly different than for frequent cannabis users, $0.61, 95\% CI [-2.32 to 3.45], p = .67$.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and frequency of CU on the expression of RA within the online sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and frequency of CU for RA was not statistically significant, $F(1, 89) = .37, p = .54$, partial $\eta^2 < .01$ (See Figure 12). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 89) = 2.64, p = .11$, partial $\eta^2 = .03$. The unweighted marginal means of RA scores for females was 27.18 ($SE = 1.17$) and 29.85 ($SE = 1.16$) for males. Mean RA scores for males were not statistically significantly different than for females, $2.67, 95\% CI [-0.60 to 5.94], p = .11$.

An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 89) = .26, p = .61$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for infrequent cannabis users was 28.09 ($SE = 1.29$) and for frequent cannabis users was 28.93 ($SE = 1.03$). Mean RA scores for infrequent cannabis users were not statistically significantly different than for frequent cannabis users, $-0.85, 95\% CI [-4.11 to 2.43], p = .61$. 


3.2.3.2.3 Treatment sample

A two-way ANOVA was conducted to determine whether there was an effect of gender and frequency of CU on the expression of IA within the treatment sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and frequency of CU for IA was not statistically significant, $F(1, 26) = 2.85$, $p = .10$, partial $\eta^2 = .10$ (See Figure 13). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 26) = .54$, $p = .47$, partial $\eta^2 = .02$. The unweighted marginal means of IA scores for females was 26.56 ($SE = 1.61$) and 28.44 ($SE = 1.98$) for males. Mean IA scores for males were not statistically significantly different than for females, 1.88, 95% CI [-3.37 to 7.12], $p = .47$.

![Figure 13](image-url)

*Figure 13.* Mean IA and RA scores for male and female infrequent vs. frequent cannabis users within the treatment sample.
An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 26) = .09, p = .77$, partial $\eta^2 < .01$. The unweighted marginal means of IA scores for infrequent cannabis users was 27.12 ($SE = 2.08$) and for frequent cannabis users was 27.88 ($SE = 1.47$). Mean IA scores for infrequent cannabis users were not statistically significantly different than for frequent cannabis users, -0.76, 95% CI [-6.01 to 4.48], $p = .77$.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and frequency of CU on the expression of RA within the treatment sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and frequency of CU for RA was not statistically significant, $F(1, 25) = .18, p = .68$, partial $\eta^2 = .01$ (See Figure 13). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, $F(1, 25) = 6.91, p < .05$, partial $\eta^2 = .22$. The unweighted marginal means of RA scores for females was 35.27 ($SE = 1.66$) and 28.44 ($SE = 2.00$) for males. Mean RA scores for males were statistically significantly lower than for females, -6.83, 95% CI [-12.18 to -1.48], $p < .05$.

An analysis of the main effect of frequency of CU was also performed which was not statistically significant, $F(1, 25) = .09, p = .77$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for infrequent cannabis users was 31.46 ($SE = 2.11$) and for frequent cannabis users was 32.24 ($SE = 1.52$). Mean RA scores for infrequent cannabis users were not statistically significantly different than for frequent cannabis users, -0.79, 95% CI [-6.13 to 4.56], $p = .77$. 

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3.3 Supplementary analyses

3.3.1 University sample

An independent-samples t test was run within the university sample to determine whether there was a significant difference in IA between non-users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. IA scores for non-users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .93$). There was a statistically significant difference in IA among non-users (22.33, $SD = 6.42$) and frequent cannabis users (25.17, $SD = 6.69$), 95% CI [-5.38 to -0.31], $t(221) = -2.22$, $p < .05$ (See Figure 14).

![Figure 14](image)

**Figure 14.** Mean IA and RA scores for non-users vs. frequent cannabis users within the university sample. Non-users had a significantly lower level of IA compared to frequent cannabis users.
Due to the significant findings, a hierarchical multiple regression was run to determine whether non-use/frequent CU was a significant predictor of IA while including the covariates of trait aggression and psychopathy. See Table 7 for full details on each regression model. The full model of trait aggression, psychopathy and non-use/frequent CU to predict IA (Model 2) was significant, $R^2 = .40$, $F(3, 195) = 12.63$, $p < .001$; adjusted $R^2 = .15$. However, the addition of non-use/frequent CU to the prediction of RA (Model 2) led to a non-significant change in $R^2$ of .00, $F(1, 195) = .17$, $p = .68$.

Table 7

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Note. *$p < .001$
An independent-samples t test was also run within the university sample to determine whether there was a significant difference in RA between non-users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. RA scores for non-users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test \( p > .05 \), and there was homogeneity of variances as assessed by Levene’s test for equality of variances \( p = .23 \). There was no statistically significant difference in RA among non-users \((24.78, SD = 6.27)\) and frequent cannabis users \((26.71, SD = 7.13)\), 95% CI \([-4.40 \text{ to } 0.54]\), \( t(220) = -1.54, p = .13 \) (See Figure 14). Due to the non-significant finding, the follow-up hierarchical regression was not run.

A two-way ANOVA was conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of IA in the university sample. Five participants in the university sample for IA were identified as outliers, as they had scores greater than 3 standard deviations above the group mean, and were removed from the analysis. IA scores for males and females were normally distributed, as assessed by Shapiro-Wilk’s test \( p > .05 \), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances \( p = .11 \).

The interaction effect between gender and non-use/frequent CU for IA was not statistically significant, \( F(1, 214) = 1.49, p = .22, \) partial \( \eta^2 < .01 \) (See Figure 15). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, \( F(1, 214) = 2.29, p = .34, \) partial \( \eta^2 = .01 \). The unweighted marginal means of IA scores for females was 23.48 \((SE = .85)\) and 25.42 \((SE = .96)\) for males. Mean IA scores for males were not statistically significantly different than for females, 1.95, 95% CI \([-0.59 \text{ to } 4.48]\), \( p = .13 \).
An analysis of the main effect of non-use/frequent CU was also performed which was statistically significant, \( F(1, 214) = 4.31, p < .05 \), partial \( \eta^2 = .02 \). The unweighted marginal means of IA scores for non-users was 23.12 (\( SE = .47 \)) and for frequent cannabis users was 25.78 (\( SE = 1.20 \)). Mean IA scores for non-users was statistically significantly different than for frequent cannabis users, -2.67, 95% CI [-5.20 to -0.13], \( p < .05 \).

![Mean Aggression Scores](image)

*Figure 15.* Mean IA and RA scores for male and female non-users vs. frequent cannabis users within the university sample.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of RA within the university sample. There were no outliers in the data as assessed by a boxplot, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test (\( p > .05 \)), and the homogeneity of
variances assumption was not violated as assessed by Levene’s test for equality of variances ($p = .36$).

The interaction effect between gender and non-use/frequent CU for RA was not statistically significant, $F(1, 218) = 2.92, p = .28$, partial $\eta^2 = .02$ (See Figure 15). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 218) = .32, p = .58$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for females was 26.07 ($SE = .78$) and 25.34 ($SE = 1.04$) for males. Mean RA scores for males were not statistically significantly different than for females, $-0.73, 95\% CI [-3.28 to 1.82], p = .58$.

An analysis of the main effect of non-use/frequent CU was also performed which was not statistically significant, $F(1, 218) = .66, p = .42$, partial $\eta^2 < .01$. The unweighted marginal means of RA scores for non-users was 25.18 ($SE = .49$) and for frequent cannabis users was 26.23 ($SE = 1.20$). Mean RA scores for non-users were not statistically significantly different than for frequent cannabis users, $-1.05, 95\% CI [-3.60 to 1.50], p = .42$.

### 3.3.2 Online sample

An independent-samples $t$ test was run within the online sample to determine whether there was a significant difference in IA between non-users and frequent cannabis users. There were no outliers in the data as assessed by a boxplot, IA scores for non-users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .62$). There was no statistically significant difference in IA among non-users ($21.89, SD = 6.64$) and frequent cannabis users ($22.81, SD = 6.61$), 95\% CI [-2.91 to 1.09], $t(225) = -.90, p = .37$ (See Figure 16). Due to the non-significant finding, the follow-up hierarchical regression was not run.
An independent-samples \( t \) test was also run within the online sample to determine whether there was a significant difference in RA between infrequent cannabis users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. RA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test \( (p > .05) \), and there was homogeneity of variances as assessed by Levene’s test for equality of variances \( (p = .38) \). There was a statistically significant difference in RA among non-users \( (25.93, SD = 7.23) \) and frequent cannabis users \( (29.04, SD = 7.29) \), 95% CI [-5.29 to -0.93], \( t(225) = -2.81, p < .01 \) (See Figure 16).

![Mean Aggression Scores for Non-Users vs. Frequent Users](image)

**Figure 16.** Mean IA and RA scores for non-users vs. frequent cannabis users within the online sample. Non-users had a significantly lower level of RA compared to frequent cannabis users.

Due to the significant findings, a hierarchical multiple regression was run to determine whether non-use/frequent CU was a significant predictor of RA while including the covariates of trait aggression and psychopathy. See Table 8 for full details on each regression model. The full
model of trait aggression, psychopathy and non-use/frequent CU to predict RA (Model 2) was significant, $R^2 = .24$, $F(3, 223) = 24.05$, $p < .001$; adjusted $R^2 = .23$. However, the addition of non-use/frequent CU to the prediction of RA (Model 2) led to a non-significant change in $R^2$ of .00, $F(1, 223) = .10$, $p = .75$.

Table 8

<table>
<thead>
<tr>
<th>Non-use/Frequent CU Predicting RA in Online Sample</th>
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<tr>
<td>Model 1</td>
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<tr>
<td>LHA</td>
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<td>SRP-III</td>
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<td>Model 2</td>
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<td>SRP-III</td>
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<td>Non-use/frequent CU</td>
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Note. *$p < .001$

A two-way ANOVA was conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of IA within the online sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and non-use/frequent CU for IA was not statistically significant, $F(1, 223) = .34$, $p = .56$, partial $\eta^2 < .01$ (See Figure 17). Therefore, an
analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, $F(1, 223) = 4.52, p < .05$, partial $\eta^2 = .02$. The unweighted marginal means of IA scores for females was 21.26 ($SE = .74$) and 23.40 ($SE = .68$) for males. Mean IA scores for males were statistically significantly different than for females, 2.14, 95% CI [0.16 to 4.13], $p < .05$.

Figure 17. Mean IA and RA scores for male and female non-users vs. frequent cannabis users within the online sample.

An analysis of the main effect of non-use/frequent CU was also performed which was not statistically significant, $F(1, 223) = .59, p = .45$, partial $\eta^2 < .01$. The unweighted marginal means of IA scores for non-users was 21.94 ($SE = .50$) and for frequent cannabis users was 22.71 ($SE = .87$). Mean IA scores for non-users were not statistically significantly different than for frequent cannabis users, -0.77 95% CI [-2.76 to 1.45], $p = .45$. 
A two-way ANOVA was also conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of RA within the online sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test \((p > .05)\), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances \((p > .05)\).

The interaction effect between gender and non-use/frequent CU for RA was not statistically significant, \(F(1, 223) = .12, p = .73\), partial \(\eta^2 < .01\) (See Figure 17). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, \(F(1, 223) = 1.31, p = .25\), partial \(\eta^2 < .01\). The unweighted marginal means of RA scores for females was 26.80 \((SE = .82)\) and 28.08 \((SE = .76)\) for males. Mean RA scores for males were not statistically significantly different than for females, 1.28, 95% CI [-.92 to 3.48], \(p = .25\).

An analysis of the main effect of non-use/frequent CU was also performed which was statistically significant, \(F(1, 223) = 7.18, p < .01\), partial \(\eta^2 = .03\). The unweighted marginal means of RA scores for non-users was 25.94 \((SE = .56)\) and for frequent cannabis users was 28.93 \((SE = .97)\). Mean RA scores for non-users were statistically significantly different than for frequent cannabis users, -2.99, 95% CI [-5.19 to -0.79], \(p < .01\).

### 3.3.3 Treatment sample

An independent-samples \(t\) test was run within the treatment sample to determine whether there was a significant difference in IA between non-users and frequent cannabis users. There were no outliers in the data, as assessed by inspection of a boxplot. IA scores for infrequent cannabis users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test \((p > .05)\), and there was homogeneity of variances as assessed by Levene’s test for equality of variances. \(t\) \(t\) test.
variances ($p = .86$). There was no statistically significant difference in IA among non-users (25.92, $SD = 6.67$) and frequent cannabis users (28.13, $SD = 6.64$), 95% CI [-7.49 to 3.07], $t(28) = -.86, p = .40$ (See Figure 18). Due to the non-significant finding, the follow-up hierarchical regression was not run.

An independent-samples $t$ test was also run within the treatment sample to determine whether there was a significant difference in RA between non-users and frequent cannabis users. There were no outliers in the data as assessed by a boxplot, RA scores for non-users and frequent cannabis users were normally distributed, as assessed by Shapiro-Wilk’s test ($p > .05$), and there was homogeneity of variances as assessed by Levene’s test for equality of variances ($p = .97$). There was no statistically significant difference in RA among non-users (36.00, $SD = 7.43$) and frequent cannabis users (32.70, $SD = 7.50$), 95% CI [-2.69 to 9.30], $t(27) = 1.31, p = .27$ (See Figure 18). Due to the non-significant finding, the follow-up hierarchical regression was not run.

![Figure 18. Mean IA and RA scores for non-users vs. frequent cannabis users within the treatment sample.](image-url)
A two-way ANOVA was conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of IA within the treatment sample. There were no outliers as assessed by the boxplots, IA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and non-use/frequent CU for IA was not statistically significant, $F(1, 26) = .31, p = .58$, partial $\eta^2 = .01$ (See Figure 19). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was also not statistically significant, $F(1, 26) = 2.20, p = .15$, partial $\eta^2 = .08$. The unweighted marginal means of IA scores for females was $28.58 (SE = 1.66)$ and $24.69 (SE = 2.03)$ for males. Mean IA scores for males were not statistically significantly different than for females, 3.89, 95% CI [-1.50 to 9.28], $p = .15$.

Figure 19. Mean IA and RA scores for male and female non-users vs. frequent cannabis users within the treatment sample.
An analysis of the main effect of non-use/frequent CU was also performed which was not statistically significant, $F(1, 26) = .91, \ p = .35$, partial $\eta^2 = .03$. The unweighted marginal means of IA scores for non-users was 25.38 ($SE = 2.14$) and for frequent cannabis users was 27.88 ($SE = 1.51$). Mean IA scores for non-users were not statistically significantly different than for frequent cannabis users, $-2.50, 95\% \ CI \ [-7.89 \ to \ 2.89], p = .35$.

A two-way ANOVA was also conducted to determine whether there was an effect of gender and non-use/frequent CU on the expression of RA within the treatment sample. There were no outliers as assessed by the boxplots, RA scores for males and females were normally distributed as assessed by Shapiro-Wilk’s test ($p > .05$), and the homogeneity of variances assumption was not violated as assessed by Levene’s test for equality of variances ($p > .05$).

The interaction effect between gender and non-use/frequent CU for RA was not statistically significant, $F(1, 25) = 1.58, \ p = .22$, partial $\eta^2 = .06$ (See Figure 19). Therefore, an analysis of the main effect for gender was performed which indicated that the main effect was statistically significant, $F(1, 25) = 12.47, \ p < .01$, partial $\eta^2 = .33$. The unweighted marginal means of RA scores for females was 37.97 ($SE = 1.61$) and 29.06 ($SE = 1.94$) for males. Mean RA scores for males were statistically significantly lower than for females, $-8.91, 95\% \ CI \ [-14.11 \ to \ -3.71], p < .01$.

An analysis of the main effect of non-use/frequent CU was also performed which was not statistically significant, $F(1, 25) = 1.02, \ p = .32$, partial $\eta^2 = .04$. The unweighted marginal means of RA scores for non-users was 34.79 ($SE = 2.05$) and for frequent cannabis users was 32.24 ($SE = 1.47$). Mean RA scores for non-users were not statistically significantly different than for frequent cannabis users, $2.55, 95\% \ CI \ [-2.65 \ to \ 7.74], p = .32$. 
CHAPTER 4 Discussion

The aim of the current study was to examine the potential relation between CU and subtypes of aggressive behaviour. Specifically, I was interested in exploring whether different levels of CU would be related to the expression of instrumental or reactive aggression. Also of interest were the roles that certain covariates, such as psychopathy and history of aggression, may play in this intricate relationship. Lastly, I was interested in the effect of one’s gender on the expression of either subtype of aggression in relation to their CU. To maximize generalizability, my search for answers to these questions was performed on samples from several populations.

What makes this study unique was my utilization of samples from three different populations and using consistent measures across all three samples. Most previous studies have only focused on a single population and the measurements used have typically been inconsistent across studies. The most disparate variables between studies included the measure of cannabis use, the definition of aggression being used, and the type of aggression under examination. This study attempted to bridge the gap left by previous research by using consistent and reliable measures of CU, CU frequency, and instrumental/reactive aggression across three main populations of interest from previous research.

The levels of CU and CU frequency were consistent with past finding in the university (Centre for Addiction and Mental Health, 2005), online (Denson & Earleywine, 2008), and treatment samples (Macdonald et al., 2008). Approximately 36% of the university sample identified themselves as current cannabis users which is higher than expected in the general population, but is consistent with this population given the average age of the participants. Approximately 21% of the online sample identified themselves as current cannabis users which is a more consistent with current cannabis use within the general public. Lastly, approximately
57% of the participants in the treatment sample identified themselves as current users which may seem high but is due to the homogeneous nature of the population and the high levels of drug use among treatment patients.

The amount of instrumental/reactive aggression found within the samples was also consistent with previous research (Kuyck, de Beurs, Barendregt, & van den Brink, 2013). Across all three samples, IA was consistently lower than RA, as expected, and it is suggested that this is due to the highly impulsive nature of RA and the planning and commitment of IA. Intercorrelations between the aggression scales and psychopathy scale are also consistent with what we would expect. The high correlation between IA and psychopathy in all 3 samples is of note, with the highest correlation of .58 within the treatment sample. This confirms the psychopathic nature of IA also seen in previous research (Glenn & Raine, 2009; Vitacco et al., 2006; Woodworth & Porter, 2002) and helps to explain the lower levels of IA seen in the current study attributable to the low prevalence of psychopathy in the general public. Surprisingly, the correlation between IA and RA was not found in the treatment sample; however, this may be due to the previously mentioned homogeneity concerns with treatment populations and the higher levels of IA seen in that population.

Regarding my first hypothesis, the relationship between CU and RA was not established. In fact, a small but significant relation between CU and RA was only found within the online sample, where cannabis users had a higher level of RA. However, when trait aggression and psychopathy were accounted for the significance was lost and the relation disappeared. Interestingly, when looking at the multiple regression for this analyses, psychopathy appears to be a stronger factor in relation to an increase in RA when compared to trait aggression. This is surprising due to the higher correlation between RA and history of aggression when compared to
psychopathy in this sample, and warrants further exploration. Further, there was no relationship found between CU and IA in any of the samples. These findings demonstrate that in the current study CU alone, both previous and current use, was not a contributing factor to either subtype of aggression, across all three samples.

Regarding my second hypothesis, the relation between frequency of CU and either subtype of aggression was also not established. The independent-samples t tests conducted on all three samples did not reveal any relation between infrequent/frequent CU and the subtypes of aggression. However, given that this analysis was conducted only on those who identified themselves as current cannabis users, and CU alone is not a determining factor in instrumental or reactive aggressive behaviour, it makes sense that there is also no dose-related effect of CU on the subtypes of aggression. This information further confirms the conclusion that CU, even when looking at infrequent versus frequent users, is not related to IA or RA.

Similar findings were revealed when looking at frequent cannabis users and non-users. Within the online sample, frequent cannabis users had significantly higher levels of IA when compared to non-users; however, this relation vanished when history of aggression and psychopathy were included in the analysis. Once again, psychopathy had a greater influence regarding IA compared to history of aggression, which is consistent with what we would expect given the relationship between IA and psychopathy. There were no other statistically significant differences between frequent cannabis use and non-use regarding IA. The results for RA are comparable. Significance was found within the online sample where frequent cannabis users had a significantly higher level of RA when compared to non-users, but trait aggression and psychopathy accounted for a greater portion of this difference and frequent CU became irrelevant. These results suggest that cannabis users and non-users engage in a relatively similar
amount of reactive aggression when accounting for the effect of trait aggression and psychopathy. Finally, no other relations were found between frequent CU/non-use and RA. This also supports the conclusion that CU frequency has no relationship with instrumental or reactive aggression.

Regarding my third hypothesis, gender may play a key role in the expression of the subtypes of aggression, but not in conjunction with CU. Analyses conducted revealed no interaction between CU and gender within any of the samples under examination. This result was consistent across all levels of CU including non-use, current use, infrequent use, and frequent use. However, when the main effect of gender was further examined some interesting results were found. In both the university and the online samples, males had a significantly higher level of IA compared to females when looking at cannabis users versus non-users. This result is consistent with the relationship previously determined with gender and psychopathy where higher rates of psychopathy is seen in males (Hare, 2003). But what is interesting is the effect that is seen in the treatment sample where males had significantly lower levels of RA when compared to females. Males are usually considered to be more impulsive in nature; however, the effect that is seen here may be due to the increase in social problems that females experience related to their drug use and the higher severity of their drug dependence when entering treatment (Brady & Randall, 1999). These findings support the idea that gender plays a key role in aggression and warrant further examination; however, cannabis is not part of this relationship.

4.1 Implications and conclusions

The results from this study have several implications regarding research methods, theoretical application, and clinical practices. Methodologically, this study highlights the importance of consistent measures when comparing samples from multiple populations. The results from this
study are strengthened by the use of the same measures across samples from three different populations. While there was no significant relation found between CU and IA/RA, the fact that this was consistent across all three samples confirms that there is indeed no meaningful relationship between these variables. Further, incorporating gender into the methods proved to be a benefit in elucidating the expression of both IA and RA within the three samples by showing that there is indeed an effect of gender on aggressive behaviour. From a theoretical standpoint, this research will help future theories revolving around CU and aggressive behaviour by further dispelling the myth that cannabis is associated with aggressive behaviour. It also helps with understanding why previous research has come to such diverse and conflicting results due to inconsistent measures of CU and aggression. Further, the failure to consider important covariates, such as psychopathy and trait aggression, may be the reason for the positive associations found in the research. This information will prove to be useful when forming subsequent theories about the lack of a relationship between cannabis and aggression. Lastly, from a clinical prospective, this information will be useful in assessing the risks associated with CU. The medical and clinical values placed on cannabis are steadily increasing as more research is being conducted in this area. This information will help with treatment plans that could incorporate the use of cannabis to reduce negative symptoms experienced by a patient, such as those suffering from PTSD or other anxiety issues who are at high risk of aggression, by dispelling the concerns that a practitioner may have about cannabis use and its association with aggression.

It is evident from this research that CU has little to no association with either instrumental or reactive aggression. The results indicate that when associations are found, they can be better explained by covariates such as psychopathy or trait aggression. It can be suggested
that the reason there is such controversy in the literature is that previous research has failed to use consistent measures across samples from multiple populations and has failed to consider psychopathy and trait aggression as important factors. The failure to include these covariates helps to explain the conflicting results seen in previous research; the study found no direct association between cannabis and aggression after those covariates have been included in the analyses.

4.2 Limitations and future directions

It should be noted that this research is not without its limitations. The first and most notable limitation relates to the treatment population. The sample from this population proved to be homogenous in ethnicity, age, SES, and social issues. Further, acquiring 68 participants within a treatment population was not an easy task; however, this sample was still under powered when compared to the other sample of the study. Ideally we would have recruited 100 or more participants within this population to reach sufficient power, and some of the trends may have achieved significance with a large sample. However, due to time constraints and length of the treatment program, achieving our goal was not possible.

Further, the measures used are another important limitation to consider. All of the measures used in this study were self-report measures that rely upon the participants’ willingness to divulge personal information. While all appropriate procedures were taken to ensure the anonymity, confidentiality, and security of the participants’ personal information, there is the possibility that the participants may have not been completely honest with their responses. However, counter measures were put in place, such as deception scales, to try to minimize the inaccurate responses. Additionally, the anonymity may have encouraged participants to complete the survey as rapidly as possible to get the compensation for participation, but counter measures
were also put in place to minimize this threat by including attentional checks and minimum response times.

Lastly, due to the fact that this study collected retrospective data, causality cannot be determined. This is primarily due to the cross sectional nature of this type of data collection. Moreover, the legal status of cannabis limits our ability to conduct experimental research with cannabis administration. With the legal status of cannabis potentially changing, it is hoped that in the near future we will be able to conduct the much needed research on cannabis by having the ability to administer cannabis in appropriate experimental studies. These studies will be crucial in gain definitive answers to questions that we have raised.

Furthermore, future research will benefit by applying the same methodologies taken here to other forms of aggressive behaviour. This includes, but is not limited to verbal aggression, social aggression, and psychological aggression. Using consistent measures of CU with the appropriate measure of aggression and conducting such research across multiple populations could prove to help solidify the conclusions that are made and would eliminate any potential discrepancies between studies like the ones mentioned in this paper. With this information in hand we are one step closer in determining if cannabis is associated with aggression and once and for all lay to rest this debate.
References


