

**The relationship between the main symptoms of ADHD
and alcohol, cannabis and nicotine**

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Thesis submitted for the degree of
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Declaration

I declare that the work presented in this thesis is my own. All research and work detailed in the text of this thesis is novel and has not been previously submitted as part of the requirements of a higher degree.

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that starts in early childhood and continues throughout the lifetime. The main symptoms of ADHD are inattention and hyperactivity/impulsivity. Even though substance misuse is one of the main comorbid conditions of ADHD, it has been an exclusion criterion in most studies of ADHD. The limited number of studies on ADHD and substance misuse measured ADHD as a whole, but in this thesis, the two main symptoms of ADHD have been divided to investigate the relationship between each symptom and developing alcohol, cannabis, and nicotine use. Studies show that individuals with ADHD and comorbid substance misuse become drug addicted at a younger age, use higher amounts of drugs, relapse is more common among them and ADHD medication treatments are less effective in them. There are common personality traits and impairments in individuals with the symptoms of ADHD and those who use alcohol, cannabis and nicotine. The studies of this thesis investigated the role of different facets of Executive Functions (EF), facets of impulsivity, emotional regulation, bipolar disorder and sleep quality in the relationship between each ADHD symptom and alcohol, cannabis, and nicotine use in typically developing participants. The results of Study 1 showed that hyperactivity/impulsivity explained additional variance after accounting for the facets of EF. Study 2 showed that inattention explained additional variance over and above the facets of impulsivity. Additionally, in studies 3, 4 and 5, emotional regulation, bipolar disorder and sleep quality were partial mediators between each ADHD symptom and some alcohol, cannabis and nicotine use scores, which are presented in detail in the next chapters.

Acknowledgement

For me as a self-funded, overseas student, this work could not have been possible without the financial support of my parents who paid all my University and accommodation fees during my whole studies.

I would like to thank my supervisors Dr. Ashok Jansari and Dr. Andrew Cooper for their support and advice during my PhD and writing this thesis: with your knowledge and guidance, I became a better and more critical researcher. I am grateful to Dr. Alice Jones, who encouraged and supported me educationally and psychologically to develop myself as a researcher: I will always remember your calm and beautiful smile, your kind compliments and encouragements, which helped me to regain my self-confidence. I would like to express my enormous gratitude to Prof. Alan Pickering, whose support, advice and wisdom was a source of inspiration enabling me to complete this journey: with your help, the entire PhD became a more pleasant and memorable chapter of my life.

My special thanks go to my mother and my father, Nikoo Hassan Moradi and Abolghassem Safaryazdi, who supported me not only during my PhD, but also during the thirty-six years of my life. Without your emotional, financial and psychological support I would not be here. I finished my PhD because of you, I reached my many goals because of you, I am Zahra because of you and I am because of you.

I am very grateful to my one and only sister, Niayesh Safaryazdi, who is always there for me like my best friend: in the happiest and saddest moments of my life you are always there, and I am the most real me beside you.

I am so thankful to Emad Eisapour for his love, patience and understanding: you came to my life making it more joyful and beautiful, and your love gave me the strength more than ever to complete the last leg of my journey.

My sincere thanks also go to three beautiful people, Maria Stavrou, Dimitra Kale and Eilish Duke, as my PhD sisters who supported me in my hardest days during my crazy PhD journey: we laughed and cried together, you were by my side during both the happiest and the hardest times and that is why I appreciate every moment and memory of being with you. I thank Fiona McCapra and Adrian Bradbury who supported me spiritually like my own parents during the years away from home: your kindness made me feel part of the family. I would also like to thank the Funds for Women Graduates (FfWG) for funding my final year accommodation fees.

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List of presentations relevant to this thesis

Safaryazdi, Z., Jansari, A., Cooper, A. (2019). The role of executive functions in the relationship between ADHD symptoms and substance misuse. Symposium presentation at the International Society for the Study of Individual Differences (ISSID). Florence, Italy.

Safaryazdi, Z., Jansari, A., Cooper, A. (2017). The role of executive functions in the relationship between ADHD symptoms and substance misuse. Poster presented at the Society for the Study of Addiction (SSA), Newcastle, UK.

Safaryazdi, Z., Jansari, A., Cooper, A. (2017). The role of executive functions in the relationship between ADHD symptoms and substance misuse. Poster presented at the 8th annual conference of the British Society for the Psychology of Individual Differences. Kent, UK.

Chapter 1

Overview

This chapter will introduce the main aspects of existing literature that have informed the investigations presented in this thesis. First, an overview of ADHD as a childhood and adulthood condition will be given, along with a description of why individuals with ADHD symptoms are particularly vulnerable to substance misuse. Second, a DSM-4 and DSM-5-based definition of substance misuse, an outline of specific substances of interest and a discussion of key terms will be provided. Third, the relationship between ADHD and substance misuse and a brief background of studies centred on this association will be discussed. Fourth, the concept of shared risk factors in individuals with ADHD symptoms and individuals with substance misuse will be introduced. Finally, the programme of investigations conducted for this thesis will be presented, with specific aims and research questions outlined. The introductory chapter does not aim to give a comprehensive review of literature on ADHD, substance misuse and the different shared impairments between these two conditions. Its objective is to provide an overview of existing knowledge in each of these areas to show why the current research is needed.

1.1. Attention deficit hyperactivity disorder (ADHD):

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder (Caroline, 2010; Sroubek et al., 2013). The symptoms start in early childhood and can continue into adulthood (Schonwald & Lechner, 2006; Silver, 2004; Sim et al., 2004).

The American Psychiatric Association (APA) published the Diagnostic and Statistical Manual of Mental Disorders (DSM), which contains standard criteria for the classification of mental disorders. It is relied upon and used by researchers, clinicians and other health providers in conjunction with other World Health Organisation (WHO) alternatives, such as Classification of Mental and Behavioural Disorders (ICD-10) (Dalal & Sivakumar, 2009). The fifth edition of DSM (DSM-5) was approved by the APA in 2012 and published in 2013. It is the first major edition in the last twenty years to have extensively revised diagnoses (DSM-5, 2013)

According to the DSM-5, ADHD is characterised by attention problems, extreme activity or problematic behaviour control inappropriate for an individual's age (APA, 2013; National Institutes of Health [NIH], 2016). Additionally, the symptoms of ADHD should be present for more than six months in more than two settings (school, home, etc.) before the age of 12 (DSM-5, 2013; Dulcan et al., 2011).

Symptoms similar to ADHD have been described in the medical literature since the 19th century (Lange et al., 2010). Since the 1970s, ADHD diagnosis and treatment have been considered controversial between clinicians, parents, teachers and the media (Parrillo, 2008). These controversies include the causes of ADHD and the use of stimulants as medications in ADHD treatment (Mayes et al., 2008). Despite being one of the most widely studied and diagnosed mental disorders, the cause of ADHD in children and adolescents is unknown in most cases (National Institute of Mental Health [NIMH], 2013).

The global community prevalence of ADHD has been reported to be 2% to 7% (5% on average) in systematic reviews (Polanczyk et al., 2014; Willcutt, 2012). Additionally, a

further 5% of children who are only sub-threshold to meet full diagnostic criteria for ADHD have significant difficulties with hyperactivity, inattention and impulsivity (Sayal et al., 2018). According to survey studies, the worldwide prevalence of clinical ADHD varies, ranging from 2.5% to 3.4% (Fayyad et al., 2007; Simon et al., 2009). Even though clinically diagnosed ADHD is increasing, it remains an under-recognised and under-diagnosed condition, especially in girls. Childhood ADHD symptoms often persist into adulthood and can be risk factors for developing other mental health disorders with adverse educational, occupational and relationship outcomes (Sayal et al., 2018).

None of the UK studies (Hsia et al., 2009; Holden et al., 2013; Jick et al., 2004; McCarthy et al., 2009; McCarthy et al., 2012; O'Leary et al., 2014; Wong et al., 2009) reported the administrative prevalence of ADHD based solely on a diagnosis. Prevalence based on prescriptions ranged from 0.003% in those under the age of 19 in 1992 (Hsia et al., 2009) to 0.92% in children aged 6 to 12 in 2008 (McCarthy et al., 2012). Between 2011 and 2012, the prescription-based prevalence of ADHD with and without diagnosis ranged from 0.19% in 6- to 17-year-olds in 1998 to 0.76% in 5- to 15-year-olds (Holden et al., 2013). According to The Health Improvement Network's (THIN) database, 0.73% of 6- to 12-year-olds, 0.57% in 13- to 17-year-olds and 0.06% in 18- to 24-year-olds have ADHD (McCarthy et al., 2012).

Holden et al.'s (2013) study found that the prevalence of diagnosed ADHD in routine practice in the UK was lower than previous reports; since 2007, both the incidence and prevalence of diagnosed ADHD have fallen in primary health care (Holden et al., 2013). Their research also indicated that both incidence and prevalence were higher in men than women (Holden et al., 2013).

In terms of gender, studies show that boys are diagnosed with ADHD three times more than girls, although the symptoms in girls are often overlooked because of the difference in symptoms between girls and boys (Crawford, 2003; Emond et al., 2009; Gershon, 2002; Singh, 2008). Typically, girls display more inattention symptoms than boys, and they are not as hyperactive (Crawford, 2003).

In terms of ADHD treatments, they vary by countries, but they usually entail changes in an individual's lifestyle, medication and counselling (NIMH, 2016). According to British guidelines, medication is the first-line treatment for children with severe symptoms and adults with ADHD. Medication is also for those with moderate symptoms who fail to improve with counselling (National Collaborating Centre for Mental Health, 2009). Nonetheless, American and Canadian guidelines recommend a combination of medication and behavioural therapy as a first-line treatment for children except for preschool-aged ones (Canadian ADHD Practice Guidelines, 2011; Centres for Disease Control and Prevention [CDC], 2015). Although the long-term effects of stimulant treatment are unclear, it is effective for up to 14 months (Arnold et al., 2015; National Collaborating Centre for Mental Health, 2009; Parker et al., 2013; Tsai & Huang, 2011). Although studies on the coping skills in adults with ADHD symptoms are very limited but previous studies show that those with diagnosed and undiagnosed ADHD develop coping skills that can help them compensate for some of their impairments (Canela et al., 2017; Liebreinz et al., 2014). For instance, using substances (Nehlin et al., 2015) such as nicotine (Liebreinz et al., 2014), physical activity (Gapin et al., 2011), changing the environment to suit the needs of the individual (environments with few stimuli) (Ramsay & Anthony, 2014) are some of the examples of coping skills that have been chosen by individuals with ADHD symptoms.

1.1.1. ADHD presentations

The term subtype in DSM-4 was changed to ‘presentation’ in the DSM-5, which denotes the fluid nature of symptoms across one’s lifespan rather than being stable traits over time. For instance, inattention is somewhat constant over time, but hyperactivity/impulsivity often changes with age (Hurtig et al., 2007). The term ‘presentation’ best reflects the evolving symptom profile of an individual’s current symptomatology. However, the term ‘subtype’ refers to more trait-like and stable characteristics (Eptein & Loren, 2013).

According to the DSM-5, there are three presentations of ADHD: (a) inattentive type with symptoms of inattention and a few or no symptoms of hyperactivity; (b) hyperactive/impulsive type with more hyperactivity and impulsivity symptoms, but few or no symptoms of inattention, which is less common; and (c) a combined type with both symptoms of inattention and hyperactivity/impulsivity (APA, 1994, 2013). However, in contrast to the general diagnosis, these presentations are not stable over time and may provide additional details for diagnosis and treatment (Willcutt et al., 2012). Due to the lack of evidence for actual presentations of ADHD, the DSM-5 indicates varying levels of inattentive and hyperactivity/impulsivity symptoms presentation (APA, 2013).

1.1.2. Main symptoms of ADHD

Common symptoms of ADHD are inattention, hyperactivity, impulsivity and disruptive behaviour (CDC, 2016; Dobie, 2012). Moreover, academic difficulties are as frequent as relationship problems in individuals with ADHD symptoms (Dobie, 2012). However, it is

difficult to define the symptoms of this disorder because it is challenging to distinguish normal levels of inattention, hyperactivity and impulsivity and elevated levels that require intervention (Ramsay, 2007). As mentioned in the previous section, ADHD is currently classified into three potential presentations, each of which has its own set of symptoms of each subgroup (Table 1.1).

Table 1.1
ADHD presentation based on DSM-5 (2013)

| ADHD presentations | Symptoms |
|---------------------------|--|
| Inattentions | Have difficulty focusing on one task Miss details and forgets things, be easily distracted and switch from one activity to another Get easily bored in a few minutes unless the task is interesting Have a problem focusing attention completing a task or learning new things. Not listening while spoken to Have daydreaming, moving slowly and get confused easily Not being able to process the information with the same speed and accuracy as others. Have trouble following instructions. Struggle to understand minute details |
| Hyperactivity/impulsivity | Squirm or fidget while seated Talk nonstop Playing with or touching everything in sight Have difficulty sitting still while eating meals, doing homework, in the classroom, and storytime. Be continuously in motion Struggle to do quiet tasks and activities Very impatient Act without regards to the negative consequences, use inappropriate comments and express emotions without limitations Have problem waiting turns and for things that they want Interrupt others' activities or conversations |
| Combined | Diagnosed when ≥ 6 symptoms of inattention and ≥ 6 symptoms of hyperactivity/impulsivity are present for ≥ 6 months. |

1.1.3. Childhood and adulthood ADHD

Evidence suggests that ADHD symptoms persist into adulthood in most cases, with different clinical and psychosocial impairments (Kooij et al., 2010). Based on the National Institute for Health and Clinical Excellence (NICE, 2018), persistent ADHD symptoms into adulthood are associated with major impairments. Longitudinal studies indicate that around two-thirds of children with ADHD symptoms continue to have the symptoms in their adolescence and adulthood (Lara et al., 2009; Rasmussen & Gillberg, 2000). However, while

some individuals display full symptoms, others appear to show ‘partial remission’ from symptoms with some continued clinical and psychosocial impairments. Faraone and colleagues (2006) conducted a meta-analysis of follow-up studies that examined the persistence of ADHD into adulthood. Their results showed that 15% of individuals retain a full diagnosis, with 50% in partial remission until the age of 25 (Faraone et al., 2006). Between 1% to 6 % of adults suffer from ADHD symptoms (Riegler et al., 2017). Meta-analyses have also demonstrated that there is a similar (5.7%) prevalence of ADHD between children and adults (Polanczyk et al., 2014; Willcutt, 2012). More recent longitudinal studies in the UK have reported higher persistence rates (79%) of ADHD symptoms into adulthood (Cheung et al., 2015; Van Lieshout et al., 2016).

The Adult Psychiatric Morbidity Survey (APMS, 2014) provides the data for ADHD in England’s general population. The Office for National Statistics’ surveys in 1993 and 2000 delivered the data of adult psychiatric morbidity in England, Scotland and Wales, but the 2007 survey only included people over the age of 16 in England. All APMS surveys used broadly consistent methods to combine the samples. Both the 2007 and 2014 surveys used a six-item adult ADHD self-report scale (ASRS) to assess inattention, hyperactivity and impulsivity in the past six months. A score of 4 or more indicated a positive screen for ADHD.

Based on the survey data in 2014, one in ten adults (9.7% with a similar rate for men and women) screened positive for ADHD (Figure 1.1). This rate was slightly higher than in the previous survey in 2007. The survey also indicated that younger adults showed more ADHD symptoms (Figure 1.2). Only 3.7% of those who tested positive for ADHD believed they had the disorder. Furthermore, even though 2.3% had been diagnosed, only 0.5% of them were

taking medication for their ADHD. In the UK, only a limited number of studies have focused on measuring the symptoms of ADHD in the general population. More non-clinical investigations are needed in the future to assess the main ADHD symptoms in adults.

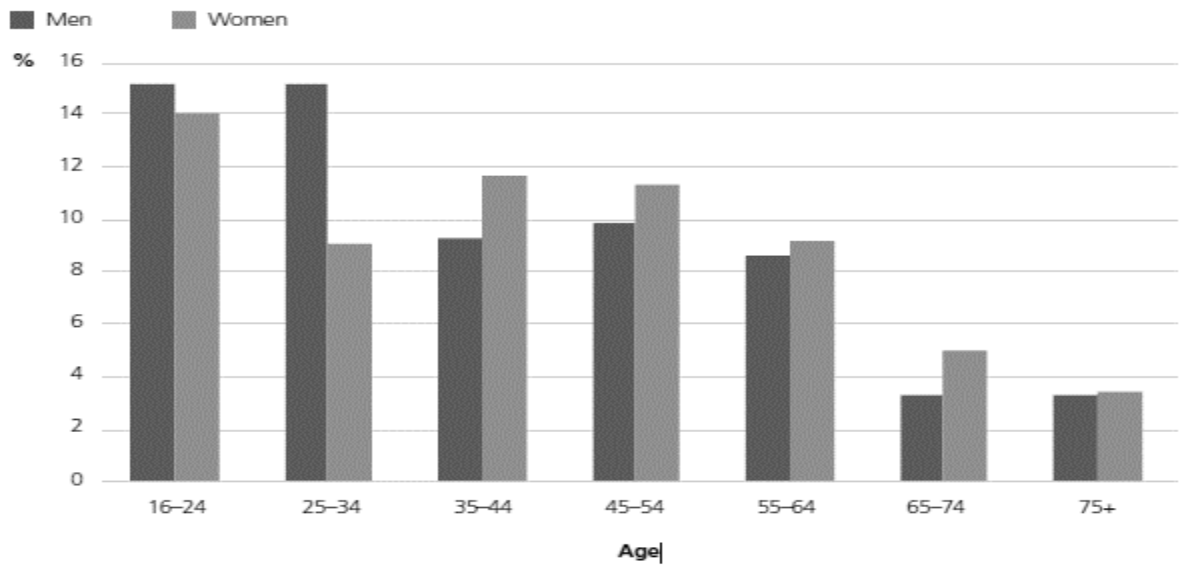


Figure 1.1: Screen positive for ADHD (score of 4 or more on the ASRS), by age and sex in England (APMS, 2014)

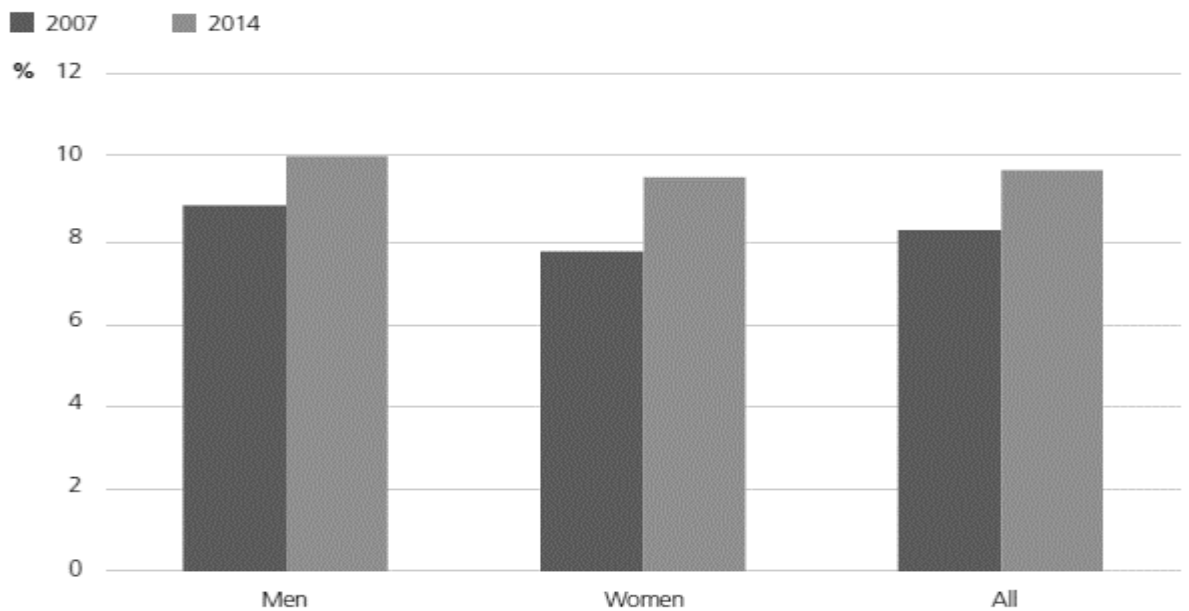


Figure 1.2: Screen positive for ADHD in 2007 and 2014, by sex in England (APMS, 2014)

Individuals with ADHD symptoms show poor social functioning (Bishop et al., 2019). Likewise, they have problems in social interactions and forming and keeping friendships compared to their peers without ADHD symptoms (Coleman, 2008). Half of all children and adolescents with ADHD symptoms are rejected by their peers compared to the 10% to 15% of children without ADHD symptoms. Those with ADHD symptoms also demonstrate difficulties in processing verbal and nonverbal language, which worsen their social interaction. Thus, they miss social cues and struggle to learn social skills (Coleman, 2008).

Another issue that individuals with ADHD symptoms face is developing substance misuse, which may result in Substance Use Disorder (SUD) (Blevins et al., 2020; Bron et al., 2013; Levin et al., 2018; Luderer et al., 2019; Tamm et al., 2013). Alcohol and nicotine misuse, along with illegal substance misuse, may start in adolescence and continue into adulthood (Bernardi et al., 2011; Gonzalez et al., 2017; Kooij et al., 2010). Over the past decade, many studies have demonstrated that the co-occurrence of ADHD and SUD is a prevalent phenomenon in adults and adolescents (Bernardi et al., 2011; Glind et al., 2014; Gonzalez et al., 2017; Kooij et al., 2010; Lee et al., 2011; Nogueira et al., 2014).

Studies reveal that individuals with ADHD symptoms and comorbid SUD become drug addicted at a younger age, use higher amounts of drugs and are hospitalised more than those without ADHD symptoms (Arias et al., 2008). Relapse is more common among people with ADHD symptoms even after successful SUD treatment (Carroll & Rounsaville, 1992). In addition, pharmacological studies suggest that the two main ADHD medications, methylphenidate and atomoxetine, are less effective in individuals with both ADHD and SUD than in those with only ADHD (Carpentier et al., 2005; Castells et al., 2011; Konstenius et al., 2010; Levin et al., 2007; McCabe et al., 2016; Thurstone et al., 2010; Wilens et al.,

2011). Thus, the next section is dedicated to the definition of substance misuse, an outline of specific substances of interest and a discussion of key terms.

1.2. Substance misuse and Substance Use Disorder (SUD)

DSM-4 classified SUD as substance-related disorders, which involved only substance or drug abuse disorders. This classification has been expanded in the DSM-5 to include gambling disorder; the section's name has been changed to substance-related and addictive disorders. An individual must meet two out of 11 criteria in a year to be diagnosed with SUD, which is a combination of substance dependence and substance abuse in the DSM-5. Within 12 months, mild SUD occurs when individuals exhibit two to three out of 11 total symptoms. Moderate SUD arises when four to five symptoms are endorsed, and severe SUD happens when six or more symptoms are noted in an individual (APA, 2013). Table 1.2 compares the symptoms of SUD in the DSM-4 and the DSM-5.

Table 1.2
Comparison of DSM-IV, DSM-5, and NSDUH Substance Use Disorder Assessment

| Characteristics | DSM-IV | DSM-5 |
|-------------------|--|--|
| Disorder Class | Substance-related disorders, included only SUDs | Substance-related and addictive disorders class now includes SUDs and gambling disorder (formerly pathological gambling) |
| Disorder Types | Abuse and dependence hierarchical diagnostic rules meant that people ever meeting criteria for dependence did not receive a diagnosis of abuse for the same class of substance | SUD, substance abuse and dependence have been eliminated in favor of a single diagnosis, SUD |
| Disorder Assessed | Substance abuse: One or more symptoms | SUD : Two out of 11 criteria clustering in a 12-month |

| | | period are needed to meet disorder threshold |
|--|---|--|
| | <ul style="list-style-type: none"> • Recurrent substance-related legal problems | <ul style="list-style-type: none"> • Dropped |
| | <ul style="list-style-type: none"> • Recurrent substance use in situations where it is physically hazardous | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance | <ul style="list-style-type: none"> • Same |
| | | <ul style="list-style-type: none"> • Added: Craving or a strong desire or urge to use the substance |
| | <p>Substance dependence: Three or more symptoms in the same 12-month period (or one symptom if dependence criteria have been met previously in the lifetime)</p> | |
| | <ul style="list-style-type: none"> • Substance is taken in larger amounts or over a longer period than was intended | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • There is a persistent desire or unsuccessful efforts to cut down or control substance use | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • Important social, occupational, or recreational activities are given up or reduced because of substance use | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • Substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by substance use | <ul style="list-style-type: none"> • Same |

| | | |
|---------------------------|---|---|
| | <ul style="list-style-type: none"> • Tolerance, as defined by either: (1) a need for markedly increased amounts of substance to achieve intoxication or desired effect or (2) a markedly diminished effect with continued use of the same amount of the substance | <ul style="list-style-type: none"> • Same |
| | <ul style="list-style-type: none"> • Withdrawal, as manifested by either: (1) the characteristic withdrawal syndrome for the substance (excludes Cannabis, Hallucinogens, and Inhalants) (2) the substance (or a similar substance) is taken to relieve or avoid withdrawal symptoms | <ul style="list-style-type: none"> • Withdrawal, as manifested by either: (1) the characteristic withdrawal syndrome for the substance (excludes Phencyclidine, Other Hallucinogens, and Inhalants) (2) the substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms Note: This criterion is not considered met for those taking opioids, sedatives, hypnotics or anxiolytics, or stimulant medications solely under appropriate medical supervision. |
| Severity | No severity criteria | Severity is assessed in terms of the number of symptoms that meet criteria: Mild: two to three symptoms Moderate: four to five symptoms Severe: six or more symptoms |
| Additional Specifications | With or without physiological dependence, early full remission, early partial remission, sustained full remission, sustained partial remission, on agonist therapy, and in a controlled environment | Early or sustained remission and if the person is in a controlled environment where access to the substance is restricted |

NOTE: DSM-IV = *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition; DSM-5 = *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition

Recurrent substance-related legal problems, such as arrests for substance-related disorderly conduct, were removed as a substance abuse criterion in the DSM-5 (APA, 2013), and a craving criterion was added. This elimination was due to low endorsement, poor fit with other items and poor discrimination of the legal problems criterion (Hasin et al., 2013). Based on the DSM-5 definition, craving is a ‘strong desire or urge to use the substance’. It also ‘makes it difficult to think of anything else’ and ‘often results in the onset of use’ (2, p. 492). It has been shown that including craving with the abuse criteria could significantly add to the diagnostic information; craving may become a target for biological treatments (Hasin et al., 2011). Hence, including craving in the DSM-5 criteria improves classification system consistency.

Another DSM-5 criterion that has undergone some changes is the dependence criterion under withdrawal. The only criteria related to the substance’s physiological effect are the withdrawal symptoms (Centre for Behavioural Health Statistics and Quality, 2016). In both the DSM-4 and the DSM-5, withdrawal is defined as an individual (a) having the characteristic withdrawal symptoms of a substance or (b) using a related or similar drug to avoid the symptoms of a specific substance.

Except for cannabis, the withdrawal criteria for all substances are unchanged in both the DSM-4 and the DSM-5. Following the publication of the DSM-4, various investigations found a cluster of symptoms related to cannabis withdrawal, which has now been included in the DSM-5 (APA, 2013). Cannabis withdrawal syndrome occurs when three or more symptoms develop within a week of stopping heavy and continued cannabis use. Symptoms include (a) depression, anger or irritability; (b) anxiety or nervousness; (c) sleep problems such as insomnia and disturbing dreams; (d) reduced appetite or weight loss; (e) restlessness;

and (f) one or more physical problems causing major discomforts such as sweating, chills, headache, shakiness and abdominal pain (Table 1.3).

Table 1.3
DSM-4 to DSM-5 Withdrawal Symptoms Comparison

| Substance | Symptoms | DSM-4 | DSM-5 |
|-----------|---|-------|-------|
| Alcohol | Two or more symptoms | √ | √ |
| | Autonomic hyperactivity | √ | √ |
| | Increased hand tremor | √ | √ |
| | Insomnia | √ | √ |
| | Nausea or vomiting | √ | √ |
| | Transient visual, tactile, or auditory hallucinations or illusions | √ | √ |
| | Psychomotor agitation | √ | √ |
| | Anxiety | √ | √ |
| | Generalized tonic-clonic seizures (formerly grand mal seizures) | √ | √ |
| Cannabis | Three or more symptoms | | √ |
| | Irritability, anger, or aggression | | √ |
| | Nervousness or anxiety | | √ |
| | Sleep difficulty (i.e., insomnia, disturbing dreams) | | √ |
| | Decreased appetite or weight loss | | √ |
| | Restlessness | | √ |
| | Depressed mood | | √ |
| | At least one of the following physical symptoms causing significant discomfort: abdominal pain, shakiness/tremors, sweating, fever, chills, or headache | | √ |
| Tobacco | Four or more symptoms | √ | √ |
| | Irritability, frustration, or anger | √ | √ |
| | Anxiety | √ | √ |
| | Difficulty concentrating | √ | √ |
| | Increased appetite | | √ |
| | Increased appetite or weight gain | √ | |
| | Restlessness | √ | √ |
| | Depressed mood | √ | √ |
| | Insomnia | √ | √ |
| | Decreased heart rate | √ | |

Note: *DSM-4 = Diagnostic and Statistical Manual of Mental Disorders, 4th edition; DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, 5th edition*

Alcohol Use Disorder

In DSM-4, alcohol abuse entails meeting one or more of four criteria, while alcohol dependence requires fulfilling three or more of seven criteria. In DSM-5, alcohol use disorder (AUD) meets two or more of 11 criteria (Centre for Behavioural Health Statistics and Quality, 2016). Moreover, in the DSM-5, the craving criterion was added, the legal problems criterion was removed, and the threshold for diagnosis was modified.

Cannabis Use Disorder

Since the initial publication of DSM-4, various investigations have found withdrawal symptoms that lead to significant clinical impairments, which are recognised in the revised DSM-5 (Hasin et al., 2008). Cannabis withdrawal symptoms in the DSM-5 include showing three or more symptoms within a week of cessation of heavy and continued cannabis use: (a) aggression, anger or irritability; (b) nervousness or anxiety; (c) sleep problems; (d) depressed mood; and (e) more than one physical problem causing substantial discomfort.

Tobacco Use Disorder

The diagnostic criteria for nicotine or tobacco use disorders have changed significantly from the DSM-4 to the DSM-5. The DSM-4 only measured nicotine dependence, and different studies used various scales, such as the Fagerstrom Nicotine Dependence Scale (Fagerstrom & Schneider, 1989), to measure nicotine dependence (Hasin et al., 2003). Investigations showed that using nicotine in dangerous situations (in bed) and continuing use with no regard to problems (not being able to work in non-smoking positions) are highly relevant due to an increase in laws prohibiting nicotine use in public areas. Thus, the DSM-5

added nicotine addiction to other SUDs. The DSM-5 refers to nicotine use as tobacco use disorder to acknowledge the departure from the previous dependence diagnosis.

Based on the Global Burden of Disease (GBD, 2016), the most prevalent SUD was AUD, with more than 100 million cases in 2016. It was followed by cannabis use disorder (22.1 million cases) and opioid use disorder (26.8 million cases), both of which were the most common illegal SUD. The GBD findings also revealed that alcohol use accounted for 99.2 million Disability-Adjusted Life-Years (DALYs); 31.8 million DALYs were attributed to drug use as a risk factor (GBD, 2016).

Statistics on drug misuse in the UK (NHS Digital, 2018)

Since the research for this thesis was conducted in the UK, it is important to know the prevalence of substance consumption and problematic substance misuse in this country. In 2017/18, about 8.5% of adults (1 in 12) aged 16 to 59 had used illegal drugs in the last 12 months, totalling 2.7 million people. The level of drug use by adults in 2015/16 was almost similar to that in 2016 (8.4%), but it was much higher 10 years earlier in 2006/07 (10.1%). Moreover, 19.2% of young adults aged 16 to 24 (1 in 5) had used only one illegal substance in the last year, amounting to around 1.1 million people (4.3% men and 1.9% women). This level was close to the reports from 2015/16 (18.0%), but the study from 2006/07 was significantly higher a decade ago (24.2% in 2005/06). Reports also show that the drug dependence level among adults in England and Wales decreased with age.

The most widely consumed drug was cannabis in 2017/18 as in previous years. Around 2.1 million people aged 16 to 59 used cannabis over a year (6.5%), which was close to the 2015/16 figures (6.7%) but significantly lower than a decade ago (8.2%). In England and

Wales, men aged 16 to 59 used cannabis twice more (9.0%) than women (4.2%) in a year. Cannabis use in 2018–2019 in England and Wales was 17% among 16- to 24-year-olds, the highest point in a decade.

Based on the latest NHS Digital report (2020), hospital admissions for drinking alcohol-related reasons in the UK were 358,000 in 2018/19, 6% higher than 2017/18 and 19% higher than 2008/09. Additionally, there were 5,698 alcohol-specific deaths in 2018, a 2% decrease from 2017 and a 7% increase from 2008. With over 14 units of alcohol per week, men and women aged 55 to 64 had the highest proportion of alcohol consumption.

The reports of NHS Digital (2019) show 489,300 hospital admissions attributable to smoking nicotine in 2017/18, representing 4% of all hospital admissions. Furthermore, smoking was responsible for 77,800 deaths in 2017, accounting for 16% of all deaths. The reports also demonstrate that 14.4% of adults aged above 18 are current smokers. Of the constituent countries, current smoking nicotine prevalence was 14.4% in England, 15.5% in Northern Ireland, 15.9% in Wales and 16.3% in Scotland. Men (16.4%) were more likely than women (12.6%) to smoke currently, and the most likely age group to smoke was 25 to 34. In addition, there are 3 million vapers in the UK in 2019, which is 5.7% of the survey respondents who are presently using an e-cigarette (Office for National Statistics, 2019).

1.2.1. Development of substance misuse to SUD and the neurobiology of alcohol, nicotine and cannabis use

Developing SUD has different stages. Initiation is the first use of alcohol and other substances. This stage typically involves peer pressure and using substances for recreational use or breaking the rules of parents or authority figures (Hser et al., 2003). The second stage is drug intake escalation, which is characterised as an increase in the person's drug use that may become excessive, overwhelming and difficult to control; the individual misses more school or work, considers taking drugs and fears losing their source of supply. They use alcohol or drugs for distinct reasons, such as to cope with their negative emotions and feelings (Edwards & Koob, 2013) or to prevent withdrawal symptoms (alAbsi et al., 2004; Simioni et al., 2012).

SUD is described as compulsive substance seeking and escalated drug use (Edwards & Koob, 2013). Excessive drug use promotes tolerance to the rewarding effects of drugs and expressing negative emotional states such as anxiety, dysphoria and irritability, which leads to losing control over intake. There is a clinical separation between recreational drug use and escalated drug intake, and compulsive drug-seeking behaviour that describes addiction in rodents and humans. A certain resilience to drug intake may be the norm in humans (Ahmed, 2010, 2012; Swendsen & Le Moal, 2011), highlighting the difference between simple drug use and developing addiction in vulnerable populations (George & Koob, 2010). Thus, exploring the neurobiology basis of different substances of abuse that are the focus of this thesis could help gain a better understanding of the impact of substances on the brain and body.

Alcohol

Dopamine (DA) is involved in the mesolimbic system circuit, affecting incentive motivation and how an individual reacts to motivational changes in the environment. Studies show an association between alcohol consumption and dopamine, which plays a role in incentive motivation. Low doses of a compound injected directly into the nucleus accumbens can block alcohol consumption by interfering with dopamine's normal activity (Hodge et al., 1997; Rassnick et al., 1992).

Additionally, using alcohol or anticipating alcohol availability produces dopamine in the nucleus accumbens, and increased dopamine in the fluid outside neurons can be used to determine it (Weiss et al., 1993). However, dopamine is an important but not essential component of alcohol reinforcement (Rassnick et al., 1993). Furthermore, in individuals with alcohol dependence, alcohol withdrawal reduces dopamine function, which can cause alcohol use relapse (Volkow et al., 2007).

The blood–brain barrier could be penetrated by ethanol, as a liposoluble neurotropic substance. Ethanol is directly toxic to the brain and can inhibit functions of the central nervous system (CNS). Alcohol dependence could cause psychological, biological and socio-environmental problems, demonstrating the aetiology and pathology of AUD. CNS neurotransmitters are crucial in the development of AUD. Previous investigations revealed various neurotransmitters involved in alcohol metabolism: dopamine, glutamate, norepinephrine, endogenous opioid transmitter, 5- hydroxytryptamine, acetylcholine and γ -aminobutyric acid (Chen & Liang, 2007).

Several studies demonstrate that the amygdala, a part of the reward circuitry, plays a significant role in the effects of alcohol on the brain. In a study, investigators examined the effect of different doses of ethanol (0–2.0 g/kg) on DA release in the brain. Their results showed that high doses of ethanol (1 and 2 g/kg) had no effect on extracellular DA levels linearly and that DA levels returned to baseline in 90 minutes (Yim et al., 2000). This outcome shows the acute tolerance of the DA release to ethanol in the nucleus accumbens (NAc), and that the ethanol-induced DA release is dependent on the ethanol concentration. Other investigations revealed that ethanol could affect Gamma aminobutyric acid (GABA) neurons and opioid receptors in the NAc, which increases DA indirectly (Adermark et al., 2011; Cowen & Lawrence, 1999; Spanagel et al., 1992).

Soder et al. (2019) tested a sample of undergraduate students to explore whether genetic markers related to increased DA neurotransmission are associated with increased alcohol use and decreased neural sensitivity to costs. They measured DA transmission score using five genetic markers linked to DA transmission. They also assessed neural sensitivity to cost using the feedback-related sensitivity (FRN) and an event-related potential implicated in neural outcome evaluation on passive evaluation and active decision-making tasks. Moreover, they determined alcohol consumption using a self-report questionnaire. In both tasks, students with higher DA transmission scores showed increased alcohol use and a more blunted FRN. Although chronic alcohol users have dopamine hyposensitivity, the results of the Soder et al. (2019) study indicated that dopamine hypersensitivity may heighten alcohol consumption in individuals who have not yet developed AUD. Other studies also found a highly significant DA-releasing effect of alcohol compared to placebo in all striatal subregions of all participants, and that alcohol use combined with its expectation induces DA

release (Boileau et al., 2003; Kegeles et al., 2018; Urban et al., 2010; Yoder et al., 2007; Yoder et al., 2016).

Serotonin is another neurotransmitter that is associated with impulsivity and alcohol consumption behaviour in rats and humans (Virkkunen & Linnoila, 1993). Alcohol use may increase serotonin release in the nucleus accumbens, which is a contributor to feelings of happiness and well-being (Young, 2007). Furthermore, alcohol increases the activity of GABA, a key inhibitory neurotransmitter. Studies suggest that chronic alcohol use increases GABA transmission, which is responsible for muscle tone regulation and motor control (Watanabe et al., 2001).

Nicotine

Nicotine dependence has three phases. First, nicotine use in humans produces a mild euphoria and a slightly pleasurable rush. It also increases arousal and relaxation and reduces fatigue (Henningfield et al., 1985). Nicotine's effects lead individuals to initiate and continue smoking, which can lead to nicotine dependence (Markou, 2008; Watkins et al., 2000). Second, nicotine dependence develops through chronic nicotine use, which causes neuroadaptations in the brain's reward system. Therefore, individuals with nicotine dependence continue smoking to avoid effective withdrawal and distressing somatic symptoms. In newly abstinent smokers, anxiety, irritability, concentration problems, depression, insomnia, weight gain and gastrointestinal discomfort have been observed (Hughes et al., 1991; Shiffman & Jarvik, 1976). The withdrawal syndrome in rats and mice is similar to that in humans, with somatic and negative affect symptoms (Malin et al., 2006;

Watkins et al., 2000). Somatic symptoms of nicotine withdrawal include facial tremors, shakes, chewing, abdominal constriction, rearing, jumping and scratching. The negative affective symptoms include anhedonia, which is a reduced responsiveness to old rewarding stimuli. Third, the probability of relapse is high for weeks, months and years in abstinent smokers. Smoking relapse, like relapse in other drug abuse, happens in the presence of a person, object or place that an individual has associated with the drug's rewarding effect (Hughes et al., 2004). Stress is a known risk factor of smoking initiation (Holliday & Gould, 2016; Huizink et al., 2009), smoking maintenance (Shaw & al'Absi, 2010) and relapse (al'Absi, 2006; al'Absi et al., 2015; Childs & De Wit, 2010; Dupont et al., 2012; Lemieux & al'Absi, 2016; McKee et al., 2015).

Nicotine activates the brain neurons' nicotine acetylcholine receptors (nAChRs), which can affect mood, cognition and body function (D'Souza & Markou, 2011). After being activated by nicotine or the endogenous neurotransmitter acetylcholine, nAChRs open a channel through which ions can pass and causes changes in the cell.

Nicotine can interact with nAChRs on neurons in the brain's mesolimbic reward system and produce rewarding effects. Dopaminergic neurons form the Ventral Tegmental Area (VTA) in the rewarding system. Dopamine is released in parts of the brain involved in emotion, memory and information processing; these regions are the nucleus accumbens (NAc), amygdala, hippocampus and Pre-Frontal Cortex (PFC). Heightened dopamine levels in the mesolimbic system increase rewarding effects. In other words, nicotine interacts with nAChRs on dopaminergic neurons and directly increases dopamine release (Balfour, 2009; Barrett et al., 2004; Koob & Volkow, 2010). Nicotine modulates dopamine release indirectly by binding to nAChRs on glutamatergic and GABAergic neurons in different brain areas,

such as the amygdala, hippocampus, NAc, PFC, pedunclopontine tegmental nucleus and ventral pallidum of the brain, starting from nicotine initiation to nicotine dependence. Consequently, a set of potential medication strategies for treating nicotine dependence control the interaction of nicotine and nAChR. Another group of treatment options affect receptors that mediate the influence of GABA, glutamate and dopamine after using nicotine.

Nicotine withdrawal decreases dopaminergic neurotransmission. In a study by Hildebrand et al. (1998), dopamine levels in the NAc decreased in nicotine-dependent rats by administering the nAChR antagonist mecamylamine rather than an inert substance. Furthermore, there was a correlation between low dopamine levels in NAc and the somatic and affective symptoms of nicotine withdrawal. Adult rats had a lower dopamine level in the NAc than adolescents (Natividad et al., 2010), probably because mesolimbic dopamine plays a bigger role in adult rats with nicotine withdrawal than adolescents. Current dopamine-based smoking cessation medications can either decrease nicotine withdrawal symptoms, inhibit nicotine's reinforcement effects or both.

Glutamate, the brain's primary excitatory neurotransmitter, plays a key role in nicotine dependence (Liechti & Markou, 2008). Nicotine binds to excitatory $\alpha 7$ -containing nAChRs on presynaptic terminals of glutamatergic neurons in various parts of the brain, including the VTA, amygdala, PFC and NAc, resulting in glutamate increase (Mansvelder & McGehee, 2002).

Acute nicotine use activates excitatory four 2-containing nAChRs on GABAergic neurons in the VTA, which causes an increase in GABA in animals with nicotine dependence. Therefore, in the beginning, the release of nicotine-induced GABA decreases

the rewarding effects of nicotine. However, four 2-containing nAChRs on GABAergic receptors are desensitised by chronic nicotine use (Mansvelder & McGehee, 2002). This desensitisation, which decreases the inhibition of dopaminergic neurons in the VTA and increases dopamine release in the NAc, should theoretically reduce nicotine-induced GABA. This process facilitates the reinforcing effects of nicotine. Compounds that increase GABAergic neurotransmission reduce both the reinforcing effects of nicotine and the reinstatement of nicotine-seeking behaviour (Markou, 2008; Vlachou et al., 2011). Hence, medications or treatments that increase GABA transmission may help individuals with nicotine dependence avoid relapse.

Cannabis

The endocannabinoid system is composed of the endogenous ligands or endocannabinoids, cannabinoid receptors and enzymes involved in the synthesis and degradation of endocannabinoids. There are many cannabinoid receptors such as the G protein-coupled receptor 55 (GPR55) bind cannabinoid ligands (Baker et al., 2006), but the two most well-known are CB1 and CB2 (Matsuda et al., 1990; Munro et al., 1993), which are seven-transmembrane protein-coupled Domain-G receptors (Pertwee et al., 2010).

Several studies have explored the cannabinoids' addictive properties and their effects on the different behavioural responses. These investigations elevated the knowledge about the neurobiology of cannabis addiction and revealed that the CB1 receptors are responsible for cannabinoid-related addicted responses. Moreover, investigating the role of CB2 cannabinoid receptors in CNS neurons revealed new possibilities. These receptors are in areas of the brain

associated with cannabis reward. Despite the low density of CB2 receptors in these regions, some research suggested that central CB2 receptors can control several behavioural responses. Delta9-tetrahydrocannabinol (THC), cannabidiol, cannabinol and delta8-tetrahydrocannabinol are the main active phytocannabinoids (compounds isolated from cannabis) (Pertwee, 2005).

The main psychoactive component of cannabis extracts is THC (Gaoni & Mechoulam, 1964), whereas phytocannabinoid and cannabidiol have no psychoactive effects but do produce anti-inflammatory responses (Iuvone et al., 2009). There are three cannabis plants classifications based on their relative THC and cannabidiol content: (a) drug type plants with a much higher than one THC/cannabidiol ratio; (b) intermediate type plants with a THC/cannabidiol ratio around one; and (c) fibre type plants with a lower than one THC/cannabidiol ratio (Hillig & Mahlberg, 2004).

In Europe, the maximum THC content for farming cannabis fibre type plants is 0.2% to 0.3% of dry matter weight. However, the THC content of the new genetically selected plant variants for recreational use is more than 20% (Pijlman et al., 2005). Additionally, an increase in the strength of the recent cannabis extracts has been found because of enhanced THC content (McLaren et al., 2008; Mehmedic et al., 2010; Pijlman et al., 2005), which may result in subjective effects of this substance. Furthermore, the impacts of cannabis may depend on a variety of factors, such as an individual's previous experience, the route of administration, and the setting in which the substance is consumed. Some of the reported subjective effects of cannabis are relaxation, mild euphoria, perceptual changes such as intensification of daily experiences and time distortion. Furthermore, anxiety and panic

attacks are among the most common opposing subjective experiences reported mostly by naive users (Green et al., 2003).

Individuals use cannabis in different ways. Cannabis can immediately enter the bloodstream through several delivery methods, such as smoking or vaping. The most common route of cannabis self-administration is the inhalation of smoked cannabis, which has immediate effects (Borodovsky et al., 2016; Knapp et al., 2018). However, a vast array of cannabis products can be used via other means (Russell et al., 2018; Spindle et al., 2019a). Another popular method of cannabis use is oral cannabis products (edibles) (Schauer et al., 2016; Steigerwald et al., 2018a), which include cannabis-infused foods and drinks (Russell et al., 2018; Spindle et al., 2019a). Besides food products, there are also oral cannabis oils and tinctures, especially for cannabidiol (CBD)-dominant products (Spindle et al., 2019). Investigations indicated that edible cannabis is perceived as being healthier than smoking cannabis, has a stronger drug effect and can be used more discreetly (Kostadinov & Roche, 2017; Lamy et al., 2016).

Cannabis vaporisers heat dried cannabis or cannabis extracts, which aerosolises cannabinoids for inhalation. Vaporisers operate at temperatures that do not combust cannabis, making them potentially less toxic for cannabis users compared to smoking cannabis (Gieringer et al., 2004; Newmeyer et al., 2017). There are also topical THC or CBD-dominant products, such as creams, gels, patches and lotions, which need a longer time to reach the bloodstream (Russell et al., 2018; Steigerwald, et al., 2018).

Acute cannabis use occurs when a person smokes cannabis in a joint, in a water pipe, with or without tobacco or in other forms in one session (Karila et al., 2014). Its toxicity is

based on the concentration of Delta 9-THC in various cannabis products (marijuana, hashish, skunk, bong and oil) (Crippa et al., 2009). Based on the plasmatic peak of Delta 9-THC, effects may develop for approximately 2 hours after taking cannabis (Costa, 2007), which are associated with euphoria, sedation, continuous laughter or talkativeness, perceptual distortion, social withdrawal and lethargy intensification of ordinary sensory experiences (Johnson, 1990). There are also physical signs such as increased appetite, blood pressure and heart rate, which start a few minutes after taking cannabis and lasts for 3 hours (Hall et al., 1998). However, chronic cannabis use causes cannabis use disorder among those who cannot control their use despite the negative consequences (Copeland & Swift, 2009; Dennis et al., 2002). Cannabis withdrawal syndromes occur in individuals who quit without seeking treatment (Budney & Hughes, 2006).

Apart from the endocannabinoid system, there are several heterologous systems involved in cannabinoid addiction. The framework that mediates the rewarding properties of all typical substances of abuse is called the mesocorticolimbic system (Di Chiara et al., 2004, Fattore et al., 2008). A crucial component of this system is dopaminergic projection, which happens in the VTA, PFC and limbic structures such as the NAc and amygdala. Cannabinoids increase dopamine levels in the VTA (Gessa et al., 1998). Furthermore, THC and the synthetic cannabinoid WIN 55,212-2 (Tanda et al., 1997) increases the extracellular dopamine concentration in the shell (not core) of the NAc, which is connected to the reinforcing properties.

Even though there are plenty of CB1 cannabinoid receptors in the brain's reward circuitry, cannabinoid agonists do not activate DA in the VTA directly due to the lack of CB1 receptors in these cells (Herkenham et al., 1991; Matsuda et al., 1993). However, the

VTA of presynaptic glutamatergic and GABAergic neurons contains CB1 receptors. Thus, excitatory and inhibitory inputs mediated by CB1 receptor activation modulate the dopaminergic neurons of the mesocorticolimbic pathway. Finally, cannabinoids increase the activity of VTA DA neurons as a result of a functional balance between glutamatergic excitatory inputs from the PFC and the effects of GABAergic inhibitory interneurons (Fattore et al., 2008; Maldonado et al., 2006).

Conversely, there is an association between withdrawal from chronic THC administration and decreased dopaminergic transmission in the limbic system like other drug addictions (Diana et al., 1998). Additionally, Tanda et al. (1999) discovered a decline in extracellular DA of the NAc shell after rimonabant precipitated THC withdrawal. Low DA tone during withdrawal is linked to abstinence symptoms, which may reduce motivation for non-drug stimuli while increasing sensitivity to abused drugs (Koob & Volkow, 2010). Nevertheless, the somatic expression of cannabinoid withdrawal does not contain the dopaminergic system activation because treatment with DA antagonists could not decrease THC physical dependence (Sañudo-Peña et al., 1999). THC stimulates the firing of mesolimbic dopamine neurons in animals, raising striatal dopamine level (Lupica et al., 2005). In humans, acute THC induces striatal dopamine release (Bossong et al., 2009; Bossong et al., 2015; Voruganti et al., 2001).

During the chronic phase of dependence, most drugs of abuse can reduce by dopamine release, resulting in a poor outcome. Giessen et al. (2017) examined striatal and extrastriatal dopamine release in participants with severe cannabis dependence but did not have any comorbid conditions, such as nicotine use. Their results indicated that excessive cannabis use with no comorbidities is connected to striatal dopamine release problems. These deficits

predict subclinical psychopathology by extending to other extrastriatal sections. In other words, severe cannabis dependence without comorbidities could cause amphetamine-induced dopamine release in the associative striatum (AST), the sensorimotor striatum (SMST) and globus pallidus. Heavy cannabis use can cause psychopathology because of decreased dopamine release in the AST (Giessen et al., 2017). Another study by Urban et al. (2012) indicated that there is no association between mild to moderate cannabis use disorder and striatal DA alterations, unlike other drug addictions. However, longer and earlier use of cannabis is associated with lower DA release in the striatum. These studies reveal a more dangerous effect of using cannabis during adolescence.

Chronic deficits of serotonin could be found in individuals with ADHD. Neuro-anatomical studies show that behavioural domains of hyperactivity and impulsivity in people with ADHD are regulated by serotonin in the orbitofrontal-striatal circuitry. They also indicated that selective serotonin reuptake inhibitors or non-stimulant drugs that affect serotonin are clinically effective in individuals with ADHD (Banerjee & Nandagopal, 2015).

Many studies revealed the functional interaction between cannabinoids and GABAergic/glutamatergic neurotransmission in the brain's reward circuit. López-Moreno et al. (2008) reported that both endogenous and exogenous cannabinoids could decrease GABAA receptor-mediated inhibitory neurotransmission. They can also reduce N-Methyl-D-aspartic acid (NMDA) and α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor-mediated excitatory effects in the limbic system.

Investigators used drug discrimination methods in rats to study the role of cholinergic mechanisms in behaviours correlated with THC's addictive nature. The results showed that

the effects of low doses of THC are potentiated by both muscarinic and nicotinic receptor activation. Solinas et al.'s (2007a, 2007b) study found that after blocking anandamide degradation with the FAAH inhibitor URB597, nicotine could produce THC-like discriminative effects dependent on CB1 receptors. Furthermore, neither scopolamine (muscarinic antagonist) nor mecamylamine (nicotinic antagonist) changed THC's discriminative effects.

Acetylcholine is a neurotransmitter involved in motor control, attention, memory and learning. Acetylcholine dysfunction can be seen in different brain disorders, such as Alzheimer's, addiction, schizophrenia and ADHD. Choline Acetyltransferase (ChAT), a presynaptic choline transporter, is the main and rate-limiting determinant of acetylcholine production in and outside of the brain. It is also upregulated in tasks that need sustained attention (English et al., 2009).

Exploring the neurobiological underpinnings of alcohol, cannabis and nicotine could better understand the association between ADHD and substance misuse in adults. Starting university can be challenging for young adults; based on the reports from NHS Digital and previous studies, university students are at a higher risk of misusing substances and developing SUD (Mohammadpoorasl et al., 2014; NHS Digital, 2019; Sommet et al., 2012; Suerken et al., 2014). The research in this thesis focused primarily on alcohol, cannabis and nicotine misuse among Goldsmiths, University of London students, which highlights the importance of investigating substance misuse in this group of individuals.

1.2.2. Substance misuse among university students

University students are more independent from their parents' supervision than before entering university. It is also a time of transition, often risky living conditions, pressure from family and educational problems (Sommet et al., 2012). Therefore, many students start using more psychoactive substances such as nicotine, alcohol and other illegal drugs (Locke et al., 2014). Many studies show that university students around the world are at a higher risk of developing SUD (Bajwa et al., 2013; Drosican, 2009; Gupta et al., 2013; Maher, 2008; Maier et al., 2013; Mohammadpoorasl et al., 2014; Sommet et al., 2012; Suerken et al., 2014). However, certain factors such as religion, living with parents and away from campus and having better health awareness play a protective role for students to develop fewer or no drug use problems (Gomes et al., 2013; Mohammadpoorasl et al., 2014; Suerken et al., 2014).

The research found that university students are at a higher risk of hazardous alcohol use (Davoren et al., 2014) and illegal drug misuse (Trevor et al., 2014) compared to other people their age in the UK. Moreover, 49% of university students have used illegal drugs at least once in their lifetime, and 21% have done so in the last month (Johnston et al., 2012). Cannabis was the most commonly consumed drug, with 47% of students having used it over their lifetime and 19% over the last month. Other common illegal drugs used by students over the last 30 days were amphetamines (4.5%), opioids (2.1%), cocaine (1.2%) and hallucinogens (1.2%). According to the Future Survey (2011), university students are more likely to use inhalants, hallucinogens, amphetamines and steroids in the last month compared to their non-university peers (Johnston et al., 2012). In addition, the Core Alcohol and Drug Survey (2011) revealed that university students use cannabis, cocaine and hallucinogens more frequently than those not in university.

Based on previous investigations, university students with ADHD have a higher risk of alcohol misuse than non-university adults. Two studies by McCabe et al. (2006, 2016) found that 90% of university students consume two to three alcoholic drinks a few times a week. Other studies and literature reviews that investigated substance misuse in university students with ADHD in UK universities showed that those with ADHD use higher amounts of substances compared to students without ADHD (Rooney et al., 2012; Sedgwick, 2018). Another study by Upadhyaya et al. (2005) tested 334 participants using the Core Alcohol and Drug Survey, which is a self-report instrument that measures substance misuse attitudes, patterns and demographic information. The Current Symptom Scale (CSS), a self-report adult ADHD scale, was also employed. The results demonstrated that compared to their peers without ADHD symptoms, university students with more symptoms of ADHD used higher amounts of nicotine and cannabis in a year, but not alcohol or other drugs. However, some (e.g. Rowland et al., 2015) proposed that the lack of ADHD diagnosis in individuals could be the reason for these different results.

In the UK, the number of studies on the use of various legal and illegal substances among university students is limited; most of the research centres on substance misuse by students from a single university or faculty. Webb et al.'s (1996) research examined second-year students at 10 UK universities and disclosed that cannabis was the most widely used drug among university students (57%), followed by amphetamines (19%), LSD (18%), magic mushrooms (16%) and amyl nitrite (15%). Another study by Newbury-Birch et al. (2001) indicated that in a sample of first-year medical students, the most commonly used illegal drugs were cannabis (51%), amyl nitrite (9%) and magic mushrooms (6%). The students at UK universities also consumed higher amounts of alcohol than their non-university

counterparts. A review of investigations measuring undergraduate drinking showed that 52% of men and 43% of women drank above the recommended weekly limits of 21 units for men and 14 units for women (Gill, 2002). However, the figures for the general population are 37% for 16-year-olds and 33% for 24-year-olds (Rickards et al., 2004). A more recent study reported that risky alcohol use is widespread among UK students, with 70% to 85% of them informing weekly binge drinking. In the UK, 63% to 84% of university students disclosed hazardous drinking, and women are catching up to men in risky alcohol use (Davoren et al., 2016).

A few UK studies focused on substance misuse among university students, some of which are outdated (Barber & Fairclough, 2006; Nixon et al., 2002; Pickard et al., 2000; Underwood et al., 2010). The UK investigation shows that many university students take drugs and poly-drugs (Bennett & Holloway, 2014). Previous research of university students in the UK has had several limitations, such as small sample sizes, limiting the generalisability of their results, or examining students from a single university or department.

Apart from these shortcomings, Ansari et al. (2015) attempted to bridge these gaps by studying the association between several variables and illegal drug use in a sample of students from seven universities in England, Wales and Northern Ireland to provide a wider picture of illegal drug use among university students around the UK. Their results indicated that more than 30% of the students reported using illegal drugs. Even though they did not inquire into the form of the illegal drug, investigations demonstrate that cannabis is the most common illegal drug in western societies (Vivancos et al., 2008; Newbury-Birch et al., 2001; Underwood et al., 2009). In a systematic review by Davoren et al. (2016), almost two-thirds

of university students in the UK and Ireland gained a score of above 8, indicating hazardous alcohol consumption.

According to the NHS Digital reports (2018), the age groups with the highest use of nicotine are 25 to 34 and then 18 to 24, which reflects the higher prevalence of using nicotine among university students. There is some data on the prevalence of nicotine use in some UK universities, but it is extremely limited. For instance, in a study conducted at the University of Birmingham, UK, the nicotine usage of 934 university participants was measured, and their results revealed that the current smoking prevalence was 14% (Bartington et al., 2020). Further investigation is required to determine the prevalence of nicotine use in different forms among university students in the UK. Thus, focusing and examining university students as a high-risk group for developing alcohol, cannabis and nicotine use may help researchers better understand this group and select the best way to assist them.

Studies on substance misuse and ADHD symptoms in university students are minimal. Many previous studies of university students with ADHD used substance misuse as an exclusion criterion. Moreover, most of the research on drug misuse in university students investigated those who had been diagnosed with ADHD, restricting the generalisability of the results (Baker et al., 2012; Rooney et al., 2012). The studies in this thesis explored the relationship between alcohol, cannabis and nicotine use and ADHD symptoms in typically developing individuals dimensionally. The next section delves into the association between ADHD and substance misuse.

1.3. Developing substance misuse and ADHD

The DSM criteria published the threshold for different disorders and problems such as ADHD, substance use, cognitive impairments or emotional dysregulation. Meeting these conditions can place a person in the category of people with problematic behaviour, resulting in impairments in their daily lives. Thus, individuals are on an impulsivity and EF performance spectrum, and all use legal or illegal substances. Different questionnaires that measure substance misuse, impulsivity, EF performance or ADHD have cutoff scores that indicate risky behaviours, which may result in poor physical/mental health outcomes. The behaviour threshold becomes problematic when it interferes with normal life or physical/mental health. However, the exact cause of the increased use of substances is unknown. Genes, peer pressure, depression, emotional distress, environmental risk factors and the drug's action may all be contributing factors (Banducci et al., 2016; Bilinski et al., 2012; Crockett et al., 2006; Kendler et al., 2003; Keyzers et al., 2020; Merikangas et al., 2000; Olsen, 2011; Overholser et al., 1997; Ruffle, 2014; Volkow, 2010).

Misusing substances, which might progress to SUD, is one of the main comorbid conditions and problems in people with ADHD. Previous literature has explored the relationship between ADHD and different substances, such as alcohol, nicotine, cannabis, cocaine, amphetamines and other illegal drugs (Blevins et al., 2020; Bron et al., 2013; Levin et al., 2018; Luderer et al., 2019; Tamm et al., 2013). Over the past decade, many studies have demonstrated that the co-occurrence of ADHD and SUD is a prevalent phenomenon in adults and adolescents (Bernardi et al., 2011; Glind et al., 2014; Gonzalez et al., 2017; Kooij et al., 2010; Lee et al., 2011; Nogueira et al., 2014). Past studies have indicated that both disorders start at an earlier age and a more severe course; the treatment outcomes are also

poorer in those with ADHD and comorbid substance misuse (Arias et al., 2008; Kollins, 2008). The prevalence of ADHD symptoms in patients with SUD is high. In a review, this prevalence ranges from 23.3% to 31% (Glind et al., 2014; van Emmerik-van Oortmerssen et al., 2012). Furthermore, 40% to 50% of individuals with ADHD symptoms have SUD (Fayyad et al., 2007). Studies show that people with ADHD symptoms and comorbid SUD become drug addicted at a younger age, use higher amounts of drugs and are hospitalised more often than those without ADHD symptoms (Arias et al., 2008). After successful SUD treatment, relapse is more common among people with ADHD symptoms (Carroll & Rounsaville, 1992). In addition, pharmacological studies indicate that the two main ADHD treatment medications, methylphenidate and atomoxetine, are less effective in individuals with ADHD and SUD compared to those who only have ADHD (Carpentier et al., 2005; Castells et al., 2011; Konstenius et al., 2010; Levin et al., 2007; McCabe et al., 2016; Thurstone et al., 2010; Wilens et al., 2011).

Brook et al. (2010) discovered that ADHD diagnosis is associated with developing higher rates of substance misuse in adulthood. Being diagnosed with ADHD during adolescence is also linked to misusing substances and then developing SUD when individuals are in their 20s and 30s. In those with ADHD, there is a positive correlation between inattention and hyperactivity/impulsivity symptoms and the risk of developing SUD later in life (Gudjonsson et al., 2012). However, discussion exists about the role of inattention symptoms versus hyperactivity/impulsivity symptoms of this condition in increasing the risk of SUD (Ernst et al., 2006; Molina & Pelham, 2003). Few investigations focused on substance misuse in individuals with ADHD symptoms rather than those with diagnosed ADHD. For instance, Daurio et al. (2017) measured the ADHD symptoms in 749 participants

using The Conners' Adult ADHD Rating Scales – Self-Report Scale: Short Version (CAARS-S:S; Conners et al., 1998) and the Alcohol Dependence Scale (ADS; Skinner & Allen, 1982) to assess alcohol dependence severity and UPPS-P (urgency [negative], perseverance, premeditation, sensation seeking and positive urgency; Lynam et al., 2006) to assess impulsivity. Their results suggested that developing AUD is more common among those with higher ADHD symptoms without a clinical ADHD diagnosis and that impulsivity mediates the relationship between ADHD and AUD.

In 2016–2017, the majority (60%) of non-opiate only individuals seeking treatment in the UK had cannabis use disorder (National Drug Treatment Monitoring System [NDTMS], 2017). Cannabis's nature and strength have evolved (ElSohly et al., 2000; Licata et al., 2005). Studies also show that it can affect mental health and cause other problems (Fergusson et al., 2002, 2006a, 2006b; Hayatbakhsh et al., 2007; Looby & Earleywine, 2007; Rey et al., 2002). Heavy cannabis use may be linked to attention problems and impaired cognitive processes in users, which is a concern that deserved further attention. Accordingly, many studies indicate that cannabis use is associated with attention impairments even in people with no intoxication (Kalant, 2004; Lundqvist, 2005; Pope et al., 2001).

Studies found that ADHD and substance misuse share common aetiological influences (Young et al., 2015). The main symptoms of ADHD usually co-occur with cannabis use disorders (Lee et al., 2011). To help future genetic studies, investigators explain the cannabis use disorder and ADHD phenomenology. There have been great concerns about young people's health, which has been affected by the growing cannabis use among them (Rey et al., 2002; von Sydow et al., 2001).

Furthermore, identifying the aspects of ADHD that may increase the risk of cannabis use problems may help in finding more treatment options for those with ADHD symptoms and cannabis misuse (Bidwella et al., 2014). Many studies have focused on the impact of cannabis use on attention, but only a few investigations have centred on how cannabis use affects ADHD symptoms. Based on Barkley and Murphy's (1998) description, adult ADHD symptoms that cause functional impairments, include being easily distracted, impaired attentional control and lack of sustained attention over long periods. Hence, it seems that cannabis users' attention problems may mirror ADHD symptoms. The relationship between early cannabis use and ADHD symptoms may thus be plausible (Fergusson et al., 2008).

Investigations found that ADHD is also a risk factor for smoking and tobacco dependence (Glass & Flory, 2010; Matthies et al., 2013). Adolescents with ADHD use an increasing amount of tobacco at a younger age and continue to do so into adulthood compared to their peers without ADHD (Breyer et al., 2014; Lambert & Hartsough, 1998). Smoking is two times more common in adults with ADHD than in the general population (Breyer et al., 2014; Lambert & Hartsough, 1998). Furthermore, those with ADHD smoke more cigarettes daily and are more reliant on tobacco than those without ADHD (Ohlmeier et al., 2007; Wilens et al., 2007). Smokers with ADHD also quit smoking more frequently but with less success and show more withdrawal symptoms than smokers without ADHD (Humfleet et al., 2005; Kollins et al., 2005; Pomerleau et al., 1995).

The main psychoactive component of tobacco is nicotine (Wonnacott et al., 2005), which mediates the relationship between smoking and cognition and affect (Darredeau et al., 2013; Harrell & Juliano, 2012; Perkins et al., 2006). Previous investigations have disclosed that nicotine strongly mediates the impact of cigarette smoking on cognitive performance and

behaviour in smokers with ADHD (Watterson et al., 2016). Even though more clarification is needed, the basic aetiology of the ADHD-nicotine association may be explained by different hypothesised mechanisms (see Section 1.3). To be more precise, shared genetic and environmental vulnerability and clinical and neurocognitive characteristics have been proposed as explanations for why people with ADHD smoke more tobacco (McClernon & Kollins, 2008). Individuals with ADHD who smoke nicotine claim that it is reinforcing and increases wakefulness, improves cognition, reduces irritability and enhances mood (Mitchell et al., 2014; Van Voorhees et al., 2012). Although smokers without ADHD describe the same effects as those with ADHD, smokers with ADHD experience stronger effects (Harrell & Juliano, 2012). They even report more satisfaction with smoking cigarettes (Van Voorhees et al., 2012).

Different approaches have been taken to explain the reason for developing substance misuse. The first focus is on the neurobiological effects of substances to explain addiction in biological terms. The second approach, which is psychological in nature, concentrates on behavioural models and individual differences. The last is the sociocultural approach, which concentrates on the cultural and environmental factors that raise the likelihood of taking substances in increasing quantities (Koob & LeMoal, 1997; Nutt, 1997).

1.3.1. Neurobiological approach: Genetics and neuroadaptation theories

Many studies on substance misuse have attempted to explore the role of genetic vulnerabilities. However, no single candidate gene directly related to substance misuse have been found (Altman et al., 1996; Ducci & Goldma, 2012). It is hypothesised that multiple

genes or incomplete manifestation of several major genes can affect substance misuse and SUD (Ducci & Goldma, 2012; Kendler, 1999; Schuckit, 1999). Several twin studies investigated the role of genetics and environment in misusing substances and developing SUD. The researchers suggested three types of influences in the trait: (a) heritability, which covers genetic influences; (b) shared environmental influences, which family members experience and make them similar to one another; and (c) none-shared environmental influences, which are unique environmental influences in a family that distinguishes the members from one another. In addition, adoption studies indicate that the correlation of a trait in adopted children pairs can be due to shared environmental influences. Comparing the results of adoption and biological siblings also shows the impact of all three influences on the trait. For instance, an association between nicotine smoking and genes that regulate dopamine has been suggested (Maes et al., 2017; Sabol et al., 1999; Sanchez-Roige et al., 2016, Sartor et al., 2015). More recently, a critical component and common factor in developing various kinds of behavioural and substance addictions is a gene called DeltaFosB (Δ FosB) (Bilinski et al., 2012; Olsen, 2011; Ruffle, 2014). The causal relationship between this gene and addiction is used as a preclinical addiction biomarker (Bilinski et al., 2012; Nestler, 2013; Ruffle, 2014).

According to studies, individuals with psychiatric conditions use alcohol and other substances at an earlier age (Arias et al., 2008; Kollins, 2008). The reason for substance use initiation in individuals with ADHD could be due to genetic, biological, mental, environmental and peer group factors. Based on previous research, using different substances at a younger age can lead to SUD in adulthood, which can be seen in individuals with ADHD (Arias et al., 2008; Kollins, 2008). It may also be attributed to other co-morbid disorders,

such as conduct disorder, which can increase the risk of SUD in individuals with ADHD later in life (Flory & Lynam, 2003; Wimberley et al., 2020). While research has shown a connection between risk factors such as comorbid conduct disorder, parental factors including socioeconomic status, SUD and mental disorders and increased risk of SUD, a higher genetic vulnerability in people with ADHD symptoms is linked to a higher risk of substance misuse (Wimberley et al., 2020).

Adoption and twin investigations have revealed that ADHD is 75% to 91% heritable (Sharp et al., 2009). Moreover, twin, adoption and family studies have demonstrated that the heritability of the interaction between genetic and environmental factors contribute to the development of alcohol dependence in more than half of the cases (Goldman et al., 2005). In addition, studies have found an association between the dopamine D2 receptor gene and substance dependence and ADHD (Wang, 2013).

Another theory of substance addiction focuses on reinforcement. Based on the view of Edwards and Koob (2013), there are two different sources of reinforcement: negative and positive reinforcement, which could produce drug-seeking motivation that leads to addiction. Positive reinforcement occurs when a stimulus heightens the probability of the following response and is connected to a state of reward. One main theoretical framework for addiction is that substances take over the brain's motivational system (Hyman et al., 2006; Robinson & Berridge, 1993), which may lead to behaviours aimed at finding and using drugs rather than focusing and gaining more adaptive goals, such as career and family (Volkow et al., 2011). Negative reinforcement happens when individuals take substances to self-medicate an existing aversive state or decrease negative emotional symptoms often associated with withdrawal, such as sleep problems, anxiety and dysphoria.

Studies have reported that people with ADHD use alcohol, cannabis and nicotine to self-medicate; substances such as nicotine may ameliorate ADHD symptoms (Levin et al., 1996; Wilens et al., 2006). The results of previous research also revealed that attentional performance in people with ADHD could be significantly improved by nicotine skin patches (Levin & Resvani, 2000). A longitudinal study by Wilens et al. (2010) found that 36% of participants with ADHD symptoms used substances to self-medicate, 25% used them to get high and 39% had unknown motivation. Thus, more than a third of their participants used cigarettes and other substances to self-medicate. Moreover, a clinical neurobiological investigation by Silva et al. (2014) indicated that individuals with ADHD symptoms and SUD had lower striatal dopamine transporter density than those with ADHD only. This difference may be due to cannabis' effect on low levels of striatal dopamine transporter density, denoting a neurobiological foundation for the self-medication theory in those with ADHD symptoms.

The most significant link between dopamine and ADHD is the use of treatment drugs for ADHD (Berridge & Devilbiss, 2011). ADHD stimulant medications manage the condition by increasing the synaptic dopamine concentrations in the striatum, which includes the reward centre, via presynaptic transporters (Greenhill et al., 1999). Drugs such as alcohol, nicotine and cannabis affect the concentration of synaptic dopamine, especially in the nucleus accumbens, the brain's reward centre (Cavacuiti, 2011). To illustrate, in a study by Brody et al. (2004), dopamine release in the ventral striatum of the nicotine addicts who smoked a cigarette during a PET scan was found to be lower than that of nicotine-dependent participants who did not smoke during the scan. Their results also revealed that there is an association between the reduced binding potential in the nucleus accumbens and putamen

and lower self-report craving. This outcome depicts the connection between craving reduction and dopamine release (see Section 1.3.1. for a review on the relationship between dopamine and substance misuse). Additionally, studies show that people with ADHD have GABAergic deficits with decreased GABA concentration. Therefore, drinking alcohol may help people with ADHD by raising GABA concentration (Edden et al., 2012). It can then be concluded that the function of these neurotransmitters in the brains of individuals with ADHD symptoms can increase their risk of AUD. Additionally, this desensitisation lowers nicotine-induced GABA release, which reduces dopaminergic neurons inhibition in the VTA and raises dopamine release in the NAc, facilitating the reinforcing effects of nicotine in individuals with ADHD symptoms. Furthermore, cannabinoids increase the activity of VTA DA neurons as a result of a functional balance between glutamatergic excitatory inputs from the PFC and the effects of GABAergic inhibitory interneurons (Fattore et al., 2008; Maldonado et al., 2006), which may be another reason why people with ADHD use an increasing amount of cannabis.

However, several investigations did not support the self-medication theory of people with ADHD symptoms. For instance, Sousa et al. (2011) indicated that the behavioural disinhibition profile of those with ADHD is consistent with smoking initiation beyond the role of self-medication. The results of another study by Dinn et al. (2004) on cigarette smoking in a sample of students aged 17 to 25 also did not concur with the self-medication model of people with ADHD symptoms. However, their findings were in line with the orbitofrontal/disinhibition model. In comparison to non-smokers, smokers performed poorly on neurocognitive tasks sensitive to orbitofrontal dysfunction; they gained significantly higher scores on behavioural disinhibition, impulsivity, and antisocial personality.

Neuroadaptation (Koob & Volkow, 2010) is another theory of substance addiction, which describes how the brain adapts to the acute effects of a substance after repeated administration. Prolonged substance administration induces changes in brain chemistry to counteract the substance's effects and can be divided into two kinds: (a) within system adaptation, which includes changes at the site of the substance's action; and (b) between system adaptation, which involves changes in mechanisms influenced by the substance's action. When substance use is stopped, there are no longer any adaptations that oppose it, and the brain's homeostasis is disrupted (Koob & Volkow, 2010). Therefore, neuroadaptation is hypothesised to cause both tolerances to the substance's effects and withdrawal after stopping use (Koob et al., 1997).

1.3.2. Psychological approaches: behavioural and individual differences theories

Psychological approaches often centre on behavioural syndromes such as compulsivity and impulsivity, which may increase the likelihood of SUD (Miller, 1980). Particularly, the focus is on impaired control over substance continuation and use despite problems. Several psychological approaches explain substance misuse and later SUD, such as learning and conditioning, cognitive and personality theories, and rational choice models. Furthermore, exploring the role of different shared parts of the brain that control cognitive performance may aid in a better understanding of the development of substance misuse in individuals with ADHD symptoms. For instance, functional MRI studies show that the PFC is a specific area of the brain that is more engaged in both ADHD and substance misuse (Arnsten, 2010; Bush, 2010; Goldstein & Volkow 2011; Korponay et al., 2017; Qin et al., 2016). Even though the

exact basis of ADHD is unknown, it is believed to be a neuro-behavioural disorder connected to structural and chemical changes in the PFC. This part of the brain is responsible for higher-order mental functions such as attention, learning and EF. Investigations suggest that the poor function of the three sub-regions of the PFC can be the cause of ADHD symptoms (Arnsten, 2010; Prince, 2008; Vance et al., 2007). The symptoms of inattention and distraction are thought to be related to the impairment of the Dorsolateral Prefrontal Cortex (DLPFC), which regulates attention (Arnsten, 2010; Chao & Knight, 1995; Woods & Knight, 1986). Furthermore, impulsivity and hyperactivity symptoms are linked to the dysfunction of the right inferior PFC (Arnsten, 2010; Aron et al., 2004). In addition, emotional responses are regulated by the ventromedial PFC (Arnsten, 2010).

Based on the Surgeon General's Report on Alcohol, Drugs and Health (2016), the basal ganglia, extended amygdala and PFC are the three main regions of the brain involved in the development and persistence of SUD. The basal ganglia are responsible for the habitual substance intake and regulate the rewarding and pleasurable effects of substance use; previous investigations have observed that individuals with ADHD symptoms reductions have lower basal ganglia volume (Emond et al., 2009). The extended amygdala, a processing centre for emotions, is involved in the anxiety, irritability and stress that accompany substance withdrawal. Research found that people with ADHD have a dysfunctional amygdala, resulting in weak impulsivity control (Tajima et al., 2016). The PFC is involved in EF, which include substance intake. control. An emerging PFC dysfunction in individuals who misuse substances (stimulants and non-stimulants) is also revealed in neuroimaging studies of cigarette smokers (Alachkov et al., 2009), those with AUD (Grüsser et al., 2004; Heinz et al., 2007), young drinkers with alcohol use and cravings (Filbey et al., 2008) and

individuals with cocaine addiction (Garavan et al., 2000). PFC is mainly involved in the ‘craving’ stage of substance misuse, which is marked by impaired EF due to disrupted PFC function. An increase occurs in the activity of the neurotransmitter glutamate, which drives substance use habits connected to cravings, disrupting dopamine influence on the frontal cortex (Kalivas, 2005, 2009). On the one hand, the Go system in the PFC is over-activated, which stimulates habit-like substance seeking. On the other hand, the under-activation of the PFC’s Stop system promotes impulsive and compulsive substance seeking (Surgeon General’s Report on Alcohol, Drugs and Health, 2016). Studies suggest that PFC dysfunction in individuals with ADHD symptoms and those with drug addiction leads to EF impairments in different tasks (Hinshaw et al., 2007; Jansari et al., 2012; Montgomery et al., 2010, 2012; Pennington & Ozonoff, 1996; Thorell, 2007). Accordingly, focusing on the link between structural differences in the PFC and the key symptoms of ADHD and substance misuse helps in understanding the neurobiology and connection of these disorders.

Behavioural theories

Behavioural theories concentrate on observable behaviours and the fact that reinforces maintain the behaviour (West, 1989). Self-administration of a substance is an example of instrumental behaviour since an individual’s activities are instrumental in gaining the substance’s effects as a consequence. There are two possible explanations for the substance’s reinforcing effects: (a) the substances’ direct impact on the brain’s reinforcement system; and (b) the substance’s effect on other reinforces such as social and sexual reinforces and behavioural outcomes such as increased attention (Altman et al., 1996).

Investigations have demonstrated the possibility of controlling the history (learning) and environmental conditions (cues) of use. Thus, these two factors are crucial in the development of substance use or misuse (Barrett & Witkin, 1986). Behaviourist theories that focus on classical conditioning also play an important role in developing and maintaining addictive behaviours. Cue exposure theory, a part of the classical conditioning theory, emphasises the importance of cues in addictive behaviours (Drummond et al., 1995; Heather & Greeley, 1990). During the administration of a substance, the presence of a cue may be more likely to produce a response, which is hypothesised to underlie craving. It can explain the reason why an individual with a history of addiction who has been abstinent for a while is experiencing strong cravings (Heather & Greeley, 1990).

Cognitive theories

Other theories may explain substance misuse and SUD, such as cognitive constructs. Based on this theory, self-regulation is a crucial factor in developing SUD. Newman and Newman (2020) defined *self-regulation* as the individual's ability to direct the course of their growth by selecting and pursuing goals and modifying goal pursuit based on environmental and personal opportunities and situations. Mimiaga et al. (2009) defined it as having an active role in engaging in a dynamic process of evaluating health threats and then addressing them using problem-solving strategies. This demands a complex interaction between personal and socio-cultural factors that affect health (Mimiaga et al., 2009). Self-regulation entails paying attention to social and physical factors, planning and considering one's goals and acting appropriately. It has been shown that excessive reliance on external structures

(such as substances) can result in addiction to gain a psychological and physical equilibrium (Mimiaga et al., 2009).

Personality theories

Researchers such as Eysenck (1956) have argued that some people have an ‘addictive personality’, which makes them more prone to addiction. According to Eysenck’s ‘psychological resource model’ (1997), the development of SUD occurred because a substance accomplishes a specific goal related to the individual’s personality profile. For those individuals, although drug use has negative consequences, it also holds benefits. Staiger et al. (2009) reported that personality influences treatment outcomes in those who misuse substances; choosing interventions that are matched to relevant personality traits might improve treatment outcomes in individuals who misuse substances. They also proposed that impulsivity-related personality traits such as behavioural disinhibition, reward sensitivity and sensation seeking are strongly associated with substance misuse. Studies have revealed that individuals with ADHD show higher impulsivity, poor judgment and more impaired EF (Wilens & Biederman, 2006; Wilens et al., 2011), leading to increased substance misuse.

1.3.3. Environmental approach

Twin studies revealed that besides genetic vulnerability to develop substance misuse, there is also a considerable environmental component (Kendler & Gardner, 1998; Kendler et

al., 1999b; Kendler et al., 2000). Investigations show that substance misuse is more common among adolescents with conduct disorder and ADHD (Arias et al., 2008; Riegler et al., 2017). Evidence also indicates that antisocial behaviours in individuals with ADHD increase substance misuse likelihood (Retz et al., 2020). Those with anxiety and depression are also more disposed to begin substance use and develop SUD at an earlier age (Cicchetti & Rogosch, 1999; Costello et al., 1999; Loeber et al., 1999).

Research found that peer attitudes towards substances are strong predictors of drug misuse among adolescents (Fergusson et al., 2002). Thus, the peer environment has a critical influence on people misusing substances and developing SUD later in life (Allen et al., 2003). Those who use substances are more likely to spend more time with peers who are drug users. In addition, investigations demonstrate that family plays a principal role in developing SUD in several ways (Bosk et al., 2021). Adolescents may be affected by their family members' substance misuse behaviours. For instance, Bosk et al. (2021) found an association between parents' substance use and their children's initiation and frequency of alcohol and cannabis use. Furthermore, older brothers' attitudes towards substance misuse behaviours are linked to their younger brothers' drug misuse behaviours (Brook et al., 1990). Hawkins et al. (1992) also reported that parents with a liberal outlook towards drug intake raise the possibility of drug use in their children. The nature of family relationships may play a role in the development of SUD. To illustrate, low levels of family bonding, poor behavioural management techniques and family conflicts can increase the risk of SUD (Hawkins et al., 1992; Lac & Crano, 2009).

An individual's sociocultural background may also affect one's substance misuse and later development of SUD. For instance, in a study of socioeconomic status and substance

misuse among Swiss young men, higher socioeconomic status was linked to alcohol and illegal substance misuse. In comparison, lower socioeconomic status was associated with more nicotine use (Charitonidi et al., 2016). Thus, people misuse substances for varying reasons, as discussed in this section. Although there is vast evidence on risk factors, no single variable has yet been identified as the central risk factor for developing substance misuse (Tarter, 2002). Single-factor causal explanations are highly unlikely because of the complex and heterogeneous nature of substance misuse.

To comprehend drug misuse and its consequences, one must acknowledge that multiple non-specific causes may lead to the same outcome. To overcome this problem, investigators are beginning to focus on multi-component models that emphasise the interaction between several factors such as biological, psychological, chronic and acute stressors (Windle, 2010). For instance, in the context of an individual's developmental stages, Zucker (2006) argues that a combination of genetic factors, socio-environmental factors and intermediate traits must be considered. Due to their complexity, analysing these theoretical models comprehensively during a PhD research programme is exceedingly difficult. However, focusing on the interactive and additive effects of individual risk factors is achievable, which will help in the refinement of complex theoretical models.

In this thesis, a psychological approach was chosen to measure individual differences and cognitive performance in participants to explore the association between each risk factor and ADHD symptoms separately and alcohol, nicotine and cannabis use. Impaired EF, impulsivity, emotional dysregulation, bipolar disorder and poor sleep quality have been selected as risk factors on the account of their consistent links to ADHD symptoms and substance misuse, which could be a manifestation of wider risks. The relationship between

these factors, ADHD symptoms and alcohol, cannabis and nicotine use, seems therefore necessary. Substance misuse has been an exclusion criterion in most past research of adults with ADHD symptoms. A few studies that explored the association between ADHD and substance misuse investigated ADHD as a single neurodevelopmental condition. Thus, there is extremely limited data on the connection between each ADHD symptom and alcohol, cannabis and nicotine use. As a result, the research for this thesis focused on the role of certain main risk factors in the relationship between the two key symptoms of ADHD (inattention and hyperactivity/impulsivity) and alcohol, cannabis and nicotine use. Furthermore, due to the dimensional and non-clinical nature of the studies in this thesis, alcohol, cannabis and nicotine use were measured dimensionally to have a full range rather than SUD.

1.4. Executive functions, impulsivity, emotional regulation, mood disorder and sleep quality in individuals with ADHD and substance misuse

1.4.1. Executive Functions (EF)

Executive functions (EF) refer to a set of cognitive processes that are essential for cognitive behavioural control. These processes help us in selecting and monitor the behaviours that enable us to achieve our objectives. Basic EF include cognitive flexibility, inhibition, attentional and inhibitory control and working memory. Planning, reasoning, problem-solving and fluid intelligence are higher-order EF that need immediate use of several basic EF (Chan et al., 2008; Diamond, 2013; Malenka et al., 2009; Meiyake &

Friedman, 2012). EF gradually develop and change and can be improved over time across an individual's lifespan. These cognitive abilities can also be adversely affected by various events in an individual's life (Diamond, 2013).

Executive functioning skills are at their peak at 20 to 29 years of age, allowing an individual to perform the most challenging mental tasks. These mental abilities begin to decline in late adulthood (De Luca et al., 2008). One of the major changes in an adult's brain is the constant myelination of neurons in the PFC, which began in adolescence (De Luca et al., 2008; Geier & Luna, 2009). The myelination process causes functional shifts in the PFC and subcortical regions such as the amygdala and nucleus accumbens and changes in dopaminergic processes such as a subcortical increase in available dopamine (Wahlstrom et al., 2010). Previous research has indicated that dopamine systems are involved in impulsive actions and substance misuse (Yates et al., 2017). Past studies and NHS Digital (2019) found that the highest rates of substance misuse (cannabis and nicotine use) are among individuals aged 16 to 34, which is also a golden age for EF skills. Therefore, it is necessary to investigate the association between EF performance and substance misuse among people in this age range, which is the focus of Study 1 in this thesis.

The connection between cognitive control (internal) and operant and classical conditioning (external) represents opposing mechanisms that complete an individual's control of stimulated behaviours (Washburn, 2016). For instance, inhibitory control is crucial for overriding stimulus-driven behavioural responses (Diamond, 2013). In executive functioning, the PFC is essential, but it is not the only part of the brain engaged in cognitive control (Alvarez et al., 2006; Diamond, 2013; Malenka et al., 2009). Previous investigations have identified the functions that are the most often involved in specific areas of the PFC:

DLPFC, which involves ‘on-line’ information processing, such as combining various dimensions of cognition and behaviour (Lezak et al., 2004). This area has been linked to verbal and design fluency, ability to continue and shift set, response inhibition, working memory, planning, reasoning, problem-solving, organisational skills and abstract thinking (Alvarez et al., 2006; Clark et al., 2008). The anterior cingulate cortex (ACC) influences emotional drives, integration and experience. Its related cognitive functions include inhibition of inappropriate responses, motivated behaviours and decision-making (Lezak et al., 2004). The orbitofrontal cortex (OFC) is responsible for monitoring ongoing behaviour and appropriate social behaviours and maintenance of set (Lezak et al., 2004). It also estimates the affective value and subjective emotional experience of sensory stimuli (Rolls & Grabenhorst, 2008). Impaired regions cause disinhibition, aggressive behaviour, sexual promiscuity, impulsivity and antisocial behaviours (Alvarez et al., 2006).

Previous studies also demonstrate that the executive function system is highly involved in handling new situations that are not covered by our ‘automatic’ psychological processes, as evidenced by the reproduction of learned schemas or set behaviours (Norman & Shallice, 2000). Norman and Shallice (2000) identified five kinds of situations in which routine behaviour does not suffice for the best performance: (a) situations that require planning or decision-making, (b) situations that need troubleshooting or error correction, (c) situations with no well-rehearsed responses or with new sequences of actions, (d) dangerous or difficult situations, and (e) situations that necessitate a strong typical response or resisting temptation.

Executive functioning is considered to be a domain-general cognitive function, but investigators have divided it into affective (hot) and cognitive (cold) aspects (Zelazo &

Miller, 2002). The distinction between hot and cold cognition indicates that EF may work differently in varying situations (Hongwanishkul et al., 2005).

Hot cognition, hypothesised to be motivated reasoning, occurs when an individual's thinking is affected by their feelings. To be more precise, hot cognition (Brand, 1985–1986) is cognition coloured by emotion. Hot cognition proposes that there is an association between cognitive and physiological arousal and that the individual is more responsive to environmental factors because it is fast, automatic and emotion-driven (Lodge & Taber, 2005). Biased and poor decision-making may result from hot cognition flaws (Huijbregts et al., 2008). Studies revealed a connection between EF performance and emotional regulation, demonstrating that those with impaired EF have weaker emotional regulation (Zelazo & Kesek, 2010). Study 3 attempted to explore the role of emotional regulation in the relationship between ADHD symptoms and substance misuse.

Cold cognition is cognitive information processing without emotional involvement, the polar opposite of hot cognition (Roiser & Sahakian, 2013). Moreover, cold cognition decisions are more likely to be based on logical and critical analysis (Kunda, 1990). Thus, when someone uses cold cognition when engaged in a task, the stimuli are not emotional, and the test result is not relevant to the person's motives (Roiser & Sahakian, 2013). Concentrating on the evidence before reporting any conclusion is an example of critical decision-making using cold cognition.

Individuals with neuropsychological conditions show deficits in both hot and cold cognition. For instance, studies indicate that people with ADHD and those who misuse substances show EF deficits (Hinshaw et al., 2007; Jansari et al., 2012; Marceau et al., 2017;

Montgomery et al., 2010, 2012; Pennington & Ozonoff, 1996; Pentz et al., 2016; Soriano-Ferrer et al., 2014; Tamm et al., 2013; Throll, 2007). People with EF deficits display poor future goal-oriented behaviours and difficulty incorporating knowledge to modify behaviour. Individuals who misuse substances have a strong desire to use different drugs regardless of the consequences (Tamm et al., 2013). Substantial substance misuse can have an intense impact on how a person handles the reinforcing properties of drugs, influence mechanisms of control and the quality of responses to decisions (Almeida et al., 2012). Studies have demonstrated an association between regular substance misuse and EF impairments (Grant et al., 2012; Piechatzek et al., 2009). However, the results of previous research are contradictory, which may be due to various EF measurements. Thus, in Study 1 of this thesis, a new virtual reality task was used to measure different EF facets and explore the connection between each facet, ADHD symptoms and alcohol, cannabis and nicotine use separately.

Investigations also found that individuals with ADHD have impairments in different EF domains (Hervey et al., 2004; Willcutt et al., 2005), including attention and response inhibition (Malloy-Diniz et al., 2007), risky decision-making (Malloy-Diniz et al., 2007; Toplak et al., 2005), working memory (Andersen et al., 2012; Schweitzer et al., 2006) and planning and shifting (Rohlf et al., 2012, van Mourik et al., 2005). Childhood ADHD is associated with increased substance misuse in adolescence and adulthood (Charach et al., 2011; Lee et al., 2011). Furthermore, as discussed in the previous section (Section 1.3), individuals with substance misuse problems often have comorbid ADHD (Wilens, 2007). The research on the relationship between EF, ADHD and substance misuse is mixed. On the one hand, some of them find no connection between substance misuse and EF impairments (Wilens et al., 2011a, 2011b), while others propose that substance misuse can predict EF

impairments even after controlling for ADHD (Fried et al., 2005). None of the previous studies has investigated the role of EF facets in the relationship between each ADHD symptom and substance misuse separately. Thus, the focus of Study 1 of this thesis was on the role of different EF facets as a multifaceted construct in the relationship between alcohol, cannabis and nicotine use and inattention and hyperactivity/impulsivity as two main symptoms of ADHD, which will be discussed in detail in Chapter 2. This research could assist future investigators in determining how each ADHD symptom is linked to each facet of EF and alcohol, cannabis and nicotine use.

1.4.2. Impulsivity

Impulsivity is the propensity to act without deliberation. Besides the importance of impulsivity in personality, it also plays a crucial role in diagnosing and comprehending different forms of psychopathology. Impulsivity may be the most common diagnostic criteria after subjective distress in the DSM (APA, 1994, 2013; Moeller et al., 2001). Given its psychological value, it is surprising that there are inconsistent conceptualisations of impulsivity. For example, Eysenck and Eysenck (1977, 1978) uncovered a multidimensional nature of impulsivity. They separated impulsiveness into four main facets: narrow impulsiveness (the tendency to act without thinking), non-planning, risk-taking and liveliness. Tellegen (1982) included a control dimension under the higher-order constraint factor. Cloninger added novelty-seeking, which measures thrill-seeking and acting on the feeling of the moment without thinking about the rules (Cloninger et al., 1991, 1993). Moreover, Depue and Collins (1999) suggest that ‘impulsivity comprises a heterogeneous cluster of lower-order traits that includes terms such as impulsivity, SS, risk-taking, novelty-

seeking, boldness, adventuresomeness, boredom susceptibility, unreliability and unorderliness' (p. 495).

Barratt and colleagues (Barratt, 1993; Gerbing et al., 1987; Patton et al., 1995; Stanford & Barratt, 1992) introduced three higher-order factors that encompass various components of impulsivity:

- Attentional impulsiveness is the ability to focus on tasks.
- Motor impulsiveness is the ability to act on the spur of the moment and perseverance.
- Non-planning is the ability to have self-control and cognitive flexibility.

The third factor was not consistently established, but the other two factors were also identified by other investigators (Luengo et al., 1991).

Newman and colleagues (Newman & Wallace, 1993; Wallace et al., 1991) mapped Eysenck's personality system on to Gray's approach/avoidance neuropsychological model (1987). Gray (1970, 1987) proposed that trait impulsivity reflects the responsiveness of the behavioural activation system (BAS) in his original formulation of reinforcement sensitivity theory (RST). In response to incentives, this system is responsible for approach motivation, whereas, in response to punishment, it is responsible for active avoidance. Individuals with high levels of impulsivity were thought to be more sensitive to conditioned reward signals, resulting in an increased tendency to seek potentially rewarding stimuli. Recent research suggests that a broader trait cluster of impulsive antisocial sensation-seeking or trait extraversion could reflect BAS activity better (Depue & Collins, 1999; Pickering & Gray, 2001; Smillie et al., 2006).

Different researchers have tried to place impulsivity in a comprehensive personality theory, but none of the proposed frameworks has received extensive acceptance. This may be because there are various reference point personality models, and there is no consensus on the number and content of personality dimensions. Whiteside and Lynam (2001) suggested a four-factor impulsivity model, based on the five-factor model of personality (McCrae & Costa, 1990) to understand and place various conceptions of impulsivity into a framework. Their decision was consistent with Zuckerman and colleagues (1991), who claimed that three- and five-factor personality models are equally strong but recommended the latter because it has more specificity.

The five-factor model of personality (McCrae & Costa, 1990) consists of five higher-order factors domains: neuroticism, conscientiousness, agreeableness, openness to experience and extraversion, with each domain having six facets. In this model, four separate facets on three different domains capture some features of impulsivity. Costa and McCrae (1992) proposed that in their personality inventory, the Revised NEO Personality Inventory (NEO PI-R), the neuroticism domain has an impulsiveness facet, while the conscientiousness domain has a self-discipline facet, both of which may measure low self-control. They stated that 'people high in impulsiveness cannot resist doing what they do not want themselves to do' and that 'people low in self-discipline cannot force themselves to do what they want themselves to do' (McCrae & Costa, p. 18).

Based on various suggestions about impulsivity, it can be concluded that impulsivity is a multifaceted construct, with many of these varieties appearing across multiple theories and classifications. One of the approaches that received empirical support was introduced by Whiteside and Lynam (2001). They attempted to identify distinct facets of personality that

had been combined and confused under the term impulsivity. They also endeavoured to bring order to the different measures and conceptions of impulsivity and introduced four underlying facets: urgency, lack of premeditation, lack of perseverance and sensation seeking.

Urgency is the first facet that is connected to the impulsiveness facet of the NEO-PI-R; it is the tendency to act impulsively in negative affect situations frequently. Negative urgency is a weak propensity for impulsive and rash actions in response to negative emotions (Cyders & Smith, 2008; Whiteside & Lynam, 2001). It is a dimension of impulsivity that can predict problematic behaviours such as substance misuse, violence, etc. better than other facets of impulsivity, such as sensation seeking (Derefinko et al., 2011; Settles et al., 2012).

Lack of premeditation is associated with the low deliberation facet of NEO-PI-R, which is a proclivity to act without regard to the action's consequences. Lack of perseverance identified with the self-discipline facet of NEO-PI-R and is an individual's inability to remain focused on a boring and difficult task. Sensation seeking is linked to the NEO-PI-R's excitement facet and refers to a propensity to seek and enjoy exciting activities and an openness to new, yet potentially dangerous experiences. This four-factor impulsivity model, known as UPPS (urgency, premeditation, perseverance and sensation seeking), was developed after factor analyses of multiple impulsivity measures. Whiteside and Lynam (2001) introduced the UPPS Impulsive Behaviour Scale as a result.

In 2007, Cyders and colleagues found a fifth disposition called 'Positive Urgency', which is the inclination to act rashly in extremely positive emotional experiences. They proposed this hypothesis based on the existence of this disposition's indirect evidence. University students drink more during celebrations than they do during the academic days

(Del Boca et al., 2004). In addition, their drinking is heavy, which may lead to unwanted sexual intercourse, heightened physical violence, dangerous driving under the influence and alcohol-related injuries and deaths (Del Boca et al., 2004). A positive mood may increase risky behaviours (Yuen & Lee, 2003). Investigations indicate that adolescents act rashly with ill-advised behaviours while they are distressed or are unusually happy (Steinberg, 2004).

Cyders et al. (2007) discovered that rash behaviour dependent on positive mood were not included in Whiteside and Lynam's (2001) factor-analysed measures of impulsivity. Thus, they developed a valid content scale; factor analysis indicated that it was unidimensional. The researchers then factor analysed the measure of positive urgency together with the other four constructs and generated a five-factor solution called UPPS-P, which stands for urgency (negative urgency), lack of premeditation, lack of perseverance, sensation seeking and positive urgency (Lynam et al., 2006). A modest correlation was found between positive urgency and sensation seeking, lack of planning and perseverance (r 's ranged from .21 to .28), and a stronger correlation with negative urgency ($r = .37$). There was a modest correlation between positive urgency and sensation seeking, lack of planning, and perseverance (r 's ranged from .21 to .28) and a more highly correlation with negative urgency ($r = .37$). In 2006, Lynam and colleagues developed the UPPS-P Impulsive Behaviour Scale, which is explained in more detail in Section 3.6.3.2 of Chapter 3 of this thesis.

Impulsivity is a major element of many disorders, including ADHD (Cortese et al., 2012; Nigg, 2001) and substance misuse (Lane et al., 2003; Madden et al., 1997). Even though the various manifestations of impulsivity may contribute to different brain networks, neurobiological findings suggested that specific brain regions are involved in impulsive behaviour (Berlin et al., 2004; Corsini, 1999; Salmond, 2005; Whelan et al., 2012). It has

been argued that genetics may also play a role in impulsive behaviours (Terracciano et al., 2011). For instance, as mentioned earlier (Section 1.4.2.), the PFC is a crucial part of the brain involved in ADHD and substance misuse; studies indicated that it is also the most ubiquitously implicated in impulsivity (Castellanos & Tannock, 2002). Damage to this area of the brain causes impaired switching between response alternatives, poor action preparation and deficits in inhibiting inappropriate responses (Bush, 2011; Franck et al., 2015; Ortiz et al., 2015; Salavert et al., 2015; Winstanley et al., 2006). Impaired PFC function may promote impulsivity, which is a core symptom of ADHD (Barkley, 1997).

In addition, impulsivity seems to be associated with all stages of SUD (De Wit, 2009; Perry & Carroll, 2008). The progression from a single dose to regular use is considered to be central to the acquisition of SUD (Perry & Carroll, 2008). There is a bidirectional relationship between substance misuse and impulsivity. Apart from the effects of impulsivity on substance misuse, studies also showed that substance misuse could increase impulsivity (Perry & Carroll, 2008). The connection between impulsivity and substance misuse could be because the bigger long-term benefits may be offset by the immediate gratification provided by the substance and that individuals with poor inhibitory control cannot overcome motivating cues, such as peer pressure (De Wit & Richards, 2004). Likewise, people who use higher amounts of alcohol, cannabis, nicotine and other illegal drugs discount the value of delayed gratification compared to those who consume less (Kollins, 2003). A more severe level of substance misuse is marked by escalation, followed by losing control, resulting in stronger substance-taking behaviour and binge substance misuse. It has been suggested that higher levels of impulsivity may increase an individual's likelihood of progressing to the escalation stage of SUD (Perry & Carroll, 2008). There is also a connection between

impulsivity and treatment, abstinence and relapse phases of SUD. According to Hershberger et al.'s (2017) meta-analysis, the results of studies that used UPPS-P to measure impulsivity facets in treatment-seeking individuals with SUD showed that higher negative urgency and lack of premeditation were associated with poorer treatment outcome. In addition, a study by Doran et al. (2007) revealed that smokers with higher Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) scores responded more to the smoking cues and gave in to the craving faster than smokers with lower impulsivity scores. Another study also suggested that people with higher impulsivity levels are more likely to remain on drugs and relapse earlier than those with lower levels of impulsivity (Perry & Carroll, 2008).

One conclusion that can be drawn from all previous literature is that impulsivity is not a unitary construct. Based on past investigations, individuals with ADHD symptoms and substance misusers both exhibit impulsive behaviours and share common regions of the brain functionality. Moreover, none of the previous studies used a comprehensive measurement of impulsivity. The multi-component perspective of impulsivity has also not been widely explored concerning individuals with ADHD symptoms and substance misuse. Thus, UPPS-P and BIS-11 were used in this thesis's Study 2 to provide a more detailed assessment of impulsivity facets. This may help to uncover processes by which facets of impulsivity act as major risk factors for developing substance misuse in people with different ADHD symptoms.

1.4.3. Emotional Regulation

Another risk factor in individuals with ADHD and those with substance misuse is emotional self-regulation, which involves two stages: (a) the ability to inhibit strong emotional reactions to various settings and (b) the continued use of self-regulatory actions such as self-soothing, diverting attention away from the provocation issue, reducing and moderating an emotion and organising emotional expression to support an individual's goals and long-term wellbeing (Barkley, 2010; Gottman & Kats, 1989; Hinshaw, 2003; Martel, 2009; Melnick & Hinshaw, 2000).

There is not a unitary standard definition for emotional regulation because of its multidimensional nature. In general, it is characterised by a lack of inhibition with strong negative and positive emotions and poor self-regulatory actions (Mitchell et al., 2012). Regulation of emotion is the ability to respond to emotional experiences in a socially acceptable and appropriately flexible manner, allowing for unplanned reactions as well as the ability to delay impulsive reactions if needed (Cole et al., 1994). It may also refer to the external and internal processes engaged in monitoring, modifying and evaluating emotional reactions (Thompson, 1994). Hence, it includes regulating one's feelings as well as those of others (Burman et al., 2015; Niven et al., 2009). Emotional regulation comprises a set of cognitive processes such as inhibiting, initiating or controlling one's state or behaviour in a specific situation: personal experiences such as feelings, cognitive abilities such as thoughts, physiological, emotional reactions such as heart rate or hormonal activities and emotional behaviours such as body and behaviour expressions. Furthermore, an individual's propensity to focus on a task and suppress inadequate behaviour is also a type of emotional regulation,

which is a significant function in life (Campbell-Sills & Barlow 2007; Geschwind et al., 2011; Gross 2002; Heilman et al., 2010).

Humans use various strategies to control their emotional experiences and expressions under distress, such as repression and suppression (Gross, 2002). These control strategies demand psychological effort. However, during stress, pleasurable or immediate goals, a conflict may occur in various regulatory goals, endangering volitional behaviour and leading to the loss of impulse control (Koole, 2009; Tice et al., 2001). In general, emotional dysregulation refers to difficulties in controlling the impact of aroused emotions on the organisation and quality of actions, interactions and thoughts. Hence, in individuals with emotional dysregulation, there is a misalignment in the response patterns between their goals, responses and modes of expression and what the social environment demands (Zeman et al., 2006).

Even though much of the research is on the three main symptoms of ADHD (inattention, hyperactivity and impulsivity) and their association with functional deficits (Anastopoulos et al., 2011; Corbisiero et al., 2013; Retz et al., 2012), more recent studies have focused on emotional regulation (Barkley & Fischer, 2010; Corbisiero et al., 2013; Reimherr et al., 2005; Shaw et al., 2014). In their review, Shaw et al. (2014) indicated that the prevalence of emotional dysregulation is 34% to 70% in adults with ADHD and 25% to 45% in children with ADHD. Additionally, emotional dysregulation impairments could predict adult impairments in different domains such as family, friends and the workplace, independent of the disorder's course (Barkley & Murphy, 2010b; Skirrow & Asherson, 2013; Stringaris & Goodman, 2009; Stringaris et al., 2010; Surman et al., 2013).

Adults and children with ADHD often show irritability, fluctuations and dysregulations in their emotions (Biederman et al., 2012; Sobanski et al., 2010; Stringaris & Goodman, 2009; Stringaris et al., 2010; Surman et al., 2011). In addition, neuroimaging studies revealed that ADHD and emotional self-regulation share common prefrontal brain networks (Hutchinson et al., 2008; Machie et al., 2007; Paloyelis et al., 2007; Valera et al., 2007). These networks link the lateral PFC, ACC and the amygdala/limbic system (Bush et al., 2000; Etkin et al., 2006; Ochsner & Gross, 2005; Paus, 2001).

On the one hand, the hyperactivity/impulsivity symptom of ADHD causes behavioural disinhibition, leading to raw and strong early emotional reactions. On the other hand, executive functioning impairments affect effortful actions required for an individual to have moderate emotional states, which are more socially acceptable, age-appropriate and better for the person's long-term wellbeing (Barkley, 1997; Castellanos et al., 2006; Martel, 2009).

Previous studies have proposed an association between impulsivity and poor EF performance in individuals with ADHD symptoms and substance misuse, which could be connected to emotional dysregulation. Emotional impulsiveness is another term for impulsivity; it is described as behaving impulsively and rashly under the pressure of positive or negative emotions (Shapiro, 1965). An individual with these behaviours reacts to a stimulus based on an immediate emotional reaction (desire or anger), regardless of the consequences (Wingrove & Bond, 1997). Past investigations suggest that emotional impulsiveness has been included in ADHD for a long time, dating from 1902 to 1976 (Barkley, 2010; Skirrow et al., 2009).

Studies indicated that individuals with ADHD often start using substances to cope with their ADHD symptoms and impaired emotional regulation (Kronenberg et al., 2015). In other words, individuals with higher ADHD symptoms misuse more substances to ‘self-medicate’. Therefore, emotional regulation may play a crucial role in misusing substances and later SUD in individuals with ADHD symptoms (Bradizza et al., 2018).

Emotional dysregulation can also affect the severity, course, treatment result and relapse of SUD. As mentioned earlier (Section 1.4.1), EF deficits are common in both individuals with ADHD symptoms and substance misusers and may be related to emotional dysregulation (Marceau et al., 2018; Wilcox et al., 2016). Marceau et al. (2018) investigated the relationship between inhibition, working memory, task-switching performance, different facets of EF and emotional dysregulation in 50 SUD treatment seekers. Their goal was based on a theoretical framework that suggested that effective emotional regulation could be linked to a person’s performance in basic EF tasks (Hofmann et al., 2012; Schmeichel & Tang, 2015). Results showed that there was a positive correlation between poor emotional regulation and inventory-based EF and personality disorder indicator scores. A deeper understanding of related neurocognitive deficits and the discovery of neurobiological substrates may serve in the assessment of more advanced strategies and the development of interventions that affect emotional dysregulation in SUD treatment. Besides basic EF performances such as working memory and inhibition, Marceau et al.’s (2018) findings revealed that task switching was uniquely connected to emotional dysregulation.

Based on common neurobiological structures (ventromedial PFC) of emotional regulation and impulsivity, it is possible that in significant emotional situations, most resources are devoted to processing emotions, with few remaining for executive functions

such as behavioural control, decision-making and cognitive analysis (Brown et al., 2012). This may explain the relationship between poorer emotional regulation and higher impulsivity in individuals who misuse substances (Jakubczyk et al., 2018).

According to studies, many people smoke to alleviate their negative affect and improve their mood, showing the importance of emotional regulation in nicotine misuse (Carter et al., 2008). Different investigations revealed the relationship between emotional dysregulation and attentional bias to smoking cues (Fucito et al., 2010), heightened nicotine craving (Szasz et al., 2012) and reduced successful cessation and early smoking initiation (Farris et al., 2016). Hence, studying emotion dysregulation may be vital in understanding why people succeed or fail to quit smoking. A main symptom of withdrawal is negative affect (Zinser et al., 1992). Poor affect regulation could raise the likelihood of addictive behaviours such as smoking to reduce negative affect. Thus, this information can shed light on the possible importance of emotional dysregulation in the smoking processes (Rogers et al., 2018). Based on SUD theories that include self-medication and negative reinforcement, emotional processes are primary factors in substance misuse and related consequences (Baker et al., 2004; Duncan, 1975; Khantzian, 1985). Another reason may be that common brain areas in emotional regulation and substance misuse increase the risk of misusing alcohol, nicotine and cannabis in people with poor emotional regulation (Damasio, 2002, Daumann et al., 2011).

In conclusion, as presented in this section, emotional regulation is associated with EF and impulsivity in individuals with ADHD symptoms and those with substance misuse (Brown et al., 2012; Fucito et al., 2010; Pedersen et al., 2016; Petit et al., 2015; Shapiro, 1965; VanderVeen et al., 2016; Watkins et al., 2015).

This section's presented studies have explored the relationship between emotional regulation and ADHD as a single neurodevelopmental condition and investigated emotional regulation and substance misuse associations separately. Study 3 of this thesis was the first attempt to examine the role of emotional regulation in the relationship between each symptom of ADHD (inattention and hyperactivity/impulsivity) and substance misuse in typically developing individuals with inattention and/or hyperactivity/impulsivity symptoms to provide a better understanding of emotional regulation as a risk factor for developing alcohol, cannabis and nicotine use.

The next sections are dedicated to mood disorder and sleep quality since many studies suggest that mood disorder is defined in part by chronic emotional dysregulation (Gross & Levenson, 1997; Kring & Werner, 2004; Lynch et al., 2001). Different investigations indicate that those who misuse substances and have mood disorders demonstrate more emotional functioning impairments (Joormann & Stanton, 2016; Witkiewitz & Marlatt, 2004). Additionally, high levels of emotional dysregulation, mood lability, anxiety and irritability have been seen in individuals with ADHD symptoms (Shaw et al., 2014). It has also been shown that emotional memory consolidation occurs, especially during Rapid Eye Movement (REM) sleep (Nishida et al., 2009). Sleep problems may impair the ability to assess vague situations, identify affective facial expressions and analyse negative emotional stimuli (Deliens et al., 2014). These cognitive impairments can lead to increased irritability, temper outbursts and emotional dysregulation (Leibenluft & Stoddard, 2013); improving sleep patterns can decrease the level of such symptoms (Waxmonsky et al., 2017).

1.4.4. Mood disorders

When the main underlying feature is a disturbance in an individual's mood, it is diagnosed as a mood disorder in the DSM and ICD (Sadock & Sadock, 2002). There are some basic subgroups of mood disorders such as elevated moods such as mania or hypomania. Depressed mood such as bipolar disorder, also called manic depression or manic-depressive disorder, are cycles of abnormally persistent high and low moods and unstable emotions. Previously it was known as manic depression, with occasional psychotic symptoms, rapid cycling and mixed states (Schacter et al., 2011). Mania is described as an abnormally elevated affect, energy and arousal. It is characterised by heightened general activation, greater affective expression and affective lability (Berrios, 2004). Even though mania is considered a depression mirror image, this heightened mood may be irritable or euphoric; stronger manic symptoms result in irritability and violence.

When an individual shows one or more major depressive episodes, they can be diagnosed with major depression disorder (MDD), also known as major depression, unipolar depression or clinical depression. If the person has a single episode, they are diagnosed with single episode major depressive disorder. Having more than one episode means that the individual can be diagnosed with recurrent major depressive disorder. In addition, unipolar depression is a kind of depression without periods of mania (Parker, 1996). Moreover, depressive disorders contain certain subtypes of less severe symptoms, such as dysthymic disorder, which is similar to depression but milder, and cyclothymic disorder, which is similar to bipolar disorder but milder (Carlson et al., 2010).

Meta-analyses have indicated that a strong predictor for developing mood disorders is gaining high scores on the personality domain neuroticism (Jeronimus et al., 2016). It has been suggested that mood disorders could be a form of evolutionary adaptation (Allen & Badcock, 2006). For example, Nesse (2000) demonstrated that a depressed mood could enhance one's coping ability in a situation where pursuing the goal will result in loss, danger or wasted energy. However, the reasons for developing bipolar disorder are unclear, but environmental and genetic factors play main roles (Anderson et al., 2012). Childhood misuse and long-term stress are examples of environmental factors (Anderson et al., 2012). Moreover, many genes with minor effects may increase the risk (Anderson et al., 2012; Goodwin, 2012). ADHD and substance misuse are also genetic and environmental factors that heighten the risk of mood disorders (Anderson et al., 2012).

Young et al. (2015) conducted a meta-analysis of eighteen studies to determine the likelihood of psychiatric co-morbidities in the incarcerated ADHD population. There was a significant overall effect size with moderate heterogeneity ($I^2 = 62.9\%$). In the youth group, the effect of ADHD on depression was not significant, but it was in adults. Adult offenders with ADHD symptoms are five times more likely to develop mood disorders, four times more for anxiety disorder and three times more for SUD than those without ADHD (Young et al., 2015). Studies show that 9.4% of adults with depression, 22.6% of adults with dysthymia and 21.2% of adults with bipolar disorder have co-morbid ADHD (Kessler et al., 2006). Untreated ADHD has been identified as a significant predictor of substance misuse and subsequent SUD (Wilens et al., 1997), with 23.1% of SUD treatment seekers having co-morbid ADHD (van Emmerik-van Oortmerssen et al., 2012).

There are strong links between ADHD and bipolar disorder, such as genetic contributions (Klassen et al., 2010). It has been suggested that both ADHD and mood disorders have similar neurobiological bases. Investigations showed that there are common brain regions involved in ADHD and other psychiatric comorbidities (Klassen et al., 2010, 2012). Differences in the activity and volume of the frontal lobe involved in emotion, attention and behaviour selection have been implicated by neuroimaging studies (Bond et al., 2012). Furthermore, studies on neurotransmitters have determined certain abnormalities in DA and norepinephrine (NE) (Bond et al., 2012; Krause, 2008) signalling (Volkow et al., 2012).

The vital neural sections that control emotional affect includes the limbic-cortical-striatal-pallidal-thalamic (LCSPT) circuits, which consists of connections between the orbital and medial PFC (OMPFC), hippocampal subiculum, ventromedial striatum, mediodorsal and midline thalamic nuclei, ventral pallidum and amygdala (Ongur et al., 2003). These parts participate in higher-order cognitive functions with instinctual information alongside environmental conditions to influence emotional regulation and mood. This is done through shared regions of the cortex involved in higher-order cognitive processes and those that regulate autonomic functions, such as the hypothalamus and the periaqueductal grey (Drevets et al., 2008). Although the neural activity in LCSPT circuits is mainly glutamatergic and is locally controlled through the GABA system (Carlson et al., 2006), the LCSPT circuit activity and its related parts can be controlled by various neuromodulators, such as endocannabinoids (Lupica et al., 2004) and different monoamines. Depression is characterised by impairments in LCSPT circuits and their controlling neurotransmitter system (Ongur, 2003).

The underlying mechanism of this effect may be due to reward processing deficits (characterised as low hedonic tone) with changed monoamine signalling, which is thought to be linked to the modulation of this circuitry (Admon & Pizzagalli, 2015; Sternat et al., 2014; Sternat & Katzman, 2016). These impairments have been observed in children with ADHD who prefer immediate rather than delayed rewards, causing increased sensitivity to reinforcement (Sagvolden et al., 1998). This parallels certain abnormal changes in reward and motivation neural pathways in depression (Dickstein et al., 2006). It can be concluded that this low hedonic tone is the main feature of depression and is caused by a shared dysfunction in monoamine signalling, especially in the ventral striatum (Sternat & Katzman, 2016). Moreover, impairments in DA and NE can be seen in individuals with ADHD and those with depression, implying shared fundamental pathophysiology (Dunlop & Nemeroff, 2007; Garnock-Jones & Keating, 2010; Nigg & Casey, 2005; Sergeant et al., 2003).

Both ADHD and bipolar disorder have similar characteristic conditions and diagnostic criteria. Since several symptoms of ADHD and bipolar disorder overlap, diagnosing both disorders is more complicated (Kent & Craddock, 2003; Wingo & Ghaemi, 2007). There are some similarities between the elevated or manic phase of bipolar disorder and ADHD, such as increased energy or being ‘on the go’, talkativeness, being easily distracted, frequently interrupting others, physical restlessness and impaired normal social inhibitions (Kessler et al., 2010).

In a study by Kim et al. (2019), in depression, there was no significant association between ADHD symptoms and anxiety and depression. However, in bipolar disorder, strong correlations between ADHD symptoms and depression, anxiety and lifetime hypomania were found. Besides certain similarities between ADHD and bipolar disorder symptoms, such as

impulsivity, inattention, hyperactivity, physical energy and behavioural and emotional lability, there are also some fundamental differences. One of the main distinctions between these disorders is that ADHD affects behaviour and attention, while bipolar disorder affects mood. Furthermore, in individuals with bipolar disorder, the symptoms cycle through different episodes of depression and mania/hypomania. Conversely, individuals with ADHD show chronic and not cycling symptoms even though they may also have mood symptoms.

This overlap between the symptoms of ADHD and bipolar disorder makes diagnosing both conditions difficult. As mentioned earlier, substance misuse is common in individuals with ADHD symptoms and those with bipolar disorder, which may be due to shared brain areas, a common genetic basis, or self-medication. Most previous studies have centred on the bipolar disorder-ADHD or bipolar disorder-substance misuse relationship. Study 4 in this thesis aimed to explore the mediation relationship between all three of these conditions. Furthermore, this research is unique in the field since it divides ADHD symptoms and examines their interaction with bipolar disorder and alcohol, cannabis and nicotine use separately. Studies have revealed an association between emotional dysregulation, mood disorders and developing substance misuse in those with ADHD symptoms (Klassen et al., 2010; Shaw et al., 2014; Young et al., 2015). However, none of them investigated whether or not those with inattention and/or hyperactivity/impulsivity use higher amounts of alcohol, cannabis and nicotine via an emotional regulation or mood disorder pathway, which was the focus of this thesis' Studies 3 and 4.

1.4.5. Sleep quality

Sleep disorder or somnipathy is a medical disorder related to the sleep pattern of an individual or an animal. Some sleep disorders can have severe negative impacts on mental, emotional, social and physical functions (Voderholzer et al., 2012). Various issues can cause sleep disorders, such as teeth grinding or night terrors (Hirshkowitz, 2004).

Sleep disorder is categorised into dyssomnias, parasomnias, circadian rhythm sleep disorders, which include sleep timing and sleep conditions caused by medical or psychological problems (Hirshkowitz, 2004). Other sleep disorders are sleeping sickness, which is caused by infection, narcolepsy and hypersomnia characterised by extreme sleepiness at inappropriate times. Insomnia occurs when a person has difficulty falling or staying asleep without a clear cause. Cataplexy happens when a person loses muscle tone while awake, and sleep apnoea occurs when an individual stops breathing during sleep. Bedwetting, sleepwalking and sleep terrors are other forms of sleep disorder (Hirshkowitz, 2004).

Kajeepeta et al. (2015) found that childhood disturbing experiences such as sexual trauma or extreme family conflict significantly raise the risk of different sleep disorders, including apnea, narcolepsy or insomnia in adulthood. Furthermore, studies indicated that idiopathic REM sleep behaviour (iRBD), a sleep disorder characterised by dream-enacting behaviour and loss of muscle atonia during REM sleep, has a genetic component. Schenck (2013) tested 632 individuals with self-report questionnaires: 50% with iRBD and 50% without. The study's results demonstrated that people with iRBD had more first-degree relatives with the same sleep disorder than those of the same age and sex but without the

disorder. Sleep disorders are also widespread in children and adults with ADHD (Díaz-Román et al., 2018; Neto & Nunes, 2017). Individuals with different substance misuses also develop poor sleep quality (Dixon et al., 2018; Roehrs & Roth, 2015). In this thesis, a full range of sleep quality was measured rather than sleep disorders to have a clearer understanding of the role of sleep quality in the relationship between each ADHD symptom and alcohol, cannabis and nicotine use in typically developing university students.

Poor sleep quality is a significant predictor of other behavioural problems in clinical, non-clinical and university student samples (Foulkes et al., 2019; Owens et al., 2009; Paavonen et al., 2009; Sung et al., 2008). Poor sleep quality has been determined as a risk factor for developing internalising and externalising disorders in many investigations. It has been suggested that normalising sleep patterns in children may improve mood and behaviour (Gregory & Sadeh, 2016). Poor sleep may also impair EF, which are important for emotional regulation, particularly in frustrating situations (Anderson & Platten, 2011; Gruber & Cassoff, 2014; Sadeh et al., 2003).

Furthermore, the top-down control regions of the brain are sensitive to the quality of sleep. For instance, sleep problems may weaken the connection between the amygdala and the medial PFC (Anderson & Platten, 2011; Gruber & Cassoff, 2014), causing increased amygdala activity in reaction to negative emotional stimuli (Yoo et al., 2007).

Research on personality and sleep quality indicated that there is an association between personality disorders with behavioural disinhibition and poor sleep and emotional dysregulation. They showed that insomnia is associated with impulsive behaviour (Asano et al., 2014). In a study, the impulsivity of 391 students was measured by a French version of

UPPS Impulsive Behaviour Scale (urgency, perseverance, premeditation and sensation seeking; Van der Linden et al., 2006, Whiteside & Lynam, 2001) Their sleep quality was evaluated by the French version of the Insomnia Severity Index (Blais et al., 1997) and their thought control ability was assessed by the French version of the Thought Control Questionnaire Insomnia-Revised (Harvey, 2001; Schmidt et al., 2009). Results revealed that the urgency and perseverance facets of impulsivity, aggressive suppression, worry strategies of thought control and insomnia severity were positively correlated. Investigators also indicated that the two strategies of thought control mediate the relationship between the two facets of impulsivity and sleep problems. These results suggest how predisposing and perpetuating factors can be related. To be more precise, specific personality traits may contribute to dysfunctional thought control strategies in some individuals, resulting in unwanted mental activity at night (Schmidt et al., 2010).

Poor sleep quality increases the risk of mental illnesses. For instance, Van Veen et al. (2017) conducted a study of 112 participants with antisocial personality disorder and borderline personality disorder. They measured subjective sleep characteristics using the Pittsburgh Sleep Quality Index (Buysse, 1988) and the Sleep Diagnosis List (SDL) (Sweere et al., 1998) and assessed impulsivity with the Barratt Impulsiveness Scale (Patton et al., 1995). Based on the findings, 53.6% of the participants had poor sleep quality, and 22.3% had severe chronic insomnia. Both sleep problems were significantly associated with impulsivity and attentional impulsiveness, which was not influenced by comorbid disorders. This study showed that active treatment of sleep problems could improve sleep quality and mental and physical health. More importantly, it can reduce the risks related to impulsive behaviours by increasing self-control.

Given that impulsivity is a major factor in developing ADHD and substance misuse and the relationship between impulsivity and sleep problems in previous investigations, it can be deduced that poor sleep quality can increase impulsive behaviours such as substance misuse in individuals with ADHD. Poor sleep quality is common among university students; past research has linked it to living with peers and adapting to a new academic schedule (Foulkes et al., 2019). This can result in poor academic performance and additional health problems such as substance misuse (Freeman et al., 2017). Moreover, misusing substances is more common among university students (Bajwa et al., 2013; Drosican, 2009; Gupta et al., 2013; Maher, 2008; Maier et al., 2013; Mohammadpoorasl et al., 2014; Sommet et al., 2012; Suerken et al., 2014). Thus, it is critical to understand the role of sleep quality in substance misuse among university students, which is the subject of this thesis' final study. Furthermore, poor sleep quality and substance misuse are more widespread in individuals with ADHD symptoms, which is why Study 5 was designed for discerning whether students with inattention or hyperactivity/impulsivity use higher amounts of alcohol, cannabis and nicotine as a result of poor sleep quality.

Depression, bipolar disorder, anxiety, substance misuse and personality disorders are the most common comorbidities of adult ADHD. Due to the significant overlap between these disorders, investigating ADHD using a dimensional instead of a categorical approach has been proposed by researchers (Katzman et al., 2017; Heidbreder, 2015). This approach is consistent with research in other areas of psychiatry, especially the National Institute of Mental Health's Research Domain Criteria (RDoC) and the DSM-5 (DSM-5, 2013), which both encourage a dimensional approach in mental disorder diagnosis and classification to help mental health research (Regier, 2007). A major driver of dimensional methods in mental

health investigations is gaining a greater understanding of the underlying functions' fundamental dimensions across the full human behaviour spectrum from normal to abnormal (RDoC, 2016). Therefore, the dimensional approach was used to measure EF, impulsivity, emotional regulation, bipolar disorder, sleep quality, ADHD symptoms and alcohol, cannabis and nicotine use in all studies of this thesis.

1.5. Aim of this thesis

The literature review in this chapter showed that adults with ADHD symptoms and those who misuse substances share common personality traits, impairments and problems. The overall goal of this thesis is to explore the role of the risk factors such as EF facets, impulsivity facets, emotional regulation, bipolar disorder and sleep quality in the relationship between inattention and hyperactivity/impulsivity and alcohol, cannabis and nicotine use in typically developing samples. To this end, 5 general aims will be pursued:

- To investigate which facet of EF predicts alcohol, cannabis and nicotine use and to examine whether inattention and hyperactivity/impulsivity symptoms separately explain additional variance in developing substance misuse after accounting for EF facets.
- To investigate which facet of impulsivity predicts alcohol, cannabis and nicotine use and to examine whether inattention and hyperactivity/impulsivity symptoms separately explain additional variance in developing substance misuse after accounting for impulsivity facets

- To explore whether emotional regulation mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.
- To explore whether bipolar disorder mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.
- To explore whether sleep quality mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.

To reach these objectives, ADHD symptoms were divided into two categories: hyperactivity/impulsivity and inattention. The groups were measured dimensionally to focus on each variable of this study across the full range of scores. This is the first research to explore the role of different risk factors in the relationship between each ADHD symptom and the use of alcohol, cannabis and nicotine separately. In Study 1, different EF facets were assessed by a new virtual reality task, as explained in Chapter 2, Section 2.6.3.8. Since this virtual reality activity could not test response inhibition as a facet of EF, the Go/NoGo task was also used. Study 1 addresses the thesis' first and second aims and attempts to answer the following questions:

- a) Do separate impulsivity facets have different relationships with alcohol, cannabis and nicotine use?
- b) Can inattention as a symptom of ADHD explain additional variance in alcohol, cannabis and nicotine use after accounting for the facets of EF?
- c) Can hyperactivity/impulsivity as an ADHD symptom cluster explain additional variance in alcohol, cannabis and nicotine use after accounting for the facets of EF?

This approach may aid in the following: (a) elucidating the relationship between each facet of EF as a risk factor and alcohol, cannabis and nicotine use; and (b) discerning whether each ADHD symptom shows a significant improvement in the proportion of explained variance in the development of alcohol, cannabis, and nicotine use, over and beyond the facets of EF. If ADHD symptoms add to the explained variance in substance misuse, other pathways from inattention or hyperactivity/impulsivity to the use of alcohol, cannabis and nicotine may exist apart from EF facets. Both EF and impulsivity have been defined in numerous ways and considered important topics in the study of substance misuse and ADHD. Bickel et al. (2012) proposed that these two multifaceted constructs are notions at widely separated ends of a shared continuum and are antipodes.

Impulsivity is another personality trait in individuals with ADHD symptoms, which may heighten the risk of substance misuse. However, Study 2's new virtual reality task could not measure its different facets. Thus, Study 3 was devoted to assessing impulsivity facets using the UPPS-P (Lynam et al, 2006) and Barratt Impulsivity Scale (BIS-11; Patton et al., 1995) for a comprehensive impulsivity model. Study 2 focuses on the first and second aims of this thesis, as well as the following research questions:

- a) Does each facet of impulsivity predict alcohol, cannabis and nicotine use in a significant way?
- b) In addition to the facets of impulsivity, does inattention as a symptom of ADHD separately add to the explained variance in the use of alcohol, cannabis and nicotine?
- c) Does hyperactivity/impulsivity as a symptom cluster of ADHD separately add to the explained variance in the use of alcohol, cannabis and nicotine?

Considering impulsivity as a multi-component in relation to ADHD and substance misuse may help to explain how impulsivity acts as a risk factor for problematic consequences. Inattention and hyperactivity/impulsivity may account for further variance in substance misuse scores. In that case, apart from impulsivity facets, there could be other pathways, from these ADHD symptoms to the use of alcohol, cannabis, and nicotine that can be explored in the future.

Research demonstrated an association between impulsivity and emotional dysregulation, which is prevalent in both ADHD and substance misuse and is further investigated in this thesis' Study 3. Increased impulsivity in emotional situations involving negative and positive moods can heighten the risk of substance misuse (Shapiro, 1965). Based on studies, people with ADHD symptoms misuse higher amounts of substances to cope with their emotional dysregulation (Kronenberg et al., 2015). Furthermore, shared brain areas in emotional dysregulation, ADHD, and substance misuse may explain the relationship between these conditions (Hutchinson et al., 2008; Machie et al., 2007; Paloyelis et al., 2007; Valera et al., 2007). Thus, examining through an emotional regulation pathway those with inattention or hyperactivity/impulsivity and alcohol, cannabis and nicotine misuse can help in the future discovery of the best treatment strategies. The following are the research questions that will be addressed:

- a) Does emotional regulation mediate the relationship between inattention and alcohol, cannabis and nicotine use?
- b) Does emotional regulation mediate the relationship between hyperactivity/impulsivity and alcohol, cannabis and nicotine use?

As mentioned earlier in this chapter (Section 1.4.4), mood disorders are defined in part by persistent emotional dysregulation. There are four main motivations for conducting Study 4: (a) the connection between mood disorder and emotional regulation, (b) the considerable prevalence of mood disorder in individuals with ADHD and those who misuse substances, (c) some similarities between bipolar disorder as a mood disorder and ADHD symptoms and (d) the presence of both depression and manic cycles in individuals with bipolar disorder.

The following are the research questions that will be addressed:

- a) Does bipolar disorder mediate the relationship between inattention and alcohol, cannabis and nicotine use?
- b) Does bipolar disorder mediate the relationship between hyperactivity/impulsivity and alcohol, cannabis and nicotine use?

Some cognitive impairments in individuals with ADHD and those who misuse substances, such as analysing negative emotions, evaluating ambiguous situations or recognising emotional facial expressions, can increase emotional dysregulation, exacerbated by poor sleep quality (Deliens et al., 2014). Moreover, certain personality disorders, such as behavioural disinhibition and impulsive behaviour (e.g. urgency and Pers), are associated with poor sleep quality (Schmidt et al., 2010). Individuals with ADHD symptoms and substance misuse show impulsive behaviours, executive dysfunction and cognitive impairments. The connection between these problems and sleep quality prompted researchers to focus on the participants' sleep quality and its relationship with ADHD symptoms and alcohol, cannabis and nicotine use. The following research questions are addressed in Study 5:

- a) Does sleep quality mediate the relationship between inattention and alcohol, cannabis and nicotine use?
- b) Does sleep quality mediate the relationship between hyperactivity/impulsivity and alcohol, cannabis and nicotine use?

Most of the previous investigations with adults with ADHD has excluded substance misuse. Studies demonstrate that risk factors such as impulsive behaviours, impaired EF, emotional dysregulation, mood disorder and poor sleep quality are shared by both people with ADHD symptoms and substance misusers. However, research on risk factors that may heighten the probability of substance misuse in adults with ADHD symptoms are limited (Baran Tatar et al., 2015; Fox et al., 2008; Goodman & Thase, 2009; Grant et al., 2004; Hinshaw et al., 2007; Hodgkins et al., 2013; Jansari et al., 2012; Montgomery et al., 2010; Montgomery et al., 2012; Roehrs & Roth, 2015; Verdejo-Garcia et al., 2008). Most of the previous studies measured ADHD as one condition. Nonetheless, this thesis was the first to investigate the role of different EF aspects, impulsivity facets, emotional regulation, bipolar disorder and sleep quality in the relationship between each ADHD symptom (inattention and hyperactivity/impulsivity) and alcohol, cannabis and nicotine use separately.

Chapter 2

2. The role of Executive functions facets in the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine

Overview

The first aim of this chapter is to summarise existing evidence regarding how different facets of Executive Functions (EF) are associated with alcohol, cannabis and nicotine use. The second aim is to examine how the main symptoms of ADHD explain additional variance in alcohol, cannabis and nicotine use after accounting for the facets of EF. Results indicated that impaired response inhibition, adaptive-thinking and time based prospective memory predicted alcohol use significantly. Nicotine use was predicted by response disinhibition and cannabis use was predicted by impaired adaptive-thinking, creative-thinking and response inhibition. In addition, results also showed that hyperactivity/impulsivity explained additional variance in alcohol, life-time cannabis and nicotine use after accounting for EF facets, which indicates that other factors than EF facets may influence alcohol, cannabis and nicotine use in those with higher hyperactivity/impulsivity, which will be explored in Chapter 3 of this thesis.

2.1. Introduction

Many investigations have shown that university students are at a higher risk of developing SUD around the world (Maher, 2008; Drosican, 2009, Mohammadpoorasl et al.,

2014; Suerken et al., 2014; Sommet et al., 2012; Bajwa et al., 2013; Maier et al., 2013; Gupta et al., 2013).

Investigations indicate that the co-occurrence of ADHD and substance misuse is highly prevalent, leading to later SUD. The symptoms of ADHD appear in early childhood, and studies show that symptoms and impairments can continue into adulthood (Lara et al., 2009; Rasmussen & Gillberg, 2000). Those with ADHD start misusing substances at an earlier age, with more severe symptoms, poorer treatment outcomes, and a higher relapse rate after treatment (Arias et al., 2008; Bernardi et al., 2011; Gonzalez et al., 2017; Kollins, 2008). As mentioned earlier in Chapter 1, research with undiagnosed adult ADHD in the UK's university population is highly limited, and further investigation is required in the future.

Impaired EF is common to both individuals with ADHD symptoms and those who misuse substances. Thus, focusing on the role of different EF facets in the relationship between each of the ADHD symptoms and the use of alcohol, cannabis and nicotine may help future research better understand people with ADHD symptoms and substance misuse. The following section discusses EF performance in individuals with ADHD symptoms and those who misuse substances.

2.2. Executive functions performance in people with ADHD and substance misuse

Executive functions are high-level control mechanisms that affect thought and behaviour regulation to successfully achieve a goal (Miyake & Friedman, 2012). Poor EF is common to both people with ADHD symptoms and substance misuse. In people who misuse

substances, impaired stimulus-driven behavioural responses to a specific rewarding stimulus control the person's addictive behaviours (Malenka et al., 2009). Many studies on the EF performance of adults with ADHD and those who misuse substances suggest that these disorders frequently overlap with their EF performance deficits (Hinshaw et al., 2007; Jansari et al., 2012; Montgomery et al., 2010; Montgomery et al., 2012).

Individuals with ADHD symptoms typically show EF difficulties (Hervey et al., 2004; Willcutt et al., 2005) such as impairments in risky decision-making (Malloy-Diniz et al., 2007; Toplak et al., 2005), attention and response inhibition (Malloy-Diniz et al., 2007), working memory (Andersen et al., 2013; Schweitzer et al., 2006) and planning and shifting (Rohlf et al., 2012; van Mourik et al., 2005). In addition to clinical heterogeneity in people with ADHD symptoms, many investigations have established heterogeneity in severity, areas and patterns of EF impairments (Gonzalez-Gadea et al., 2013; Willcutt et al., 2005).

Although recent studies demonstrate neuropsychological deficits in people with ADHD symptoms compared to typically developing individuals (Fair et al., 2012), some results of different studies on the EF performance of people with ADHD symptoms are still unclear. Some individuals with ADHD symptoms show severe EF deficits, while others depict EF performance comparable to those without ADHD symptoms (Willcutt et al., 2005). Weyandt et al. (2013) indicated that university students with ADHD symptoms ($n = 24$) demonstrate clinically significant EF deficits on rating scales compared to those without ADHD ($n = 26$). Other studies show that EF deficits can persist in adulthood even after ADHD symptoms have decreased (Gau et al., 2009; Seidman, 2006).

Investigations on the relationship between social functions and EF performance also show inconsistencies. For instance, while some studies have found a correlation between EF

and social functions (Kofler et al., 2011; Tseng & Gau, 2013), this link was not supported when other combined EF tests were used in children and adolescents with ADHD symptoms (Biederman et al., 2004; Huang-Pollock et al., 2009). A study by Tseng and Gua (2013) showed differences in EF tests performances between individuals with ADHD and with or without social dysfunction. On the other hand, Biederman et al. (2004) found no difference in the EF performance of individuals with ADHD symptoms with and without social dysfunction. These inconsistent results reflect the need for further research into the relationship between ADHD and EF performance.

Impaired EF is also standard in people who misuse substances (Finn et al., 2002). Investigations have revealed a two-way relationship between impaired EF and substance misuse. Hence, EF deficits can both be a pre-existing vulnerability and a consequence of substance use (Bernardin et al., 2014; Squeglia et al., 2012). Studies also suggest that people with more severe EF deficits have a higher risk of substance misuse (Finn et al., 2002). These deficits make them more likely to have substance misuse problems (Day et al., 2013; Nigg et al., 2006) and benefit less from treatment (Bates, 2000).

Some researchers suggest that this relationship may have multiple pathways. For instance, impaired set-shifting or information updating can contribute to substance misuse by making it harder for an individual to choose a coping strategy instead of misusing substances (Day et al., 2014). It is also possible that response inhibition deficits might make it difficult for an individual to resist joining friends who engage in substance misuse (Day et al., 2014).

Some longitudinal investigations show that individuals with EF deficits can develop heavy drinking (more than 4 drinks a day) (Squeglia et al., 2012; Wetherill et al., 2013).

Their findings reveal that EF deficits can cause vulnerabilities that may heighten the risk of alcohol consumption. Evidence also supports a link between EF impairments and relapse to alcohol use among alcohol abstainers (Day et al., 2014). Consequently, research indicates that EF could be relevant in the initiation and maintenance of alcohol use and successful abstinence.

An alternative suggestion is that substance use or misuse results in EF deficits (Bernardin et al., 2014; Cohen et al., 1997; Courtney et al., 2013; Fama et al., 2004; Field et al., 2010; Jones et al., 2013; Maharasingam et al., 2013; Noël et al., 2012; Pitel et al., 2007, 2008). Previous studies have shown that alcohol misuse can damage the brain (Fein et al., 2002; Mason et al., 2005; Meyerhoff et al., 2005). It causes grey matter atrophy in the cerebellum, limbic system and front-parietal lobes (Harris et al., 2008; Oscar-Berman & Marinkovic, 2007; Oscar-Berman et al., 2009). It also causes white matter reduction, specifically in the fronto-cerebellar pathways, which causes impairments in problem-solving, working memory and shifting (Sullivan et al., 2003). As a result of these brain network problems, people with AUD may have cognitive impairments including visuo-perceptive, attentional deficits (Bernardin et al., 2014), emotional and interpersonal problems (D'Hondt et al., 2014; Marinkovic et al., 2009; Maurage et al., 2016) and poor memory (Pitel et al., 2014).

Current AUD models display increased sensitivity to alcohol-related stimuli, and executive dysfunction is a critical issue in AUD (Field & Cox, 2008; Klein et al., 2013). People with AUD make risky decisions and prefer the immediate reward over alcohol consumption's negative consequences (Bechara et al., 2005; Camchong et al., 2014; Noël et al., 2010). Inhibition deficits persist long after quitting alcohol use and have a crucial role in

relapse (Jones et al., 2013). Cognitive recovery is usually seen after a year; the first months of abstinence are important because the executive abilities remain impaired, and the risk of relapse is high (Stavro et al., 2013). Benedek et al. (2017) found that alcohol reduces executive control. Although it can impair divergent thinking (solving problems with various possible solutions), it may improve creative problem solving or convergent thinking (solving problems with a single correct answer). There is an association between divergent thinking and working memory and cognitive disinhibition at the EF level (Benedek et al., 2012, 2014; De Dreu et al., 2012; Zabelina et al., 2012).

While some studies show that cannabis use can cause EF impairments, the evidence is equivocal (Broyd et al., 2016; Pope et al., 2001). For instance, Broyd et al. (2016) found in their review that acute and chronic cannabis use decreases attentional control, but a definitive conclusion could not be drawn due to insufficient data. Some studies also reported the harmful effects of chronic cannabis use on cognitive flexibility during changing environmental context (Fontes et al., 2011; Lane et al., 2007). However, other investigations found no difference in attentional control (Pope et al., 2001), cognitive flexibility and memory span (Fisk & Montgomery, 2008) between chronic cannabis users and non-users.

Studies indicate that cannabis use causes impairments in prospective memory and EF, which may persist beyond acute intoxication (Montgomery et al., 2012). Deficits in attention, complex mental processes, motor skills and reaction time have been reported by previous investigations (Montgomery et al., 2012). Acute cannabis use also doubles the risk of collision in drivers (Bedard et al., 2007). Heavy cannabis use may induce acute functional psychosis, which is similar to schizophreniform disorder and may persist for a long time even after intoxication in some cases (Karila et al., 2014).

However, there are conflicting opinions about the association between cannabis use and cognitive disorders (Iversen, 2005; Pope et al., 2001; Pope et al., 2003; Solowij, 1995). Early onset of use seems to be linked to cognitive impairments (Meier, 2012). Grant et al. (2012) indicated that even young cannabis users between 18 and 29 years of age with no mental health issues and SUD or any other illegal substance misuse showed cognitive impairments. These deficits persist for a few days (Pope et al., 2001), a few months or even longer (Bolla et al., 2005; Solowij, 1995) after cessation of cannabis use. Two meta-analyses indicated that any neurocognitive deficit associated with cannabis misuse or its withdrawal symptoms are limited to the first 25 days of abstinence (Schreiner et al., 2012). In young adults, chronic cannabis misuse can also impair verbal reasoning (Wadsworth et al., 2006), verbal n-back (Herzig et al., 2014) and working memory, but not spatial working memory (Becker et al., 2014; Grant et al., 2012) or digit span (Gruber et al., 2012; Macher & Earleywine, 2012). In some investigations, working memory deficits continued for a few weeks after stopping cannabis use (Medina et al., 2007) but resolved after long periods of abstinence (Hanson et al., 2010; Hanson et al., 2014; Ranganathan & D'Souza, 2006; Thames et al., 2014; Winward et al., 2014).

In several studies, the main result of cannabis intoxication is attention deficit. Reports indicate that acute cannabis use impairs focused, divided and sustained attention in a dose-dependent manner (Anderson et al., 2010; Bedi et al., 2013; D'Souza et al., 2004; D'Souza et al., 2008; D'Souza et al., 2008b; Hunault et al., 2009; Kollins et al., 2015; Schoedel et al., 2012; Theunissen et al., 2015; Wesnes et al., 2010). Some investigators believe that tolerance development among some daily users may cause their reduced impairments (Ramaekers et

al., 2009; Ramesh et al., 2013; Schwoppe et al., 2012). Chronic cannabis users have a higher attentional bias to cannabis-related stimuli (Beraha et al., 2013; Morgan et al., 2010).

Testing the EF performance of nicotine users also shows different results (Dawkings et al., 2007; Jansari et al., 2013). Flaudias et al. (2016) assessed individuals with moderate and heavy nicotine dependence using the Stroop test and Hayling tests. Their results revealed that response inhibition was the only EF facet that has significantly predicted nicotine dependence. However, in Jansari et al.'s (2013) study, 2 hours of nicotine abstinence improved EF and prospective memory in smokers. These discrepancies may be due to the use of different EF tests or variations in the sample.

In light of the inconsistent results of investigations on the relationship between EF and ADHD and substance misuse, further research is needed to gain better insights into these connections. Finding the association between each symptom of ADHD and different facets of EF and between alcohol, cannabis and nicotine use and EF will help future investigations in determining the best treatment for every individual. Measuring EF as a multifaceted construct is one of the most critical issues in this area. As mentioned earlier in this section, different EF tests yield different outcomes (Biederman et al., 2004; Huang-Pollock et al., 2009; Kofler et al., 2011; Tseng & Gau, 2013). Thus, using a test that can measure real-world EF performance across different groups of people is really important.

2.3. Assessment of Executive functions

EF refers to a set of mental processes described in the previous chapter (Section 1.4.1) (Baddeley et al., 1997). Since there is no final agreement on the functional

construction of the executive system (Burgess & Simons, 2005; Chan et al., 2008), there is a wide range of theories relating to EF's definition. Some recommend that a unitary structure is ideal for EF, while others proposed a multifaceted and diverse executive system (Miyake et al., 2000; Miyake & Friedman, 2012). Furthermore, there is little consensus among researchers on EF's conceptual issues, such as in defining which daily tasks need purported cognitive domains activation and which do not. Given the broad spectrum of skills that EF appears to encompass, a diverse collection of tools has been developed to measure EF in children and adults.

Two of the most commonly used tools are the Wisconsin Card Sorting Test (WCST) (Grant & Berg, 1948) and the Controlled Oral Word Association Test (COWAT) (Deutsch, 1995). The WCST assesses the ability to respond to feedback and follow the rules, while the COWAT evaluates verbal fluency. It has been assumed for decades that these two tasks reliably measure cognitive functions. However, investigations indicate that although individuals with different brain injuries or mental disorders (e.g. acquired brain injury) typically perform on the traditional standard EF tasks, they show various EF impairments in their everyday lives (Damasio, 1996; Eslinger & Damasio, 1985). For instance, Eslinger and Damasio (1985) investigated a patient with bilateral ablation of the orbital and lower mesial frontal cortices. The patient had intense changes in his behaviour for eight years. His performance in his personal and professional responsibilities was poor. However, his measurable intelligence was superior, and his performance in neurologic and neuropsychological tests was intact; hence, he was considered a 'malingerer'. There are different theories as to why standard clinical EF tests fail to detect impairment in some individuals.

One proposed reason is the distinction between hot EF (functions involved in emotions and social behaviour regulation) and cold EF (processes involved in automatic logical abilities such as attention, cognitive flexibility, resistance to interference) (Grafman & Litvan, 1999; Zelazo & Kesek, 2010). Most tasks measure isolated parts of the system, so a single measurement would not be enough to detect different problems in an individual entirely. Due to the broad range of clinical measurements, investigators suggested using various tests to find EF impairments in clinical and non-clinical samples (Bennett et al., 2005).

One central point is that traditional tests such as WCST and COWAT measure different EF dimensions and are administered independently (Jansari et al., 2013). However, given that the executive system directs various cognitive subsystems, this questions whether traditional assessments are valid. Evaluating EF's functionality and analysing it singularly and as independent systems cannot appropriately represent the whole system's functioning reliability (Jansari et al., 2013). In other words, traditional measurements cannot challenge and assess cognitive abilities in the same way as in real life.

Besides the need for using multiple forms of assessment, it has been suggested that the tests may not be valid (Jansari et al., 2013). Chaytor and Schmitter-Edgecombe (2003) have argued in their neuropsychological research review that a test's ecological validity is a crucial point, considering that a core purpose of the clinical and non-clinical studies is to learn more about daily life functioning to make appropriate recommendations.

This argument is consistent with Shallice and Burgess' (1991) assertion that the assessments do not resemble the actual tasks with which someone might struggle. It has also been argued that standard tests are too linear and simple: (a) focus is only on one task at a time; (b) task initiation is prompted rather than self-generated; (c) planning for a required

behaviour for completing a task happens in seconds or minutes, while in real life, this occurs over a long time, ranging from seconds to months; (d) and prioritising between completing the tasks is minimal or non-existent (Jansari et al., 2014). Generally, these assessments tolerate 'little resemblance to the ill-structured activities that characterise modern lives' (Bennett et al., 2005, p. 606).

In psychology, *ecological validity* is a measure of how an investigation's findings could be generalised to a real-world setting (Jansari et al., 2014). Ecological validity is the main proposed reason for individuals' average performance on standard EF tasks and their poor performance in daily life (Chaytor & Schmitter-Edgecombe, 2003). Historical examination shows that ecological validity is evident in various experimental research dimensions, such as the nature of the investigational setting, the target stimuli and the researcher's response employed as the measure. However, no clear criteria for evaluating an investigation's ecological validity have been proposed.

New tests have been developed to overcome these problems. For instance, Shallice and Burgess (1991) have devised a real-world paradigm that changed the traditional paper-and-pencil approach in neuropsychological tests. The tasks included multiple shops in a big and busy shopping centre, and those who displayed average performance on conventional tests were considerably impaired in the real-world task. Shallice and Burgess' study supported the clinical observation of participants who did well on established cognitive tests but struggled with real-life and daily activities. The researchers also focused on the necessity of developing practical tasks for higher cognitive functions and introduced a new paradigm to neuropsychological and cognitive investigations called the Multiple Errands Task (MET) (Shallice & Burgess, 1991).

Several studies have created different clinical versions of MET (Knight et al., 2002; Knight & Henman, 2003). Although these approaches successfully assessed cognitive functions in the real world, they have limitations as general clinical tools for various reasons. Despite their higher ecological validity, the clinician's control in these methods was limited. For example, emotionally stressful situations, such as accidents or misunderstandings during a task, can affect an individual's performance. Moving participants to a suitable setting, which is vital for assessment and rehabilitation centres, could be time-consuming and expensive. Additionally, unless the environments are designed for people with motor deficits, such individuals could have specific difficulties that may affect the assessment. The Behavioural Assessment of Dysexecutive Syndrome (BADS) (Wilson et al., 1996) was combined with the multiple errands paradigm elements to address some of these issues. The BADS measures EF deficits, and its ecological validity has been measured and confirmed by investigators (Wilson et al., 1998).

Virtual Reality (VR) technology has been employed to reduce the challenges of using ecological validity assessments while decreasing pragmatic concerns. This technology is a 3D computer environment that can replicate real-world physical surroundings, objects and events. VR has been proven to be a reliable tool for measuring EF for different reasons (Brooks & Rose, 2003; Rizzo & Kim, 2005). To begin with, in comparison to a taxing but unsafe real-world situation and to a safe yet non-taxing traditional paper-and-pencil measurements, assessing the participant in a virtual world allows for the observation of their behaviour and performance in a safe and rich environment. Moreover, the researcher can control the variables during an experiment, which is not possible in real life. The researcher can add, remove or alter the settings or variables to measure and observe a targeted situation or performance. Furthermore, participants agree that VR tools, which are detached from their

daily problems, can be more engaging than paper-and-pencil tests. The results of VR assessments are also replicable in different settings and populations, and they are sensitive to cognitive functions in the same way as real-world tasks (Pugnetti et al., 1998).

Various studies used VR to measure EF and included different environments, samples, measurement types and various EF dimensions (Table 2.1). For instance, Rand et al. (2009) created the Virtual Multiple Errands Test (VMET), which could differentiate patients' performance with post-stroke problems and healthy controls. Their results were correlated with the original MET (Knight et al., 2002).

Table 2.1
Review of investigations using VR to measure EF (Jansari et al., 2014)

| Authors | Participants | Environment | Measures | EF |
|---|---|--|--|------------------------------|
| Zalla, Plassiart, Pillon, Grafman & Sirigu (2001) | Acquired Brain Injury | Home/Morning routine | Action sequences & mistakes | Action planning |
| Elkind, Rubin, Rosenthal, Skoff & Prather (2001) | Healthy Normal Controls (age range 18-74) | Beach/Sorting beach apparel and refreshments | WCST-type categorisation and errors | Unclear |
| McGeorge et al (2001) | Acquired Brain Injury | Office | Time taken; quality of action plan | Planning |
| Titov & Knight (2005) | Acquired Brain Injury | Street | Ability to complete delayed intentions | Prospective remembering |
| Klinger, Chemin, Lebreton, & Marie (2006) | Parkinson's Disease | Supermarket | Number of errors; time selecting items | Shifting attention, planning |
| Carelli, Morganti, Weiss, Kizony & Riva (2008) | Healthy elderly | Supermarket | Number of errors; time selecting items | Shifting attention, planning |
| Josman, Klinger & Kizony (2008) | Stroke, MCI, Schizophrenia | Supermarket | Time take; distance | Strategy |

| | | | | |
|-------------------------------------|-----------------------|---------------------|--|--|
| | | | travelled; number of actions | |
| Rand, Rukan, Weiss & Katz (2009) | Acquired Brain Injury | Supermarket | Number of mistakes; rule breaks; non efficiency mistakes | Multi-tasking; strategy-formation; planning; selection |
| Reger, Parsons, Gahm & Rizzo (2010) | Military personnel | Middle Eastern city | Serial Attention Test; Stroop time and errors | Attention |

Despite the progress, there is an important limitation in terms of the collected dependent measures in such investigations. For instance, when Rand et al. (2009) and Klinger et al. (2006) used a VR task based on Shallice & Burgess' (1991) original shopping task, they had a limited analysis of the number of errors such as those in task completions, rule breaks and 'non-efficiency' mistakes. Although their VMET was able to differentiate between patients and healthy individuals, it is difficult to ascertain whether their measures can be extended to real-life problems that form the dysexecutive syndrome pattern in terms of targeted rehabilitation.

Elkind et al. (2001) likewise developed a VR assessment, which was an electronic version of the WCST and prone to the same problems as this paper-based neuropsychological test. Titov and Knight (2005) created a virtual street scene that could only score participants' efficiency if they could remember and record the required information. Thus, it could only calculate a narrow set of measures related to prospective memory and none of the other EF dimensions.

Based on the limitations and strengths of the VR tests, some investigators proposed a number of required qualities to create a more sensitive and informative measurement of EF (Jansari et al., 2014):

- A more realistic environment with non-linear overall goals should be developed.
- Tasks should be used to assess a number of abilities in parallel.
- The solutions to the tasks' problems should not be apparent.
- A task should not be extremely short to be comparable to real-world tasks when measuring planning and behaviour.
- It should be a safe and rich environment, with the investigator having control over the variables.
- It should also be useful for those with motor disabilities.

Thus, to avoid the limitations of the previous VR tasks and to provide a task that could measure different facets of EF, a new VR task was developed.

2.4. The Jansari assessment of Executive Functions (JEF[©])

Jansari et al. (2014) conducted a study based on previous investigations using VR to develop an ecologically valid EF assessment. They attempted to reduce the limitations of the dependent measures by creating the task in a way that participants would have to complete certain non-linear tasks. Participants were asked to plan and prioritise their actions to reach the required goals.

Their study had two main aims: (a) to develop a new real-world task that distinguishes between individuals with EF deficits and healthy controls, which will help in neuropsychological and vocational rehabilitation; and (b) to evaluate if EF performance differences can be measured in a VR task. Jansari and his colleagues conducted two studies to achieve these objectives: one in which a real-life task was generated and managed with a small sample of six participants and matched controls, and the other in which the assessment

was redeveloped in a VR environment with a larger sample of 17 individuals with Acquired Brain Injury (ABI) and matched healthy controls.

Their task was called the Jansari assessment of Executive Functions (JEF[®]). In the first study, the real-life version of JEF[®] was used, and participants could move around the office and complete the required tasks. The performance of ABI patients and healthy controls were compared. Notably, as an inclusion criterion, all ABI patients performed at the borderline level or higher on the BADS, and only one participant performed poorly on the Brixton or Hayling tasks (Burgess & Shallice, 1997). Besides the overall measurement, analysis of participant cognitive abilities indicated that JEF[®] could identify differences in a particular EF. A VR version of the study with a larger sample revealed that the difference between the two groups of ABI and healthy controls were preserved, probably due to the more extensive data (Jansari et al., 2014). Based on the participants' intact performance on the two famous clinical tests, the primary implication of these results is that JEF[®] can detect the real-world EF deficits described by participants and is ecologically valid. It can also contribute significantly to the clinical assessment of individuals with EF deficits and preserved IQ.

The research's main strength was that it operationally and clearly defined and investigated the executive constructs to aid future investigations based on their results. The assessment measured the main functions that professionals working in the rehabilitation of people with EF deficits could decipher. The evaluation included tasks that measured one construct at least twice, assisting in the creation of an executive ability profile for each participant. Their performance would be assessed through a corresponding three-level scoring system (please refer to Section 2.6.3.8).

JEF[®] has been used in many studies to explore the impact of nicotine and cannabis use on EF performance in healthy individuals. By using JEF[®], Jansari et al. (2012) found that nicotine use improved overall JEF[®] performance, time-based prospective memory and event-based prospective memory only in smokers. Action-based prospective memory was enhanced in both non-smokers and smokers. Non-smokers outperformed smokers in both selective thinking and adaptive thinking (Jansari et al., 2012).

Montgomery et al. (2012) discovered that cannabis users performed worse in JEF[®] than non-users. Cannabis users were also more impaired in the JEF[®] subscales of planning, time-based prospective memory and event-based prospective memory, lending credence to the notion that cannabis usage causes poor prospective memory and executive functioning (Montgomery et al., 2012).

In another study, Montgomery et al. (2011) demonstrated that alcohol intoxication affects different aspects of EF, such as planning, prioritisation, creative thinking and adaptability subscales of JEF[®] (Montgomery et al., 2011). This research also suggests that the impairments may be present at modest doses of alcohol without the subject becoming intoxicated.

Based on previous research, JEF[®] is an ecologically valid assessment that identifies real-world impairments in those who misuse substances or brain damage. The current investigation focused on the role of EF in the relationship between ADHD symptoms and substance misuse. JEF[®] also could help in measuring the daily EF performance of adults with ADHD symptoms in a virtual reality environment. This is the first study to use JEF[®] to assess different facets of EF in adults with ADHD symptoms and substance misuse.

Using a dimensional assessment instead of diagnostic categorisation, the current research attempted to determine which EF facets could increase the risk of developing

alcohol, cannabis and nicotine use and which ADHD symptoms could predict these substance misuses above and beyond the EF facets in a group of undergraduate students.

2.5. Aim of the study

This study had two objectives: (a) to explore the role of different EF facets in the use of alcohol, cannabis and nicotine and whether any of the facets can predict higher levels of alcohol, cannabis and nicotine use in a typically developing university students; and (b) to determine which ADHD symptoms could explain variance in alcohol, cannabis and nicotine use after accounting for EF facets.

According to previous research discussed earlier, it is hypothesized that those who consume alcohol would be impaired in their planning, prioritisation, creativity and adaptability (Montgomery et al., 2011). In addition, those who use cannabis would have impaired planning as well as time-based and event-based prospective memory facets of EF, while those who use nicotine would improve their action-based, time-based and event-based prospective memory facets of EF (Montgomery et al., 2012, Jansari et al., 2012).

In addition, previous studies have shown that people with ADHD symptoms have poorer attention and response inhibition (Malloy-Diniz et al., 2007), working memory (Andersen et al., 2013; Schweitzer et al., 2006) and planning and shifting skills (Rohlf et al., 2012; van Mourik et al., 2005). Therefore, those with ADHD symptoms are hypothesised to be more impaired in response inhibition, working memory, planning and shifting.

Furthermore, as mentioned earlier in this thesis, ADHD symptoms are associated with other deficits in addition to EF impairments, such as risky and impulsive behaviours (Barkley, 1997), emotional dysregulation (Hetchman et al., 2016), mood disorders (Kessler et al., 2006) and poor sleep quality (Díaz-Román et al., 2018; Neto & Nunes, 2017). These

problems are also related to the increasing alcohol, cannabis and nicotine use. Hence, it is supposed that ADHD symptoms would substantially explain the variances in the use of alcohol, cannabis and nicotine than EF deficits. This study is unique in the field since it attempts to find the relationship between alcohol, cannabis and nicotine use, EF facets and each ADHD symptom cluster separately.

Although the results of an investigation by Moehring et al. (2019) have shown that 12.3% of participants in the general population were at risk of risky alcohol misuse after being tested by AUDIT, in another study, 14.9% of undergraduate students yielded a score of 5 or higher in AUDIT, which was a cutoff score in the investigation and indicated risky alcohol use (Abayomi et al., 2013). Davoren et al. (2016) found that among investigations that used AUDIT, 62.8% of the students in 2003 to 84% in 2014 reported harmful alcohol use in the UK. However, the prevalence of substance misuse in the UK shows a decline in the use of alcohol, cannabis and nicotine in this population over the years (NHS Digital, 2018; please refer to Section 1.2). The results of previous studies with university students and non-university student samples show significant differences, which indicates higher levels of risky alcohol use in university students.

Investigators measured the cannabis use of 229 undergraduate students with CUDIT-R and found that 49% of them had cannabis use disorders. Their results indicated a rise in the CUDIT-R scores as individuals' diagnostic severity increased: no diagnosis ($n = 115$, $M = 4.06$, $SD = 3.23$), mild ($n = 54$, $M = 9.52$, $SD = 4.06$), moderate ($n = 31$, $M = 13.52$, $SD = 5.40$) and severe ($n = 27$, $M = 19.11$, $SD = 6.10$) (Schultz et al., 2019). Thus, there is a diverse range of substance misuse scores in different studies around the world, which may be attributed to varying cutoff scores for substance use questionnaires. Other factors that may influence alcohol, cannabis and nicotine use include genetics, social factors and time of the

study conducted (Soder et al., 2019, De Wit & Richards, 2004, Allen et al., 2003, Kim et al., 2017; Song et al., 2020). The percentage of risky alcohol and cannabis use, frequency and the numbers of daily nicotine use and alcohol and cannabis dependence have been measured for the current sample and will be discussed in Section 2.8.

2.6. Methods

2.6.1. Participants

There were 90 adult participants with a mean age of 23.83 years ($SD = 7.33$, range = 18 to 47 years old). Based on the results of the substance misuse questionnaires, five outliers were excluded from the study (more detailed information in the Section 2.7.1). The statistical analysis was conducted on data from 85 participants (72 first year undergraduate university students from the psychology department and 13 students from other departments at the same university), 76.5% (65) of whom were women. This study contained online questionnaires and a face-to-face session. Participants were recruited from Goldsmiths, University of London's Department of Psychology's Research Participation Scheme and received course credits for their participation. Social media networks, such as the student Facebook page, were employed to recruit participants. Additionally, the department notice boards were used for advertising the study (Appendix 16). Any participant with a major psychiatric disorder (e.g. bipolar affective disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder) and/or major physical health problems (e.g. brain injury), those with an age less than 18 years old, and those who cannot read and understand English were excluded. Participants signed an online consent form confirming that they were 18 or older and agreed to participate in the study. All participants completed the measures anonymously.

2.6.2. Procedure

This study was approved by the Department of Psychology's ethics committee at Goldsmiths, University of London (Ethics Reference Numbers: PS230516ZSS; please see Appendix 12 for the approved Ethical Approval Form). This study contained two parts, the first consisted of online questionnaires to measure ADHD symptoms and the use of alcohol, cannabis and nicotine. The second part involved JEF[®] and Go/NoGo tests which are both computer based tasks. The information about the study and the link of it was on the Research Participation Scheme page that was accessible to students at the Department of Psychology of Goldsmiths, University of London and the Facebook page of Goldsmiths, University of London. Participants could email the researcher with any question they had about the study. On the first page of the online section, specific additional details about the number and length of the questionnaires, ethical issues and the study's overall focus were provided. Participants had to fill out an online consent form confirming that they are 18 or older and agreed to be involved in the study; otherwise, they could not answer the questionnaires. Moreover, any question that they did not want to answer could be skipped. At the end of the survey, there was a debrief page with detailed information about the study. They completed the ADHD and substance misuse questionnaires online. After completing the online part, participants were asked to come to the lab to complete the Go/ NoGo task and JEF[®].

2.6.3. Measures

2.6.3.1. The Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993)

This questionnaire was developed as a screening instrument for harmful alcohol use as part of a six-country WHO collaborative project (Australia, Bulgaria, Kenya, Mexico, Norway and the USA). It contains 10 questions regarding domains of alcohol use, drinking behaviour and alcohol-related problems. Questions were chosen from a 150-item original assessment schedule that were administered to 1,888 individuals who joined representative primary health care services. Within the original assessment, there were several scales. Ninety-two per cent of individuals diagnosed with hazardous alcohol use had a total AUDIT score of 8 or higher, while 94% of those with no alcohol misuse had a score of less than 8. Saunders et al. (1993) selected the questions in each scale based on total correlation coefficient, heterogeneity, concerns shown by family and health workers, and the simplest way of asking about alcohol consumption frequency. The questions are based on the participants' drinking habits: 'How often do you have a drink containing alcohol?' or 'How many standard drinks containing alcohol do you have on a typical day when drinking?'. Questions regarding possible alcohol dependence or the likelihood of AUD are also asked: 'During the past year, how often have you failed to do what was normally expected of you because of drinking?' or 'During the past year, how often have you needed a drink in the morning to get yourself going after a heavy drinking session?'. Moreover, this questionnaire inquires about harmful alcohol use: 'Have you or someone else been injured as a result of your drinking?' and 'Has a relative or friend, doctor or other health worker been concerned about your drinking or suggested you cut down?'. Each question has a score ranging from 0 to 4, with a total possible score of 40. A score of 8 or more indicates harmful or hazardous drinking, while a score of 13 or more in women and 15 or more in men denote possible alcohol dependence. Based on the evidence, the first three questions focus on the concept of 'alcohol consumption' and the rest of the questions (4 to 10) reflect 'alcohol use problems'.

This questionnaire is a simple method for early detection of alcohol use problems (please see Appendix 4 for the full questionnaire). The Cronbach alpha for the current study was .80.

2.6.3.2. The Alcohol Use Questionnaire (Mehrabian, Rusell, 1978);

This 12-item questionnaire asks about the weekly amount and type of alcoholic drinks consumed over the past six months (please see Appendix 5 for the full questionnaire).

Questions cover the frequency and quantity of alcohol consumption and the speed of drinking and the number of times the individual has been intoxicated in the past six months.

For instance, it asks, ‘On how many days per week do you drink wine, or any wine type product e.g. sherry, port martini (at least one small glass)?’ or ‘On those days you do drink wine (or similar), about how many glasses (pub measure) do you drink?’. This questionnaire was used by previous investigations, such as Wigg and Stafford (2016), Higgs et al. (2008) and Stafford and Dodd (2013) and showed validity and reliability. The AUQ score is calculated as Item 3 + Item 6 + Item 9 + (4× Item 10) + Item 11 + (0.2 × Item 12). The results showed that both measures were strongly correlated with alcohol consumption and other alcohol use behaviour questions. In the current study, the Cronbach alpha was .75.

2.6.3.3. The Cannabis Use Disorder Identification Test-Revised (CUDIT-R; Adamson et al., 2010); CUDIT-R is the revised version of the original CUDIT.

Adamson et al. (2010) developed the CUDIT-R, which contains eight items covering four domains: consumption, cannabis problems, dependence and psychological features (please see Appendix 6 for the full questionnaire). For instance, the first question is about the individual’s cannabis consumption, which asks, ‘How often do you use cannabis?’ The scores of the answers range from 0 (Never) to 4 (Daily or almost daily), and a total score is obtained by adding the scores of all items. Along with the original CUDIT’s confirmed psychometric suitability, the revised version is shorter and has good psychometric properties.

It also has high sensitivity (91%) and specificity (90%) (Adamson et al. 2010). A score of 8 or higher indicates hazardous cannabis use, while a score of 12 or more suggests possible cannabis use dependence. The Cronbach alpha of this measurement for the current study was .80.

2.6.3.4. The Cannabis Use Questionnaire (CAN; this questionnaire was chosen based on the previous study by Stautz, Dinc and Cooper, 2017);

This is a 9-item measure inquires about the individual's cannabis use (smoked through a pipe, bong (water-filtered pipe) or cannabis cigarette (joint or spliff), baked into food and eaten or made into a tea and drunk; please see Appendix 7 for the full questionnaire). To illustrate, lifetime cannabis use was highlighted through the question, 'Have you ever used cannabis before?', while the frequency of cannabis used over the past year was measured by asking, 'In the last year, on how many separate days have you used cannabis?'. The participant can choose from nine options ranging from 'Never' to 'Daily or nearly daily'. The Cronbach alpha for the current study was .59.

2.6.3.5. The Nicotine Use Questionnaire (Centres for Disease Control and Prevention in the Office on Smoking and Health [CDC/OSH], 1998);

There are four key questions on the current nicotine questionnaire, whose scores are added to obtain the overall nicotine use score. The reasons for only selecting a subset of questions were time and relevance; these four questions were chosen to measure only smoking quantity and frequency. In the current investigation, the following multiple-choice questions reflect nicotine use problems and frequency over the past month: 'About how many cigarettes have you smoked in your entire life?', 'Have you ever smoked cigarettes daily, that is, at least one cigarette every day for 30 days?', 'During the past 30 days, on how many days did you smoke cigarettes?' and 'During the past 30 days, on the days that you

smoked, how many cigarettes did you smoke per day?'. The calculated Cronbach alpha for the current investigation was .78. This Cronbach alpha value shows that this shortened test has an adequate internal consistency estimate of test score reliability.

2.6.3.6. *Go/ No-go task (Kindlon, Mezzacappa, Earls, 1995);*

This is a PEBL (Psychology Experiment Building Language) task that requires executing or inhibiting a motor response. PEBL is a free software system for psychological laboratory testing (Mueller & Piper, 2014). Go/NoGo contains a sequential presentation of letters that begins with a 2×2 array of 4 stars. A single P or R letter is presented in one of the squares for 500 milliseconds and a 1,500-millisecond inter-stimulus interval (Figure 2.1).

The task has two phases. The first phase involves 160 trials in which participants press a button when they see the target letter P and withhold their response to the non-target letter R. The second phase is the opposite of the first, in which the participants respond to the target letter R but not to the letter P. Together, the two phases have 320 trials. The task starts with a brief practise session to ensure that all participants fully comprehend the task. The ratio of target and non-target letters in both conditions are 80:20 each. Behavioural performance of the individual is assessed by calculating four values from the two phases: (a) correct responses to the target letter (Go); (b) errors of omission to the target letter; (c) errors of commission (responding to the non-target letter); (d) correct rejections to the non-target letter. Additionally, reaction and its variability to the target letter are calculated for each participant. Based on previous investigations, Go errors are considered as an indicator of inattention, while No-go errors and reaction to Go responses are indicators of impulsivity (Barkley, 1991; Halperin et al., 1991). In the current study, the number of commission errors (pressing a key when a non-target letter is displayed) was regarded as an indicator of the response inhibition problem.

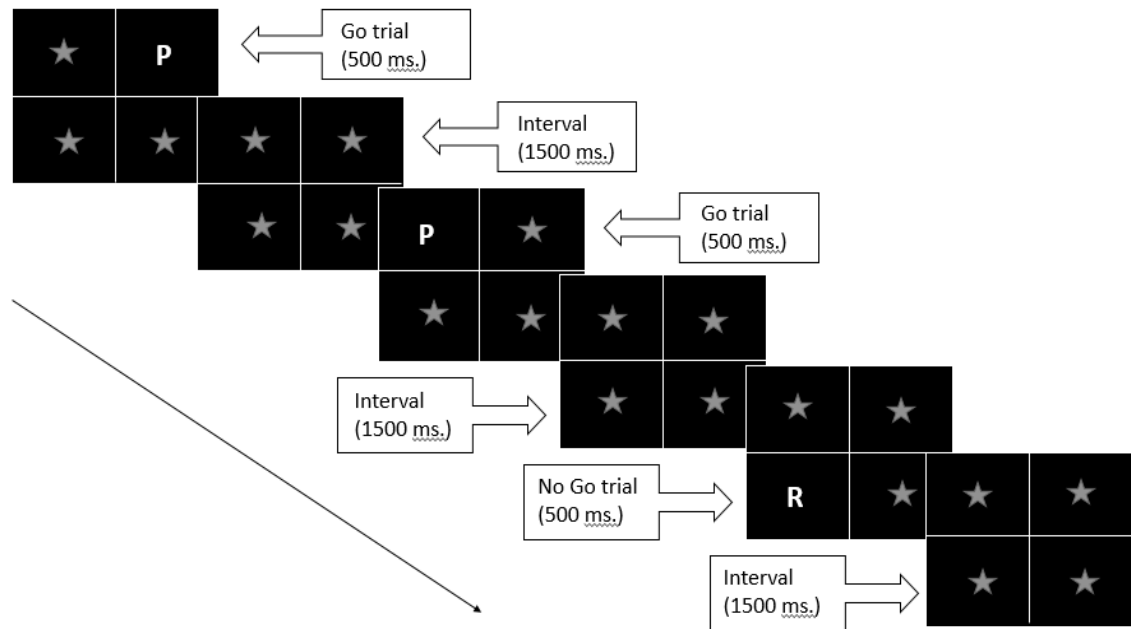


Figure 2.1: The stimulus of the Go/NoGo task. The target letter (P in the first trial and R in the second trial) appears for 500 ms; then, an interval lasting 1500 ms was presented; participants should respond to the target letter as soon as possible after the stimulus appeared and inhibit their respond to the non-target letter.

2.6.3.7. ADHD Self-Report Scale (ASRS; Kessler et al., 2007);

This is an 18 item self-report questionnaire that measures adult ADHD inattentive symptoms and hyperactive/impulsive symptoms. This scale was developed to be consistent with DSM-4 criteria. However, according to Bastiaens and Galus (2018), it performs equally well in identifying individuals with ADHD symptoms based on the DSM-5. Part A assesses inattention symptoms by asking questions such as, ‘How often are you distracted by the activity or noise around you?’. Part B measures hyperactivity/impulsivity symptoms of ADHD by asking queries such as, ‘How often do you interrupt others when they are busy?’. Each part has nine questions. Participants must choose among five answers: ‘Never’, ‘Rarely’, ‘Sometimes’, ‘Often’ and ‘Very often’ (please see Appendix 9 for the full

questionnaire). A score of 0–16 indicates that an individual is unlikely to have ADHD, while 17–23 means likely to have ADHD and 24 or above suggests highly expected to have ADHD (Kessler et al., 2007). Cronbach alpha for ASRS-IA (Part A) for the current investigation was .84 and for ASRS-HI (Part B) was .85.

2.6.3.8. The Jansari assessment of Executive Functions (JEF[®]; Jansari et al., 2014);

This is a 40-minute virtual reality task. It involves an office environment where the participants spend their first day of work. During the JEF[®] testing session, the scenario is given to participants to become familiar with their role, the virtual environment, and how to navigate it. They are provided a list of tasks titled ‘Manager’s tasks for completion’, which they must complete for the office manager, who is away and unable to oversee their work. This list includes arrangements for collecting certain items from the post, setting up chairs and tables for the meeting and turning on the coffee machine when the first person arrives. Besides the tasks enumerated at the start of the testing session, there will be a number of virtual and hard copy memos given mid-session that will require participants to perform additional tasks or amend a current one. There are two rooms: one is the participant’s office (Figure 2.2), and the other is the meeting room (Figure 2.3). All the activities must be completed in these rooms, which are linked by a corridor. JEF[®] examines eight aspects of EF concurrently: planning, prioritisation, selective thinking, adaptive thinking, creative-thinking, as well as action-based, event-based and time-based prospective memory. Each construct is measured by two tasks.

For ‘planning’, the participant is expected to arrange objects and events in a logical order, but not in order of importance. The two tasks include (a) writing a plan of action based on the ‘manager’s tasks for completion’ list and (b) arranging the furniture in the meeting room. In ‘prioritisation’, the participant orders events based on their importance. The two

tasks involve organising (a) the order of certain agenda topics that will be discussed in the meeting and (b) the order of the cleaner's jobs. For 'selective thinking', choosing between two or more options based on acquired knowledge is required. The participant must determine (a) the post company that should send a letter or parcel based on its size and importance and (b) the company that should send the parcel signalled by Memo 3. To engage in 'creative thinking', the participant finds solutions to problems using methods that are not obvious or specified. The two tasks involve (a) covering the graffiti on the whiteboard, which is in permanent ink and (b) repairing the leaking crack in the ceiling above the coffee machine. For 'adaptive thinking', the participant is required to regain goals when the conditions of success are changed. They must (a) replace the broken overhead projector and (b) send the port through another means in the absence of a company postman. In 'action-based prospective memory', the participant is asked to remember a task cued by a stimulus connected to an action that the participant is already performing. This involves (a) updating the post diary after sending each post and (b) recording any equipment breakage on a note for the manager. For 'event-based prospective memory', the participant should remember to do a task cued by an external stimulus. They must (a) record fire alarms during the session and (b) turn on the coffee machine before the first guest arrives. In 'time-based prospective memory', the participant must remember to perform a task at a specific time in the future. They should (a) turn on the overhead projector 10 minutes before the meeting and (b) make a note for the manager whether or not the company postman arrived at a specific time.

Based on previous investigations by Jansari et al., a set of scoring criteria has been developed. A score of 0 equals no attempt or unacceptable performance. A score of 1 reflects satisfactory performance, while a score of 2 denotes excellent or perfect performance. The overall score is reached by adding the scores from each subscale. Each raw score is converted

to a percentage for that construct, and the percentages are then averaged for the final total performance. The Cronbach alpha for this measurement was .67.



Figure 2.2: Screen Capture of Virtual Reality Office Environment (©Ashok Jansari, September 2014).



Figure 2.3: Screen Capture of Virtual Reality Meeting Room Environment (©Ashok Jansari, September 2014)

2.6.4. Data analysis strategy

Data were analysed using IBM SPSS version 20. Pearson correlation was conducted to examine the connection between the variables of the study and the results are presented in Table 2.5. Hierarchical linear regression was carried out to find out, if different facets of EF can predict alcohol and nicotine use and, to explore whether the symptoms of ADHD can explain more variance in alcohol and nicotine use problems after accounting for all EF facets. An alpha level of $p = .05$ was used for significance testing.

Due to the high number of zero scores in CUDIT-R and CAN questionnaires among 85 participants of this study, the scores of these tests were assessed as dichotomous values. Participants were asked if they use cannabis or not, which showed if they are current cannabis users and in another question, they were asked if they have ever used cannabis,

which indicated their lifetime cannabis use. Those who answered yes got the value of 1 and those with no cannabis use got 0 value. Therefore, in summary, hierarchical linear regression was used for alcohol and nicotine use while logistic regression was used for cannabis use. Odd ratios are presented, which reflects the change in the likelihood of usage with one standard deviation increase in the predictor.

2.7. Results

2.7.1. Outliers and multi-collinearity

First the outliers in the data were checked. To find out if there were outliers in the data of the current study or not, the maximum and the minimum values in the Residual Statistics table of regression analysis were checked. The minimum value of ≤ -3.29 or a maximum value of ≥ 3.29 shows that there are outliers in the data (Tabachnick & Fidell, 2013). Standardized residual values for each of the participants were checked. Based on the scores of ADHD and substance misuse questionnaires, five participants were identified as outliers with scores of more than 3.29 on alcohol, cannabis and nicotine use questionnaires. After deleting any value equal to or above 3.29 or equal to or below -3.29 as the outliers, an analysis of standard residuals was carried out again, which showed that the data contained no outliers. Tests to see if the data met the assumption of collinearity indicated that multi-collinearity was not a concern (VIF <10 and the Tolerance >0.1). Based on the kurtosis and skewness of the AUDIT (kurtosis = .50, skewness = 1.02), the scores were square rooted (SQRT (AUDIT+1)) and the AUDIT was used in the analysis (kurtosis = -.97, skewness = .401). The scores of AUQ (kurtosis = 1.7, skewness = .92) and NIC (kurtosis = 1.03, skewness = 1.11) were also Log transformed (LG10 (AUQ or NIC + 1) and AUQ (kurtosis =

-.32, skewness = -.85) and NIC (kurtosis = .51, skewness = .16) was calculated and used in the analysis.

2.7.2. The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

The percentage of alcohol, cannabis, and nicotine use frequency and quantity are presented in Table 2.2. In AUDIT, the frequency of alcohol use was asked using the question “How often do you have a drink containing alcohol?”. The participant could choose between 5 options and the percentage of each answer is presented in the table below. The quantity of alcohol use was asked by “How many standard drinks containing alcohol do you have on a typical day when drinking?” and there were 5 options, which are shown in the same table. Based on the results, 27.1% of participants showed hazardous alcohol use by gaining a score of 8 or more in AUDIT. In addition, 6.2% of women had a score of 13 or above indicating alcohol use dependence, while none of the men had a score of 15 or more, indicating no alcohol dependence in men.

In AUQ, the frequency of getting drunk after drinking alcohol was measured by “How many times have you been drunk in the last 6 months?”. Data showed that 67.1% of participants have never been drunk in the last 6 months. In addition, the frequency of wine or wine type products, beer or cider and spirits were measured, which are presented in Table 2.3.

In CUDIT-R, cannabis frequency and quantity were measured by asking the participants “How often do you use cannabis?” and “How many hours were you stoned when

you had been using cannabis?” and the the results show that 83.5% of the participants have never used cannabis and 2.4% use 4 or more times a week. Additionally, 87.1% of the participants have never been stoned but 1.2% of them were stoned for 5 to 6 hours when they used cannabis (Table 2.2). Based on the score of 8 or more for hazardous cannabis use and 12 or more for cannabis use disorder as the cut off for this questionnaire, 6% of participants had hazardous cannabis use and cannabis dependence was among 2.4% of the participants. In CAN, lifetime cannabis use was measured by asking “Have you ever used cannabis in your life?” and the frequency of cannabis use in the last year was measured by asking “In the last year, on how many separate days have you used cannabis?”. The latter could choose between nine options ranging from Never (0) to Daily or nearly daily (7). Results show that 36.5% of the participants had used cannabis in their lives and 5.6% of them had used cannabis daily or nearly daily over the past year (Table 2.2).

In NIC, nicotine frequency was measured by asking “During the past 30 days, on how many days did you smoke cigarettes?” and the nicotine quantity was asked by the question “During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?” and the results show that 9.4% of participants smoked all 30 days of the last month and 1.2% of them smoked more than 20 cigarettes per day (Table 2.2).

As mentioned in Chapter 1, gaining a score of 24 or above on ASRS shows that the individual is highly likely to have ADHD. In this study, 25% of the university students had this score.

Table 2.2
The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

| | Frequency | Percent | Hazardous use (%) | Dependence (%) |
|--------------|-----------|---------|-------------------|------------------------|
| AUDIT | | | 27.1 | Women = 6.2 Men = 0 |

| | | |
|---|----|----------------------------|
| AUDIT frequency | | |
| (How often do you have a drink containing alcohol?) | | |
| Never | 29 | 34.1 |
| Monthly or less | 19 | 22.4 |
| 2-4 times a month | 15 | 17.6 |
| 2-3 times a week | 14 | 16.5 |
| 4 or more times a week | 8 | 9.4 |
| AUDIT quantity | | |
| (How many standard drinks containing alcohol do you have on a typical day when drinking?) | | |
| 1 or 2 | 47 | 55.3 |
| 3 or 4 | 20 | 23.5 |
| 5 or 6 | 9 | 10.6 |
| 7 to 9 | 1 | 1.2 |
| 10 or more | 0 | 0 |
| CUDIT-R | | 6 2.4 |
| CUDIT-R frequency | | |
| (How often do you use cannabis?) | | |
| Never | 71 | 83.5 |
| Monthly or less | 8 | 9.4 |
| 2-4 times a month | 4 | 4.7 |
| 2-3 times a week | 0 | 0 |
| 4 or more times a week | 2 | 2.4 |
| CUDIT-R quantity | | |
| (How many hours were you stoned when you had been using cannabis?) | | |
| Less than 1 | 74 | 87.1 |
| 1 or 2 | 6 | 7.1 |
| 3 or 4 | 4 | 4.7 |
| 5 or 6 | 1 | 1.2 |
| 7 or more | 0 | 0 |
| CAN | | |
| Lifetime cannabis use | | |
| (Have you ever used cannabis in your life?) | | |
| Yes | 31 | 36.5 |
| No | 54 | 63.5 |
| Cannabis quantity in the last year | | |
| (In the last year, on how many separate days have you used cannabis?) | | |
| Never | 65 | 76.5 |
| Once every 12 months | 1 | 1.2 |
| Once every 2 to 3 months | 4 | 4.7 |
| Once a month | 0 | 0 |
| 2 to 3 times a month | 1 | 1.2 |
| Once a week | 2 | 2.4 |
| 2 to 3 times a week | 2 | 2.4 |
| 4 to 5 times a week | 5 | 5.9 |
| Daily or nearly daily | 5 | 5.9 |
| NIC | | |

NIC frequency

(During the past 30 days, on how many days did you smoke cigarettes?)

| | | |
|---------------|----|------|
| 0 days | 67 | 78.8 |
| 1 to 2 days | 3 | 3.5 |
| 3 to 5 days | 0 | 0 |
| 6 to 9 days | 1 | 1.2 |
| 10 to 19 days | 2 | 2.4 |
| 20 to 29 days | 4 | 4.7 |
| All 30 days | 8 | 9.4 |

NIC quantity

(During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?)

| | | |
|--|----|------|
| I did not smoke cigarettes during the past 30 days | 68 | 80.0 |
| Less than 1 cigarette per day | 1 | 1.2 |
| 1 cigarette per day | 1 | 1.2 |
| 2 to 5 cigarettes per day | 7 | 8.2 |
| 6 to 10 cigarettes per day | 6 | 7.1 |
| 11 to 20 cigarettes per day | 1 | 1.2 |
| More than 20 cigarettes per day | 1 | 1.2 |

Note: AUDIT = Alcohol Use Identification Test; CUDIT-R = Cannabid Use Identification Test-Revised; NIC = Nicotine Use Test

Table 2.3
The frequency of wine and wine type products, beer or cider and spirits

| Days in a week | Wine | | Beer or cider | | Spirits | |
|----------------|-----------|------|---------------|------|-----------|------|
| | Frequency | % | Frequency | % | Frequency | % |
| 0 | 51 | 60 | 59 | 69.4 | 60 | 70.6 |
| 1 | 19 | 22.4 | 14 | 16.5 | 20 | 23.5 |
| 2 | 7 | 8.2 | 1 | 1.2 | 3 | 3.5 |
| 3 | 4 | 4.7 | 1 | 1.2 | 2 | 2.4 |
| 4 | 1 | 1.2 | 8 | 9.4 | 0 | 0 |
| 5 | 1 | 1.2 | 1 | 1.2 | 0 | 0 |
| 6 | 0 | 0 | 1 | 1.2 | 0 | 0 |
| 7 | 2 | 2.4 | 0 | 0 | 0 | 0 |

2.7.3. Correlation between variables

Table 2.4 shows the descriptive statistics of all the scores of the study. In the current study, men were coded ‘1’ and women were coded ‘2’. The participants comprised of 31 (36.5%) lifetime cannabis users and 54 (63.5%) non-lifetime cannabis users and 16.5% of the sample were current cannabis users.

Pearson correlation was calculated between the variables and results demonstrated that age was significantly negatively correlated with adaptive thinking ($r = -.25, n = 76, p = .03$) and time based prospective memory ($r = -.25, p = .03$) constructs of JEF[®], response inhibition of Go/ No-Go ($r = -.28, p = .01$) and alcohol use ($r = .352, n = 69, p = .003$).

Analyzing the correlation between the symptoms of ADHD and EF showed that hyperactivity/impulsivity symptoms of ADHD and creative thinking construct of JEF[®] ($r = -.22, n = 85, p = .04$) were negatively correlated. Inattention symptoms of ADHD were significantly negatively correlated with selective thinking construct of JEF[®] ($r = -.31, n = 83, p = .003$), and response inhibition ($r = .30, n = 83, p = .01$). Alcohol use was significantly positively correlated with both hyperactivity/impulsivity ($r = .27, n = 76, p = .02$) and

inattention ($r = .25, n = 75, p = .03$). There was also a statistically significant positive correlation between nicotine use and hyperactivity/impulsivity ADHD symptom ($r = .21, n = 85, p = .05$). Cannabis use was not correlated with ADHD symptoms. Tables 2.4 and 2.5 shows more detailed descriptive statistics, demographic information and correlation between variables of this study.

Table 2.4
Descriptive statistics and demographic information

| | <u><i>n</i></u> | <u>Range</u> | <u>Minimum</u> | <u>Maximum</u> | <u>Mean</u> | <u>Std. Deviation</u> |
|--------------------------------------|-----------------|--------------|----------------|----------------|-------------|-----------------------|
| Age | 76 | 29 | 18 | 47 | 23.83 | 7.33 |
| JEF [©] _TOTAL | 85 | 50 | 50 | 100 | 75.72 | 12.12 |
| JEF [©] -Planning | 85 | 95 | 17 | 100 | 68.68 | 27.62 |
| JEF [©] -Prioritization | 85 | 75 | 25 | 100 | 83.53 | 19.50 |
| JEF [©] -Selective thinking | 85 | 100 | 0 | 100 | 85.29 | 21.23 |
| JEF [©] -Creative-thinking | 85 | 100 | 0 | 100 | 57.68 | 34.45 |
| JEF [©] -Adaptive-thinking | 85 | 100 | 0 | 100 | 58.53 | 32.86 |
| JEF [©] -Action BPM | 85 | 50 | 50 | 100 | 85.59 | 20.91 |
| JEF [©] -Event BPM | 85 | 50 | 50 | 100 | 90.29 | 17.29 |
| JEF [©] -Time BPM | 85 | 75 | 25 | 100 | 76.18 | 23.11 |
| Go_No Go | 85 | 28 | 0 | 28 | 10.82 | 6.63 |
| ADHD_HI | 85 | 25 | 2 | 27 | 13.21 | 5.85 |
| ADHD_IA | 83 | 31 | 2 | 33 | 16.23 | 5.82 |
| ADHD_TOTAL | 83 | 55 | 5 | 60 | 29.34 | 10.57 |
| AUDIT | 76 | 20 | 0 | 20 | 4.76 | 4.1 |
| AUDIT_trans | 76 | 3.58 | 1.00 | 4.58 | 2.1740 | 1.02 |
| AUQ_trans | 82 | 1.92 | 0 | 1.92 | 1.0711 | .53 |
| Nic_trans | 85 | 20 | 0 | 1.32 | 5.11 | 6.15 |

Note: JEF[©]_TOTAL = Jansari assessment of Executive Function; Action BPM = Action-based prospective memory; Event-BPM = Event based prospective memory; Time-BPM = Time based prospective memory; Go_No Go = Response inhibition task; ASRS_TOTAL = Adult ADHD Self-report Scale; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire (alcohol frequency);

Table 2.5
Pearson correlation between ADHD, EF and substance misuse variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | |
|---|--------|-------|-------|-------|------|--------|-------|-------|------|--------|------|-------|--------|--------|-------|--------|-------|----|--|
| 1.Age | 1 | | | | | | | | | | | | | | | | | | |
| 2.Gender | -.32** | 1 | | | | | | | | | | | | | | | | | |
| 3.JEF ^o _total | -.16 | .12 | 1 | | | | | | | | | | | | | | | | |
| 4. JEF ^o _planning | .00 | -.04 | .50** | 1 | | | | | | | | | | | | | | | |
| 5. JEF ^o _prioritization | .00 | .10 | .32** | .21 | 1 | | | | | | | | | | | | | | |
| 6. JEF ^o _selective thinking | -.06 | -.12 | .42** | .12 | .00 | 1 | | | | | | | | | | | | | |
| 7. JEF ^o _creative-thinking | .06 | .02 | .50** | .11 | .00 | .03 | 1 | | | | | | | | | | | | |
| 8. JEF ^o _adaptive-thinking | -.25* | .10 | .72** | .10 | .02 | .16 | .35** | 1 | | | | | | | | | | | |
| 9. JEF ^o _actionBPM | -.11 | .05 | .21 | -.02 | .21* | -.06 | -.15 | .04 | 1 | | | | | | | | | | |
| 10. JEF ^o _eventBPM | -.05 | .29** | .42** | .08 | -.15 | .15 | .10 | .25* | .00 | 1 | | | | | | | | | |
| 11. JEF ^o _timeBPM | -.25* | .15 | .68** | .28** | .13 | .35** | -.01 | .54** | .03 | .38** | 1 | | | | | | | | |
| 12.Go_Nogo | -.28* | .05 | -.22 | -.08 | .11 | -.14 | -.21* | -.10 | .16 | -.30** | -.20 | 1 | | | | | | | |
| 13.ADHD_total | -.05 | .16 | -.16 | -.02 | .19 | -.24* | -.21 | -.15 | .03 | -.09 | -.03 | .27* | 1 | | | | | | |
| 14. ADHD_IA | -.06 | .12 | -.20 | -.03 | .20 | -.32** | -.17 | -.16 | .02 | -.14 | -.11 | .30** | .902** | 1 | | | | | |
| 15. ADHD_HI | -.01 | .12 | -.12 | -.01 | .16 | -.11 | -.23* | -.12 | .04 | -.03 | .04 | .18 | .904** | .631** | 1 | | | | |
| 16.AUDIT_NEW | .35** | -.27* | .01 | .06 | .03 | .09 | .00 | .03 | .00 | -.08 | -.17 | .13 | .285* | .254* | .274* | 1 | | | |
| 17.AUQ_NEW | .17 | -.04 | .09 | -.07 | -.03 | .11 | .06 | .20 | -.04 | .13 | .09 | -.08 | .145 | .139 | .108 | .514** | 1 | | |
| 18.Nic_NEW | .23 | -.17 | .07 | .00 | .14 | .09 | .11 | -.01 | .04 | -.11 | -.03 | .12 | .209 | .145 | .215* | .642** | .268* | 1 | |

Note: JEF_TOTAL = Jansari assessment of Executive Function; Action BPM = Action based prospective memory; Event BPM = Event based prospective memory; Time BPM = Time based prospective memory; Go_No Go = Response inhibition task; ASRS_TOTAL = Adult ADHD Self-Report Scale; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire (alcohol frequency); CUDIT-R = Cannabis Use Identification Test; CAN-FREQ = Cannabis Use Frequency.

**Correlation is significant at the 0.001 level (2 tailed) / * Correlation is significant at the 0.05 level (2 tailed)

2.7.4. The role of EF in the relationship between ADHD symptoms and alcohol use

The results of hierarchical regression showed that in the first step, age positively predicted AUDIT significantly ($F(2, 65) = 4.75, p < .05; \beta = 0.28$) and accounted for 13% of the variation in AUDIT scores ($R^2 = .13, F \text{ change} = 4.75, p = .01$). In step 2, adding EF facets to the regression model explained an additional 20% of the variation in AUDIT ($R^2 = .2, F \text{ change} = 1.83, \text{Sig } F \text{ change} = .08$). Among different constructs of EF, adaptive thinking ($F(11, 56) = 2.46, p < .05; \beta = 0.31$), time based prospective memory ($\beta = 0.33$) and response inhibition ($\beta = 0.33$) predicted AUDIT score significantly. This means that lower scores in adaptive thinking and time based prospective memory and more commission errors as poor response inhibition predicted higher scores in AUDIT. The ASRS-HI score of the ADHD questionnaire (ASRS) explained additional variance in AUDIT significantly after accounting for EF facets. It accounted for 12% of the variation ($R^2 = .45, F \text{ change} = 11.85, p = .001, \beta = .39$). These results indicated that the individuals with a higher score on ASRS-HI also had more AUDIT score after accounting for EF facets.

The facets of EF could not predict AUQ in a significant way. The two subscales of ADHD also could not predict AUQ above and beyond the EF facets, but the total score of ASRS was the only significant predictor and accounted for 7.3% of the variance in AUQ score ($R^2 = .18, F \text{ change} = 5.17, p = .027, \beta = .30$) (Table 2.6 & 2.7).

Table 2.6
Hierarchical regression model predicting AUDIT scores from ADHD symptoms and EF.

| Step | Predictor | R^2 | R^2 change | β (Standardized Coefficient) |
|------|-----------|-------|--------------|------------------------------------|
| 1 | Age | .13 | .13 | -.28* |

| | | | | |
|---|--|-----|-----|-------|
| | Gender | | | -0.16 |
| 2 | JEF [®] _Planning | .33 | .20 | .33 |
| | JEF [®] _Prioritization | | | .01 |
| | JEF [®] _Selective- thinking | | | .10 |
| | JEF [®] _Creative- thinking | | | .09 |
| | JEF [®] _Adaptive- thinking | | | .31* |
| | JEF [®] _Action BPM | | | .32 |
| | JEF [®] _Event BPM | | | -.03 |
| | JEF [®] _Time BPM | | | .33* |
| | Go/ No Go | | | .33* |
| 3 | ASRS_HI | .44 | .12 | .39** |
| | ASRS-IA | .43 | .10 | .38 |

Note: AUDIT = Alcohol Use Disorder Identification Test; ASRS_HI = Hyperactivity/impulsivity score of Adult ADHD Self-Report Scale; ASRS-IA = inattention score of Adult ADHD Self-Report Scale. Go/ No Go= response inhibition task.
* $p < .05$ / ** $p < .001$

Table 2.7

Hierarchical regression model predicting AUQ scores from ADHD symptoms and EF.

| Step | Predictor | R^2 | R^2 change | β (Standardized Coefficient) |
|---------|--------------------------------------|---------|--------------|------------------------------------|
| 1 | Age | .02 | .02 | .15 |
| | Gender | | | .00 |
| 2 | JEF [©] _Planning | .11 | .09 | -.11 |
| | JEF [©] _Prioritization | | | -.03 |
| | JEF [©] _Selective-thinking | | | .10 |
| | JEF [©] _Creative-thinking | | | -.00 |
| | JEF [©] _Adaptive-thinking | | | .09 |
| | JEF [©] _Action BPM | | | .08 |
| | JEF [©] _Event BPM | | | .09 |
| | JEF [©] _Time BPM | | | .03 |
| | Go/ No Go | | | .24 |
| | 3 | ASRS_HI | .19 | .07 |
| ASRS_IA | | .17 | .05 | .28 |

Note: AUQ = Alcohol Use Questionnaire; ASRS_HI = Hyperactivity/impulsivity score of Adult ADHD Self-Report Scale; ASRS-IA = inattention score of Adult ADHD Self-Report Scale; Go/ No Go= response inhibition task.

* $p < .05$ / ** $p < .001$

2.7.5. The role of EF in the relationship between ADHD symptoms and nicotine use

Hierarchical regression results demonstrate that age and gender accounted for 9.5% of the variation in nicotine use significantly ($F(2, 71) = 3.73, p < .05$). Adding EF facets to the model explained an additional 14.7% of the variation in nicotine use, which was not statistically significant, but response inhibition facet of EF predicted nicotine use in a significant way ($\beta = 0.25; t(84) = 2.04; p < .05$). This indicated that those with more errors on

the response inhibition task had more nicotine use problems. Introducing ADHD symptoms to the regression model explained an additional 8% of the variation in nicotine use significantly above and beyond the EF facets. Hyperactivity/impulsivity symptoms of the ADHD was the variable that predicted nicotine use significantly ($\beta=0.3$; $t(84) = 2.13$; $p<.05$) (Table 2.8). Participants with higher ASRS-HI symptoms had more nicotine use after accounting for EF facets.

Table 2.8
Hierarchical regression model predicting nicotine scores from ADHD symptoms and EF.

| Step | Predictor | R^2 | R^2 change | β (Standardized Coefficient) |
|---------|--------------------------------------|-------|--------------|------------------------------------|
| 1 | Age | .09 | .09 | .19 |
| | Gender | | | -.19 |
| 2 | JEF [©] _Planning | .24 | .15 | -.08 |
| | JEF [©] _Prioritization | | | .19 |
| | JEF [©] _Selective-thinking | | | .09 |
| | JEF [©] _Creative-thinking | | | .12 |
| | JEF [©] _Adaptive-thinking | | | .06 |
| | JEF [©] _Action BPM | | | .03 |
| | JEF [©] _Event BPM | | | -.05 |
| | JEF [©] _Time BPM | | | -.06 |
| | Go/ No Go | | | .25* |
| | 3 | | | ASRS_IA |
| ASRS_HI | | .30* | | |

Note: Nicotine= nicotine use questionnaire; ASRS_HI = Adult ADHD Self-Report Scale hyperactivity/impulsivity score; ASRS_IA = Adult ADHD Self-Report Scale Inattention score; Go/ No Go= response inhibition task.

* $p < .05$ / ** $p < .001$

2.7.6. The role of EF in the relationship between ADHD symptoms and cannabis use

Logistic regression results in predicting current cannabis use indicated that the addition of age and gender in the first block ($\chi^2 = 3.79, p > .05$ with $df = 2$) did not improve fit over the null model significantly and they explained 8% of the variation in cannabis use, which was not statistically significant. Adding EF facets in the second block ($\chi^2 = 16.25, p > .05$ with $df = 11$) did not improve fit over the null model and explained 38.2% of the variation in the outcome, which was not statistically significant. Adaptive thinking and response inhibition were the two facets that contributed to the prediction model significantly so, a one-unit increase in adaptive thinking score increased the odds of cannabis use 1.04 times and one-unit increase in response inhibition score increased the odds of cannabis use 1.17 time when other variables are controlled. In the third block, adding ADHD symptoms did not improve fit over the null model significantly (Table 2.9).

Another logistic regression analysis was conducted to predict lifetime cannabis use using age and gender, EF facets and ADHD symptoms as predictors. Age and gender were not significant predictors for lifetime cannabis use ($\chi^2 = 2.09, p > .05$ with $df = 2$). Adding EF facets did not improve fit over the null model ($\chi^2 = 10.38, p > .05$ with $df = 11$) in a significant way. Creative-thinking was the only facet of EF that could contribute to the prediction model significantly and the results showed that when one-unit increase occurred in creative-thinking, the odd ratio is 1.18 times as large and therefore the person is 1.18 times more likely to use cannabis in life. ADHD symptoms did improve fit over the null model significantly ($\chi^2 = 8.96, p > .05$ with $df = 2$). Nagelkerke's R^2 of 0.31 showed a moderate relationship between ADHD symptoms and lifetime cannabis use. The Wald criterion

indicated that hyperactivity/impulsivity symptoms of ADHD made a significant contribution to prediction (Wald= 6.19, $p < .05$). The results showed that a one-unit rise in hyperactivity was associated with 1.19 times greater odds of lifetime cannabis (Table 2.10).

Table 2.9

Logistic Regression Models Predicting CUDIT-R from ADHD Symptoms and EF.

| | Block 1 | Block 2 |
|--|---------------|---------------|
| Likelihood Ratio Test χ^2 | 12.78 | 1.31 |
| Nagelkerke's Pseudo R2 | .23 | .25 |
| Independent Variable (Standardized) | ExpB (95% CI) | ExpB (95% CI) |
| JEF [©] _Planning | 1.01 | 1.01 |
| JEF [©] _Prioritization | 1.01 | 1.01 |
| JEF [©] _Selective-thinking | 1.02 | 1.03 |
| JEF [©] _Creative-thinking | 1.01 | 1.01 |
| JEF [©] _Adaptive-thinking | 1.02* | 1.02* |
| JEF [©] _Action BPM | .99 | .99 |
| JEF [©] _Event BPM | 1.02 | 1.01 |
| JEF [©] _Time BPM | .97 | .97 |
| Go/ NoGo | 1.12* | 1.12* |
| ASRS_IA | | .96 |
| ASRS_HI | | 1.08 |

Note: CUDIT-R= Cannabis Use Disorder Identification Test; Action BPM= Action based prospective memory, Event BPM= Event based prospective memory, Time BPM= Time based prospective memory; GO/ NoGo = Response inhibition task; ASRS_IA= Inattention subscale of adult ADHD Self-Report Scale; ASRS_HI= Hyperactivity/impulsivity subscale of adult ADHD Self-Report Scale.

* $p < .05$ / ** $p < .00$

Table 2.10

Logistic Regression Models Predicting life time cannabis use from ADHD Symptoms and EF.

| | Block 1 | Block 2 |
|--|---------------|---------------|
| Likelihood Ratio Test χ^2 | 7.57 | 7.18 |
| Nagelkerke's Pseudo R2 | .12 | .22 |
| Independent Variable (Standardized) | ExpB (95% CI) | ExpB (95% CI) |
| JEF [®] -Planning | 1.01 | 1.01 |
| JEF [®] -Prioritization | 1.00 | 1.00 |
| JEF [®] -Selective-thinking | 1.02 | 1.02 |
| JEF [®] -Creative-thinking | 1.02* | 1.02* |
| JEF [®] -Adaptive-thinking | 1.00 | 1.01 |
| JEF [®] -Action BPM | 1.00 | 1.00 |
| JEF [®] -Event BPM | .99 | .98 |
| JEF [®] -Time BPM | 1.00 | .99 |
| Go/ NoGo | 1.03 | 1.02 |
| ASRS_IA | | .97 |
| ASRS_HI | | 1.15* |

Note: CUDIT-R= Cannabis Use Disorder Identification Test; Action BPM= Action based prospective memory, Event BPM= Event based prospective memory, Time BPM= Time based prospective memory; GO/ NoGo = Response inhibition task; ASRS_IA= Inattention subscale of adult ADHD Self-Report Scale; ASRS_HI= Hyperactivity/impulsivity subscale of adult ADHD Self-Report Scale.

* $p < .05$ / ** $p < .001$

2.8. Discussion

This study aimed to examine which EF facets predict alcohol, cannabis and nicotine in typically developing adults. A secondary purpose was to assess whether inattention or hyperactivity/impulsivity explains additional variance in alcohol, cannabis and nicotine use after accounting for EF facets. The results of this investigation showed that age is a negative, significant predictor of alcohol misuse, implying that there is a negative correlation between the participants' age and alcohol consumption, indicating that by getting older, there would be a decline in alcohol misuse. This is consistent with previous research that found first-year

undergraduates imbibe more alcohol than second- and third-year undergraduates (Bewick et al., 2008). Using JEF[®] and the Go/NoGo task revealed that response inhibition, adaptive thinking, and time-based prospective memory were the three EF facets that predicted alcohol misuse scores of the participants.

Based on the outcomes of the present study and previous investigations, inhibition of behaviour is one of the most essential functions that require behaviour regulation (Diamond, 2013). *Response inhibition* is an individual's ability to inhibit their impulses and responses to stimuli and select a more suitable reaction and behaviour to attain their goals (Diamond, 2013; Ilieva et al., 2015). Research indicate that self-control is an important aspect of inhibitory control, which is impaired in people with ADHD and those who misuse substances (Diamond, 2013; Koob & Volkow, 2010; Malenka et al., 2009). In a 4-year follow-up study, Rubio et al. (2008) discovered that heavy drinkers show greater impairment than controls in the stop-signal task, and this poor response inhibition predicts their degree of alcohol use. Moreover, Weafer et al. (2010) found an association between response inhibition deficits, poor attention inhibition and high risk-taking, and the frequency and quantity of alcohol use in two groups of adults with and without ADHD symptoms. It can be concluded that those with poor self-control show more impaired response inhibition in situations that involve alcohol or other drugs.

The findings of this study also denoted that there is a connection between alcohol consumption and adaptive thinking; this JEF[®] subscale needs *cognitive flexibility* (Jansari et al., 2014), which has been defined as the ability to respond to environmental changes by shifting attention between unrelated tasks (Myake et al., 2000). These results are consistent with previous investigations with people who consume alcohol, which revealed that the adaptive thinking subscale of JEF[®] is a significant predictor of alcohol consumption in those

with an alcoholic drink (0.4 g/kg) relative to individuals with a placebo (Montgomery et al., 2011). However, the study of Montgomery et al. (2011) has shown that creative thinking, prioritisation and event-based prospective memory are impaired in those with a dose of 0.4 g/kg alcohol among 40 participants aged 18 to 25 years. These three facets of EF were not impaired in the participants of the current investigation. This may be because Montgomery et al. (2011) measured the effect of modest doses of alcohol in participants to determine the immediate impacts, while in the current study, participants were not taking any alcoholic drink before or during the task.

Although previous researchers such as George et al. (2005) showed that acute alcohol use negatively affects working memory in social drinkers, the present study found that the time-based prospective memory subscale of JEF[®] specifically predicts higher alcohol misuse in participants. Thus, response inhibition, adaptive thinking and time-based prospective memory were statistically significant predictors of alcohol use in the current research.

An association between adaptive thinking (which requires cognitive flexibility), memory and response inhibition has been proposed by investigators (Diamond, 2013). In other words, Diamond (2013) expanded the definition of cognitive flexibility, which is the ability to change spatial or inter-personal perspectives. To do so, a person must inhibit the previous perspective and create a new one in memory. This means that cognitive flexibility depends on inhibition control and working memory. Studies using fMRI found various brain parts that are involved in cognitive flexibility (Laber et al., 2008). These brain regions, including the PFC, basal ganglia, ACC and posterior parietal cortex, are also involved in working memory and inhibition control, supporting the idea that cognitive flexibility is influenced by working memory and inhibition control (Miyake et al., 2000). These findings can explain the results of the current study. According to Diamond (2013), people with more

inhibition deficits and poorer memory are less capable of inhibiting a previous perspective and creating a new one in order to have a flexible response to a new situation, which predicts their use of alcohol. Longitudinal investigations are required for future studies to establish the exact cause-and-effect relationship between these EF facets and alcohol use.

In the current investigation, the hyperactivity/impulsivity added to the variance of AUDIT and AUQ scores above and beyond the three EF facets, which were measured by JEF[®]. This means that the model significantly improved the proportion of explained variance in the alcohol use score based on the hyperactivity/impulsivity score of ADHD symptoms. Hence, after accounting for impaired cognitive flexibility, response inhibition, and prospective memory, hyperactivity/impulsivity added to alcohol consumption variance over and beyond these EF facets. Previous investigations showed that university students with ADHD symptoms misuse drugs and alcohol at a higher rate than their peers without ADHD symptoms (DuPaul et al., 2009; Murphy et al., 2002). The present study found that the hyperactivity/impulsivity predict higher levels of alcohol use in university students through pathways other than EF deficits, which will be explored in Chapter 3.

In this study, age and gender were also significant positive predictors of nicotine use. The only EF facet that predicted nicotine use in participants was response inhibition. To measure response inhibition, the stop-signal task was used in previous investigations. Studies using this task indicated that longer stop-signal reaction time predicts nicotine dependence in people with ADHD symptoms four years later (Groenman et al., 2015). Furthermore, Anokhin and Golosheykin (2016) also demonstrated that impaired frontal neural activity is associated with response inhibition deficits, increasing the risk of nicotine dependence.

The current study results contributed to the field by showing that the hyperactivity/impulsivity symptom of ADHD explained a statistically significant additional

variance in nicotine use outside the EF facets. Thus, the facets of EF are not the only significant predictors of nicotine use in individuals with more serious ADHD symptoms. Hence, in individuals with greater hyperactivity/impulsivity, there may be pathways other than response inhibition that increase nicotine smoking, as investigated in Study 2 (Chapter 3) of this thesis. As mentioned earlier, there is a connection between response inhibition and attention deficit (Weafer et al., 2010). Thus, those with the inattention symptom of ADHD use higher amounts of nicotine from the response inhibition pathway, while those with the hyperactivity/impulsivity symptom use more nicotine for other reasons than response inhibition. However, further studies are required to reveal the exact cause-and effect-relationship between the two ADHD symptoms, response inhibition and nicotine use.

Despite the conflicting results from previous investigations, the present study demonstrated that adaptive thinking and response inhibition were statistically significant predictors of the current cannabis use. Literature shows that cannabis use, especially its acute use, affects cognitive flexibility (Curran et al., 2002; Morrison et al., 2009; Pattij et al., 2008; Weinstein et al., 2008). However, the results of studies on the effect of non-acute cannabis use on cognitive flexibility are inconsistent. A study on dose-related effects of cannabis use by Bolla et al., (2002) demonstrated a positive correlation between poorer cognitive flexibility and higher cannabis use frequency in heavy cannabis users than moderate and occasional users. Even though the results of human studies are not consistent, it is hypothesised that the relationship between non-acute cannabis use and impaired cognitive flexibility could be mediated by various factors such as the age of onset or the frequency of cannabis use (Mechoulam et al., 2007; Pope et al., 2003). However, the present study measured university students' use of different forms of cannabis in the last month and over the previous six months.

Contrary to the results for alcohol and nicotine use, ADHD symptoms could not explain additional variance in current cannabis misuse after accounting for EF facets. Thus, based on this study's results, it could be concluded that poorer adaptive thinking (cognitive flexibility) and impaired response inhibition are the EF facets that predict cannabis use. The reason for using cannabis in people with ADHD symptoms could have different reasons. First, cannabis could be used in self-medication to cope with cognitive impairments (Moitra et al., 2015). Second, response inhibition is associated with increased risk-taking and decreased self-control (Cheng & Lee, 2012), which are significantly linked to cannabis use (Bolla et al., 2002; Lane et al., 2005; Pope et al., 2003). Third, shared brain regions in individuals with ADHD and substance misuse may heighten response disinhibition, resulting in impaired adaptive thinking that predicts cannabis use (please see Chapter 1, Section 1.3).

Age and gender were not significant predictors of lifetime cannabis use, but the only EF facet that was a crucial indicator of lifetime cannabis use was creativity. Creative thinking is another facet of EF that also needs cognitive flexibility. The creative thinking subscale of JEF[®] measures the individual's ability to find a solution that is not immediately noticeable in the situation; they should think creatively to solve the problem (Jansari et al., 2014). Divergent thinking in people with low creativity could be increased by acute cannabis use (Schafer et al., 2012). Furthermore, investigations suggest that low-potency cannabis use does not affect creativity, whereas high-potency cannabis use can impair divergent thinking (Kowal et al., 2015). In an interesting recent study, LaFrance and Cuttler (2017) tested 412 sober cannabis users and 309 non-users using cannabis consumption, personality self-report and objective creativity questionnaires. Their results revealed that sober cannabis users were more creative than non-users. These effects were abolished after controlling for higher levels of openness to new experiences in cannabis users. Hence, it showed that being open to new

experiences is the main factor in cannabis consumption. The current study discovered that participants with higher levels of creativity are more likely to use cannabis. Being open to new experiences is a definition of sensation seeking (Jung et al., 2018), which predicts substance misuse and is discussed in Chapter 3.

After incorporating the symptoms of ADHD into the model, the hyperactivity/impulsivity symptom predicted lifetime cannabis use above and beyond the creative thinking subscale of JEF[©]. In an investigation by Boot et al. (2017), participants with higher hyperactivity/impulsivity levels reported being more creative in daily life. Consequently, it can be inferred that creativity predicts higher levels of cannabis use in those with inattention symptom, but not in those with higher hyperactivity/impulsivity. Furthermore, hyperactivity/impulsivity added to the proportion of variation in cannabis use beyond the creativity facet of EF. Apart from creativity, there could be other pathways that raise the likelihood of using more cannabis in individuals with higher hyperactivity/impulsivity symptom of ADHD.

The current investigation attempted to determine which aspects of EF contribute to the relationship between ADHD symptoms and high rates of alcohol, cannabis and nicotine use in a group of participants using a dimensional assessment of symptoms rather than diagnostic categorisation. Moreover, using a new virtual reality task to measure different facets of EF, employing various questionnaires to measure wider aspects of substance misuse such as the frequency and quantity of alcohol use, different forms of alcoholic drinks, cannabis use, lifetime cannabis use and nicotine use, and analysing the relationship between each of these variables and two ADHD symptom clusters separately, make this study unique. Suggesting that inattention predicts the use of alcohol, cannabis and nicotine through an EF pathway also makes this a novel research. In line with this, attentional interventions such as

cognitive-behavioural therapy (CBT) (Lopez et al., 2018) or mindfulness (Smalley et al., 2009) may decrease EF impairments in individuals with inattention problems, potentially lowering their risk of alcohol, cannabis and nicotine use.

In the current study, 27.1% of the participants showed hazardous alcohol use, which was lower than the reported rates in previous studies presented in Chapter 1 and Section 2.5 of the present chapter. The reason of lower risky alcohol use in the current sample could be smaller sample size in this study compared to previous investigations. This also could be due to the reported decline in substance misuse in the UK by the NHS-Digital (2018).

Additionally, although the lifetime cannabis use was among 36.5% of the sample, 16.5% were current cannabis users and 6% of the them had hazardous cannabis use. In 2018–2019, cannabis use in England and Wales among 16 to 24-year-olds was 17%, which is the highest point in a decade. The present study asked about current and lifetime cannabis use and the current cannabis use in the present study is close to the reports of NHS-Digital (2018-19). In the present study the frequency of using nicotine over the last month and the number of daily cigarettes were asked, 9.4% of them smoked nicotine for the entire 30 days of the past month (Table 2.2). Further investigations are required to measure risky nicotine use and nicotine dependence in university students in the UK.

2.9. Limitations and future directions

Even though this study demonstrates important findings about ADHD symptoms, EF and the use of alcohol, cannabis and nicotine, it has some limitations. First, using a new virtual reality task may not be easy for everyone. Some participants, especially older adults, may struggle to complete the task not because of their poor EF but perhaps due to unfamiliarity with computerized tasks.

Secondly, this was a cross-sectional study that measured the different variables in a population at a specific point in time. This form of research does not illustrate a cause-and-effect relationship between the variables. Cross-sectional studies often cannot include data on other variables that might affect the relationship between the hypothesised cause and effect (Mann, 2003). Therefore, longitudinal studies are recommended in this area to explore a cause-and-effect relationship between the variables of this research.

The questionnaires used in this study are not diagnostic tools. Additionally, there is an argument in the literature that the cutoff should be higher for university students than for non-student peers due to normative patterns of substance intake at university (Bewick et al., 2008; Dawson et al., 2004; Kypri et al., 2005, Webb et al., 1998). In a study by Bewick et al. (2008), university students' alcohol consumption declined over the course of their undergraduate studies. Students reported drinking more alcohol during their first year than in their second or third years. This could be a limitation for the current study mainly because the majority of participants were first-year undergraduate students, and the cutoff for 'at risk' alcohol consumption level should be higher for them.

Furthermore, the nicotine questionnaire had been shortened. Shortening questionnaires may lead to low reliability and loss of accuracy, and the shortened version may not have the same psychometric properties (Kleka & Paluchowski, 2017; Smith et al., 2000). However, in the current study, the Cronbach alpha for the shortened questionnaire revealed good reliability (Cronbach alpha > .70).

Another limitation is that the study participants were not matched in terms of ethnicity, income, or the number of men and women. Additionally, underreporting of alcohol use could often be a problem due to poor recollection of recent drinking episodes (Stockwell et al., 2004). To measure fluctuations in the quantity and frequency of drinking across time,

the Timeline Follow Back methods (Sobell & Sobell, 1992) are recommended. Moreover, the information entered by participants may not be entirely truthful because of social desirability bias, especially on the illegal substance misuse questionnaire (cannabis use).

2.10. Conclusion

To conclude, this study indicated which EF facets significantly predict the use of alcohol, cannabis and nicotine. It also revealed that hyperactivity/impulsivity can explain the variance in substance misuse above and beyond those EF facets. Hence, it can be determined that hyperactivity/impulsivity is correlated with developing different substance misuse for reasons beyond EF impairments; these issues will be explored in Studies 2, 3, 4 and 5 of this thesis.

To our knowledge, this is the first study to examine the role of different EF facets in the relationship between the ADHD symptom clusters and alcohol, cannabis, and nicotine use using a new virtual reality task. To measure the predictive capacity of each ADHD symptom separately after accounting for the facets of EF, it is also the first research to divide ADHD symptoms into two groups: inattention and hyperactivity/impulsivity. In this study, rather than a categorical approach, a dimensional approach was used in measuring the symptoms of ADHD and the use of alcohol, cannabis and nicotine in the participants to have a full range.

Another important variable in this relationship is ‘impulsivity’, which is believed to be a multifaceted personality trait common to both people with ADHD and substance misusers. As previously mentioned, JEF[®] measures different facets of EF, but it cannot assess different facets of impulsivity; thus, the following chapter centres on the role of

different facets of this construct in the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine.

Chapter 3

3. The role of impulsivity in the relationship between ADHD symptoms and substance misuse

Overview

This chapter summarises existing evidence regarding how different facets of impulsivity are associated with alcohol, cannabis, and nicotine use. It also intends to present the study's results on how the main symptoms of ADHD explain additional variance in alcohol, cannabis and nicotine use after accounting for impulsivity facets. The BIS-motor, negative urgency and sensation seeking subscales predicted alcohol misuse, while inattention contributed to the proportion of explained variance in alcohol misuse over and above the facets of impulsivity. Similarly, BIS-motor, negative urgency and sensation seeking predicted nicotine use, and inattention explained additional variance in nicotine misuse after accounting for impulsivity facets. Furthermore, cannabis use was predicted by BIS-motor, nonplanning, negative urgency and sensation seeking subscales. Both ADHD symptoms also indicated cannabis use over and above impulsivity facets. Thus, individuals with hyperactivity/impulsivity symptoms might use alcohol, cannabis and nicotine through an impulsivity pathway. Those with inattention symptoms use more alcohol, cannabis and nicotine from other routes than through the facets of impulsivity. Study 1 of this thesis showed that inattention symptom did not add to the proportion of explained variance in substance misuse after accounting for EF facets, suggesting other pathways for developing substance misuse in those with inattention symptom.

3.1. Introduction

According to the World Health Organisation (WHO), a person older than 19 years of age is considered an adult. Moreover, adolescence is a period of transition from childhood to adulthood that roughly refers to the time from pubertal onset and typically spans from 12 to 18 years of age. However, according to Jaworska and MacQueen (2015), the period of adolescence has been expanded to include young adulthood (up to 25 years of age). Impulsive behaviours mostly happen during the young adulthood period (Casey, 2015; Stone et al., 2012). For this reason, various studies have identified several elements that can increase the rate of impulsive behaviours in young adults: genetics, brain development, life transitions and peer group influences (Chambers & Potenza, 2003; Quinn et al., 2011). Impulsivity as a multifaceted construct can be described in different ways, including (a) impulsive action, which is a lack of behavioural restraint without regard for the negative consequences of the actions; and (b) impulsive choice, which is impaired self-control or inability to delay satisfaction (Grant & Chamberlain, 2014; Perry & Carroll, 2008; Weafer et al., 2014). There is still debate about the nature and the number of the fundamental dimensions of impulsivity as a multidimensional trait. Prominent models introduce several dimensions, as presented in Chapter 1 (Berg et al., 2015; Dawe & Loxton, 2004; Depue & Collins, 1999; Lynam et al., 2006; Potenza & Taylor, 2009; Sharma et al., 2013; Steinberg, 2008; Whiteside & Lynam, 2001).

Even though the association between ADHD symptoms and increased rates of substance misuse is well established, little is known about the risk factors that explain this association. ADHD is a complex condition with various comorbidities, cognitive impairments and personality traits, all of which may heighten the risk of substance misuse (Kessler et al.,

2006; Martel et al., 2010; Seidman, 2006). As previously discussed, impulsivity increases in those with various neuropsychiatric disorders, such as ADHD and substance misuse, than in the general population, in addition to EF facets as risk factors for developing substance misuse and shared impairments in ADHD (Moeller et al., 2001). This trait overlap supports arguments that impulsivity explains the higher rates of substance misuse in individuals with ADHD (Iacono et al., 2008). Although these theoretical models proposed that shared impulsivity traits explain the association between ADHD symptoms and increased substance misuse, there has been relatively little experimental support for this hypothesis. Some investigations have attempted to explore the risk factors that may increase the likelihood of substance misuse in people with ADHD, which will be discussed in the next section (Section 3.2). These studies presented certain facets of impulsivity that contribute to alcohol, cannabis and nicotine use in those with ADHD symptoms, but their impulsivity measurement has limitations. None of them used a comprehensive impulsivity model to identify the main personality aspects linked to the relationship between ADHD symptoms and substance misuse.

Since various characteristics may result in impulsive action, it is crucial to investigate which facets of impulsivity may raise the risk of substance misuse in those with ADHD symptoms. Previous investigations found shared impulsivity-related traits in individuals with ADHD and substance misuse, including response disinhibition, high sensation seeking and emotional impulsiveness (Kotov et al., 2007; Roberts et al., 2011; Verdejo-Garcia et al., 2007). Although these studies indicate that both groups have common impulsivity characteristics, none of them analyses how these traits contribute to the connection between increased substance misuse and ADHD symptoms. The present study explored the

relationship between substance misuse, facets of impulsivity and ADHD symptoms clusters. The UPPS-P and BIS-11 measured impulsivity facets to provide specific information about the personality processes contributing to alcohol, cannabis and nicotine use among those with ADHD symptoms.

3.2. Impulsivity and ADHD

ADHD symptoms can persist into adulthood. Individuals with this disorder have impairments in reward and emotional processing, working memory, time processing and inhibitory control (Bramham et al., 2012; Cummins et al., 2011; Finke et al., 2011; Ibáñez et al., 2011; Marx et al., 2011; O'Brien et al., 2010; Pasini et al., 2007; Valko et al., 2010; Wilbertz et al., 2012; Willcutt et al., 2005). However, several studies have shown that impulsivity and selective attention in adults with ADHD symptoms improve with age (Bramham et al., 2012).

Different investigations indicate that individuals with ADHD show higher levels of impulsivity in a variety of tasks (Solanto, 1998, 2002). Those with ADHD symptoms perform poorly on the Continuous Performance Task (CPT), denoting that they have attentional and impulse control deficits (Winstanley et al., 2006). Adults with ADHD symptoms have slower reaction times and more omission errors, which means that they do not press the key button on 'go' trials on the Go/NoGo task (please refer to Section 2.6.3.6 of Chapter 2). This finding indicates their poor attentional ability (Epstein et al., 2003). Additionally, individuals with ADHD symptoms produce more commission errors (pressing the key button in 'no-go' trials on the Go/NoGo task), demonstrating weak behavioural

inhibition. Studies also demonstrate that children with ADHD are slower in inhibiting their responses in the stop-signal reaction time (SSRT) task. They cannot inhibit their 'go' response on the 'no-go' trials of Go/NoGo task (Nigg, 1999, Purvis & Tannock, 2000, Schachar & Logan, 1990, Schachar et al., 1995).

Individuals with ADHD symptoms make more impulsive decisions in delayed discounting tasks (Schweitzer & Sulzer-Azaroff, 1995; Solanto et al., 2001; Sonuga-Barke et al., 1992, 1996). They choose smaller, instantaneous rewards rather than bigger, more delayed ones. Some studies have shown that people with ADHD symptoms can wait for a reward if they cannot shorten the testing session by selecting a minor yet immediate reward (Schweitzer & Sulzer-Azaroff, 1995; Solanto et al., 2001; Sonuga-Barke et al., 1992, 1996). Therefore, investigators suggest that impulsive choice reveals an individual's strong desire to avoid delay (Sonuga-Barke, 2002, 2003). Furthermore, functional expressions in adults with ADHD, such as impulsive behaviours, low concentration and hyperactivity, might be due to their delay-aversion (Sonuga-Barke, 2002, 2003). Nonetheless, adults with ADHD symptoms can wait for greater rewards under certain conditions, implying a double dissociation between behavioural disinhibition and a preference for delayed rewards (Sonuga-Barke et al., 1992, 1996). In addition, there is no correlation between the level of inhibitory control (as measured by SSRT) in individuals with ADHD symptoms and their preference for larger rewards (as evaluated by delayed discounting tasks) (Solanto et al., 2001). These two measures are highly diagnostic, suggesting that inhibitory control deficits and strong impulsive choice are primary symptoms of ADHD (Winstanley et al., 2006).

Although the exact cause of ADHD is not well known, investigations revealed that PFC dysfunction could be a contributing factor (Castellanos & Tannock, 2002). Individuals with

ADHD have abnormalities in their OFC (Hesslinger et al., 2002; Itami & Uno, 2002). The frontal cortex dysfunctionality in individuals with ADHD symptoms, such as attentional dysfunction and distractibility, is supported by studies of individuals with brain injuries or frontal cortex diseases (Chao & Knight, 1995; Shue & Douglas, 1992; Wilkins et al., 1987; Woods & Knight, 1986). Even rats and monkeys with PFC damage show attentional impairments (Arnsten, 1997, 1998; Chao & Knight, 1995; Muir et al., 1996; Passetti et al., 2002; Woods & Knight, 1986).

Besides prefrontal disorder in individuals with ADHD symptoms, subcortical regions of the basal ganglia can also play a key role in the development of this condition (Rieger et al., 2003; Sergeant, 2000). According to the results of neuroimaging studies with children with ADHD symptoms, abnormalities in the frontostriatal circuits may cause impaired impulse control during the Go/NoGo task (Vaidya et al., 1998). Frontostriatal circuits connect the frontal lobes and the basal ganglia (striatum), which mediates motor, cognitive abilities and behavioural functioning in the brain (Alexander et al., 1986). Therefore, these ADHD research results demonstrate that frontal cortex deficits can lead to impulsive behaviours. Neuropsychological studies show the involvement of the PFC in preparing to act, switching between tasks and inhibiting inappropriate responses (Brass & Von Cramon, 2002; Dove et al., 2000; Mecklinger et al., 1999; Rogers et al., 1998; Shallice & Burgess, 1991; Sohn et al., 2000). It has been suggested that damage to the right inferior frontal gyrus could account for poor response inhibition (Aron et al., 2003). Moreover, patients with damage to their ventromedial frontal cortex have impaired decision-making and impulsive social behaviour. They make riskier choices on the Iowa Gambling Task (IGT) (Bechara et al., 1994). They also bet more in the presence of normal probability judgements (Manes et al., 2002). Due to

their higher impulsive choices, individuals with ADHD symptoms develop riskier behaviour without considering the negative consequences of their actions, such as substance misuse, which may later progress to SUD. Therefore, investigating the association between impulsivity facets and ADHD symptoms is essential when considering the shared brain regions in ADHD and impulsivity.

A quarter of individuals with SUD are diagnosed with adult ADHD (van Emmerik-van Oortmerssen et al., 2012). High levels of impulsivity characterise both conditions. For instance, chronic cocaine users have higher motor and cognitive (decision-making) impulsivity than non-users (Coffey et al., 2003; Fillmore & Rush, 2002; Heil et al., 2006). People who misuse substances show impairments in their reward processing, working memory and attention, denoting a cognitive deficit overlap in individuals with ADHD symptoms and substance misuse (Hester & Garavan, 2004; van Holst & Schilt, 2011; Verdejo-Garcia et al., 2006). Based on this overlap, adults with ADHD with higher impulsivity may be more at risk of substance misuse later in life than those with lower impulsivity levels (Crunelle et al., 2013). One prominent hypothesis in ADHD research is that primary cognitive and EF deficits may cause ADHD symptoms. Their combination with reward and motivational impairments is thought to be crucial in the pathophysiology of ADHD (Sonuga-Barke, 2003; Willcutt et al., 2005). The current study is the first to explore impulsivity as a multifaceted construct in the relationship between ADHD symptoms and alcohol, cannabis and nicotine use.

It has been found that different dimensions of impulsivity may be involved in substance misuse. Previous research indicated that reward sensitivity and disinhibition of responding to a stimulus are two emerging dimensions that are uniquely engaged in substance misuse

(Gullo et al., 2014; Hamilton et al., 2015; Hamilton et al., 2015; King et al., 2014; Sharma et al., 2013; Stautz et al., 2017). As a result, the next section is dedicated to reviewing the previous research on the relationship between different facets of impulsivity and substance misuse.

3.3. Impulsivity and substance misuse

There is a bidirectional relationship between impulsivity and substance misuse. To be more precise, trait impulsivity during development is a major cause of substance misuse. Increased impulsivity in adults will raise the likelihood of substance misuse and, subsequently, SUD (De Wit, 2006). Moreover, chronic and acute substance misuse may heighten impulsive behaviours, leading to increased substance misuse (De Wit, 2006).

Lower dopamine auto-receptor binding in the midbrain and more striatum amphetamine-induced dopamine release are associated with high impulsive behaviours in the human brain (Buckholtz et al., 2010). Furthermore, there is a negative correlation between the grey matter of the OFC and impulsivity in healthy individuals (Matsuo et al., 2009). However, in those with addiction, using stimulants continuously may worsen impulsive behaviour and probably modify its neural underpinning (Ersche et al., 2010). In neuroimaging studies, individuals with cocaine addiction had significantly smaller striatum, amygdala and PFC (Barrós-LoCERTALES et al., 2011; Makris et al., 2004; Matochik et al., 2003; Tanabe et al., 2009). These brain regions are associated with the main aspects of the addiction cycle, such as reinforcement learning, craving and inhibitory control (Koob & Volkow, 2010). These parts

of the brain shared by substance misuse and impulsivity highlight the importance of examining different facets of impulsivity as risk factors for substance misuse and, later, SUD.

Jeffrey Gray has proposed a critical biological theory of personality called reinforcement sensitivity theory (1972, 1990). He created a human personality model that involved two brain biological systems (please refer to Section 1.4.2 of Chapter 1). Gullo and Dawe (2008) used the example of two cars braking at different speeds to explain reward sensitivity on Gray's Behavioural Approach System (BAS; Gray, 1975). If both vehicles have the same effective brakes, the faster one (stronger impulsive) would need a longer time to stop (inhibitory control). Based on this hypothesis, Padmala and Pessoa (2010) tested 22- ± 3-year-old adults ($n = 35$) using a stop-signal task (Go/NoGo) and found that rewarding 'go' phase answers increased impulsive responses. Participants with increased impulsivity showed higher reward-driven response disinhibition and reduced activity of the inferior frontal gyrus and other related regions of the brain.

Researchers suggest that reward sensitivity, which negatively correlates with successful inhibition of responses in an individual, is a critical mechanism of impulsive behaviour (Dawe & Loxton, 2004; Gray, 1975; Padmala & Pessoa, 2010). Reward sensitivity results in more reward-seeking (Dawe et al., 2004; Ernst et al., 2006; Hamilton et al., 2015; Potenza & Taylor, 2009). High sensitivity to positive reinforcement and motivation may increase the risk of substance misuse (e.g. alcohol dependence, Dawe et al., 2004).

Disinhibition or 'rash' impulsiveness is another crucial dimension of impulsivity involved in substance misuse (Dawe & Loxton, 2004; Dawe et al., 2004). It occurs when an individual does not consider the adverse outcomes of the actions, reducing response

inhibition (Dawe et al., 2004; Ernst et al., 2006; Hamilton et al., 2015; Potenza & Taylor, 2009). This dimension of impulsivity was supported by Barratt (1993), Eysenck (1993), Zuckerman (Zuckerman & Kuhlman, 2000) and Cloninger (1987) models (Dawe & Loxton, 2004). Moreover, various studies showed that disinhibition increases the risk of alcohol use by decreasing the ability to inhibit drinking regardless of the harmful effects (Dawe et al., 2004; Ernst et al., 2006; Hamilton et al., 2015; Potenza & Taylor, 2009).

The previous arguments do not mean that there are no other impulsivity dimensions associated with substance misuse. Stautz et al. (2017) claimed that other facets would not increase risky alcohol usage during adolescence and young adulthood without having a relationship with reward sensitivity and/or response disinhibition.

Negative urgency, a dimension of UPPS and UPPS-P (Section 1.4.2, Chapter 1), is a unique facet of trait impulsivity related to negative affect, which can increase impulsive decisions. Furthermore, investigations revealed that major depressive disorder causes inhibitory control deficits (Snyder, 2013; Tice et al., 2001; Whiteside & Lynam, 2001). Negative affect can also decrease reward sensitivity (Gullo & Stieger, 2011), although there is some contradictory evidence about the connection between trait urgency and young adult substance misuse. Some investigations have found no positive correlation between urgency and substance misuse after controlling other impulsivity facets (Gullo et al., 2014; Lopez-Vergara et al., 2016).

Various personality scales measure different impulsive behaviours, such as novelty seeking, behaviour control and response disinhibition (Dawe et al., 2004). Many cross-sectional investigations studied the relationship between these constructs and substance misuse.

Although there were various samples with diverse measures of impulsivity, a significant connection between substance misuse and impulsivity was found (Baker & Yardley, 2002; Ersche et al., 2010; Johnson et al., 2003; Jorm et al., 1999; McGue et al., 2001; Shillington & Clapp, 2002; Simons & Carey, 2002; Soloff et al., 2000). For instance, in cross-sectional studies, those with higher substance misuse scored higher on novelty and sensation seeking (Battaglia et al., 1996; Grau & Ortet, 1999; Pidcock et al., 2000; Sher et al., 1995, 2000; Wills et al., 1994). Similarly, Malmberg et al. (2010) determined that sensation seeking as a dimension of impulsivity was indicative of ever misusing a substance in adolescents aged 11 to 15. Therefore, sensation seeking raises the risk of early substance misuse. Other studies also established that childhood impulsiveness is associated with adulthood substance misuse (Howard et al., 1997; Masse & Tremblay, 1997; Tarter et al., 2004). Even cross-sectional studies with psychotic patients indicated that a lifelong history of substance misuse is linked to impulsivity and sensation seeking (Dervaux et al., 2001; Gut-Fayand et al., 2001; Liraud & Verdoux, 2000; Van Ammers et al., 1997). Hence, these studies denote that sensation seeking, in its broadest sense, is significantly associated with adult and adolescent substance misuse. However, facets of impulsivity other than sensation seeking have received less attention. The present study attempted to address this problem by using the UPPS-P and BIS-11 to assess the impulsivity facets, alongside aspects of alcohol, cannabis and nicotine use in a university student sample.

3.4. The relationship between impulsivity, ADHD and substance misuse

The precise relationship between ADHD and substance misuse is unclear. Even when there is no comorbid disorder, ADHD remains an independent risk factor for developing

various psychoactive substance misuses such as alcohol, cannabis, nicotine, amphetamine, cocaine, etc. (see Section 1.3 of Chapter 1) (Biederman et al. 1995; Milberger et al. 1997). Comparatively, 15.2% of adults with ADHD symptoms meet the criteria for substance misuse three times more than those without ADHD symptoms (Kessler et al., 2006). Even though the connection between ADHD and substance misuse is well established, little is known about the factors that explain this relationship. ADHD is a complex condition with many related cognitive impairments, personality traits and comorbidities, all of which may increase the risk of substance misuse and, later, SUD (Kessler et al., 2006; Martel et al., 2010; Seidman, 2006). Sharing common personality traits, such as impulsivity, supports the hypothesis that higher impulsivity may increase the likelihood of substance misuse in individuals with ADHD (Dick et al., 2010; Iacono et al., 2008; Verdejo-Garcia et al., 2008). While these theoretical models suggest that impulsivity can explain the relationship between ADHD and higher substance misuse, there is still a lack of experimental evidence to support this.

Few studies have focused on the factors that may elevate the risk of substance misuse in adults with ADHD symptoms. For instance, Roberts et al. (2014) worked with 361 undergraduate students aged 20 to 25. They used the UPPS-P to measure impulsivity facets and the Conners Adult ADHD Rating Scale-Self-Report: Long version (CAARS-S:L; Conners et al., 1999) to assess ADHD symptoms. Moreover, they investigated substance misuse by asking participants to report their substance use pattern over the past year via a life history calendar and AUDIT. Their results indicated that negative urgency, sensation seeking and premeditation accounted for the variance in the relationship between hyperactivity/impulsivity and risky alcohol drinking. Furthermore, negative urgency

accounted for the association between hyperactivity/impulsivity and the risk of nicotine and cannabis use.

Weafer et al. (2010) used self-report and behavioural assessments to measure three dimensions of impulsivity in adults with ADHD and healthy controls. They intend to predict self-reported alcohol use through poor response inhibition, poor attentional inhibition and increased risk-taking. Their results indicated that attentional inhibition predicted alcohol consumption only in the group of participants with ADHD. They hypothesised that particular forms of behavioural disinhibition may contribute to the high rates of substance misuse in adults with ADHD. Using the Sensation Seeking Scale-V, Rooney et al. (2012) found high rates of alcohol misuse in university students with ADHD symptoms (Zuckerman, 1994). Even though previous investigations showed that some facets of impulsivity might predict substance misuse in adults with ADHD symptoms, their impulsivity measures are limited. None of them provided a comprehensive impulsivity assessment (Roberts et al., 2014). As previously mentioned, impulsivity is a multifaceted construct (Nigg, 2001). Moreover, the dysfunctionality of some personality processes and cognitive functions can cause impulsive action (Whiteside & Lynam, 2003).

Personality-based impulsivity models have attempted to identify different factors that lead to rash and unplanned actions (Dick et al., 2010). The UPPS-P Impulsive Behaviour Scale (Lynam et al., 2006) assesses five impulsive behaviour-related traits that were discussed in Chapter 1 (Section 1.4.2). When compared to other tests, UPPS-P is a more comprehensive measurement of personality traits that result in impulsive actions (Roberts et al., 2014). This impulsivity test examines the contribution of specific personality traits and impulsivity in neuropsychological disorders that involve rash behaviours (Anestis et al.,

2007). Previous investigations showed that some facets of impulsivity measured by the UPPS or UPPS-P predicted alcohol, cannabis and nicotine use among university students. Jones and colleagues (2014) assessed 400 university students aged 18 to 25 using the UPPS (Whiteside & Lynam, 2001), Student Alcohol Questionnaire-Revised (SAQ; Engs & Hanson, 1994) and Drinking Motivation Questionnaire (DMQ; Cooper, 1994). They investigated the relationship between alcohol intake, impulsivity facets, drinking motives and the propensity to engage in alcohol-related risky behaviours. Their results revealed associations between sensation seeking, negative urgency and premeditation, reasons for drinking and the tendency to participate in alcohol-related risky behaviours. Thus, their findings concluded that high negative urgency levels and severe drinking consequences could be valuable predictors of alcohol misuse among university students.

Given that various characteristics may lead to impulsive behaviour, it is crucial to discern specific facets of impulsivity that contribute to the connection between ADHD symptoms and substance misuse in adults. Adults with ADHD symptoms and substance misuse exhibit different impulsive-related traits, including impaired inhibitory control, high level of sensation seeking and an inclination to act impulsively in response to strong emotions (Roberts et al., 2011; Kotov et al., 2010; Verdejo-Garcia et al., 2007). While the investigations indicated that both groups of participants have certain impulsive characteristics, neither study compared the contribution of these traits and the relationship between ADHD and substance misuse (Roberts et al., 2014). Moreover, none of the previous research studied the connection between ADHD symptoms, facets of impulsivity and the use of alcohol, cannabis and nicotine separately.

The current study was an attempt to determine which facets of impulsivity accounted for the relationship between inattention and hyperactivity/impulsivity and the use of alcohol, cannabis and nicotine. A multifaceted assessment approach can extend previous research that has only measured impulsivity as a unitary construct (Rooney et al., 2012). ADHD symptoms were measured using a dimensional assessment rather than diagnostic categorisation based on the dimensional structure of the symptom clusters of ADHD (Hinshaw, 1994). In addition, having symptoms of ADHD is linked to functional impairments even in those who do not meet the full criteria for ADHD (Bussing et al., 2010). The UPPS-P was used to evaluate various impulsivity facets and their contribution to the use of alcohol, cannabis and nicotine in individuals with ADHD symptoms. The BIS-11 was also employed to measure three other facets of impulsivity (see Section 1.4.2 of Chapter 1). Moreno et al. (2012) used the BIS-11 to assess the aspects of impulsivity and unveiled an association between motor impulsiveness and cannabis use. In another investigation, Jakubczyk et al. (2018) disclosed the association between the nonplanning subscale of BIS-11 and AUD. In previous studies, the nonplanning subscale was a major predictor of heavy smoking (Ryan et al., 2013; Skinner et al., 2004). Both UPPS-P and BIS-11 were chosen to provide a more comprehensive assessment of impulsivity and to measure different facets of impulsivity seen in individuals with ADHD that could increase the risk of substance misuse and later SUD.

3.5. Aim of this study

This research intends to determine which facet of impulsivity can predict participants' use of alcohol, cannabis and nicotine by employing the UPPS-P and BIS-11. It also aims to explore the relationship between those facets and the sample's inattention and

hyperactivity/impulsivity. Another objective of the study is to discern whether the symptom clusters of ADHD can explain additional variance in the use of alcohol, cannabis and nicotine after accounting for impulsivity facets.

Based on the previously presented literature review (Sections 2.1 and 3.1), cannabis is the most commonly used substance among university students (Monitoring the Future Survey, 2011; Newbury-Birch et al., 2001; Webb et al., 1996). In comparison to the non-student population, these students use higher amounts of alcohol, nicotine and other drugs, such as magic mushroom, LSD, cocaine, amphetamine, etc. (Bennett & Holloway, 2014; Edden et al., 2012; Edwards & Kendler, 2012; Home Office, 2012; Humphreys et al., 2011; Molina et al., 2007; NHS Digital, 2019). Thus, in a study with a much larger sample size ($n = 380$) than the previous one ($n = 90$), focusing on the role of different facets of impulsivity in the relationship between each ADHD symptom and alcohol, cannabis and nicotine use may help in gaining a better view of ADHD and substance use.

Based on past research's findings (see Section 3.4), it is hypothesised that negative urgency, sensation seeking and premeditation would be key predictors of alcohol use, while negative urgency would be a major determiner of nicotine and cannabis use in the current study. Moreover, the BIS-11 nonplanning subscale is thought to be a strong indicator of alcohol and nicotine use, whereas motor impulsiveness would mainly suggest cannabis use. No research has been done to explore whether inattention or hyperactivity/impulsivity may explain the unique variance in substance misuse after accounting for the facets of impulsivity; the current study is the first to investigate this. Since the hyperactivity/impulsivity symptom and various impulsivity facets overlap, it is hypothesised

that after accounting for impulsivity facets, the inattention symptom will clarify additional variance in alcohol, cannabis and nicotine.

3.6. Methods

3.6.1. Participants

Participants were 380 individuals aged 18 and above with a mean age of 22.01 ($SD = 7.43$) that have been recruited from Goldsmiths, University of London's Department of Psychology's Research Participation Scheme; the majority of participants were women (81.5%). In the previous study, students from the second and third year of the university were also recruited. Because of the lack of budget to pay the participants for their time, only first year undergraduates were recruited in this study and they received course credits for their participation. G*Power 3.1.9.7 (Faul et al., 2007, 2009) was used to measure this study's statistical power and sample size which for this study was 166. Larger sample size increases the power of the study so, more participants were recruited for this study. Any participant with major psychiatric disorder (e.g. bipolar disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder) and/ or major physical health problems (e.g. brain injury) were excluded. Thirty-nine participants were deleted because of incomplete questionnaires. Participants signed an online consent form confirming that they were 18 or older and agreed to participate in the study. All participants completed the measures anonymously.

3.6.2. Procedure

This study was approved by the ethics committee of the Department of Psychology at Goldsmiths, University of London (Ethics Reference Number: PS260116ZSS) (please see Appendix 13 for the approved Ethical Approval Form). The information about the study and the link for it was on the Research Participation Scheme page that is accessible to first-year undergraduate students at the Department of Psychology at Goldsmiths, University of London, along with researcher's email address. Studies were completed in exchange for course credit. Participants could email the researcher with any question they had about the study. On the first page of the online section, specific additional details about the number and length of the questionnaires, ethical issues and the study's overall focus were provided.

Participants had to fill out an online consent form confirming that they are 18 or older and agreed to be involved in the study; otherwise, they could not answer the questionnaires. Moreover, any question that they did not want to answer could be skipped. At the end of the survey, there was a debrief page with detailed information about the study.

3.6.3. Measures

Due to the fact that the studies of this thesis were recruiting participants at almost the same time as Study 1 (Chapter 2), the same questionnaires were used to measure ADHD symptoms, the use of alcohol, cannabis and nicotine (please see Chapter 2, Section 2.6.3). The Cronbach alpha for each questionnaire of the current study are as follows: ASRS-IA = .82, ASRS-HI = .82, AUDIT = .85, AUQ = .762, Nicotine = .30, CUDIT-R = .89, CAN = .72.

3.6.3.1. *Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995);*

Barratt and the International Society for Research on Impulsivity believed that impulsivity is a multifaceted construct thus developed BIS-11 as a multi-dimensional measurement. A principal components analysis on the scores of the current version of the BIS-11 was defined by Patton et al (1995) manuscript, which was gathered from 248 psychiatric inpatients and 412 university students. This questionnaire measures some facets of impulsivity and it includes 30 items, which are scored to yield the three second-order (attentional, motor, non-planning) and 6 oblique first-order factors (attention and cognitive instability for attentional factor, motor and perseverance for motor factor, self-control and cognitive complexity for the non-planning factor of impulsivity). Participants should answer the questions by choosing between 'Rarely/Never', 'Occasionally', 'Often' or 'Almost always/Always'. And the answers have scores from 1 to 4 (please see Appendix 10 for the full questionnaire). A common question asked about this questionnaire is what score can be used as highly impulsive? Previous investigations used the total score of 74, one standard deviation above the mean presented by Patton et al. (1995) for high impulsiveness. On the other hand, Stanford et al. (2009) recommended the cutoff score of 72 or higher, which suggest that the scale has a good concurrent validity. The total score between 52 and 71 on BIS-11 reflects normal limits of impulsiveness. The Cronbach alpha of the total score of BIS-11 for the current study was .85. The Cronbach alpha of each subscale is as follows: BIS-attentional = .74, BIS-motor = .66, BIS-non-planning = .73.

3.6.3.2. *The Impulsive Behaviour Scale (UPPS-P; Lynam et al, 2006);*

This is a 59 item self-report scale, which is a revised version of the original one created by Whiteside and Lynam (2001). It can be used in both adults and adolescents and it

has been used to measure the levels of impulsivity in different groups of patients such as those with drug and alcohol misuse (Whiteside & Lynam, 2003), individuals with risky behaviours, problems with sustained motivation and ADHD (Miller, Derefinko, Lynam, Milich, & Fillmore, 2010) and gambling (Miller, Flory, Lynam, & Leukefeld, 2003). It measures five facets of impulsivity such as premeditation, perseverance, sensation seeking and positive urgency and negative urgency (please refer to Section 1.4.2). Lack of premeditation is a failure to consider consequences of behaviour before acting (e.g. “When I am very happy, I can’t seem to stop myself from doing things that can have bad consequences”). Lack of perseverance is when the individual is not able to follow boring or difficult tasks (e.g., “I tend to give up easily”). An inclination to seek excitement and new experiments is sensation seeking (e.g., “I’ll try anything once”). To act impulsively during negative affect is negative urgency (e.g. “I have trouble resisting my cravings”) and during positive affect is positive urgency (e.g. “When I am in a great mood, I tend to get into situations that could cause me problems”). Items are measured using a four point Likert-type response format, from 1 (I agree strongly) to 4 (I disagree strongly) and most of the items are reverse coded. This scale may help the researchers to understand the reasons for the impulsive behaviours in different individuals. For instance, if two people have the same impulsive behaviours (e.g. gambling) with different reasons such as to distract from negative emotions or to stay in positive emotion, it is likely that they respond differently to a treatment. Smith et al (2007) compared UPPS-P responses with interview data and found that the interview data and questionnaire data had the same factor structure of impulsivity traits. In addition, the assessment of each construct across methods had convergent validity and there was discriminant validity between the various constructs. The Cronbach alpha for the total score of the UPPS-P of the current investigation was .74. The Cronbach alpha for each

subscale is as follows: negative urgency = .86, positive urgency = .92, lack of premeditation = .86, lack of perseverance = .54, sensation seeking = .87.

3.6.4. Data analysis strategy

Data were analysed using IBM SPSS version 20. Pearson correlation was conducted to examine the connection between the variables of the study and the results are presented in Table 3.4. An alpha level of $p = .05$ was used for significance testing. Hierarchical linear regression was carried out to find out first, to investigate whether different facets of impulsivity can predict alcohol, nicotine and cannabis use significantly and second, to explore whether the symptoms of ADHD can explain more variance in alcohol, nicotine and cannabis use after accounting for all impulsivity facets.

3.7. Results

3.7.1. Outliers and multi-collinearity

An analysis of standard residuals showed that the data contained no outliers (Std. Residual Min > -3.29 , Std. Residual Max < 3.29). There was a strong significant correlation between BIS-Attention and both inattention and hyperactivity/impulsivity. Therefore, this dimension of the BIS questionnaire was excluded from the model to minimize the multi-collinearity.

To correct for slight positive skew in the AUQ (skewness = 1.03, kurtosis = 1.26) and CUDIT-R (skewness = 2.03, kurtosis = 5.31) and CAN (skewness = 1.4, kurtosis = 3.1) scores were log transformed (new = $LG10(\text{old} + a)$; $a = 1$) and the skewness and kurtosis

became as follows: AUQ (skewness = -.09, kurtosis = -.44) and CUDIT-R (skewness = -.34, kurtosis = -.40) and CAN (skewness = .07, kurtosis = -1.01).

3.7.2. The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

The same questions as Section 2.7.2 were asked to measure the frequency and quantity of alcohol, cannabis and nicotine use, which are presented in Table 3.1. Based on the data, 28.4% of the participants gained a score of 8 or more on AUDIT, which reflects hazardous alcohol use. 8.1% of women gained a score of 13 or above and 19.7% of men got the score of 15 or above on AUDIT, which is a cutoff score for each gender for alcohol dependence.

The quantity and frequency of wine and wine type products, beer or cider and spirits also presented in Table 3.2, which were measured by AUQ. Results showed that 8.2% of the participants got a score of 8 or above on CUDIT-R, which shows hazardous cannabis use and 4.4% got the score of 12 or above, which shows possible cannabis use disorder (Table 3.1).

The results also indicated that 24.3% of the university students of this study had a score of 24 or above in ASRS, which demonstrates the highly likelihood of having ADHD.

Table 3.1

The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

| | Frequency | Percent | Hazardous use (%) | Dependence (%) |
|--|-----------|---------|-------------------|---------------------------|
| AUDIT | | | 28.4 | Women = 8.1 Men = 19.7 |
| AUDIT frequency (How often do you have a drink containing alcohol?) | | | | |
| Never | 98 | 28.7 | | |
| Monthly or less | 79 | 23.2 | | |
| 2-4 times a month | 90 | 26.4 | | |
| 2-3 times a week | 61 | 17.9 | | |
| 4 or more times a week | 13 | 3.8 | | |
| AUDIT quantity (How many standard drinks containing alcohol do you have on a typical day when drinking?) | | | | |
| 1 or 2 | 180 | 52.8 | | |
| 3 or 4 | 96 | 28.2 | | |
| 5 or 6 | 54 | 15.8 | | |
| 7 to 9 | 9 | 2.6 | | |
| 10 or more | 2 | .6 | | |
| CUDIT-R | | | 8.2 | 4.4 |
| CUDIT-R frequency (How often do you use cannabis?) | | | | |
| Never | 256 | 75.1 | | |
| Monthly or less | 53 | 15.5 | | |
| 2-4 times a month | 16 | 4.7 | | |
| 2-3 times a week | 0 | 0 | | |
| 4 or more times a week | 16 | 4.7 | | |
| CUDIT-R quantity (How many hours were you stoned when you had been using cannabis?) | | | | |
| Less than 1 | 74 | 87.1 | | |
| 1 or 2 | 6 | 7.1 | | |
| 3 or 4 | 4 | 4.7 | | |
| 5 or 6 | 1 | 1.2 | | |
| 7 or more | 0 | 0 | | |
| CAN | | | | |
| Lifetime cannabis use (Have you ever used cannabis in your life?) | | | | |
| Yes | 170 | 49.9 | | |
| No | 171 | 50.1 | | |
| Cannabis quantity in the last year (In the last year, on how many separate days have you used cannabis?) | | | | |
| Never | 223 | 68.3 | | |
| Once every 12 months | 23 | 6.7 | | |
| Once every 2 to 3 months | 32 | 9.4 | | |

| | | |
|-----------------------|----|-----|
| Once a month | 11 | 3.2 |
| 2 to 3 times a month | 15 | 4.4 |
| Once a week | 7 | 2.1 |
| 2 to 3 times a week | 7 | 2.1 |
| 4 to 5 times a week | 3 | .9 |
| Daily or nearly daily | 10 | 2.9 |

NIC

NIC frequency

(During the past 30 days, on how many days did you smoke cigarettes?)

| | | |
|---------------|-----|------|
| 0 days | 237 | 69.5 |
| 1 to 2 days | 24 | 7.0 |
| 3 to 5 days | 10 | 2.9 |
| 6 to 9 days | 13 | 3.8 |
| 10 to 19 days | 10 | 2.9 |
| 20 to 29 days | 21 | 6.2 |
| All 30 days | 26 | 7.6 |

NIC quantity

(During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?)

| | | |
|--|-----|------|
| I did not smoke cigarettes during the past 30 days | 238 | 69.8 |
| Less than 1 cigarette per day | 20 | 5.9 |
| 1 cigarette per day | 17 | 5.0 |
| 2 to 5 cigarettes per day | 42 | 12.3 |
| 6 to 10 cigarettes per day | 11 | 3.2 |
| 11 to 20 cigarettes per day | 11 | 3.2 |
| More than 20 cigarettes per day | 2 | .6 |

Note: AUDIT = Alcohol Use Identification Test; CUDIT-R = Cannabid Use Identification Test-Revised; NIC = Nicotine Use Test

Table 3.2

The frequency of wine and wine type products, beer or cider, spirits

| Days in a week | Wine | | Beer or cider | | Spirits | |
|----------------|-----------|------|---------------|------|-----------|------|
| | Frequency | % | Frequency | % | Frequency | % |
| 0 | 214 | 62.8 | 216 | 63.3 | 224 | 65.7 |
| 1 | 69 | 20.2 | 84 | 24.6 | 87 | 25.6 |
| 2 | 35 | 10.3 | 26 | 7.6 | 19 | 5.6 |
| 3 | 10 | 2.9 | 10 | 2.9 | 4 | 1.2 |
| 4 | 10 | 2.9 | 4 | 1.2 | 5 | 1.5 |
| 5 | 2 | .6 | 1 | .3 | 1 | .3 |
| 6 | 0 | 0 | 0 | 0 | 0 | 0 |
| 7 | 0 | 0 | 0 | 0 | 0 | 0 |

3.7.3. Descriptive statistics and the correlations between variables

Table 3.3 shows the mean, standard deviation and the number of participants for each variable. Men were coded '1' and women were coded '2'. The Pearson correlation analysis between the variables of this study showed that inattention symptom of ADHD was correlated with all dimensions of BIS-11 and UPPS-P. It was also correlated significantly with all substance misuse questionnaire scores. The hyperactivity/impulsivity symptom of ADHD was correlated significantly with all the dimensions of the two impulsivity questionnaires and also with the two alcohol tests, nicotine use questionnaire and CUDIT-R, but it was not correlated with CAN. However, ADHD-total score was correlated with all impulsivity dimensions and substance misuse questionnaires scores. The scores of AUDIT, AUQ, NIC, and CAN were correlated with all facets of impulsivity except BIS-nonplanning. CUDIT-R score was correlated with all facets of impulsivity in this study (Table 3.4).

Due to high correlation between BIS-attention and inattention symptom of ADHD ($r=0.64$, $n=340$, $p < 0.001$), hyperactivity/impulsivity symptom of ADHD ($r=0.65$, $n=340$, $p < 0.001$) and ADHD-total ($r=0.73$, $n=340$, $p < 0.001$), BIS-attention was deleted from the analysis.

Table 3.3
Descriptive statistics and demographic information

| | Mean | Std. Deviation | <i>n</i> | Range | Minimum | Maximum |
|-----------------|-------|----------------|----------|-------|---------|---------|
| Age | 22.01 | 7.43 | 295 | 63 | 18 | 81 |
| ASRS_IA | 17.65 | 5.72 | 341 | 34 | 0 | 34 |
| ASRS_HI | 13.59 | 5.81 | 341 | 36 | 0 | 36 |
| ASRS_total | 31.24 | 10.26 | 341 | 70 | 0 | 70 |
| UPPS_NU | 29.15 | 6.74 | 341 | 36 | 12 | 48 |
| UPPS-Prem | 21.65 | 5.26 | 341 | 28 | 11 | 39 |
| UPPS_Pers | 21.50 | 3.43 | 340 | 20 | 13 | 33 |
| UPPS_SS | 28.01 | 7.46 | 341 | 36 | 12 | 48 |
| UPPS_PU | 42.24 | 8.39 | 341 | 38 | 18 | 56 |
| BIS_attentional | 17.19 | 4.01 | 340 | 20 | 8 | 28 |
| BIS_motor | 21.66 | 4.34 | 341 | 25 | 12 | 37 |
| BIS_nonplanning | 24.97 | 4.99 | 341 | 29 | 11 | 40 |
| AUDIT | 5.47 | 5.67 | 339 | 29 | 0 | 29 |
| AUQ | 4.52 | 2.07 | 340 | 95 | 0 | 95 |
| NIC | 21.19 | 14.90 | 334 | 49 | 0 | 49 |
| CUDIT-R | .69 | .38 | 341 | 23 | 0 | 23 |
| CAN | .70 | .71 | 340 | 83 | 0 | 83 |

Note: ADHD = Attention Deficit Hyperactivity Disorder; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = NU; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; sensation seeking = Sensation-seeking; BIS = Barratt Impulsiveness Scale; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

Table 3.4

Pearson correlation between ADHD, impulsivity and substance misuse variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | |
|----------------|--------|--------|--------|--------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|----|--|
| 1-Age | 1 | | | | | | | | | | | | | | | | | | |
| 2-Gender | -.21** | 1 | | | | | | | | | | | | | | | | | |
| 3-ASRS_IA | -.17** | -.04 | 1 | | | | | | | | | | | | | | | | |
| 4-ASRS_HI | -.06 | -.04 | .58** | 1 | | | | | | | | | | | | | | | |
| 5-ASRS_total | -.12* | -.04 | .89** | .89** | 1 | | | | | | | | | | | | | | |
| 6-UPPS_NU | -.18** | .04 | .55** | .43** | .55** | 1 | | | | | | | | | | | | | |
| 7-UPPS_Prem | -.04 | -.07 | .37** | .35** | .41** | .50** | 1 | | | | | | | | | | | | |
| 8-UPPS_Pers | -.08 | -.09 | .44** | .20** | .36** | .27** | .54** | 1 | | | | | | | | | | | |
| 9-UPPS_SS | .09 | .22** | .13* | .13* | .15** | .17** | .19** | .00 | 1 | | | | | | | | | | |
| 10-UPPS_PU | .19** | .11 | -.40** | -.51** | .52** | .63** | .53** | .20** | .34** | 1 | | | | | | | | | |
| 11-BIS_atte | -.14* | -.04 | .64** | .65** | .73** | .53** | .41** | .32** | .16** | .48** | 1 | | | | | | | | |
| 12-BIS_motor | -.03 | -.10 | .40** | .42** | .46** | .53** | .56** | .23** | .28** | .52** | .50** | 1 | | | | | | | |
| 13-BIS_nonplan | -.21** | .03 | .53** | .35** | .50** | .57** | .65** | .48** | .16** | .51** | .51** | .55** | 1 | | | | | | |
| 14-AUDIT | .07 | -.22** | .25** | .29** | .31** | .31** | .28** | .15** | .26** | .30** | .26** | .35** | .21** | 1 | | | | | |
| 15-AUQ | .01 | -.16** | .19** | .15** | .19** | .23** | .20** | .12* | .25** | .16** | .13* | .25** | .10 | .70** | 1 | | | | |
| 16-NIC | .16** | -.15* | .20** | .12* | .18** | .23** | .15** | .14* | .19** | .12* | .12* | .26** | .07 | .49** | .56** | 1 | | | |
| 17-CUDIT-R | -.13* | -.07 | .65** | .58** | .69** | .43** | .38** | .32** | .22** | .45** | .50** | .35** | .39** | .29** | .23** | .27** | 1 | | |
| 18-CAN | .13* | -.17** | .16** | .08 | .13* | .15** | .13* | .11* | .21** | .07 | .11* | .23** | .09 | .43** | .50** | .57** | .23** | 1 | |

Note: ADHD = Attention Deficit Hyperactivity Disorder; IA = Inattentive; HI = Hyperactive/Impulsive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking; BIS = Barratt Impulsiveness Scale; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Question

3.7.4. The role of impulsivity in the relationship between ADHD symptoms and alcohol use

The results of this study show that 28.4% of the participants gained a score of 8 or above in AUDIT, which shows hazardous alcohol use. The hierarchical linear regression analysis for alcohol use ($n = 341$) indicated that at stage one, after adding gender and age, gender contributed significantly to the regression model, ($F(2,290) = 9.41, p < .05, \beta = -.24$) and accounted for 6.1% of the variation in AUDIT ($R^2 = .06, F \text{ change} = 9.41, p = .00$). Adding BIS dimensions to the second step showed that BIS-motor contributed to the model significantly ($F(4,288) = 14.24, p < .05, \beta = .28$) and accounted for 10.4% of the variation in AUDIT ($R^2 = .16, F \text{ change} = 17.95, p = .00$). This means that participants with higher motor impulsiveness gained higher score on AUDIT. Adding the two main symptoms of ADHD showed that ASRS-IA contributed significantly ($F(5,287) = 13.70, p < .05, \beta = .20$) and accounted for 2.8% of the variation above and beyond BIS dimensions ($R^2 = .19, F \text{ change} = 9.79, p = .00$) (Table 3.6). After accounting for the subscales of BIS-11, the ASRS-IA score predicted higher scores on AUDIT.

Table 3.5
Hierarchical regression model predicting AUDIT scores from the two ADHD symptoms and BIS subscale scores.

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .16 | .10 | .28 | .00 |
| | BIS-nonplanning | | | .07 | .27 |
| 2 | BIS-motor | .19 | .03 | .23 | .00 |
| | BIS-nonplanning | | | .01 | .88 |
| | ASRS-IA | | | .20 | .00 |
| | ASRS-HI | | | .12 | .07 |

Note: AUDIT = Alcohol Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Table 3.6
Hierarchical regression model predicting AUDIT scores from ADHD-total score and BIS subscale scores.

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .16 | .10 | .28 | .00 |
| | BIS-nonplanning | | | .07 | .27 |
| 2 | BIS-motor | .19 | .03 | .23 | .00 |
| | BIS-nonplanning | | | .01 | .93 |
| | ASRS-total | | | .09 | .20 |

Note: AUDIT = Alcohol Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Two separate hierarchical regression analyses were conducted to see the contribution of each impulsivity dimension to the model predicting AUDIT. Adding UPPS-P sub-scales to the model indicated that negative urgency and sensation seeking were the two dimensions that contributed to the model significantly ($F(7,284) = 10.81, p < .05, \beta(SS) = .17, \beta(NU) = .22$). These two sub-scales accounted for an additional 15.1% of the variation ($R^2 = .21, F$ change = 10.89, $p = .000$). The results show that those with higher negative urgency and sensation seeking scores had more AUDIT scores. ADHD total score did not contribute to the model significantly (Table 3.7), but the ASRS-IA predicted 1.3% more variance over and above the UPPS-P dimensions ($R^2 = .22, F$ change = 4.72, $p = .03$). (Table 3.8).

Table 3.7

Hierarchical linear regression model predicting AUDIT scores from the two ADHD symptoms and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .21 | .15 | .22 | .00 |
| | UPPS-Prem | | | .08 | .28 |
| | UPPS-Pers | | | .02 | .80 |
| | UPPS-SS | | | .18 | .00 |
| | UPPS-PU | | | .06 | .43 |
| 2 | UPPS-NU | .22 | .01 | .01 | .31 |
| | UPPS-Prem | | | .31 | .28 |
| | UPPS-Pers | | | .97 | .14 |
| | UPPS-SS | | | .00 | .28 |
| | UPPS-PU | | | .97 | .31 |
| | ASRS-IA | | | .74 | .03 |
| | ASRS-HI | .07 | .28 | | |

Note: AUDIT = Alcohol Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

Table 3.8

Hierarchical linear regression model predicting AUDIT scores from the ADHD- total score and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .21 | .15 | .22 | .00 |
| | UPPS-Prem | | | .08 | .28 |
| | UPPS-Pers | | | .02 | .80 |
| | UPPS-SS | | | .18 | .00 |
| | UPPS-PU | | | .06 | .43 |
| 2 | UPPS-NU | .22 | .01 | .18 | .02 |
| | UPPS-Prem | | | .08 | .27 |
| | UPPS-Pers | | | .01 | .83 |
| | UPPS-SS | | | .17 | .00 |
| | UPPS-PU | | | .02 | .83 |
| | ASRS-total | | | .15 | .33 |

Note: AUDIT = Alcohol Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

The results of hierarchical linear regression showed that gender predicted AUQ in a significant way ($F(2,291) = 4.93, p < .05, \beta = .18$) and it accounted for 3.3% of the variance ($R^2 = .03, F$ change = 4.93, $p = .00$). Adding BIS sub-scales to the model showed that BIS-motor accounted for 5% of the variance in predicting AUQ score ($R^2 = .08, F$ change = 7.89,

$p = .00$). In the third step, inattention symptom predicted AUQ scores above and beyond this impulsivity facet ($R^2 = .10$, F change = 2.67, $p = .07$, β (ASRS-IA) = .17) (Table 3.9 and 3.10). This means that after accounting for the BIS-11 subscales, those with higher inattention symptom used alcohol more frequently.

Table 3.9
Hierarchical linear regression model predicting AUQ scores from ADHD symptoms and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .08 | .05 | .25 | .00 |
| | BIS-nonplanning | | | -.05 | .46 |
| 2 | BIS-motor | .10 | .02 | .24 | .00 |
| | BIS-nonplanning | | | -.12 | .11 |
| | ASRS-IA | | | .17 | .03 |
| | ASRS-HI | | | -.02 | .73 |

Note: AUQ = Alcohol Use Questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Table 3.10
Hierarchical linear regression model predicting AUQ scores from ADHD-total score and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .08 | .05 | .25 | .00 |
| | BIS-nonplanning | | | -.05 | .46 |
| 2 | BIS-motor | .09 | .01 | .00 | .00 |
| | BIS-nonplanning | | | .21 | .21 |
| | ASRS-total | | | .08 | .08 |

Note: AUQ = Alcohol Use Questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Adding UPPS-P dimensions to the second step of the model predicted higher AUQ scores ($F(7,265) = 6.53$, $p < .05$, β (SS) = .20, β (NU) = .24) with negative urgency and sensation seeking (table 3.11 and 3.12). None of the ADHD scores explained additional variance in AUQ after accounting for the facets of impulsivity.

Table 3.11

Hierarchical linear regression model predicting AUQ scores from the two ADHD symptoms and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .14 | .10 | .25 | .00 |
| | UPPS-Prem | | | .07 | .38 |
| | UPPS-Pers | | | .00 | .97 |
| | UPPS-SS | | | .21 | .00 |
| | UPPS-PU | | | .10 | .21 |
| 2 | UPPS-NU | .14 | .00 | .23 | .00 |
| | UPPS-Prem | | | .07 | .38 |
| | UPPS-Pers | | | .02 | .82 |
| | UPPS-SS | | | .21 | .00 |
| | UPPS-PU | | | .12 | .15 |
| | ASRS-IA | | | .04 | .66 |
| | ASRS-HI | .04 | .60 | | |

Note: AUQ = Alcohol Use Questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

Table 3.12

Hierarchical linear regression model predicting AUQ scores from ADHD-total score and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .14 | .10 | .25 | .00 |
| | UPPS-Prem | | | .07 | .38 |
| | UPPS-Pers | | | .00 | .97 |
| | UPPS-SS | | | .21 | .00 |
| | UPPS-PU | | | .10 | .21 |
| 2 | UPPS-NU | .14 | .00 | .22 | .00 |
| | UPPS-Prem | | | .07 | .37 |
| | UPPS-Pers | | | -.02 | .81 |
| | UPPS-SS | | | -.21 | .00 |
| | UPPS-PU | | | .12 | .14 |
| | ASRS-total | .07 | .33 | | |

Note: AUQ = Alcohol Use Questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

3.7.5. The role of impulsivity in the relationship between ADHD symptoms and nicotine use

Hierarchical linear regression results showed that adding gender and age in the first step accounted for 4.4% of the variance ($R^2 = .04$, F change = 6.48, $p = .002$) in NIC significantly

($F(2,285) = 6.48, p < .05, \beta(\text{age}) = .12, \beta(\text{gender}) = .14$). The two BIS subscales predicted significant additional variance ($R^2 = .10, F \text{ change} = 9.14, p = .000$), with BIS-motor significantly predicting higher NIC scores ($F(4,283) = 7.99, p < .05, \beta = .27$). In the third step, inattention symptom of ADHD contributed to the regression model significantly ($F(6,281) = 7.02, p < .05, \beta = .23$). This ADHD symptom, but not the total score accounted for 2.9% of the variance in NIC scores after accounting for facets of impulsivity ($R^2 = .13, F \text{ change} = 4.66, p = .010$) (Tables 3.13 and 3.14). Participants with higher inattention symptoms of ADHD had more nicotine use after accounting for the subscales of BIS-11.

Table 3.13

Hierarchical linear regression model predicting NIC scores from the two ADHD symptoms and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|------|
| 1 | BIS-motor | .10 | .06 | .27 | .00 |
| | BIS-nonplanning | | | -.06 | .38 |
| 2 | BIS-motor | .13 | .03 | .27 | .00 |
| | BIS-nonplanning | | | -.15 | .056 |
| | ASRS-IA | | | .23 | .00 |
| | ASRS-HI | | | -.11 | .13 |

Note: NIC = Nicotine use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Table 3.14

Hierarchical linear regression model predicting NIC scores from ADHD-total score and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .10 | .06 | .27 | .00 |
| | BIS-nonplanning | | | -.06 | .38 |
| 2 | BIS-motor | .11 | .01 | .24 | .00 |
| | BIS-nonplanning | | | -.09 | .20 |
| | ASRS-total | | | .10 | .14 |

Note: NIC = Nicotine use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Adding UPPS-P dimensions to a separate hierarchical linear regression indicated that higher negative urgency and sensation seeking predicted higher NIC scores significantly ($F(7,279) = 7.01, p < .05, \beta(SS) = .15, \beta(NU) = .28$) and they accounted for 10.3% of the variance ($R^2 = .15, F \text{ change} = 6.77, p = .000$). After adding UPPS-P dimensions, ADHD symptoms could not predict NIC significantly (Tables 3.15 and 3.16).

Table 3.15

Hierarchical linear regression model predicting NIC scores from the two ADHD symptoms and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .15 | .10 | .30 | .00 |
| | UPPS-Prem | | | -.04 | .58 |
| | UPPS-Pers | | | .11 | .10 |
| | UPPS-SS | | | .16 | .01 |
| | UPPS-PU | | | .08 | .33 |
| 2 | UPPS-NU | .15 | .00 | .28 | .00 |
| | UPPS-Prem | | | -.04 | .62 |
| | UPPS-Pers | | | .09 | .19 |
| | UPPS-SS | | | .16 | .01 |
| | UPPS-PU | | | .08 | .37 |
| | ASRS-IA | | | .05 | .52 |
| | ASRS-HI | -.02 | .80 | | |

Note: NIC = Nicotine use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

Table 3.16

Hierarchical linear regression model predicting NIC scores from ADHD-total score and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .15 | .10 | .30 | .00 |
| | UPPS-Prem | | | -.04 | .58 |
| | UPPS-Pers | | | .11 | .10 |
| | UPPS-SS | | | .16 | .01 |
| | UPPS-PU | | | .08 | .33 |
| 2 | UPPS-NU | .15 | .00 | .29 | .00 |
| | UPPS-Prem | | | -.04 | .58 |
| | UPPS-Pers | | | .10 | .13 |
| | UPPS-SS | | | .16 | .01 |
| | UPPS-PU | | | .09 | .30 |
| | ASRS-total | | | .03 | .71 |

Note: NIC = Nicotine use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

3.7.6. The role of impulsivity in the relationship between ADHD symptoms and cannabis use

The results of a hierarchical linear regression showed that age predicted cannabis use significantly ($F(2,290) = 4.14, p < .05, \beta = -.16$) and it accounted for 2.8% of the variance in CUDIT-R score ($R^2 = .03, F \text{ change} = 4.14, p = .02$). In the second step BIS-motor and BIS-non-planning predicted significant additional 15.7% of the variance in CUDIT-R scores ($F(4,288) = 16.27, p < .05, \beta(\text{BIS-motor}) = .22, \beta(\text{BIS-non-planning}) = .23$) ($R^2 = .18, F \text{ change} = 27.65, p = .000$). Adding ADHD symptoms to the third step accounted for an additional 34.6% of the variance in CUDIT-R scores ($R^2 = .53, \beta(\text{ASRS-IA}) = .49, \beta(\text{ASRS-HI}) = .20$). So, both ADHD symptoms and the ADHD-total score ($R^2 = .52, \beta = .69$) predicted cannabis use significantly above and beyond the facets of BIS-11 (Tables 3.17 and 3.18).

Table 3.17

Hierarchical linear regression model predicting CUDIT-R scores from the two ADHD symptoms and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .18 | .16 | .22 | .00 |
| | BIS-nonplanning | | | .24 | .00 |
| 2 | BIS-motor | .53 | .35 | .06 | .24 |
| | BIS-nonplanning | | | .03 | .52 |
| | ASRS-IA | | | .49 | .00 |
| | ASRS-HI | | | .30 | .00 |

Note: CUDIT-R = Cannabis Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Table 3.18

Hierarchical linear regression model predicting CUDIT-R scores from ADHD-total score and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .18 | .16 | .22 | .00 |
| | BIS-nonplanning | | | .24 | .00 |
| 2 | BIS-motor | .52 | .34 | .04 | .43 |
| | BIS-nonplanning | | | .00 | .98 |
| | ASRS-total | | | .69 | .00 |

Note: CUDIT-R = Cannabis Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

The next hierarchical linear regression results showed that negative urgency and sensation seeking contributed to the model significantly ($F(7,284) = 17.36, p < .05, \beta(SS) = .20, \beta(NU) = .17, R^2 = .30$) and accounted for an additional 27.2% of the variance in CUDIT-R scores. Adding ADHD symptoms scores separately ($F(9,282) = 38.21, p < .05, \beta(ASRS-IA) = .45, \beta(ASRS-HI) = .27, R^2 = .55$) and as a total score ($F(8,283) = 42.06, p < .05, \beta(ASRS-total) = .64, R^2 = .54$) also predicted cannabis use significantly and accounted for additional 25% of the variance in CUDIT-R score (Tables 3.19 and 3.20).

Table 3.19

Hierarchical linear regression model predicting CUDIT-R scores from the two ADHD symptoms and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .30 | .27 | .17 | .01 |
| | UPPS-Prem | | | .01 | .83 |
| | UPPS-Pers | | | .19 | .10 |
| | UPPS-SS | | | .10 | .04 |
| | UPPS-PU | | | .28 | .21 |
| 2 | UPPS-NU | .55 | .25 | .04 | .51 |
| | UPPS-Prem | | | .03 | .55 |
| | UPPS-Pers | | | .02 | .67 |
| | UPPS-SS | | | .09 | .05 |
| | UPPS-PU | | | .10 | .10 |
| | ASRS-IA | | | .46 | .00 |
| | ASRS-HI | .27 | .00 | | |

Note: CUDIT-R = Cannabis Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

Table 3.20

Hierarchical linear regression model predicting CUDIT-R scores from ADHD-total score and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .30 | .27 | .17 | .01 |
| | UPPS-Prem | | | .01 | .83 |
| | UPPS-Pers | | | .19 | .06 |
| | UPPS-SS | | | .10 | .00 |
| | UPPS-PU | | | .28 | .21 |
| 2 | UPPS-NU | .54 | .24 | .01 | .85 |
| | UPPS-Prem | | | .02 | .69 |
| | UPPS-Pers | | | .05 | .29 |
| | UPPS-SS | | | .09 | .03 |
| | UPPS-PU | | | .08 | .21 |
| | ASRS-total | | | .64 | .00 |

Note: CUDIT-R = Cannabis Use Disorder Identification Test; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; NU = Negative Urgency; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking;

A hierarchical linear regression model was fit predicting the CAN from different dimensions of BIS, UPPS-P and ASRS scores. In the first step, gender predicted CAN score significantly ($F(2,292) = 5.40, p < .05, \beta = .14, R^2 = .03$). Both age and gender accounted for 3.6% of the variance in CAN scores. Adding BIS sub-scales to the second step of the model accounted

for an additional 4.6% of the variance in CAN score with BIS-motor predicting more CAN score ($F(4,290) = 6.48, p < .05, \beta = .22, R^2 = .08$). Adding ADHD symptoms to the third step showed that ASRS-IA, ASRS-HI and ASRS-total score accounted for 0% of the variance in CAN scores (Tables 3.21 and 3.22).

Table 3.21

Hierarchical linear regression model predicting CAN score from the two ADHD symptoms and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .07 | .06 | .16 | .02 |
| | BIS-nonplanning | | | .10 | .12 |
| 2 | BIS-motor | .07 | .00 | .16 | .02 |
| | BIS-nonplanning | | | .09 | .20 |
| | ASRS-IA | | | .02 | .71 |
| | ASRS-HI | | | -.02 | .79 |

Note: CAN = Cannabis Use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

Table 3.22

Hierarchical linear regression model predicting CAN score from ADHD-total score and BIS subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|-----------------|-------|--------------|------|-----|
| 1 | BIS-motor | .07 | .05 | .16 | .02 |
| | BIS-nonplanning | | | .10 | .12 |
| 2 | BIS-motor | .07 | .00 | .16 | .02 |
| | BIS-nonplanning | | | -.10 | .15 |
| | ASRS-total | | | .01 | .93 |

Note: CAN = Cannabis Use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; BIS = Barratt Impulsiveness Scale

The results of a separate hierarchical regression show that adding UPPS-P dimensions to the second step, after age and gender in the first step, accounted for significant additional 4% of the variance in CAN scores, which was significant ($F(7,286) = 2.37, p < .05, R^2 = .05$).

ADHD symptoms and ADHD-total could not predict higher scores significantly in this questionnaire after adding UPPS-P sub-scales (Table 3.23 and 3.24). Thus, based on the data

analysis, not BIS-11 nor UPPS-P could not predict cannabis use in participants using CAN and the symptoms of ADHD did not explain additional variance in predicting CAN score over and above the facets of impulsivity.

Table 3.23

Hierarchical linear regression model predicting CAN score from the two ADHD symptoms and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .05 | .04 | .13 | .09 |
| | UPPS-Prem | | | .06 | .45 |
| | UPPS-Pers | | | -.00 | .90 |
| | UPPS-SS | | | -.10 | .11 |
| | UPPS-PU | | | .01 | .88 |
| 2 | UPPS-NU | .05 | .00 | .11 | .19 |
| | UPPS-Prem | | | .06 | .43 |
| | UPPS-Pers | | | .02 | .74 |
| | UPPS-SS | | | .09 | .12 |
| | UPPS-PU | | | .02 | .80 |
| | ASRS-IA | | | .04 | .59 |
| | ASRS-HI | -.01 | .90 | | |

Note: CAN = Cannabis Use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; Negative Urgency = NU; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking.

Table 3.24

Hierarchical linear regression model predicting CAN score from ADHD-total score and UPPS-P subscales scores

| Models | Predictors | R^2 | R^2 change | Beta | Sig |
|--------|------------|-------|--------------|------|-----|
| 1 | UPPS-NU | .05 | .04 | .13 | .09 |
| | UPPS-Prem | | | .06 | .45 |
| | UPPS-Pers | | | -.00 | .90 |
| | UPPS-SS | | | -.10 | .11 |
| | UPPS-PU | | | .01 | .88 |
| 2 | UPPS-NU | .05 | .00 | .11 | .15 |
| | UPPS-Prem | | | .06 | .44 |
| | UPPS-Pers | | | -.01 | .79 |
| | UPPS-SS | | | -.10 | .11 |
| | UPPS-PU | | | .02 | .75 |
| | ASRS-total | | | .04 | .50 |

Note: CAN = Cannabis Use questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; UPPS = Urgency Premeditation Perseverance Sensation-seeking; Negative Urgency = NU; PU = Positive Urgency; PRE = (lack of) Premeditation; PER = (lack of) Perseverance; SS = Sensation-seeking

3.8. Discussion

This study is the first to explore the involvement of different impulsivity facets in the relationship between each ADHD symptom separately and the use of alcohol, cannabis and nicotine by examining a spectrum of symptoms in a typically developing population. The

results of this investigation indicated that certain facets of impulsivity predict the use of alcohol, cannabis and nicotine and that ADHD symptoms accounted for unique variance in alcohol, cannabis and nicotine use.

BIS-motor was found to be a significant predictor of high scores in all substance use questionnaires in the current analysis. *Motor impulsiveness* is the ability to act without thinking and on the spur of the moment (Bari et al., 2016; Patton et al., 1995). The result of the current study is consistent with the findings of some previous investigations (Balodis et al., 2010; Bjork et al., 2004; Chivers et al., 2016; García-Montes et al., 2009; Goudriaan et al., 2007; Lawrence et al., 2009; Mitchell et al., 2005). For instance, Moreno et al. (2012) discovered that participants with higher cannabis and alcohol use scored higher on the BIS-11 motor subscale than the control group.

Different factors can explain the connection between motor impulsiveness and substance misuse. Previous studies show that motor impulsiveness is significantly correlated with inhibition control (Enticott et al., 2006). Moreover, Caswell et al. (2013) found that the underlying mechanisms of impulsivity's motor dimension are engaged in inhibitory control. In one investigation, people who smoked cannabis were slower in the stop-signal task than the participants who smoked placebo (Ramaekers et al., 2006), indicating more motor impulsiveness in those with cannabis misuse (Prashad & Filbey, 2017). High motor impulsiveness may be specifically associated with impaired response inhibition in those who misuse substances.

Shared parts of the brain may also explain the relationship between motor impulsiveness and substance misuse. Neuroimaging studies have highlighted changes in cannabis-related brain networks (Batalla et al., 2013). Investigators found a stronger functional connection between the cortical (PFC) and subcortical (substantia nigra and

subthalamic nucleus) regions of the brain in those with cannabis misuse, resulting in more effort to stop an ongoing response. This finding may also explain the previously mentioned association between motor impulsiveness and response inhibition (Filbey & Yezhuvath, 2013). Cannabis misuse also reduces the neural effectiveness of the motor adaptation part of the brain, which is located in the PFC and basal ganglia (Gentili et al., 2015). In other studies, people with chronic cannabis misuse were shown to have reduced anterior cingulate activity during inhibitory control (Gruber & Yurgelun-Todd, 2005; Hester et al., 2009). In those with AUD, maladaptive changes in the medial PFC (a brain part that monitors ongoing actions) were detected. Deficits in this area lead to more compulsive reactions (Wang et al., 2016). Ventrolateral PFC and DLPFC are involved in response inhibition, presumably exerting executive control over the motor response (Criaud & Boulinguez, 2013). A meta-analysis of 24 fMRI investigations using a stop-signal task reported that the posterior cingulate cortex is the brain region chiefly involved in response inhibition (Cieslik et al., 2015). Nicotine smokers show deficits in the area of the brain that is engaged in motor impulsiveness and response inhibition (Potvin et al., 2015). It can be inferred that there is a significant association between acting quickly without thought and impaired response inhibition in impulsive actions and increased use of alcohol, cannabis and nicotine. This conclusion is also consistent with the results of Study 1, which found that response inhibition significantly predicted the use of alcohol, cannabis and nicotine. Therefore, people who misuse substances are more impaired in their response inhibition and have a higher motor impulsiveness score. Longitudinal investigations are needed to explore the cause-and-effect relationship between response inhibition, motor impulsiveness and substance misuse. Interventions such as mindfulness or cognitive rehabilitation therapies have been shown to

help increase response inhibition in those with ADHD or substance misuse (Andreu et al., 2018; Bailey et al., 2019; Yazdanbakhsh et al., 2018).

Nonplanning is another subscale of BIS, which measures the lack of future planning and forethought (Meule, 2013). The current results indicated that the BIS-11 nonplanning subscale predicted cannabis misuse. Other studies also found that those with cannabis use have higher total scores in self-report questionnaires that measure impulsivity, such as BIS-11 (Gruber et al., 2011; Silveri et al., 2011). Additionally, prospective research outcomes include higher scores in cognitive and motor impulsivity (Silveri et al., 2011) and declined orientation indexed in the BIS-nonplanning subscale (Churchwell et al., 2010). Shared parts of the brain could be one reason for developing cannabis misuse in those with a higher score on the BIS-nonplanning subscale. For instance, Churchwell et al. (2010) demonstrated that a volume reduction in the medio-orbital PFC was seen in those with cannabis misuse and individuals with higher scores on the BIS-nonplanning subscale.

The negative urgency and sensation seeking subscales of the UPPS-P were the other dimensions of impulsivity that predicted further substance misuse. Some investigations have proposed possible reasons for the association between negative urgency and alcohol misuse. For example, Cyders et al. (2014b) demonstrated a connection between negative urgency and increased inhibitory brain activity during negative emotion. The ventromedial PFC's activity can mediate the relationship between urgency and alcohol misuse during alcohol-related cues (Cyders et al., 2014a). Thus, those with a higher negative urgency score have more impaired response inhibition during negative emotions, which predicts increased alcohol consumption.

According to Anestis et al. (2007), negative urgency is still strongly correlated with alcohol use behaviours when negative affect is controlled. Thus, its effects on drinking cannot be explained solely by affect. Concentrating on the reasons why people with higher

negative urgency drink alcohol can help us better comprehend the association between alcohol use and negative urgency. Drinking motives refer to an individual's motivations for consuming alcohol (Cooper et al., 1992a, 1992b; Cooper, 1994). Previous studies suggest that there are two affective motives: enhancing positive affect and coping with adverse ones, both of which may account for negative urgency's impact on alcohol use (Carey & Correia, 1997; Jones et al., 2014; Kuntsche et al., 2008; Merrill & Read, 2010; Read et al., 2003). Anthenien et al. (2017) found that those with more positive expectations about alcohol use outcome are more motivated to drink, connoting that there are significant indirect influences on negative urgency and alcohol consumption. Jones et al. (2014) also reported that urgency is related to different motives for drinking and the tendency to engage in alcohol-related risky behaviours. They recommended that screening for high levels of urgency and severe drinking consequences could be useful predictors of problems related to alcohol misuse among university students in the UK. The researchers discovered that people with higher negative urgency had a stronger desire to consume alcohol to minimise negative feelings and increase positive ones. People with riskier behaviours may display more coping and enhancement motives during negative emotions. Moreover, negative urgency mediated alcohol use and coping and enhancement motives (Adams et al., 2012; Fischer et al., 2004; Jones et al., 2014).

Based on the current study's results and previous investigations, this strong association between alcohol use and negative urgency may be attributed to different factors. One of them is using alcohol to cope with negative emotions through riskier drinking and failing to adopt other coping strategies (Merrill & Read, 2010; Smith et al., 2007). The other might be a result of poor response inhibition in negative emotional situations. The results of Study 1 showed that response disinhibition was a significant predictor of alcohol use.

Whiteside et al. (2005) used the UPPS (Whiteside & Lynam, 2001) to measure four dimensions of impulsivity in a group of young adults. Among the subscales of the UPPS, urgency significantly predicted nicotine dependence. Other studies also showed that higher levels of urgency can predict elevated levels of craving (Billieux et al., 2007). Urgency can also indicate the increased risk of daily smoking (Lee et al., 2015) and its severity (Pang et al., 2014).

In another study, a positive association between smoking, negative affect, psychological distress and emotional dysregulation has been found in adolescents (Gutman et al., 2011; Hu et al., 2008). These studies revealed that adolescents with higher urgency have higher expectations from smoking in terms of negative and positive reinforcement properties. They are motivated to smoke during both negative and positive moods, which increases their expectation of smoking's effects on affect or mood improvement.

Negative urgency predicted cannabis use as reflected in the current study (Kaiser et al., 2012; Robinson et al., 2014). Kaiser et al. (2012) demonstrated that negative urgency was a major predictor of alcohol, nicotine and cannabis use. The current research revealed a critical link between negative urgency and impulsive personality traits and the use of alcohol, cannabis and nicotine. Exploring how negative urgency can lead to impulsive behaviours is necessary to grasp the connection between negative urgency and substance misuse. Even though previous investigations reported an association between negative urgency and risky behaviour, the reason for impulsive behaviour in situations with negative affect is not apparent. It might be that some individuals use dangerous behaviours, such as substance misuse, as a coping mechanism for their negative affect. However, impulsive behaviour may not have a coping purpose, but the negative emotion might interfere with cognitive processes and result in poor risk evaluation (Shields et al., 2016). Thus, if negative urgency represents

risky behaviour to cope with negative affect, it could be helpful to teach the individual alternative coping strategies instead of misusing substances. If those individuals cannot use specific cognitive processes when distressed, working on problem-solving skills and being more mindful of emotions and impulses can be beneficial. The most considerable risk for those who struggle to cope with their symptoms is that they may choose an unhealthy or inappropriate coping strategy, leading to more problems in their daily lives, such as developing SUD or increased substance misuse, with all their negative implications (Jones et al., 2014). Therefore, selecting the best coping strategy with the help of a professional could decrease the risk.

Sensation seeking is the individual's propensity to enjoy and follow exciting, new, novel and complex experiences, even if they are physically or socially risky or dangerous (Jung et al., 2018; Zuckerman, 1975, 1994). Studies have shown cross-sectional and longitudinal connections between high sensation seeking and increased substance misuse and its consequences, including nicotine, alcohol and cannabis use (Arnett, 1996; Crawford et al., 2003; Kaynak et al., 2013; Keyes et al., 2015; MacPherson et al., 2010; Malmberg et al., 2012; Miles et al., 2001; Patrick & Schulenberg, 2010; Schulenberg et al., 1996). Sensation seeking is a strong indicator of alcohol use (Jones et al., 2014; MacPherson et al., 2010) and binge drinking among adults and adolescents (Doumas et al., 2017; Sargent et al., 2010; Strautz & Cooper, 2013). It is also the strongest predictor of substance misuse in university students (Jaffe & Archer, 1987) and poly-drug use in cross-cultural samples (Pokhrel et al., 2010). There is a significant association between sensation seeking and early onset (Malmberg et al., 2012), higher substance misuse levels (Bekman et al., 2010; Crawford et al., 2003; Urbán et al., 2008) and increased SUD (Adams et al., 2012; Alterman et al., 1990; Jaffe & Archer, 1987; Meil et al., 2016, Ortin et al., 2012; Stautz & Cooper, 2013). Long-

term substance misuse is uncommon among university students, but investigations indicate that sensation seeking is the strongest predictor of substance misuse (Dvorak & Day, 2014b; Meil et al., 2016; Spillane et al., 2010; Stautz & Cooper, 2013).

A recent study found a connection between sensation seeking, binge drinking and cannabis use in women in their mid to late 20s, but not in men (Evans-Polce et al., 2018). This correlation suggests that young adults of different genders have different risk factors for substance misuse. Sensation seeking is also a strong predictor of substance misuse in women. The current study's findings may be influenced by the fact that the majority of the participants were women.

It has been suggested that, as with other facets of impulsivity and EF, sensation seeking is governed by the frontal regions of the brain (Joseph et al., 2009; Santesso et al., 2008). According to research, the association between sensation seeking and heightened substance misuse is because of higher levels of physiological reward responses due to immature cognitive control (located in the PFC) for substance misuse behaviours (Bardo et al., 1996; Kaynak et al., 2013). The relationship between sensation seeking and later-developed SUD in adolescence and adulthood can be mediated by earlier initiation of substance misuse (Charles et al., 2016; Steinberg, 2008). Many longitudinal studies indicate that increased sensation seeking can raise the risk of substance misuse in adolescence (Crawford et al., 2003; MacPherson et al., 2010) and young adulthood (Knafo et al., 2013). Thus, helping people with higher sensation seeking levels and more impulsive actions, such as substance misuse, find a safer, healthier, exciting and novel activity (sports, adventurous trips to help others, etc.) can reduce the risk of developing substance misuse.

The current study's results contributed to the field by revealing that ASRS-IA, ASRS-HI and ASRS-total scores significantly predicted alcohol, nicotine, and cannabis use. While

BIS-motor, negative urgency and sensation seeking were impulsivity facets that predicted AUDIT and AUQ scores in the current study, the inattention predicted alcohol use over and above these facets of impulsivity. This ADHD questionnaire (ASRS) score showed a substantial improvement in the proportion of the explained variance in developing alcohol usage after accounting for impulsivity facets. This outcome could denote that higher inattention scores raise the risk of alcohol use in ways other than impulsivity. Therefore, those with higher inattention scores use more alcohol for reasons other than high BIS-motor, sensation seeking and negative urgency. People with higher hyperactivity/impulsivity symptom also use more alcohol through higher BIS-motor, sensation seeking and negative urgency score pathways.

The facets of impulsivity that predicted cannabis use were BIS-motor, BIS-non planning and negative urgency. Both ADHD symptoms also predicted cannabis use over and above the aspects of impulsivity, indicating that there could be other factors that increase the likelihood of developing cannabis dependence in people with higher ADHD symptoms.

BIS-motor, negative urgency and sensation seeking subscales predicted the NIC questionnaire's scores. Moreover, the inattention symptom of ADHD showed a significant improvement in the proportion of the explained variance in developing nicotine use. It could be established that this ADHD symptom may increase the risk of nicotine use through a non-impulsivity pathway. Similar to the results of alcohol use, those with hyperactivity/impulsivity symptoms of ADHD use higher amounts of nicotine through the BIS-motor, sensation seeking, and negative urgency pathways, whereas those with more inattention symptoms of ADHD use more nicotine via other routes. These findings are noteworthy because motor impulsiveness, sensation seeking and negative urgency are not generally considered part of the ADHD phenotypes. The present study suggested that

behaviours related to these facets of impulsivity can explain some of the impairments associated with ADHD symptoms.

In Chapter 2 of this thesis, Study 1 investigated EF facets in the relation between ADHD and substance misuse. Adaptive thinking (cognitive flexibility), time-based prospective memory and creative thinking were found to be other pathways that could raise the risk of misusing alcohol, cannabis and nicotine in individuals with ADHD symptoms. Studies 1 and 2 of this thesis have provided helpful information about the relationship between ADHD, impulsivity, EF and substance misuse. In these two studies, hyperactivity/impulsivity accounted for a particular variance in cannabis and nicotine use after accounting for the facets of EF. Additionally, inattention explained additional variance in alcohol, nicotine and cannabis use over and above the aspects of impulsivity. This result means that those with inattention symptom will develop other substance misuses through the EF facet pathways, while those with hyperactivity/impulsivity symptoms of ADHD can develop them via the impulsivity facet routes. In these studies, the spectrum of symptoms in a typically developing population is being examined, not the of ADHD. As a result, investigating each individual's personality traits and impairments could be more effective in discerning the best treatment option for them to minimise their risk of substance misuse. Future longitudinal investigations are recommended to determine the cause-and-effect relationship between the variables of this study.

This study was the first to divide the symptoms of ADHD into two main categories: inattention and hyperactivity/impulsivity. Additionally, each facet of impulsivity was measured separately using two tests (UPPS-P and BIS-11). Alcohol, cannabis and nicotine were found to be the most commonly taken substances by university students. The results of this study shed light on the relationship between each facet of impulsivity and substance

misuse, as well as whether each ADHD symptom accounts for a statistically significant amount of variance in substance misuse after impulsivity facets are taken into account.

In AUDIT-based investigations, 62.8% of students reported hazardous alcohol use in 2003, rising to 84% in 2014 (Davoren et al., 2016). A cross-sectional survey of students at a Northern England university in 2010 using a university-wide sampling frame reported that 82% of the students received an AUDIT score of 8 or above, which was higher than previous years (Beenstock, 2010). In 2011, Heather et al. (2011) measured hazardous drinking across seven universities in the UK, finding that 60.6% of the participants reported hazardous alcohol consumption. Another study of university students in North West England found that 71.2% had engaged in hazardous drinking (Gunby et al., 2012). After providing questionnaires to sports venues at ten UK universities, research with sports students showed that 84% of the sample registered hazardous alcohol use (O'Brien et al., 2014). These studies report higher proportions of dangerous alcohol use among university students across the UK than the current research (28.4%), which may be attributed to different reasons including the year of the studies, the number of women and men in the sample and the time of the year that the study was conducted. Previous investigations show that men report higher rates of hazardous alcohol use than women (Beenstock et al., 2010; Faulkner et al., 2006), whereas the participants of the current study were mainly women (81.5%).

Past studies reveal that cannabis was the most commonly used drug among university students in the UK (57%) (Webb et al., 1996). Furthermore, Newbury-Birch et al. (2001) indicated that the most widely used illegal drug among a sample of first-year medical students was cannabis (51%). According to NHS Digital, cannabis usage among 16- to 24-year-olds in England and Wales was 17% in 2018–2019, the highest level in a decade. The current study results found that 8.2% of the participants yielded a CUDIT score of 8 or

higher, connoting hazardous cannabis use. Moreover, 24.9% of participants were current cannabis users and 49.9% of the sample had lifetime cannabis use. The difference between the current cannabis use rates in the present study's sample and previous investigations could be due to the year of the investigations. Based on NHS reports, the prevalence of cannabis misuse has declined in the past decades (NHS Digital, 2018). The percentage of hazardous cannabis use in the current sample was closer to the NHS Digital reports.

Moreover, the present research discovered that 7.6% of participants smoked nicotine for all 30 days of the past month, but harmful nicotine use was not measured. The reports of the NHS (2019) show that adults aged 25 to 34 (19%) were the most likely to smoke, followed by those aged 18 to 24 (17%). In a study conducted at the University of Birmingham, UK, the nicotine usage of 934 university participants was assessed; their results showed that the current smoking prevalence was 14% (Bartington et al., 2020). The disparity in rates between the present investigation and Bartington et al.'s (2020) study may be due to the latter having a larger number of participants ($n = 38000$) than the former ($n = 380$). Their sample population was more diverse, involving undergraduates, postgraduates and university employees, while the present study only recruited first-year psychology undergraduates. Furthermore, no cutoff score for nicotine use in the sample was used in the current study, and more research on nicotine use in university students is required. Data on nicotine frequency and quantity over the past 30 days is presented in Table 3.1.

3.9. Limitations

This study provided valuable information about the role of different facets of impulsivity in the relationship between ADHD and the use of alcohol, cannabis and nicotine.

The study's large sample size ($n = 380$) was one of its strengths but having a sample of first-year undergraduates limits the generalisability of the results to a more extensive and diverse population. Participants of this study were primarily women; hence, studying the same number of men and women in future studies is recommended to improve the diversity of the sample.

Furthermore, this was a cross-sectional research with several advantages: (a) does not manipulate variables; (b) allows investigators to test numerous characteristics at once; (c) assess the prevailing features in a population; (d) provides information about what is going on in a given population; and (e) describes characteristics that exist in a community.

However, this kind of study is unable to determine a cause-and-effect relationship between variables and only measures different variables and conditions at a specific point in time (Mann, 2003). Although longitudinal investigations are comparably more effective in determining causal relationships, they are more expensive and time-consuming, which would pose significant challenges for a PhD investigation with limited time and budget. Thus, future research should focus on longitudinal and clinical studies with a more diverse sample. In addition, the lack of data on the sample's ethnicity prohibited comparisons between ethnic groups. No information on socioeconomic status was collected as well. Future analyses could explore how ethnicity and sociocultural factors influence the associations observed here.

A further limitation was assessing emotional dysregulation, substance misuse or ADHD symptoms based solely on self-reports. Many studies have found that participants' accounts of their own experiences are biased (Devaux & Sassy, 2016). Self-reports' social desirability bias may be reduced by using interviews and questionnaires with friends and family members (Althubaiti, 2016).

Finally, the current study's hypotheses were non-directional, implying that the independent variables would influence the dependent variables. Since their directions were not specified, they do not indicate whether the relationships are positive or negative. Thus, future investigations into the direction of the effects among variables should use one-tailed hypotheses.

3.10. Conclusion

On the one hand, the present study's findings convey that alcohol and nicotine misuse can be predicted by motor impulsiveness, negative urgency and sensation seeking. Coupled with these three subscales, BIS-nonplanning, on the other hand, predict cannabis misuse. This research extends the literature by showing that the inattention symptom accounted for unique variance in substance misuse in the sample over and above the facets of impulsivity, denoting that those with inattention symptom misuse higher amounts of alcohol, cannabis and nicotine through other pathways apart from the impulsivity facet route.

Past investigations revealed that people with ADHD use a variety of substances to cope with their symptoms (Rabiner et al. 2008) and live an improved daily life. In certain situations, they may prefer smaller but immediate rewards (substance misuse) to bigger yet delayed incentives (suitable coping strategy). They also have various impairments in their EF, decision-making and emotional regulation, leading them to use higher amounts of substance without regard to its negative consequences. This probability could result in substance misuse and increased problems in their daily lives. Hence, the following chapter concentrates on emotional dysregulation as a common issue among adults with ADHD symptoms and substance misuse.

Chapter 4

4. The role of emotional regulation in the relationship between ADHD symptoms and substance misuse

Overview

This chapter describes a cross-sectional analysis of first-year undergraduate students (aged 18 and above). The study's main aim was to explore whether emotional regulation mediates the relationship between the two ADHD symptom clusters (inattention and hyperactivity/impulsivity) and alcohol, cannabis, and nicotine use. Results showed that although emotional regulation was a partial mediator between hyperactivity/impulsivity and alcohol and cannabis use, it was not a full mediator in the model. Emotional regulation also did not mediate the relationship between hyperactivity/impulsivity and nicotine use. Furthermore, inattention, emotional regulation and any substance misuse scores did not have a mediation relationship.

4.1. Introduction

In the previous chapters, the role of EF and impulsivity facets in the relationship between ADHD symptoms and substance misuse was investigated. After accounting for EF facets, the results revealed that hyperactivity/impulsivity explained the unique variance in substance misuse. In contrast, inattention explained additional variance in substance misuse after accounting for impulsivity facets. The present study was an attempt to explore other pathways from ADHD symptom clusters to substance misuse. It investigated whether ADHD

symptom clusters could predict alcohol, cannabis and nicotine use via an emotional regulation pathway.

Individuals with ADHD symptoms exhibit emotional dysregulations in their daily lives (Baran Tatar et al., 2015). ADHD is also a comorbid condition for most people who misuse substances (Kober 2014; Yildirim & Goka, 2005). Thompson (1994) defines *emotional regulation* as the ability to adapt an emotional state to support goal-oriented and adaptive behaviours. *Emotional dysregulation* then refers to impaired adaptive processes that result in behaviours that prevent a person from reaching their goals. These behaviours include (a) emotionally inappropriate and extreme emotional expressions and experiences, (b) fast and impaired emotional shifts and (c) inattention to emotional stimuli (Richard-Lepouriel et al., 2016; Shaw et al., 2014). Emotional dysregulation causes a person to be quick to anger, excitable and more prone to extreme mood lability and potentially socially inappropriate emotional expressions (Shaw et al., 2014). This chapter focuses on the connection between emotional dysregulation, ADHD symptoms and the use of alcohol, cannabis and nicotine.

4.2. Emotional regulation and ADHD symptoms

Emotional regulation difficulties are present in 25% to 45% of children with ADHD symptoms and 30% to 70% of adults with ADHD symptoms (Barkley & Fischer, 2010; Shaw et al., 2014; Stringaris & Goodman, 2009; Surman et al., 2013). Wehmeier et al. (2010) indicated that people with ADHD symptoms who do not have emotional dysregulation have fewer difficulties in their relationships, family and academic life and occupational achievements than those who do. Since it causes social and occupational impairments

(Barkley & Murphy, 2010), growing evidence from neuroimaging, clinical and genetic investigations recommends that emotional dysregulation should be one of the main components of ADHD, at least in a subgroup of individuals (Retz et al., 2012a, 2012b). Longitudinal studies are likely to be significant in understanding the development and interaction of emotional dysregulation and the main symptoms of ADHD (Banaschewski et al., 2012; Shaw et al., 2014; Skirrow & Asherson, 2013).

Many investigators agree that, in addition to the other two main symptoms of ADHD, emotional dysregulation is a third element (Shaw et al., 2014). Even though emotional dysregulation has been a core diagnostic feature of ADHD in some studies (Barkley & Fischer, 2010; Vidal et al., 2014), many individuals with ADHD symptoms do not appear to exhibit any significant emotional dysfunction (Shaw et al., 2014). Therefore, it is not currently a part of the DSM-5 criteria.

One study has found an association between all facets of ADHD and emotion dysregulation (Becker et al., 2006). However, most research suggests a link between emotional lability (irritable moods with volatile and changeable emotions), the hyperactivity/impulsivity ADHD symptom and the combined type of ADHD in children and adults, but not the inattentive type (Skirrow & Asherson, 2013; Sobanski et al., 2010). Furthermore, Reimherr and colleagues (2015) indicated that individuals with higher hyperactivity/impulsivity symptoms of ADHD had more emotional dysregulation symptoms than those with higher inattention symptoms. Emotional dysregulation may be used as a diagnostic criterion for the hyperactivity/impulsivity variant of ADHD.

Even though there is mixed evidence from other studies, it proposes that those with ADHD and emotional dysregulation may form a distinct genetic group, with siblings of probands with impaired emotional regulation and ADHD also presenting high rates of ADHD symptoms and emotional lability (Shaw et al., 2014). There are two proposed hypotheses based on the underlying pathophysiological mechanisms of emotional dysregulation. The first is the ‘dyscontrol hypothesis’, which states that emotional dysregulation in ADHD is associated with EF impairments in those with ADHD and that top-down inhibitory process deficits will result in impaired emotional reactions, even though emotional processing per second would be as expected (Posner et al., 2014). The second is the ‘affectivity hypothesis’, which asserts that dysfunctions in bottom-up circuits responsible for processing emotional stimuli, such as the amygdala, OFC and the ventral striatum, contribute to abnormal emotional processing and emotional dysregulation in those with ADHD symptoms (Lenzi et al., 2018). In light of the contradictory findings of previous investigations on the relationship between emotional dysregulation and inattention and hyperactivity/impulsivity, the present study was an attempt to explore the connection between each ADHD symptom cluster, emotional regulation and substance misuse.

Researchers used different tools to measure emotional regulation in adults and children with ADHD symptoms. According to Banaschewski et al. (2012), Shaw et al. (2014) and Sorensen et al. (2011), the most common tools to assess emotional regulation in people with ADHD are the Conners Rating Scales (Conners et al., 1998), the Child Behaviour Checklist (CBCL) (Achenbach & Dumenci, 2001) and the Behaviour Rating Scale of Executive Function (BRIEF) (Gioia et al., 2002).

4.3. Emotional regulation and substance misuse

In the US, the Substance Abuse and Mental Health Services Administration (Results from the 2012 National Survey on Drug Use and Health, 2013) reported that most university students drink alcohol in a month, with 40% binge drinking. Alcohol misuse leads to poor academic performance (Martinez et al., 2014; Singleton, 2007), accidents (Shults et al., 2009) and greater risky behaviours (Hahn et al., 2015a, 2015b).

Heather et al (2011) used the AUDIT to evaluate 770 undergraduates from seven UK universities in a cross-sectional study. The results indicated that 65% of men and 58% of women scored 8+ on the AUDIT, meaning harmful or hazardous drinking. Moreover, 40% of the participants were hazardous drinkers, 11% were harmful drinkers and 10% were likely to have alcohol-use dependence.

There is a notable link between emotional dysregulation and substance misuse symptoms (Aldao et al., 2010; Aldao & Nolen-Hoeksema 2010; Gaher et al., 2013; Gratz & Tull, 2010). Maladaptive coping strategies and increased impulsive behaviour during emotional distress represent decreased emotional flexibility and may indicate a change in preference from self-control to affect regulation (Tice et al., 2001). In emotional situations, people who misuse substances have been reported to show more impulse control disorders than those who do not misuse substances (Kisa et al., 2005).

Researchers discovered a relationship between emotional dysregulation and alcohol misuse (Gaher et al., 2014; Hahn et al., 2015a, 2015b; Simons & Gaher, 2005). Additionally, research into potential mediators of emotional dysregulation and alcohol misuse has begun (Cavicchioli et al., 2019; Dragan, 2015). In one study, positive metacognitions, which defines

as an awareness of one's own thought processes (Metcalf & Shimamura, 1994), but not negative ones, were found to mediate the relationship significantly. Simons et al. (2017) tested 435 university students using the Difficulties in Emotion Regulation Scale (DERS). Their results showed that when alcohol is used to reduce negative emotions or increase positive ones, alcohol misuse and subsequent AUD increase. Furthermore, Dvorak et al. (2014a) measured alcohol use and emotional regulation difficulties in 1,758 university students. Their findings indicated that emotional dysregulation and lack of emotional clarity is positively associated with alcohol-related consequences.

Based on NHS Digital Statistics (2018), 14.4% of the English population currently smokes. This figure fell from 19.8% in 2011 to 14.9% in 2017, a decline of around 1.8 million adult smokers in six years. Adults aged 25 to 34 were the most likely to smoke (19%), followed by 18 to 24 (17%). The majority in this age group are university students. Those who are 65 and over (8%) were the least likely to smoke.

The nicotine withdrawal symptoms experienced by smokers during their quit attempt may be one factor that makes quitting smoking highly difficult. They are most likely to relapse due to withdrawal symptoms (Fiore et al., 2008). Negative affect is one of the symptoms (Hughes et al., 1990) that smokers have reported more than non-smokers (Kassel et al., 2003). According to studies, smokers claim that smoking helps them cope with their emotional distress; it can also reduce their negative affect, a strong smoking trigger (Copeland et al., 1995; Piper et al., 2004).

Based on the tobacco negative affect model, the propensity to experience negative affect and emotional dysregulation together cause quitting difficulties (Baker et al., 2004; Brown et

al., 2005; Kenford et al., 2002). More recent studies support this theory by showing that emotional dysregulation is associated with smoking behaviour (Adams et al., 2012; Johnson et al., 2012). Emotional dysregulation predicts smoking in people with depression, impaired cognitive processes and anxiety sensitivity (Adams et al., 2012; Johnson et al., 2012). Johnson and McLeish (2016) indicated that higher negative affect is linked to a greater desire to smoke to alleviate negative emotions. Additionally, quitting smoking is problematic because it raises negative feelings, and managing negative emotions becomes more challenging without it. Therefore, emotional dysregulation is an essential target for smoking cessation interventions (Johnson & McLeish, 2016).

Self-medication and negative-reinforcement theories of substance misuse indicate that emotional processes and related disturbances are primary contributors to substance misuse and its consequences (Baker et al., 2004; Duncan, 1976; Khantzian, 1985). Low emotional awareness (Thorberg et al., 2009) and emotional dysregulation (Sher & Grekin, 2007) are two emotional factors that have received attention. Emotional awareness is considered to have two dimensions: (a) the extent of attention to emotions and (b) emotional clarity, which refers to identifying and understanding the types and sources of emotions (Boden & Berenbaum, 2012; Boden et al., 2013; Coffey et al., 2003; Gohm & Clore, 2000, 2002). Low emotional awareness, particularly poor emotional clarity, is an independent and significant predictor of psychopathologies, including SUD (Berenbaum et al., 2006; Berenbaum et al., 2012; Boden & Berenbaum, 2012; Thorberg et al., 2009).

According to the National Health Service (NHS, 2017) and Substance Abuse and Mental Health Services Administration (SAMHSA, 2011), cannabis is the most widely used illegal substance in the UK and worldwide. Different forms of emotional dysregulation are linked to

cannabis misuse (Simons & Carey, 2002). An association between emotional clarity and cannabis use has also been found (Dorard et al., 2008; Limonero et al., 2006). Poor emotional clarity has been connected to higher cannabis consumption (Limonero et al., 2006) and misuse (Dorard et al., 2008). Researchers demonstrated that individuals with and without co-occurring psychopathology use cannabis to cope with their negative and unpleasant emotional experiences (Bonn-Miller et al., 2008; Bonn-Miller et al., 2011). In a study by Bonn-Miller et al. (2008) of 136 young adult cannabis users, there was a significant relationship between the total DERS score and cannabis use coping motives. Further investigation revealed that emotional regulation difficulties were complete mediators in the relationship between post-traumatic stress symptom severity and cannabis use coping motives (Bonn-Miller et al., 2011).

There could be reasons other than self-medication for the relationship between cannabis use and emotional regulation. To illustrate, the previous investigation of this thesis (Chapter 3) measured different facets of impulsivity in the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine. The results showed that cannabis use was significantly predicted by negative urgency, which is described as acting impulsively in negative emotional situations. According to some studies, shared parts of the brain, such as PFC and the amygdala, are involved in negative urgency, emotional regulation and substance misuse, specifically cannabis misuse (Bardo et al., 2018; McQueeney et al., 2011; Stephanou et al., 2016). This finding may be one of the reasons why people with emotional regulation deficits misuse cannabis. Thus, it can be concluded that impairments in the function of those shared brain areas can increase the risk of cannabis misuse and the likelihood of emotional dysregulation and increased impulsive actions, especially in negative emotional situations.

Examining the relationship between ADHD, impaired emotional regulation and substance misuse seems necessary, based on their shared brain parts and genetic basis. As presented earlier in this chapter, past studies have proposed correlations between each of these conditions separately. They have revealed relationships between ADHD and emotional dysregulation, ADHD and substance misuse, as well as substance misuse and emotional dysregulation relationships separately. Still, none of them has explored the connections between all three conditions. As a result, the present study is the first to investigate whether emotional dysregulation mediates the relationship between ADHD symptom clusters and substance misuse.

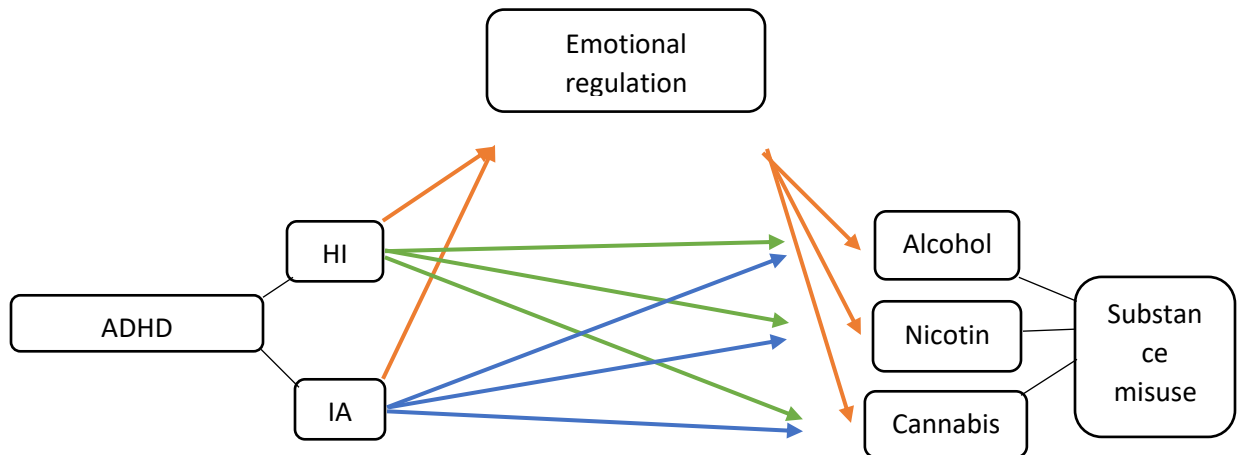
4.4. Aims of this study

The main goal of this research is to investigate whether emotional regulation mediates the relationship between ADHD symptom clusters (hyperactivity/impulsivity and inattention) and the use of alcohol, cannabis and nicotine in typically developing university students. A four-step regression model is designed to analyse the mediation relationship between the variables in this analysis:

- to determine if each ADHD symptom significantly predicts emotional regulation on its own
- to explore whether emotional regulation indicates the use of alcohol, cannabis and nicotine
- to investigate how far symptoms of ADHD predict the use of alcohol, cannabis and nicotine.

- to examine how the predictive capacity of ADHD symptoms changes when emotional regulation is present in predicting the use of alcohol, cannabis and nicotine (Figure 4.1)

If ADHD symptoms are not significant predictors of substance misuse in the presence of emotional regulation, there is complete mediation in the model. If both ADHD symptoms and emotional regulation are substantial predictors of alcohol, cannabis and nicotine use, there is partial mediation between the variables. This study is the first to explore the role of emotional dysregulation as a mediator between hyperactivity/impulsivity and inattention symptoms and the use of alcohol, cannabis and nicotine separately. Based on the preceding sections of this chapter, it is hypothesised that the hyperactivity/impulsivity symptom of ADHD significantly predicts emotional regulation. In contrast, emotional regulation would predict alcohol, cannabis and nicotine use. In the third step, both ADHD symptom clusters would significantly predict substance misuse scores. Additionally, since the mediation analysis' fourth step is an exploratory research question, no specific prediction is made. Figure 4.1 provides a summary of the hypothesised relations between these variables.



- The direct effect of ASRS-HI on alcohol, cannabis and nicotine misuse.
- The direct effect of ASRS-IA on alcohol, cannabis and nicotine misuse.
- The indirect effect of ASRS-HI and IA on the use of alcohol, cannabis and nicotine via emotional regulation.

Figure 4.1: The relationship between the different variables of this study. Based on previous literature, ADHD can predict all three, alcohol, nicotine and cannabis use significantly. Some studies showed the relationship between each ADHD symptom and these three substance misuses. The current study is an attempt to find if emotional regulation can mediate each ADHD subtype and alcohol, nicotine and cannabis use significantly.

4.5. Methods

4.5.1. Participants

Two hundred and six students aged 18 and above (mean age of 22.36, $SD = 5.62$) were recruited from Goldsmiths, University of London’s Department of Psychology’s Research Participation Scheme; 74% ($n = 154$) of participants were women. Due to a shortage of funds to compensate them for their time, only first-year undergraduates were recruited in this study and granted course credits for their participation. G*Power3.1.9.7 was used to measure this study’s statistical power and sample size. The calculated sample size was 119 individuals;

more participants were recruited to increase the study's statistical power. Students with a major psychiatric disorder (e.g. schizophrenia, borderline personality disorder and obsessive-compulsive disorder) and/or major physical health problems (e.g. brain injury) were excluded. Participants signed an online consent form confirming that they were 18 or older and agreed to participate in the study. All participants completed the measures anonymously.

4.5.2. Procedure

The ethics committee of Goldsmiths, University of London's Department of Psychology approved this study (Ethics Reference Number: PS130617ZSS; please see Appendix 14 for the approved Ethical Approval Form). Participants were recruited from Goldsmiths, University of London's Department of Psychology's Research Participation Scheme. The study's information and its link were on the Research Participation Scheme page, along with the researcher's email address. Participants could email the researcher with any question they had about the study. On the first page of the online section, specific additional details about the number and length of the questionnaires, ethical issues and the study's overall focus were provided. Participants had to fill out an online consent form confirming that they are 18 or older and agreed to be involved in the study; otherwise, they could not answer the questionnaires. Moreover, any question that they did not want to answer could be skipped. At the end of the survey, there was a debrief page with detailed information about the study. Depending on their answers, each respondent took 30 to 40 minutes to complete the questionnaire.

4.5.3. Measures

ASRS was employed to measure ADHD symptoms. The AUDIT and AUQ were used to assess alcohol use, frequency and quantity. The CUDIT-R and CAN questionnaires evaluated cannabis use, frequency and quantity in the participants of this study. The Cronbach's alpha for the measures in the current study are as follows: ASRS-IA = .86, ASRS-HI = .88, ASRS-total = .90, AUDIT = .93, AUQ = .69, NIC = .85, CUDIT-R = .92 and CAN = .72.

4.5.3.1. Behaviour Rating Inventory of Executive Function - Adult Version (BRIEF-A; Roth et al., 2005);

The Behaviour Rating Inventory of Executive Function – Adult Version (BRIEF-A) was used in this study to measure participants' emotional regulation and cognitive performance in daily life. The self-report form is designed for adults aged 18 to 90 who are healthy or have a wide variety of psychiatric, systemic, neurological and developmental disorders. It contains 75 items within nine non-overlapping scales and an overall function measure (Global Executive Composite [GEC]) based on theoretical and statistical considerations.

The BRIEF-A has two summary index scales: Behavioural Regulation Index (BRI) and Metacognition Index (MI). The BRI comprises four subscales: Inhibit, Shift, Emotional Control and Self-Monitor, while the MI has five subscales: Initiate, Working Memory, Plan/Organise, Task Monitor and Organisation of Materials. The inventory also has three validity scales: Negativity, Infrequency and Inconsistency. The negativity validity scale measures the level to which an individual answers selected items in an unusually negative manner, while the infrequency validity scale assesses the level to which adults endorse items

in an abnormal fashion. Lastly, the inconsistency validity scale indicates the level to which the respondent answers similar items in an inconsistent manner.

In the current investigation, the ‘emotional control’ subscale was used to measure emotional regulation and had 10 items. A total emotional regulation score was determined by adding up the scores of each item and calculating a ‘*t*’ score using the BRIEF-A manual script tables. The Cronbach’s alpha for the measurement of emotional regulation in this study was .97.

4.5.4. Data analysis strategy

Data were analysed using IBM SPSS version 20 and Pearson correlation was conducted to examine the connection between the variables of the study and the results are presented in Table 4.4. To find out whether emotional regulation is a mediator between ADHD symptoms and substance misuse, the following subscale scores of the ASRS were used: inattention symptom of ADHD (ASRS-IA), hyperactivity/impulsivity symptom of ADHD (ASRS-HI). For each of these categories, a four-step multiple regression analysis was done to test whether emotional regulation mediates each ASRS score and the three substance misuses or not. In Step One, each ADHD facet (predictor) must predict emotional regulation (mediator). In Step Two, emotional regulation must predict the use of alcohol, cannabis and nicotine (criterion) significantly. In Step Three, each facet of ADHD must predict the use of alcohol, cannabis and nicotine significantly. In the last step, the relationship between the predictor and the criterion must shrink in the presence of the mediator, which shows a full mediation in the model. If both predictor and mediator predict the criterion significantly in the last step,

there is a partial mediation in the model. In addition, the Sobel test and PROCESS macro were used to measure the magnitude of the indirect effect between the variables of this study.

4.6. Results

4.6.1. Outliers and multi-collinearity

An analysis of standard residuals for all variables showed that the data contained no outliers (Std. Residual Min >-3.29 , Std. Residual Max <3.29). The assumption of collinearity for all variables indicated that multi-collinearity was not a concern (VIF value is less than 10 and the Tolerance is more than 0.1).

To correct for slight positive skew (1.04) (kurtosis = .29) in the AUDIT scores (mean = 10.55, $SD = 8.6$), AUQ scores (mean = 36.66, $SD = 36.3$) (skewness = 1.47, kurtosis = 2.34), CUDIT-R (mean = 4.19, $SD = 7.6$) (skewness = 1.82, kurtosis = 2.31) and CAN (mean = 14.52, $SD = 17.24$) (skewness = 1.11, kurtosis = 1.16) scores were log transformed (new = $LG10(\text{old} + a)$; $a = 1$). The new skewness and kurtosis for all of these variables became as follows: AUDIT (skewness = -.32, kurtosis = -.26), AUQ scores (skewness = -.69, kurtosis = -.22), CUDIT-R (skewness = .24, kurtosis = -1.29) and CAN (skewness = -.26, kurtosis = -1.7).

4.6.2. The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

To measure alcohol, cannabis and nicotine use frequency and quantity in the participants, the same questions as Chapter 2 were asked and the percentage of frequency and quantity of use are presented in Table 4.1.

Results indicated that 50.2% of participants showed hazardous alcohol use by getting the score of eight or above and 27.3% of women gained the score of 13 or above and 44.2% of men got 15 or above on AUDIT, which shows possible alcohol dependence. The percentage of hazardous alcohol use in this study was the highest among other studies of this thesis. This could be because of differences between the first year undergraduates of this year or the time of the recruitment, which was during the fresher's week and the beginning of the academic year. The student use higher amounts of alcohol during this time of the year. In addition, 25.5% of the participants got the score of 8 or more on CUDIT-R, which shows hazardous cannabis use and 20.9% of the students got the score of 12 or more on CUDIT-R that reflects possible cannabis use disorder.

In AUQ, the frequency of getting drunk after drinking alcohol was measured by "How many times have you been drunk in the last 6 months?". Data showed that 67.1% have never been drunk in the last 6 months. In addition, the frequency of wine or wine type products, beer or cider and spirits were measured, which are presented in Table 4.2.

The results also indicated that 25.2% of the sample of this study had a score of 24 or above in ASRS, which demonstrates the high likelihood of having ADHD.

Table 4.1

The percentile of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

| | Frequency | Percent | Hazardous use (%) | Dependence (%) |
|--|-----------|---------|-------------------|----------------------------|
| AUDIT | | | 50.2 | Women = 27.3 Men = 44.2 |
| AUDIT frequency (How often do you have a drink containing alcohol?) | | | | |
| Never | 75 | 35.5 | | |
| Monthly or less | 32 | 15.2 | | |
| 2-4 times a month | 35 | 16.6 | | |
| 2-3 times a week | 46 | 21.8 | | |
| 4 or more times a week | 18 | 8.5 | | |
| AUDIT quantity (How many standard drinks containing alcohol do you have on a typical day when drinking?) | | | | |
| 1 or 2 | 108 | 51.2 | | |
| 3 or 4 | 56 | 26.5 | | |
| 5 or 6 | 35 | 16.6 | | |
| 7 to 9 | 6 | 2.8 | | |
| 10 or more | 1 | .5 | | |
| CUDIT-R | | | 25.6 | 20.9 |
| CUDIT-R frequency (How often do you use cannabis?) | | | | |
| Never | 142 | 67.3 | | |
| Monthly or less | 18 | 8.5 | | |
| 2-4 times a month | 31 | 14.7 | | |
| 2-3 times a week | 9 | 4.3 | | |
| 4 or more times a week | 6 | 2.8 | | |
| CUDIT-R quantity (How many hours were you stoned when you had been using cannabis?) | | | | |
| Less than 1 | 156 | 73.9 | | |
| 1 or 2 | 22 | 10.4 | | |
| 3 or 4 | 23 | 10.9 | | |
| 5 or 6 | 4 | 1.9 | | |
| 7 or more | 1 | .5 | | |
| CAN | | | | |
| Lifetime cannabis use (Have you ever used cannabis in your life?) | | | | |
| Yes | 102 | 48.3 | | |
| No | 104 | 49.3 | | |
| Cannabis quantity in the last year (In the last year, on how many separate days have you used cannabis?) | | | | |

| | | |
|--------------------------|-----|------|
| Never | 124 | 58.8 |
| Once every 12 months | 11 | 5.2 |
| Once every 2 to 3 months | 10 | 4.7 |
| Once a month | 6 | 2.8 |
| 2 to 3 times a month | 11 | 5.2 |
| Once a week | 9 | 4.3 |
| 2 to 3 times a week | 10 | 4.7 |
| 4 to 5 times a week | 14 | 6.6 |
| Daily or nearly daily | 11 | 5.2 |

NIC

NIC frequency

(During the past 30 days, on how many days did you smoke cigarettes?)

| | | |
|---------------|-----|------|
| 0 days | 127 | 60.2 |
| 1 to 2 days | 13 | 6.2 |
| 3 to 5 days | 4 | 1.9 |
| 6 to 9 days | 7 | 3.3 |
| 10 to 19 days | 11 | 5.2 |
| 20 to 29 days | 20 | 9.5 |
| All 30 days | 24 | 11.4 |

NIC quantity

(During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?)

| | | |
|--|-----|------|
| I did not smoke cigarettes during the past 30 days | 127 | 60.2 |
| Less than 1 cigarette per day | 11 | 5.2 |
| 1 cigarette per day | 12 | 5.7 |
| 2 to 5 cigarettes per day | 29 | 13.7 |
| 6 to 10 cigarettes per day | 17 | 8.1 |
| 11 to 20 cigarettes per day | 8 | 3.8 |
| More than 20 cigarettes per day | 2 | .9 |

Note: AUDIT = Alcohol Use Identification Test; CUDIT-R = Cannabid Use Identification Test-Revised; NIC = Nicotine Use Test

Table 4.2
The frequency of wine and wine type products, beer or cider, spirits

| Days in a week | Wine | | Beer or cider | | Spirits | |
|----------------|-----------|------|---------------|------|-----------|------|
| | Frequency | % | Frequency | % | Frequency | % |
| 0 | 113 | 53.6 | 117 | 55.5 | 114 | 54.0 |
| 1 | 50 | 23.7 | 40 | 19.0 | 53 | 25.1 |
| 2 | 16 | 7.6 | 21 | 10.0 | 17 | 8.1 |
| 3 | 11 | 5.2 | 10 | 4.7 | 10 | 4.7 |
| 4 | 3 | 1.4 | 2 | .9 | 6 | 2.8 |
| 5 | 2 | .9 | 2 | .9 | 4 | 1.9 |
| 6 | 1 | .5 | 0 | 0 | 0 | 0 |
| 7 | 10 | 4.7 | 14 | 6.6 | 2 | .9 |

4.6.3. Descriptive statistics and the correlations between variables

Tables 4.3 and 4.4 show the descriptive statistics and Pearson correlations of the current study. Men were coded as ‘1’ and women were coded as ‘2’ in this study. Higher score in emotional dysregulation was correlated significantly with the symptoms of ADHD (r (ASRS-total) = .60, r (ASRS-IA) = .43, r (ASRS-HI) = .62, $p < .001$) and alcohol use ($r = .44$, $p < .001$), cannabis use ($r = .46$, $p < .001$) and nicotine use ($p < .05$). There was also a significant correlation between emotional regulation and gender ($r = .18$, $p < .05$). ADHD symptoms were correlated significantly with all types of substance misuses in this study ($p < .001$).

Table 4.3
Descriptive statistics and demographic information

| | Mean | Std. Deviation | <i>n</i> | Minimum | Maximum | Range |
|---------------|-------|----------------|----------|---------|---------|-------|
| Age | 22.36 | 5.62 | 206 | 18 | 41 | 23 |
| ER | 58.14 | 13.10 | 206 | 38 | 89 | 51 |
| ADHD_total | 34.88 | 12.28 | 206 | 0 | 72 | 72 |
| ADHD_IA | 18.99 | 6.68 | 206 | 0 | 36 | 36 |
| ADHD_HI | 15.89 | 7.32 | 206 | 0 | 36 | 36 |
| AUDIT-trans | .9349 | .36 | 206 | 0 | 39 | 39 |
| AUQ-trans | 1.32 | .55 | 206 | 0 | 206 | 206 |
| CUDIT-R-trans | .57 | .51 | 206 | 0 | 33 | 33 |
| CAN-trans | .87 | .70 | 206 | 0 | 84 | 84 |
| NIC | 20.35 | 17.94 | 206 | 0 | 52 | 52 |

Note: ADHD = Attention Deficit Hyperactivity Disorder; IA = Inattentive; HI = Hyperactive; ER = Emotional Regulation score of BRIEF-A questionnaire; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

Table 4.4
Pearson correlation between ER, ADHD scores and the use of alcohol, cannabis and nicotine scores

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|--------------|--------|--------|-------|-------|-------|-------|-------|-------|-------|-------|----|
| 1-Age | 1 | | | | | | | | | | |
| 2-Gender | -.25** | 1 | | | | | | | | | |
| 3- ER | .09 | -.17* | 1 | | | | | | | | |
| 4-ASRS-total | .02 | -.21** | .60** | 1 | | | | | | | |
| 5-ASRS-IA | -.12 | -.06 | .43** | .86** | 1 | | | | | | |
| 6-ASRS-HI | .15* | -.30** | .62** | .89** | .53** | 1 | | | | | |
| 7-AUDIT | .12 | -.28** | .44** | .71** | .56** | .67** | 1 | | | | |
| 8-AUQ | .17* | -.28** | .35** | .60** | .48** | .56** | .82** | 1 | | | |
| 9-CUDIT-R | .19** | -.28** | .46** | .65** | .49** | .63** | .68** | .59** | 1 | | |
| 10-CAN | .19* | -.21** | .37** | .54** | .45** | .50** | .63** | .56** | .90** | 1 | |
| 11-NIC | .17* | -.14* | .18* | .26** | .19** | .26** | .46** | .47** | .50** | .58** | 1 |

Note: ER = Emotional regulation score of BRIEF-A questionnaire; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire

4.6.4 Does emotional regulation mediate the relationship between ASRS-IA and substance misuse?

A) AUDIT:

In this study, 50.2% of the participants gained a score of 8 or above in AUDIT, which indicates risky alcohol use. Additionally, the results of the multiple regression in the first step of the mediation analysis showed that ASRS-IA predicted emotional regulation in a significant way ($R^2 = .38$, $F(1,204) = 127.43$, $p < .001$) and it accounted for 38.4% of the variance ($\beta = .62$, $p < .001$). In the second step, emotional regulation predicted AUDIT scores significantly and accounted for 19.8% of the variance ($R^2 = .2$, $F(1,204) = 50.31$, $p < .001$) ($\beta = .44$, $p < .001$). In the third step, ASRS-IA predicted AUDIT score significantly ($R^2 = .45$, $F(1,204) = 17.84$, $p < .001$). It accounted for 45.4% of the variance ($\beta = .67$, $p < .001$) in AUDIT scores. The last step showed that controlling for the mediator (emotional regulation), ASRS-IA scores were still a significant predictor of AUDIT scores, ($\beta = .65$, $t(206) = 9.801$, $p < .001$). The indirect effect of ASRS-IA on AUDIT scores via emotional regulation problems was .13, with a 95% confidence interval, which did not include zero; that is to say, the effect was significantly greater than zero at $\alpha = .05$. The Sobel test was conducted and did not find partial mediation in the model ($z = 0.50$, $p = .62$) (Table 4.5).

B) AUQ

In the first step of the mediation model, independent variable, ASRS-IA, predicted emotional regulation as the mediator significantly and accounted for 38.4% of the variance ($\beta = .62$, $p < .001$). The second step of the multiple regression shows that emotional regulation

is a significant predictor AUQ ($R^2 = .12$, $F(1,204) = 28.82$, $p < .001$). It accounted for 12.4% of the variance ($\beta = .35$, $p < .001$) in AUQ scores. The third step of the mediation model revealed that ASRS-IA predicts AUQ scores significantly ($R^2 = .32$, $F(1,204) = 94.42$, $p < .001$) and accounted for 31.6% of the variance in AUQ ($\beta = .56$, $p < .001$). The last step indicated that after controlling emotional regulation as the mediator, ASRS-IA is still a significant predictor of AUQ score ($\beta = .56$, $t(206) = 7.56$, $p < .001$). The indirect effect of ASRS-IA on AUQ scores via emotional dysregulation was .2, with a 95% confidence interval, which included zero at $\alpha = .05$. The Sobel test was conducted and the results showed that emotional regulation is not even a partial mediator between ASRS-IA and AUQ ($z = 0$, $p = 1$) (Table 4.5).

C) CUDIT-R

In this study, 23.78% of the participants gained a score higher than 8 on the CUDIT-R, which indicates risky cannabis use. The first step of the multiple regression was the same as AUDIT and AUQ, ASRS-IA predicted emotional dysregulation significantly ($R^2 = .38$, $F(1,204) = 127.43$, $p < .001$) and accounted for 38.4% of the variance ($\beta = .62$, $p < .001$). The second regression analysis indicated that emotional dysregulation is a significant predictor of CUDIT-R scores ($R^2 = .22$, $F(1,204) = 56.24$, $p < .001$) ($\beta = .46$, $p < .001$) and accounted for 21.6% of the variance in CUDIT-R. The third step of the mediation model revealed that inattention symptom of ADHD predicts CUDIT-R significantly ($\beta = .64$, $p < .001$). It accounted for 40.5% of the variance ($R^2 = .40$, $F(1,204) = 138.63$, $p < .001$). In the fourth step, ASRS-IA was still a significant predictor of CUDIT-R scores after controlling emotional regulation as the mediator ($\beta = .56$, $t(206) = 8.24$, $p < .001$). The indirect effect of

ASRS-IA on CUDIT-R scores via emotional dysregulation was .50, with a 95% confidence interval, which included zero at $\alpha = .05$. The Sobel tests results showed that emotional regulation is not a partial mediator between ASRS-IA and CUDIT-R scores ($z = 1.32, p = .18$) (Table 4.5).

D) CAN

In step 1 of the mediation model, our IV, ASRS-IA, predicted the mediator, emotional regulation, in a significant way. Step 2 indicated that emotional regulation as the mediator predicted CAN score significantly ($R^2 = .14, F(1,204) = 33.06, p < .001$) and accounted for 14% of the variance in CAN ($\beta = .37, p < .001$). The results of the third step demonstrate that ASRS-IA is a significant predictor of CAN score ($R^2 = .25, F(1,204) = 68.60, p < .001$) and it accounted for 25.2% of the variance ($\beta = .50, p < .001$). The last step of the multiple regression showed that after controlling the mediator, inattention symptom of ADHD is still a significant predictor of CAN score ($\beta = .44, t(203) = 5.7, p < .001$). The indirect effect of ASRS-IA on CAN score via emotional dysregulation was .60, with a 95% confidence interval, which included zero at $\alpha = .05$. The results of the Sobel test also show that emotional regulation is not a partial mediator between these two variables ($z = 1.24, p = .21$) (Table 4.5).

E) NIC

The results of the first step of the mediation model were the same as the previous substances. In the second step, emotional regulation predicted nicotine use significantly ($R^2 =$

.03, $F(1,204) = 6.6, p < .001$) ($\beta = .18, p < .05$) and accounted for 3.1% of the variance in NIC scores. The third step of the mediation analysis indicated that ASRS-IA can predict nicotine use significantly ($R^2 = .07, F(1,204) = 14.50, p < .001$). It accounted for 6.6% of the variance in nicotine use ($\beta = .26, p < .001$). The result of the fourth step revealed that after controlling the mediator, emotional regulation, the inattention symptom of ADHD is still a significant predictor ($\beta = .24, t(206) = 2.78, p < .001$). The indirect effect of ASRS-IA on NIC scores via emotional dysregulation was .41, with a 95% confidence interval, which included zero at $\alpha = .05$. The Sobel test results showed that emotional regulation is not a partial mediator between ASRS-IA and nicotine use ($z = 0.32, p = .75$) (Table 4.5).

Table 4.5

Four steps mediation analysis of emotional dysregulation between ASRS-IA and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|-----------|--------------------|-------|--------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-IA | ER | .38** | 127.43 | .62** | .49 | .13 | -.31 | .56 |
| 2 | ER | AUDIT | .20** | 50.31 | .44** | | | | |
| 3 | ASRS -IA | AUDIT | .45** | 169.84 | .67** | | | | |
| 4 | ASRS -IA | AUDIT | .45** | 84.90 | .64** | | | | |
| | ER | | | | .04** | | | | |
| 1 | ASRS -IA | ER | .38** | 127.43 | .62** | 0 | .02 | -.78 | .83 |
| 2 | ER | AUQ | .12** | 28.82 | .35** | | | | |
| 3 | ASRS -IA | AUQ | .32** | 94.42 | .56** | | | | |
| 4 | ASRS -IA | AUQ | .32** | 46.98 | .55** | | | | |
| | ER | | | | .05** | | | | |
| 1 | ASRS -IA | ER | .38** | 127.43 | .62** | 1.32 | .50 | -.15 | 1.15 |
| 2 | ER | CUDIT-R | .22** | 56.24 | .46** | | | | |
| 3 | ASRS -IA | CUDIT-R | .40** | 138.63 | .63** | | | | |
| 4 | ASRS -IA | CUDIT-R | .41** | 71.32 | .56** | | | | |
| | ER | | | | .11** | | | | |
| 1 | ASRS -IA | ER | .38** | 127.43 | .62** | 1.24 | .60 | -.38 | 1.57 |
| 2 | ER | CAN | .14** | 33.06 | .37** | | | | |
| 3 | ASRS -IA | CAN | .25** | 68.08 | .50** | | | | |
| 4 | ASRS -IA | CAN | .26** | 35.28 | .43** | | | | |
| | ER | | | | .10** | | | | |
| 1 | ASRS -IA | ER | .38** | 127.43 | .62** | .32 | 4.18 | -22.30 | 31.47 |
| 2 | ER | NIC | .03** | 6.57 | .17** | | | | |
| 3 | ASRS -IA | NIC | .07** | 14.50 | .25** | | | | |
| 4 | ASRS -IA | NIC | .07** | 7.27 | .24** | | | | |
| | ER | | | | .02** | | | | |

Note: ASRS = ADHD Self-Report Scale; ER = Emotional dysregulation; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

4.6.5 Does emotional regulation mediate the relationship between ASRS-HI and substance misuse?

A) AUDIT

The first step of the mediation analysis showed that hyperactivity/impulsivity symptom of ADHD is a significant predictor of emotional regulation ($R^2 = .18$, $F(1,204) = 46.3$, $p < .001$) and accounted for 18.5% of the variance in emotional regulation scores ($\beta = .43$, $p < .001$). The second step indicated that the mediator can predict AUDIT scores significantly ($\beta = .445$, $p < .001$). It accounted for 19.8% of the variance ($R^2 = .2$, $F(1,204) = 50.31$, $p < .001$). The third step revealed that ASRS-HI predicted AUDIT in a significant way and accounted for 31.5% of the variance in AUDIT scores ($R^2 = .31$, $F(1,204) = 93.7$, $p < .001$) ($\beta = .56$, $p < .001$). In the fourth step of the mediation model, both the mediator and ASRS-HI as the independent variable were statistically significant predictors of AUDIT scores (β (ASRS-IA) = .45, $t(206) = 7.32$, $p < .001$). In this case, the effect size was .57, with a 95% confidence interval, which did not include zero; that is to say, the effect was significantly greater than zero at $\alpha = .05$. The Sobel test results indicated that emotional regulation is a partial mediator between hyperactivity/impulsivity symptom of ADHD and AUDIT ($z = 3.11$, $p = .001$) (Table 4.6).

B) AUQ

The first step of the mediation model for the relation between ASRS-HI, emotional regulation and AUQ scores was the same as the AUDIT results. Step 2 of the model showed that emotional regulation is a significant predictor of AUQ ($R^2 = .12$, $F(1,204) = 28.82$, p

<.001) and accounted for 12.4% of the variance ($\beta = .35, p < .001$). In Step 3, ASRS-HI predicted AUQ significantly ($R^2 = .23, F(1,204) = 61.62, p < .001$) and accounted for 23.2% of the variance in AUQ scores ($\beta = .48, p < .001$). The last step of the mediation analysis revealed that after controlling for emotional regulation as the mediator, ASRS-HI was still a statistically significant predictor of AUQ score ($\beta = .40, t(206) = 6.05, p < .001$). The indirect effect between ASRS-HI and AUQ via emotional regulation showed that the effect size was .63, with a 95% confidence interval, which did not include zero; that is to say, the effect was significantly greater than zero at $\alpha = .05$. The Sobel test was conducted and the results showed that emotional regulation is a partial mediator between ASRS-HI and AUQ ($z = 2.21, p = .03$) (Table 4.6).

C) CUDIT-R

The results of the first step of the mediation model were the same as previous sections and ASRS-HI was a significant predictor of emotional regulation as the mediator. In the second step, emotional regulation predicted CUDIT-R scores significantly ($R^2 = .22, F(1,204) = 56.24, p < .001$) and accounted for 21.6% of the variance ($\beta = .46, p < .001$). In the third step, hyperactivity/impulsivity symptom of ADHD predicted CUDIT-R in a statistically significant way ($R^2 = .24, F(1,204) = 65.5, p < .001$) and accounted for 24.3% of the variance in CUDIT-R scores ($\beta = .49, p < .001$). The last step indicated that in the presence of a mediator, both ASRS-HI as the independent variable and emotional regulation as the mediator were significant predictors of CUDIT-R scores (β (ASRS-IA) = .36, $t(206) = 5.61, p < .001$, β (ER) = .31, $t(206) = 4.84, p < .001$). The indirect effect between ASRS-HI and CUDIT-R via emotional regulation showed that the effect size was 1.02, with a 95%

confidence interval, which did not include zero; that is to say, the effect was significantly greater than zero at $\alpha = .05$. The Sobel test was conducted and found partial mediation in the model ($z = 4.45, p < .001$) (Table 4.6).

D) CAN

In the first step of the mediation analysis, ASRS-HI predicted emotional regulation significantly like previous sections. Step 2 of the model indicated that emotional regulation can predict CAN score ($R^2 = .14, F(1,204) = 33.06, p < .001$) ($\beta = .37, p < .001$). It accounted for 13.9% of the variance in CAN score. The third step showed that ASRS-HI is a significant predictor of CAN ($R^2 = .20, F(1,204) = 52.33, p < .001$) and the fourth step revealed that after controlling the emotional regulation, hyperactivity/impulsivity symptom of ADHD could still predict CAN score significantly ($\beta = .36, t(206) = 5.28, p < .001$). The indirect effect between ASRS-HI and CAN score had a .99 effect size with a 95% confidence interval, which did not include zero; that is to say, the effect was significantly greater than zero at $\alpha = .05$. The Sobel test results showed that emotional regulation is a partial mediator between HI and CAN score ($z = 2.74, p = .006$) (Table 4.6).

E) NIC

It has been mentioned in the previous sections that hyperactivity/impulsivity symptom of ADHD is a significant predictor of emotional regulation. The second step of the mediation analysis indicated that emotional regulation can predict nicotine use significantly ($\beta = .18, p < .05$). It accounted for 3.1% of the variance in NIC scores ($R^2 = .03, F(1,204) = 6.6, p$

<.001). The third step showed that ASRS-HI is a statistically significant predictor of NIC ($R^2 = .04$, $F(1,204) = 7.94$, $p < .001$) and accounted for 3.8% of the variance in nicotine use ($\beta = .19$, $p < .001$). In the last step it has been shown that none of emotional regulation as the mediator and ASRS-HI as the independent variable predicted nicotine use significantly (β (ASRS-IA) = .14, $t(206) = 1.90$, $p > .05$, β (ER) = .11, $t(206) = 1.51$, $p > .05$) (Table 4.38). The indirect effect size between ASRS-HI and NIC was .13 with a 95% confidence interval, which included zero at $\alpha = .05$. The Sobel test results demonstrate that emotional regulation is not a partial mediation in the model ($z = 1.47$, $p = .14$) (Table 4.6).

Table 4.6

Four steps mediation analysis of emotional dysregulation between ASRS-HI and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|----------------|--------------------|-------|-------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-HI | ER | .18** | 46.28 | .43** | 3.11* | .57* | .21 | .91 |
| 2 | ER | AUDIT | .20** | 50.31 | .44** | | | | |
| 3 | ASRS -HI | AUDIT | .31** | 93.67 | .56** | | | | |
| 4 | ASRS -HI ER | AUDIT | .37** | 58.47 | .45** .25** | | | | |
| 1 | ASRS -HI | ER | .18** | 46.28 | .43** | 2.20* | .63* | .13 | 1.14 |
| 2 | ER | AUQ | .12** | 28.82 | .35** | | | | |
| 3 | ASRS -HI | AUQ | .23** | 61.62 | .48** | | | | |
| 4 | ASRS -HI ER | AUQ | .26** | 35.23 | .40** .17** | | | | |
| 1 | ASRS -HI | ER | .18** | 46.28 | .43** | 4.49** | .1.02* | .50 | 1.56 |
| 2 | ER | CUDIT-R | .22** | 56.24 | .46** | | | | |
| 3 | ASRS -HI | CUDIT-R | .24** | 65.49 | .49** | | | | |
| 4 | ASRS -HI ER | CUDIT-R | .32** | 48.09 | .36** .31** | | | | |
| 1 | ASRS -HI | ER | .18** | 46.28 | .43** | 2.74* | .99* | .35 | 1.69 |
| 2 | ER | CAN | .14** | 33.06 | .37** | | | | |
| 3 | ASRS -HI | CAN | .20** | 52.33 | .45** | | | | |
| 4 | ASRS -HI ER | CAN | .24** | 32.68 | .35** .22** | | | | |
| 1 | ASRS -HI | ER | .18** | 46.28 | .43** | 1.47 | 13.23* | -6.69 | 32.09 |
| 2 | ER | NIC | .03* | 6.57 | .17** | | | | |
| 3 | ASRS -HI | NIC | .04* | 7.94 | .33** | | | | |
| 4 | ASRS -HI ER | NIC | .05* | 5.14 | .14** .11** | | | | |

Note: ASRS -HI = Hyperactivity/Impulsivity symptom of ADHD; ER = Emotional dysregulation; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

4.7. Discussion

This study initially aimed to examine the role of emotional regulation in the relationship between ADHD symptoms and substance misuse. Specifically, it intended to determine whether emotional regulation mediates the relationship between inattention and hyperactivity/impulsivity and the use of alcohol, cannabis and nicotine in a typically

developing sample of young adults. Results indicated that emotional regulation is a partial mediator between hyperactivity/impulsivity and alcohol and cannabis use.

A four-step regression model analysis was used to explore the mediation relationship between the variables of the present study. In the first step, both inattention and hyperactivity/impulsivity significantly predicted emotional regulation deficits. Even though the majority of studies focused on the core symptoms of ADHD, such as inattention, hyperactivity and impulsivity (Corbisiero et al., 2013; Retz et al., 2012), there has been a recent emphasis on emotional regulation (Corbisiero et al., 2013; Reimherr et al., 2005; Shaw et al., 2014). In studies with children and adults with ADHD symptoms, emotional dysregulation, fluctuations and irritability were observed (Biederman et al., 2012; Surman et al., 2011). Shaw et al. (2014) found that 34% to 70 % of adults with ADHD symptoms had emotional dysregulation. Moreover, emotional dysregulation predicted the course of symptoms and deficits in adults with ADHD in different domains, such as family, friends, workplace, etc. (Barkley & Murphy, 2010b; Skirrow & Asherson, 2013; Stringaris & Goodman, 2009; Stringaris et al., 2010; Surman et al., 2013). Earlier concepts of adult ADHD (Able et al., 2007; Surman et al., 2013) included emotional dysregulation as a defining feature, concurring with clinic-based investigations, even though more population-based studies are needed (Shaw et al., 2014). However, undiagnosed ADHD participants had the same results, though those with more ADHD symptoms demonstrated increased emotional regulation deficits (McQuade & Breaux, 2017). Research on the association between emotional regulation and each ADHD symptom cluster, one of this study's focal points, is scarce and contradictory.

There are shared parts of the brain in emotional dysregulation and ADHD, which may explain the co-occurrence of these two conditions. For instance, many investigations have discovered hyperactivity in the amygdala during the perception of fearful expressions in those with ADHD symptoms (Brotman et al., 2010; Marsh et al., 2008). This amygdala hyperactivity has also been seen in individuals with ADHD symptoms during delayed reward processing, which is consistent with the findings of delay aversion behavioural studies (Scheres et al., 2007; Stoy et al., 2011; Ströhle et al., 2008). Another critical part of the brain in emotional regulation and reward representations is the OFC, which has connections to the amygdala, the thalamus and multiple cortical regions (O'Doherty et al., 2001; Phillips et al., 2008). Some investigations proposed that people with ADHD have abnormalities in orbitofrontal activity during reward anticipation and receipt (Overmeyer et al., 2001; Plessen et al., 2006). The third part of the brain involved in the bottom-up circuitry is the ventral striatum, which mediates positive affect and reward processing (Knutson et al., 2001). Functional neuroimaging investigations reported decreased ventral striatum responsiveness in people with ADHD symptoms during the anticipation and receipt of rewards (Shaw et al., 2014). Thus, shared brain regions in ADHD and impaired emotional regulation deficits may explain the association between these two conditions.

The number of studies involving typically developing adults with ADHD symptoms, especially university students, is minimal. Nonetheless, the current study's results in the first step of the mediation analysis are consistent with past research findings. This research added to the field by separating the two main facets of ADHD and investigating the relationship between each of them as the predictive variable and emotional regulation as the mediator in the model in a typically developing sample.

Individuals with inattention and those with hyperactivity/impulsivity symptom of ADHD had more significant emotional dysregulation. However, the correlation between the hyperactivity/impulsivity symptom of ADHD and emotional regulation was higher than that of the inattention symptom of ADHD (Table 4.4), which may be attributed to the association between emotional regulation and impulsivity, as discussed in Section 1.4.3 of this thesis. Schreiber et al. (2012) tested 194 healthy young adults aged 18 to 29 years old. Their results indicated that those with higher scores on impulsivity were more impaired in their emotional regulation. It is suggested that a lack of adaptive emotional regulation strategies may cause more impulsive behaviours (Morrell et al., 2010; Selby et al., 2008; Whiteside & Lynam, 2003).

Moreover, investigators revealed that although hyperactivity/impulsivity and inattention are both predictors of depression, hyperactivity/impulsivity is a stronger predictor of emotional regulation problems and depressive symptoms than inattention (Seymour et al., 2014). Hyperactivity/impulsivity is a significant indicator of emotional lability (rapid mood-changing) or facial recognition, especially sad expressions, in individuals with ADHD (Baran Tatar et al., 2015; Miller et al., 2011; Skirrow & Asherson, 2013). Even children with higher hyperactivity/impulsivity symptom of ADHD are more impaired in their emotional regulation. Researchers discovered no connection between inattention symptoms and emotional regulation (Maedgen & Carlson, 2010).

Walcott and Landau (2010) found that greater response disinhibition is linked to inadequate emotional regulation. This outcome could explain the connection between hyperactivity/impulsivity symptoms of ADHD and emotional dysregulation (Berlin & Bohlin 2002). Hence, people with higher hyperactivity/impulsivity symptoms perform worse in

response inhibition tasks, which is an indicator of emotional regulation issues. However, further research is required to fully grasp the cause-and-effect relationship between each ADHD symptom and emotional regulation.

The second objective of this study was to examine whether emotional regulation predicts the use of alcohol, cannabis and nicotine. Emotional regulation was a statistically significant predictor of all substance use scores. The results of the second step of the regression analysis in the present study are consistent with previous studies on the relationship between emotional dysregulation and substance misuse. For instance, in a study with 674 university students, those with higher emotional dysregulation showed more alcohol related problems (Kim & Kwon, 2020). Moreover, Aurora and Klanecky (2016) tested 200 university student and found a positive association between impaired emotional regulation and alcohol use in their sample. Zimmermann et al. (2017) have conveyed that negative affect dysregulation is both a risk factor for developing cannabis misuse and a consequence of cannabis misuse. Besides, it has been proven that using impaired emotional strategies more frequently is associated with early smoking initiation, heightened smoking urges and failure during abstinence (Fucito et al., 2010; Szasz et al., 2012). The theoretical model revealed that individuals with recurrent emotional dysregulations agree that abstaining from using more nicotine helps them regulate their emotions (Baumeister & Heatherton, 1996; Khantzian, 1997; Yucel et al., 2007). This outcome may be due to shared brain regions that are activated during nicotine smoking and emotional regulation. Many neuroimaging studies demonstrate that nicotine smokers have PFC dysfunctions, which are associated with cognitive-emotional regulation and result in general emotional dysregulation (Lubman et al., 2004; McRae et al., 2010; Mocaiber et al., 2011; Moratti et al., 2011; Ochsner et al., 2004; Sutherland et al., 2012).

Several factors can explain the relationship between emotional regulation and substance misuse. First, those with more emotional regulation problems use higher amounts of alcohol, cannabis or nicotine to cope with their emotions (Bonn-Miller et al., 2008; Bonn-Miller et al., 2011; Piper et al., 2004; Simons et al., 2017). Investigations showed that coping-motivated alcohol use is the most problematic motive for alcohol consumption; it is associated with alcohol misuse and alcohol-related consequences (Cooper, 1994; Kuntsche et al., 2005). According to studies, 43.9% of undergraduates who consume alcohol experience negative consequences that can affect their academic life (Adlaf et al., 2005; Perkins, 2002). Studies also showed that individuals who smoke assert that it helps them cope with their emotional distress and negative affect (Copeland et al., 1995; Piper et al., 2004). Moreover, previous studies have proposed a connection between coping motives and cannabis use (Bonn-Miller et al., 2008). With regards to these results, studies have revealed that various treatment strategies, such as learning appropriate coping strategies and mindfulness, could help people who misuse substances to cope with emotional dysregulation (Guendelman et al., 2017; Prakash et al., 2015).

According to Study 2 of this thesis and other investigators (Jones et al., 2014), negative urgency as a facet of impulsivity predicts the use of alcohol, cannabis and nicotine significantly. negative urgency pertains to acting impulsively in negative emotional situations and associated with emotional regulation deficits (Bardo et al., 2018; McQueeney et al., 2011; Stephanou et al., 2016). As presented in Section 1.4.3, there are activated brain regions common in negative urgency, emotional regulation and substance misuse, increasing the likelihood of alcohol, cannabis and nicotine misuse or, later, SUD in those with emotional dysregulation. Nonetheless, these conditions can be improved by medication or other cognitive therapies. Some investigations also showed that substance use expectancies have

lasting impacts on subsequent rates of consumption and issues associated with substance misuse. Alcohol expectancies are cognitions related to the effect of alcohol use, which can be positive or negative (Brislin et al., 2020; Kuntsche & Kuntsche, 2018). Positive expectancies can predict risk factors including early initiation, risky drinking patterns and increased alcohol consumption or AUD (Cranford et al., 2009, 2010; Jester et al., 2015; Settles et al., 2014; Windle et al., 2008).

This research's third intention was to discern whether or not each ADHD symptom cluster predicted the use of alcohol, cannabis and nicotine. As independent variables, both ADHD scores were significant predictors of all substance misuse scores. This result was consistent with previous studies that found people with ADHD use more alcohol, cannabis and nicotine (Elkins et al., 2018; Martínez-Luna et al., 2017). The current investigation contributes to the existing literature by separately measuring both facets of ADHD (hyperactivity/impulsivity and inattention) and analysing the relationship between each facet and alcohol, cannabis and nicotine use. In Table 4.4, the correlation between hyperactivity/impulsivity and each substance misuse score was stronger than the correlation between inattention and substance misuse.

The fourth goal was to examine whether or not the relationship between the facets of ADHD and substance misuse weakens in the presence of emotional regulation as a mediator. According to the findings of this study, emotional regulation was not a complete mediator between ADHD symptoms and substance misuse scores. In the present study's sample, Sobel test results revealed that emotional regulation was a partial mediator between the hyperactivity/impulsivity symptom of ADHD and AUDIT, AUQ, CUDIT-R and CAN scores.

Emotional regulation was a partial mediator because, in the final phase of the regression model, both the hyperactivity/impulsivity symptom of ADHD and emotional regulation were significant predictors of alcohol and cannabis use. This result means that emotional regulation accounts for some of the relationships between hyperactivity/impulsivity, alcohol and cannabis use. It also implies that apart from a significant relationship between emotional regulation–hyperactivity/impulsivity and emotional regulation–alcohol and cannabis use, hyperactivity/impulsivity and alcohol and cannabis use have a direct connection. The last step of the analysis showed that emotional regulation was not a partial mediator between the inattention symptom and the use of alcohol, cannabis and nicotine. It can be hypothesised that other variables can mediate this relationship; these factors are common to both those with ADHD and substance misuse. Consequently, the following study (Chapter 5) examines the role of mood disorders, which were introduced in Section 1.4.4 of this thesis, to determine whether they can considerably mediate the relationship between each ADHD symptom and the use of alcohol, cannabis and nicotine.

In this study, 50.2% of participants yielded an AUDIT score of 8+, denoting hazardous alcohol use, while 25.5% of them received an 8+ CUDIT-R score, representing dangerous cannabis use. As stated in Section 1.2 of Chapter 1, 63% to 84% of university students in the UK reported hazardous drinking, with women nearly equal to men in risky alcohol use (Davoren, 2016). Based on a study of second-year students at 10 UK universities, cannabis was the most commonly used drug among university students (57%) (Webb et al., 1996).

In comparison to Studies 1 and 2 of this thesis, the present study's findings are similar to those of Davoren's systematic review of alcohol use among university students. The percentage of those with cannabis use was also higher in this study than in Studies 1 and 2.

This outcome may be due to two reasons:

(1) This study had a larger sample ($n = 206$) than the first study ($n = 85$), indicating its statistical power.

(2) The timing of the studies can affect the participants' responses to the questionnaires, resulting in varying percentages of participants with alcohol or cannabis use (Kim et al., 2017; Song et al., 2020).

The current study was conducted shortly after Christmas and New Year, asking about the participants' alcohol, cannabis and nicotine use in the previous month or six months, which may have influenced their responses to the substance use questionnaires. The current research and the subsequent studies, which were conducted almost simultaneously, centred on a combination of mood disorders and sleep quality and had the highest rates of substance misuse among the studies of this thesis.

4.8. Limitations

Besides the findings reported in this chapter, the research has some limitations that should be considered. It was a non-clinical study, recruiting typically developing participants. Studying individuals clinically diagnosed with ADHD and substance misuse may help future research investigate the relationship between the variables at a more clinically significant level.

Due to this study being a self-funded PhD research with time and budget constraints, recruitment could only include first-year undergraduates. This factor limited the extent to which the results can be generalised to a larger population (Creswell, 2014), such as older adults or those without a university education. Moreover, a cross-sectional study, used to measure the different variables in a population at a specific point in time, limits the results. It is not possible to show the causal relationship between the variables or to include data on other variables that might affect the association between the hypothesised cause and effect (Mann, 2003). Using within-subject designs that test the same subjects each time, longitudinal studies may reduce confounding variables and provide a clearer picture of the impact of potential mediators.

Participants' responses may have been influenced by fatigue and boredom experienced while completing online questionnaires. The study's design allowed the participants to answer questions in several sessions, but this may have inadvertently led to more incomplete questionnaires than expected. Further research should include longitudinal studies with clinical and a more diverse population. Employing more face-to-face sessions or shorter questionnaires may also decrease the likelihood of fatigue.

The BRIEF-A measured emotional regulation in the participants of this study. However, as previously mentioned, other emotional factors such as emotional awareness, emotional lability (rapid mood-changing) or facial recognition have been shown as significant predictors of substance misuse in past research (Baran Tatar et al., 2015; Miller et al., 2011; Skirrow & Asherson, 2013; Sobanski et al., 2010). Emotional face recognition tests or other computerised tasks can be used to measure these emotional factors and their links to

substance misuse and the two ADHD symptoms. Moreover, using a nicotine use questionnaire with a cutoff score may help assess risky nicotine use in the sample.

Furthermore, the predominance of women in this study may affect the substance misuse rates in the sample. Based on NHS Digital and previous investigation reports, men consume more alcohol, cannabis, and nicotine than women (Davoren, 2016; NHS Digital, 2018, 2019), indicating that evidence from a more gender-balanced sample may be more accurately generalised.

In light of these limitations, it is evident that the current findings require replication in a study with a more balanced gender ratio sample, assessed longitudinally to investigate the causal relationship between the variables.

4.9. Conclusion

This study attempted to explore the role of emotional regulation in the connection between ADHD symptoms and the use of alcohol, cannabis and nicotine. The results revealed that emotional regulation partially mediated the relationship between hyperactivity/impulsivity and alcohol and cannabis use. Thus, those with more hyperactivity/impulsivity symptoms of ADHD use higher amounts of alcohol and cannabis via an emotional dysregulation pathway; the factors that may explain this relationship were presented earlier in this section (please see Section 4.7). This outcome can help future interventions consider emotional regulation when discerning the best way to treat individuals with more hyperactivity/impulsivity symptoms and substance misuse. Various treatment strategies have been suggested to help those with ADHD symptoms and emotional

dysregulation; these include CBT (Mongia & Hechtman, 2012), mindfulness, derived partly from dialectical behaviour therapy (Wander et al., 1981), and dietary interventions, given that low levels of omega-3 fatty acids are associated with electrophysiological anomalies during emotion processing in ADHD (Gow et al., 2013).

Other common disorders and problems in people who misuse substances and those with ADHD symptoms may mediate the relationship between the inattention symptom of ADHD and the use of alcohol, cannabis and nicotine. Mood disorder is one of these impairments, which would be explored in the next chapter.

Chapter 5

5. Does mood disorder mediate the relationship between ADHD symptoms and substance misuse?

Overview

Study 4 found emotional regulation as a partial mediator between hyperactivity/impulsivity and alcohol and cannabis use; thus, the current study could investigate other pathways linking the two ADHD symptoms clusters to substance misuse. The study outlined in this chapter sought to examine the role of bipolar disorder as a mood disorder in the relationship between ADHD and substance misuse. A total of 223 first-year undergraduate students completed measures of bipolar disorder, ADHD symptoms and alcohol, cannabis and nicotine use. The main aim of this study was to explore whether bipolar disorder mediates the connection between the two ADHD symptom clusters (i.e. inattention and hyperactivity/impulsivity) and alcohol, cannabis and nicotine use. The results show that bipolar disorder is a partial mediator between inattention and alcohol, cannabis and nicotine use; however, it only plays the same role between hyperactivity/impulsivity and the use of alcohol and cannabis, but not nicotine.

5.1. Introduction

The main underlying feature of mood or affective disorder is a disturbance in an individual's mood (Sadock, 2002). Maudsley, an English psychiatrist, introduced the primary category of affective disorder (Berrios, 1985; Lewis, 1934). It was later replaced by mood disorder since it is the underlying and long-term emotional state, while affective disorder refers to one's external expression observed by others (Sadock, 2002).

There are different types of mood disorders. When a person has one or more episodes of major depression, they are diagnosed with Major Depressive Disorder (MDD). It is also called clinical depression, major depression or unipolar depression. Global epidemiological studies found that the prevalence of depressive disorder is around 5% (Ferrari et al., 2013) worldwide and is reported across the lifespan (Evans et al., 2021; Ferrari et al., 2013). The Adult Psychiatric Morbidity Survey, a survey of mental health and wellbeing carried out in England (2014) by NatCen Social Research, in collaboration with the University of Leicester for NHS Digital, reported similar numbers in the UK with 3.8% of adults aged 16 to 64 having had experienced depressive episodes (4.3% of women and 3.2% of men). Women are more likely than men to suffer from major depression (Vizard et al., 2017). Previous investigations also show that in England, 24% of women and 13% of men are diagnosed with depression at some point in their lives (Craig et al., 2015). In children, prevalence increased with age. Just 0.3% of 5- to 10-years-olds, 2.7% of 11- to 16-year-olds and 4.8% of 17- to 19-year-olds met the clinical criteria for depression (Vizard et al., 2017).

Another form of mood disorder is Substance-Induced Mood Disorder (SIMD), which occurs when the aetiology of a mood disorder is directly affected by a psychoactive drug or chemical substance or when a mood disorder follows substance intoxication or withdrawal (Diagnostic and Statistical Manual of Mental Disorders [DSM-5], 2013). Moreover, people with comorbid substance misuse can have the mood disorder, which has features of manic, hypomanic, mixed and depression (Revadigar & Gupta, 2021). A mood disorder can be induced by many substances (Patten et al., 2004; Schuckit et al., 2007).

Bipolar and related disorders as mood disorders that describe an unstable emotional condition with cycles of abnormal and persistent high and low mood, which is the focus of this study and will be subsequently discussed (DSM-5, 2013; Schacter et al., 2011). Bipolar

disorder is also called manic depressive disorder, previously known as manic depression (Stöppler, 2013). This condition is highly heritable (Johansson et al., 2019). Studies show that if one parent has bipolar disorder, the likelihood of their offspring receiving a diagnosis is 15% to 30%. Still, if both parents have the disorder, the probability of diagnosis rises to 50% to 75% (Abbel & Ey, 2009). Moreover, McManus et al.'s (2016) study reported that 2% of participants screened positive for bipolar disorder on the Mood Disorder Questionnaire (MDQ). Men's rates were slightly higher than women's. The highest rate of bipolar disorder was observed in women aged 16 to 24 (3.4%) and in men aged 16 to 44 (3%) (Mental Health Statistics for England, 2014). The proportion who tested positive for bipolar disorder decreased with age, with 16- to 24-year-olds having the highest percentage. This age group consists primarily of university students (Higher Education Statistics Agency, 2017/18), indicating the importance of studying mood disorders and comorbid conditions in this demographic.

Investigations reveal that some individuals with ADHD also have mood disorders (Anderson et al., 1987; Kessler et al., 2006; Kunwar et al., 2007; Tzang et al., 2009). ADHD can significantly impact one's health and functioning in different life domains (Brod et al., 2012; Das et al., 2012; De Zwaan et al., 2012; Ebejer et al., 2012). There are strong links between ADHD and bipolar disorder, such as genetic contributions (Klassen et al., 2010). As mentioned earlier in Section 1.4.4 of this thesis, both ADHD and bipolar disorder have similar characteristics and diagnostic criteria. Since several of their symptoms overlap, diagnosing these disorders is more complicated (Kent & Craddock, 2003; Wingo & Ghaemi, 2007). There are some parallels between the elevated or manic phase of bipolar disorder and ADHD, such as increased energy or being 'on the go', talkativeness, high distractibility,

frequent interruptive behaviours, physical restlessness and impaired normal social inhibitions (Kessler et al., 2010). Besides certain similarities in symptoms, such as impulsivity, inattention, hyperactivity, physical energy and behavioural and emotional lability, there are also some fundamental differences between ADHD and bipolar disorder (Kim et al., 2019). ADHD affects behaviour and attention, while bipolar disorder affects mood. Furthermore, in individuals with bipolar disorder, the symptoms cycle through different episodes of depression and mania/hypomania. Conversely, individuals with ADHD have chronic rather than cycling symptoms, even though they may also have mood symptoms (Kim et al., 2019). Since the symptoms of ADHD and bipolar disorder overlap, diagnosing both conditions is difficult.

Risk-taking behaviours also characterise bipolar disorder, and symptoms are exacerbated by misusing alcohol and other substances (Holmes et al., 2009). However, the high prevalence of comorbidity between substance misuse and psychiatric disorders does not necessarily demonstrate causation between them, even if one became apparent before the other (National Institute on Drug Abuse [NIDA], 2018). On the one hand, to be diagnosed, the symptoms of a psychiatric disorder must progress to a specific level as described in the DSM (currently DSM-5, 2013). On the other hand, substance misuse can occur sooner than other mental disorders due to subclinical symptoms and imperfect recollection of substance misuse, making it confusing to determine which came first (NIDA, 2010).

The relationship between ADHD and substance misuse is crucial. In existing studies focusing on the relationship between these two conditions, results are not as straightforward as expected. In a study by Estévez et al. (2015), 5,677 men (mean age 20 years) were tested using the ADHD Self-Report Scale (ASRS) and the Cohort Study on Substance Use Risk

Factors (C-SURF). The connection between ADHD and substance misuse was examined through alcohol, nicotine and cannabis use. Results indicated no statistically significant difference in lifelong alcohol use between individuals with and without ADHD symptoms. However, those with ADHD symptoms reported early or very early age of alcohol use onset and were more likely to develop AUD. Moreover, men with ADHD symptoms started using nicotine at an earlier age than those without ADHD symptoms. In addition, nicotine dependence was more likely in those with ADHD symptoms compared to the comparison group. Young men with ADHD symptoms also reported using more cannabis and other illegal drugs than those without ADHD. They started using cannabis at an earlier age and were less likely to be non-users than those without ADHD symptoms. They also had greater levels of cannabis dependence than the control group.

Although many investigations indicate associations between ADHD symptomology and substance misuse, the findings are inconsistent (Kousha et al., 2011; Lee et al., 2011). Some studies have found no correlation between ADHD and substance misuse, whereas others discovered connections (Galera et al., 2013; Lee et al., 2011; Madsen & Dalsgaard, 2014). One of the reasons for the inconsistent results could be the lack of representative sample or limited sample sizes (Charach et al. 2011; Lee et al. 2011). Therefore, while most evidence points to high rates of comorbidity, it is unclear if ADHD symptoms can predict substance misuse or if other variables mediate the relationship.

Study 3 showed that emotional regulation is a partial mediator between hyperactivity/impulsivity and alcohol and cannabis use, revealing a part of the connection between ADHD symptoms and substance misuse. Hence, the present study attempted to investigate other possible pathways linking ADHD symptom clusters to substance misuse. Literature has disclosed associations between emotional regulation and mood disorders. As

mentioned in Chapter 4, emotional regulation involves first detecting and evaluating stimuli and regulating an individual's emotional response to these stimuli (Phillips et al., 2003). Impaired emotional regulation may lead to pathological mood states such as bipolar disorder (Phillips et al., 2003b). Bipolar disorder is distinguished mainly by acute dysfunctional mood states, alternating between mania (bipolar disorder Type 1) or hypomania (bipolar disorder type 2) and depression (Critchley, 2003). This specific mood lability proposes a possible neural network dysfunction in emotional regulation.

Bipolar disorder is a primary mood disorder, and many fMRI studies use emotional regulation tasks (Townsend & Altshuler, 2012). A primary abnormality of bipolar disorder is the inability to regulate emotion, resulting from the disorder's acute mood states. Structural and functional imaging investigations have shown specific impairments in the frontal-limbic regions. These findings suggest that impairments in these circuits cause the manic and depressive symptoms seen in bipolar disorder (Altshuler & Townsend, 2012; Strakowski et al., 2005).

The key neural substrates of an emotional regulation circuit are the amygdala, medial PFC, ventral lateral PFC and anterior cingulate cortex (Phillips et al., 2003). Neuroimaging studies have revealed that the amygdala and insula are engaged in normal emotion processing, while the medial and lateral regions of ventral lateral PFC are involved in mood regulation (Baker et al. 1997; Northoff et al., 2000) and associative emotional memory functions (Bookheimer, 2002; Prince, 2003).

There are limited numbers of functional imaging studies during mania since it is challenging to keep manic patients still during scanning. However, individuals with bipolar disorder have been reported to have enlarged amygdala and increased activation (Altshuler et al., 1998; Brambilla et al., 2003; Yurgelun-Todd et al., 2000). Moreover, higher limbic

activation in response to emotional stimuli is one of the most consistent findings (Altshuler & Townsend, 2012).

Based on the results of previous neuroimaging research, there are shared brain regions in emotional regulation, bipolar disorder, ADHD and substance misuse. Consequently, this thesis's Study 4 intended to explore the role of bipolar disorder as a mediator between each ADHD symptom separately and substance misuse. It was likewise driven by (a) the association between emotional regulation (Study 4's focus) and bipolar disorder as a mood disorder and (b) the ADHD-bipolar disorder and substance misuse-bipolar disorder connections. In the following sections, the relationship between bipolar disorder, ADHD symptoms and substance misuse will be examined.

5.2. ADHD and mood disorders

Untreated ADHD is one of the most common neurobehavioural disorders in treatment-seeking children and adults (American Academy of Paediatrics. Sub-committee on Attention-Deficit/Hyperactivity Disorder, 2001; Greenhill et al., 2002). It has considerable social and personal costs in one's life, including driving safety, criminal activity, academic and occupational underachievement and personal relationship problems (Biederman et al., 2006; Seidman et al., 2006).

Individuals with ADHD show a high rate of co-occurring disorders such as conduct disorder, Oppositional Defiant Disorder (ODD), mood and anxiety disorder and substance misuse (Biederman et al., 2006). Over the past decade, interest in ADHD and mood disorders has surged. About 13% of adults with mood disorders have comorbid ADHD; conversely, 38% of individuals with ADHD show comorbid mood disorders (Kessler et al., 2006). Adult ADHD and mood disorders both have a high prevalence, a lengthy illness course, low case

detection, a multifactorial aetiology, a high rate of comorbidity, significant financial costs and illness burden and considerable interpersonal and occupational problems (McIntyre et al., 2010). Furthermore, neuroimaging studies reveal that both disorders share common neural circuits and brain regions (Spencer et al., 2007; Seidman et al., 2005).

ADHD doubles the likelihood of comorbid depressive disorder (Jensen et al., 1993; Kessler et al., 2006); however, ADHD stimulant treatment may decrease the risk of anxiety and depression over time (Biederman et al., 2009). Adults with depression exhibit ADHD symptoms at a rate of about 5% to 12%, compared to around 4% in healthy controls (Alpert et al., 1996; Joo et al., 2012; Kessler et al., 2006; McIntyre et al., 2010). Conversely, 9% to 25% of individuals with ADHD experience depressive symptoms compared to 1% to 8% of those without ADHD (Chen et al., 2013; Fischer et al., 2007; Kessler et al., 2003; Kessler et al., 2006).

Research also supports the co-occurrence of ADHD and bipolar disorder (NICE, 2014). Based on previous studies, the prevalence of ADHD in children with bipolar disorder ranges from 57% to 98%, whereas the rate of bipolar disorder in children with ADHD is 22% (Faraone et al., 1997; Singh et al., 2006). There continues to be much controversy about the validity of concurrent diagnoses of ADHD and severe mood instability or bipolar disorder (Wilens et al., 2010). ADHD is typically characterised by cognitive and hyperactivity/impulsivity symptoms, while bipolar disorder is associated with mood instability, psychosis, persistent irritability/rage and lack of response to structure (Wilens et al., 2003). Individuals who exhibit both groups of symptoms may suffer from both ADHD and bipolar disorder (Wilens et al., 2003).

In an investigation by Tamam et al. (2008), people with a history of childhood ADHD and those with adulthood ADHD showed an earlier age-onset of bipolar disorder. They had

earlier affective or depressive episodes than individuals without ADHD. Moreover, individuals with bipolar disorder and comorbid ADHD had more axis I psychiatric disorders such as AUD or panic disorder than those with just bipolar disorder. Axis I psychiatric disorders, introduced by the DSM, include mood disorders, anxiety disorder, eating disorders, SUD, psychotic disorders and dissociative disorders (DSM-5, 2013).

Studies show that 6% to 15% of men and women with bipolar disorder have comorbid ADHD (Cordera et al., 2014). Other investigations indicate an even higher prevalence of ADHD in individuals with bipolar disorder (e.g. McIntyre et al., 2010; Nierenberg et al., 2005; Perugi et al., 2013; Wingo & Ghaemi, 2007). The incidence of bipolar disorder in adults with ADHD is reported to be 20% by epidemiological research; it can even reach 50% in individuals with ADHD if bipolar disorder symptoms are considered (Halmoy et al., 2010; Kessler et al., 2006; McGough et al., 2005). Many different hypotheses have been proposed to explain the association between ADHD and bipolar disorder, ranging from shared genetic factors to overlapping dimensions such as impulsivity (Cordera et al., 2014; Youngstrom et al., 2010). Those with comorbid ADHD and bipolar disorder show an early onset of bipolar disorder, more episodes of depression-manic and only depression, fewer periods without symptoms and poorer treatment outcomes (Karaahmet et al., 2013; Nierenberg et al., 2005; Tamam et al., 2008).

It is worth noting that the prevalence of ADHD symptoms in those with bipolar disorder (21.2%) is higher than those with depression (9.4%) (Kessler, 2006). Furthermore, both ADHD and bipolar disorder have similar characteristic conditions and diagnostic criteria; their symptoms also overlap, making diagnosis more complicated (Kent & Craddock, 2003; Wingo & Ghaemi, 2007). On the one hand, the two conditions' similarities, such as increased energy, high distractibility, physical restlessness and impaired response

inhibition, led some researchers to find statistically significant associations between bipolar disorder and ADHD, but not depression (Kessler et al., 2010; Kim et al., 2019). On the other hand, some fundamental differences between these two disorders must be noted; bipolar disorder has cycling symptoms and mainly influences mood, while ADHD has chronic symptoms and primarily impacts behaviour. Furthermore, individuals with both disorders have more psychiatric comorbidities such as anxiety and substance misuse (Tamam et al., 2008). Although studies revealed an association between ADHD and bipolar disorder, none investigated the relationship between each ADHD symptom cluster and bipolar disorder separately. Hence, one of Study 4's aims was to explore the relationship between each symptom of ADHD and bipolar disorder.

Wilens (2014) claimed that 30% of adults with ADHD symptoms used substances for recreational pleasure, but 70% did so to cope with their symptoms, get better sleep or improve their mood. Moreover, individuals with undiagnosed ADHD self-medicate with different substances to be productive (Sherman et al., 2007). Epidemiological studies show that mood disorder is 4.7 times more prevalent in those who misuse substances than the general population (Ross et al., 2016). Research also found a strong association between depression and substance misuse (Bolton et al., 2009; Davis et al., 2005).

Since ADHD and substance misuse share genetic and neurobiological characteristics, it can be argued that treatments for both conditions could be combined (Arcos-Burgos et al., 2012; Frodl, 2010). Particularly, untreated ADHD in individuals with poor concentration and high impulsive behaviours may interfere with substance misuse treatment, causing worse outcomes (Ercan et al., 2003). In summary, previous investigations revealed that, on the one hand, mood disorders could play a crucial role in developing substance misuse; on the other hand, adults with ADHD symptoms exhibit more mood disorders than the general

population. However, research on mood disorders, especially bipolar disorder, in adults with ADHD symptoms and comorbid substance misuse is limited. Therefore, further investigations on the role of bipolar disorder as a mood disorder in the relationship between ADHD symptoms and substance misuse is imperative.

5.3. Substance misuse and mood disorders

Researchers have revealed the association between mental disorders and substance misuse. Three scenarios should be considered in their relationship. First, there are risk factors that increase the likelihood of comorbid mental conditions and substance misuse. According to research, psychiatric disorders and substance misuse share common genetic vulnerabilities (Volkow, 2010). For instance, early onset of cannabis use or repeated drug administration raises the risk of mental disorders in adulthood (Markou et al., 1998; McEwen, 2000; Volkow, 2010). In addition, genetic factors may influence a person's response to a substance and whether or not it can provide them with a positive feeling (Ducci & Goldman, 2012). Environmental factors, such as stress and trauma, may cause genetic changes that can be passed down through generations and may be linked to the development of substance misuse and mental disorders (Nielsen et al., 2012).

Second, mental disorders and substance misuse are related. Studies have identified some psychiatric disorders that increase the probability of developing substance misuse and SUD (Baigent, 2012; Kelly & Daley, 2013). For instance, people with mental disorders self-medicate using alcohol and other substances (Khantzian, 1985; Markou et al., 1998; Santucci, 2012). In addition, brain changes in people with psychiatric conditions may heighten the rewarding effects of a substance, motivating the individual to continue using it (Markou et al., 1998; Merikangas et al., 2008; Santucci, 2012).

Third, substance misuse and SUD alter the brain, thereby increasing the risk of developing psychiatric disorders (Markou et al., 1998; McEwen, 2000). Gómez-Coronado et al.'s (2018) review explored the shared neurobiological mechanisms between mood disorder, as a mental disorder, and substance misuse. They found that neurotransmitters, such as dopamine, GABA, and glutamate and their receptors, the central corticotropin-releasing hormone, hypothalamic–pituitary–adrenal axis activation, oxidative stress and inflammation are all involved (Gómez-Coronado et al., 2018). Given the high comorbidity of psychiatric conditions and substance misuse, research should better understand their bidirectional relationship. In particular, the neurobiological mechanisms underlying the connection between mood disorders and substance misuse have not yet been thoroughly explored; further in-depth investigations are required.

Studies indicate that mood disorders are the most common psychiatric comorbidities among people who misuse substances (Quello et al., 2005). Individuals who misuse substances are twice as likely to develop mood and anxiety disorders (Volkow, 2010). Mood disorders may increase a person's vulnerability to substance misuse and addiction; thus, its diagnosis and treatment may mitigate the risk of SUD later on (Volkow, 2010). The inverse may be true, with diagnosis and treatment of substance misuse lessening the severity of mood disorders while heightening treatment efficacy (Volkow, 2010).

If depression and substance misuse are considered as separately occurring conditions, they might be attributed to certain neurobiological, genetic and environmental factors. However, the underlying mechanisms of both conditions are not well understood when they co-occur (Volkow, 2004). Four main hypotheses may explain comorbid substance misuse and depression (Nunes & Levin, 2008). First, the self-medication theory proposes that individuals with depression use substances repeatedly to alleviate their depressive symptoms,

resulting in SUD (Markou et al., 1998). Second, taking different substances or experiencing withdrawal symptoms after ceasing to use them may lead to depression (substance-induced depression) (Anand et al., 2019). Third, depressive symptoms may stem from a substance-abusing lifestyle (Nunes & Levin, 2008). Lastly, since both conditions are thought to have common psychological or biological backgrounds, co-occurrence is high (Quello et al., 2005). Regardless of their order of occurrence, one may maintain or exacerbate the other (Nunes & Levin, 2008).

Regarding nicotine use, the NHS reports (2018–19) state that smoking rates rise in tandem with the severity of mental health symptoms. Moreover, individuals with psychiatric conditions smoke significantly more, have considerably heightened levels of nicotine use disorders and are consequently at greater risk of smoke-related harm. Smoking in the general population of the UK has fallen from 27% in the mid-1990s to 14.9% in 2017 (NHS Digital, 2018; Office of National Statistics, 2017). However, data from Public Health England indicates that 40.5% of individuals with severe mental health problems smoke (Public Health England, 2015), a percentage that has remained constant for the last 20 years (Szatkowski & McNeill, 2015). The Health Survey for England (HSE, 2019) reported long-term decreases in the prevalence and average daily smoking of individuals without a longstanding mental health problem or taking psychoactive medication. Despite the possibility of a reduced prevalence among those taking psychoactive drugs, their data show no decline in people with mental disorders.

Some investigators found a significant association between common mental disorders, such as mania and depression, and nicotine dependence (Breslau et al., 2004; Grant et al., 2004). They revealed that smoking nicotine is more prevalent in individuals with mood and anxiety disorders than those with other multiple psychiatric illnesses or without

any psychiatric disorder (Goodwin et al., 2008; McCabe et al., 2004). It has been proposed that those with bipolar disorder have an increased susceptibility to nicotine smoking (Gutiérrez-Rojas et al., 2008; Moylan et al., 2013). The underlying mechanisms of the association between nicotine use and bipolar disorder are bidirectional (Cassidy et al., 2002; Plante & Winkelman, 2008). Therefore, although it is correct that nicotine use is a risk factor for bipolar disorder in most people, caution is recommended when interpreting these findings and suggesting causality.

Investigators have suggested different reasons for people to begin and continue smoking. As explained in Chapter 1, nicotine is absorbed into the bloodstream as tobacco is smoked. Nicotine stimulates dopamine production, which is associated with pleasurable feelings (Novak et al., 2010; Pomerleau & Pomerleau 1984). Smokers develop regular smoking patterns, ensuring a steady release of dopamine; they start to crave nicotine when its level decreases in their bloodstream. Thus, they experience ‘stress’ until the craving is relieved. Smokers also often describe this sense of relief as ‘relaxing’; they need an increasing amount of nicotine to feel ‘normal’ (Markou, 2008; Prochaska, 2011; Ratschen et al., 2011; Watkins et al., 2000). In this regard, cyclical dopamine dysregulation has been suggested as the primary mechanism underlying bipolarity (Evins et al., 2014). Previous studies have shown that individuals with depression and nicotine use self-medication to reduce negative emotion and increase the positive affect more than those who use nicotine but are not depressed (Lerman et al., 1996). Additionally, nicotine replacement decreases the post-cessation negative affect in people with depression (Kinnunen et al., 1996). However, it is unclear whether these results apply to individuals with nicotine use and bipolar disorder. The link between smoking and bipolar disorder requires further investigation.

Genetic vulnerability and common engaged regions of the brain are other possible reasons for higher smoking rates in people with mood disorders. Smoking may lead to changes in neurophysiology that reveal an underlying vulnerability to affective episodes. Consistent with this hypothesis, chronic nicotine use may increase the risk of depression by desensitising nicotinic acetylcholine receptors in the limbic system (Mineur & Picciotto, 2009). Furthermore, long-term nicotine use can raise the likelihood of depression by influencing serotonin pathways in the hippocampus (Balfour & Ridley, 2000). Even though there has not been a systematic study of this phenomenon at the onset of bipolar disorder, it has been proposed that ceasing nicotine use may precipitate an affective episode in some individuals with bipolar disorder who use nicotine (Heffner et al., 2011).

The high prevalence (20% to 55%) of current depression in treatment-seeking individuals who misuse substances is a concern (Burns et al., 2005; Mcketin et al., 2011; Nunes et al., 2004; Rounsaville et al., 1991; Teesson et al., 2005). Although depression has been linked to poorer treatment outcomes (Dodge et al., 2005; Greenfield et al., 1998; Hasin et al., 2002; McKay et al., 2002; Teesson et al., 2008), the development and assessment of behavioural interventions for depression have received less empirical attention. Individuals with depression and substance misuse have a more prolonged and more severe illness course, poorer social and occupational functioning, higher frequency of health service use, greater Post-traumatic Stress Disorder (PTSD) risk, more suicidal behaviours and more relapse of their substance misuse or SUD (Havard et al., 2006; McLellan et al., 1983; Teesson et al., 2008).

Mood disorders and AUD can co-occur and are two prevalent conditions that start in adolescence and continue into adulthood (Kessler et al., 2007a, 2012). Investigations with adolescents indicate that those with depression show almost double the rates of alcohol

misuse (65%) than individuals without a mental disorder (34%) (Lawrence et al., 2015). Similarly, the severity of depressive symptoms could be a strong predictor of mid-adolescence alcohol misuse frequency (Scholes-Balog et al., 2015). Nevertheless, when parents' attitudes towards substance misuse were taken into account, the relationship between depression and alcohol misuse in adolescents was no longer significant (Scholes-Balog et al., 2015). A UK cohort study on the association between mood and alcohol disorders tested 7,100 adolescents and found that alcohol misuse between the ages of 13 and 15 is linked to depressive symptoms at 17 and above (Edwards et al., 2014). Birrell et al. (2016) indicated that bipolar disorder is a unique risk factor for first-time alcohol use in the general population, with significant developmental timing interactions.

Although the relationship between cannabis use and mood disorders remains unclear (Hall & Degenhardt, 2009; Lev-Ran et al., 2013), cross-sectional investigations report the comorbidity of cannabis use and depression as a global health burden (Chen et al., 2002; Grant, 1995; Whiteford et al., 2013). Moreover, the evidence from longitudinal studies on cannabis use and depression is conflicted. Many studies have discovered a significant association between baseline cannabis use and future depression (Bovasso, 2001; Fergusson & Horwood, 1997). However, other investigations have found no difference between cannabis users and non-users in developing depression later in life (Brook et al., 2002; Degenhardt et al., 2011). Lev-Ran et al.'s (2013) meta-analysis revealed that there might be a correlation between cannabis use, especially heavy use, and depression, but confounding factors may limit the causation.

Studies on the association between bipolar disorder and cannabis use are much fewer than those on depression. However, the association between cannabis use and bipolar disorder is strong in some cross-sectional studies (Cerullo & Strakowski, 2007; Etain et al.,

2012). Moreover, several longitudinal investigations also indicate a relationship between cannabis use baseline and potential bipolar disorder (Baethge et al., 2008; Henquet et al., 2006; Tijssen et al., 2010). It has also been suggested that individuals with bipolar disorder may use cannabis to reduce symptoms of their disorder's manic/hypomanic and depressive stages (Bizzarri et al., 2007, 2009); however, longitudinal studies do not support these claims (Bolton et al., 2009).

Reviewing previous studies in this section reveals a discrepancy in the literature on the connection between bipolar disorder and substance misuse. Moreover, none of the past research examined the role of bipolar disorder in the relationship between each ADHD symptom cluster and the use of alcohol, cannabis and nicotine. Therefore, further research into bipolar disorder's ability to mediate between (a) inattention and substance misuse and (b) between hyperactivity/impulsivity and substance misuse is needed.

5.4. Mood disorders, ADHD and substance misuse among university students

Many mental health problems, especially mood disorders, peak between ages 18 to 25; this is a vulnerable developmental period in a person's life (Kessler et al., 2007; Royal College of Psychiatrists, 2011). For many people, this stage coincides with attending university, a critical life event that can be highly stressful for some students (Conley et al., 2014; Kessler et al., 2007; NHS, 2020). Those with pre-existing mental health issues can experience a worsening of their symptoms during this phase (McLafferti et al., 2017), affecting academic performance (Auerbach et al., 2016; Zivin et al., 2009). Others can develop psychopathology due to stressful university life and adapting to a new environment

(Bewick et al., 2010; Eisenberg et al., 2007). Besides adjusting to university life, transitioning to adult life could also be challenging (McLafferty et al., 2017). For some older students, being away from family or friends for the first time, shifting from a more structured school environment to less structured university life or having other responsibilities, such as caring for family or work commitments, can exacerbate mood disorders and mental health problems (Keeling, 2003). Since significant life events can cause distress in some people, researchers argued that being a university student is an emotional period that can lead to a sense of alienation, exclusion and loss (Christie et al., 2008; Johnson et al., 2008; Pompili et al. 2009).

In the UK, the prevalence of mental health problems such as depression, bipolar disorder or suicidality is high among university students (Bewick et al., 2010; Cooke et al., 2006). For instance, first-year undergraduate students reported that their anxiety levels were extremely high during their first year; they also disclosed having poorer psychological wellbeing during their time at university relative to their pre-entry level (Bewick et al., 2010; Cooke et al., 2006). In another study, about 25% of first-year students had clinical psychological distress (Topham et al., 2011). In addition, increased enrolment in UK universities could be related to a rise in psychological problems, with 17.3% of students having mental health issues (McLafferty et al., 2017). The development or continuation of mood disorders during university is not uncommon (Demery et al., 2012). Based on previous literature, stress has been cited as a potential causal factor in developing depressive disorders (Dienes et al., 2006; Kim et al., 2007; Post & Leverich, 2006). Furthermore, Hlastala et al. (2000) argued that a stressful life event is more likely to trigger a bipolar disorder episode in young adults with greater psychological vulnerability.

Some students develop mental health disorders for the first time while at university. In a study of 739 Ulster University students in Northern Ireland (2017), the prevalence of lifetime and 12-month mental health problems, substance misuse, ADHD and suicidality were remarkably high, with more than 50% of the undergraduate students reporting a lifelong mental disorder (McLafferty et al., 2017). As discussed in Chapter 1, Section 1.2 of this thesis, university students use more alcohol, cannabis and nicotine than their non-student peers. Moreover, the highest prevalence of bipolar disorder is among individuals aged 16 to 24, most of whom are university students (APMS, 2014). Additionally, people with ADHD symptoms consume higher amounts of alcohol, cannabis and nicotine (Arias et al., 2008). Thus, based on the previous studies and using a dimensional approach for the present study, it is likely to detect a distribution of scores of bipolar disorder, ADHD and substance misuse in the current study's sample.

Thus, the motivations for conducting the current study are as follows: (a) higher rates of substance misuse and mood disorders, specifically bipolar disorder, in university students; (b) associations between ADHD and bipolar disorder, ADHD and substance misuse and bipolar disorder and substance misuse; and (c) the gap in the literature concerning the role of bipolar disorder in the relationship between inattention and hyperactivity/impulsivity and substance misuse.

5.5. Aims of this study

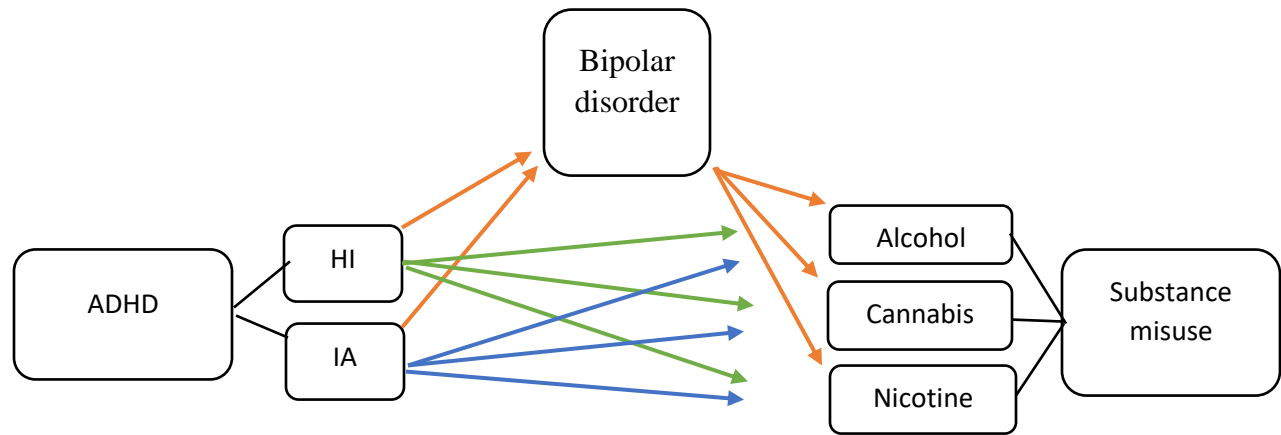
This research is the first to separate the two chief symptom clusters of ADHD (hyperactivity/impulsivity and inattention), intending to investigate the relationship between each ADHD symptom, bipolar disorder and substance misuse, separately in a typically developing university students. This study's main objective is to explore whether bipolar

disorder, as a mood disorder, could mediate the connection between different ADHD symptoms and alcohol, nicotine and cannabis use in a sample of undergraduate students.

A four-step regression model was employed to examine the role of bipolar disorder in mediating these two conditions. The first step investigated whether ADHD symptoms, as independent variables, predicted bipolar disorder significantly, while the second phase determined whether bipolar disorder, as a mediator, predicted substance misuse significantly. The third step then explored whether each ADHD symptom indicates the use of alcohol, cannabis and nicotine significantly. The last step examined whether or not the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine weakens in the presence of bipolar disorder as a mediator (Figure 5.1).

To establish that bipolar disorder fully mediates the relationship between ADHD symptoms and substance misuse, the effect of ADHD symptoms on substance misuse controlling for bipolar disorder should be zero. Otherwise, bipolar disorder is considered a partial mediator in the model. If bipolar disorder is a crucial risk factor for substance misuse in individuals with ADHD, a novel treatment for these people with comorbid bipolar disorder, as a mood disorder and substance misuse may be developed in the future.

According to previous investigations, it is hypothesised that bipolar disorder predicts alcohol, cannabis and nicotine use in the model's second step. In line with Study 4's findings, it is presumed that in the analysis's third step, both ADHD symptom clusters predict alcohol, cannabis and nicotine use significantly. Since this research is the first to investigate the association between ADHD symptoms, bipolar disorder and substance misuse separately, the first and fourth steps of the regression analysis were exploratory research questions and no specific prediction is made.



- The direct effect of ASRS-HI on alcohol, cannabis and nicotine misuse.
- The direct effect of ASRS-IA on alcohol, cannabis and nicotine misuse.
- The indirect effect of ASRS-HI and IA on the use of alcohol, cannabis and nicotine via bipolar disorder as a mood disorder.

Figure 5.1: The relationship between the different variables of this study. Based on previous literature, ADHD can predict all three, alcohol, nicotine and cannabis use significantly. Some studies showed the relationship between each ADHD symptom and these three substance misuses. The current study is an attempt to find if mood disorder can mediate each ADHD subtype and alcohol, nicotine and cannabis use significantly.

5.6. Methods

5.6.1. Participants

Two hundred and seventy participants were recruited through the Department of Psychology's Research Participation Scheme at Goldsmiths, University of London. The non-completion of the questionnaires resulted in the exclusion of 47 participants. Consequently, there were a total of 223 participants, all of whom were over 18 years old, with a mean age of 19.29 years ($SD = 6.10$). Moreover, women comprised 75.1% of the participants ($n = 169$). This study only recruited first-year undergraduates, who were given course credits for their participation.

Given that this study was combined with Chapter 6's analysis, participants were recruited and tested simultaneously. G*Power3.1.9.7 calculated a sample of 119 participants for both studies. More participants were recruited for this study and the subsequent research ($n = 270$) to increase their statistical power. Individuals with major psychiatric disorders, such as schizophrenia, borderline personality disorder, obsessive-compulsive disorder, and significant physical health problems, such as brain injury, were excluded. Participants had to confirm that they were 18 or older and agreed to participate in the study via an online consent form. All of them completed the measures anonymously.

5.6.2. Procedure

The Department of Psychology's ethics committee at Goldsmiths, University of London, approved this study (Ethics Reference Number: PS130617ZSS) (please see Appendix 15 for the sanctioned Ethical Approval Form). Participants were recruited through the Department of Psychology's Research Participation Scheme. The study's details and link were advertised on the Research Participation Scheme page, along with the researcher's email address. Participants could email the researcher and ask any question about the study. More information about the questionnaires' number of items and duration, ethical issues and the study's overall focus was on the first page of the online survey. The questionnaires were designed in a way that participants could skip a question they did not want to answer. There was also a debrief page with detailed information about the study at the end of the survey for participants.

5.6.3. Measures

The measures (AUDIT, AUQ, CUDIT-R, CAN, NIC, and ASRS) used in the previous chapters were employed to assess substance misuse and ADHD symptoms. For the complete details, please see Section 2.6.3. The Cronbach alpha values for the current study's measurements are as follows: ASRS-IA = .89; ASRS-HI = .89; ASRS-total = .93; AUDIT = .95; AUQ = .74, NIC = .86; CUDIT-R = .99; CAN = .68.

5.6.3.1. *The Mood Disorder Questionnaire (MDQ) (Hirschfeld et al., 2000);*

A team of psychiatrists and researchers developed the MDQ to address the need for an appropriate and precise diagnosis of bipolar disorder. It is a short questionnaire that is freely accessible. Previous investigations show that the MDQ has a satisfactory level of accuracy and specificity, allowing it to identify seven out of ten people with bipolar disorder while excluding nine out of ten individuals who do not have this disorder (Hirschfeld et al., 2000). There are five main questions to which the participant must respond with a 'yes' or 'no' (although Question 1 has 13 sub-questions). Answering 'yes' to seven or more of the 13 items under the first question, responding with 'yes' to the second question and choosing 'moderate' or 'serious' for the third question are all indicative of a person having a mood disorder (please see Appendix 11 for the complete questionnaire). In a meta-analysis of the MDQ's validity, investigators discovered that summary sensitivity was .62 and summary specificity was .85 at the standard cutoff value of 7 (Wang et al., 2015). No diagnostic accuracy differences were observed after adjusting for various clinical correlates in investigations of Eastern and Western countries. The MDQ was also found to have good overall diagnostic accuracy (Wang et al., 2015). For the current study, the calculated Cronbach Alpha for this scale was .82.

5.6.4. Data analysis strategy

Data were analysed using IBM SPSS version 20. Pearson correlation was conducted to examine the connection between the variables of the study and the results are presented in Table 5.4. The ADHD scores were divided into two categories: inattention (ASRS-IA) and hyperactivity/impulsivity (ASRS-HI), which are the main ADHD symptoms. A four-step mediation analysis was conducted for each category, which aimed (a) to investigate the connection between different ADHD symptoms and BD; (b) to consider the link between the use of alcohol, cannabis and nicotine and BD; (c) to investigate whether ADHD facets predict the use of alcohol, cannabis and nicotine; and (d) to explore whether the relationship between the ADHD symptoms and substance misuse diminishes in the presence of BD. Furthermore, the Sobel test and PROCESS macro were used to measure the indirect effect between the variables of this study.

5.7. Results

5.7.1. Outliers and multi-collinearity

An analysis of standard residuals showed that the data contained seven outliers in cannabis and nicotine use (Std. Residual Min >-3.29 , Std. Residual Max <3.29), which were deleted from the data. The assumption of collinearity indicated that multi-collinearity was not a concern (VIF value is less than 10 and the Tolerance is more than 0.1).

In this study to correct for slight positive skew (1.71) (kurtosis = 2.60) in the AUDIT scores, AUQ scores (skewness = 2.14, kurtosis = 4.55) and CUDIT-R (skewness = 1.63, kurtosis = 1.75) and CAN (skewness = 1.04, kurtosis = 1.54) scores were log transformed

(new = LG10 (old + a); a= 1). The skewness and kurtosis of each questionnaire after transformation were as follows: AUDIT scores (skewness = -.27, kurtosis = .08), AUQ scores (skewness = -.45, kurtosis = -.36) and CUDIT-R (skewness = .05, kurtosis = -.74) and CAN (skewness = -.66, kurtosis = -.87)

5.7.2. The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

Table 5.1 presents the frequency and quantity of alcohol, cannabis and nicotine use by percentage. With the AUDIT, alcohol use frequency was determined by the question ‘How often do you have a drink containing alcohol?’, while the quantity of alcohol was established by asking, ‘How many standard drinks containing alcohol do you have on a typical day when drinking?’. The participant had five options to choose from, with the percentage of each answer listed in the table below. Based on the results, 32.9% of participants showed hazardous alcohol use by gaining an AUDIT score of 8 or higher. In addition, 19.1% of women and 16% of men indicated alcohol use dependence with a score of 13 or more for women and 15 or more for men.

Moreover, the frequency and quantity of cannabis use were measured by asking the participants, ‘How often do you use cannabis?’ and ‘How many hours were you stoned when you had been using cannabis?’. Table 5.1 summarises the results by calculating the percentage of each answer in the sample. Results revealed that 20% of participants had hazardous cannabis use, and 15.1% were dependent on it. The CAN also assessed cannabis use throughout a lifetime and the previous year; results are likewise included in Table 5.1.

The frequency of nicotine use was discerned by asking ‘During the past 30 days, on how many days did you smoke cigarettes?’, while the quantity of nicotine use was gleaned by inquiring, ‘During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?’. The percentage of each answer is also given in Table 5.1.

According to this study’s findings, 15.5% of the participants met the MDQ’s positive screen criteria for bipolar disorder (seven or more ‘yes’ answers to the first 13 questions; ‘yes’ response to the second question; choosing ‘moderate’ or ‘serious’ for the third question). Additionally, 22.7% of the sample had an ASRS score of 24 or higher, indicating a high probability of having ADHD.

Table 5.1

Percentage of alcohol, cannabis and nicotine use frequency, quantity and hazardous use and dependence.

| | Frequency | Percent | Hazardous use (%) | Dependence (%) |
|--|-----------|---------|-------------------|----------------------------|
| AUDIT | | | 32.9 | Women = 19.1 Men = 16.0 |
| AUDIT frequency (How often do you have a drink containing alcohol?) | | | | |
| Never | 84 | 37.3 | | |
| Monthly or less | 33 | 14.7 | | |
| 2-4 times a month | 35 | 15.6 | | |
| 2-3 times a week | 41 | 18.2 | | |
| 4 or more times a week | 30 | 13.3 | | |
| AUDIT quantity (How many standard drinks containing alcohol do you have on a typical day when drinking?) | | | | |
| 1 or 2 | 123 | 54.7 | | |
| 3 or 4 | 60 | 26.7 | | |
| 5 or 6 | 33 | 14.8 | | |
| 7 to 9 | 6 | 2.7 | | |
| 10 or more | 1 | .4 | | |
| CUDIT-R | | | 20.0 | 15.1 |
| CUDIT-R frequency (How often do you use cannabis?) | | | | |
| Never | 148 | 65.8 | | |
| Monthly or less | 32 | 14.2 | | |
| 2-4 times a month | 25 | 11.1 | | |
| 2-3 times a week | 8 | 3.6 | | |
| 4 or more times a week | 10 | 4.4 | | |
| CUDIT-R quantity (How many hours were you stoned when you had been using cannabis?) | | | | |
| Less than 1 | 181 | 80.4 | | |
| 1 or 2 | 27 | 12.0 | | |
| 3 or 4 | 10 | 4.4 | | |
| 5 or 6 | 3 | 1.3 | | |
| 7 or more | 2 | .9 | | |
| CAN | | | | |
| Lifetime cannabis use (Have you ever used cannabis in your life?) | | | | |
| Yes | 118 | 52.6 | | |
| No | 105 | 46.7 | | |
| Cannabis quantity in the last year (In the last year, on how many separate days have you used cannabis?) | | | | |
| Never | 130 | 57.8 | | |
| Once every 12 months | 7 | 3.1 | | |
| Once every 2 to 3 months | 11 | 4.9 | | |
| Once a month | 6 | 2.7 | | |
| 2 to 3 times a month | 7 | 3.1 | | |
| Once a week | 10 | 4.4 | | |
| 2 to 3 times a week | 22 | 9.8 | | |
| 4 to 5 times a week | 20 | 8.9 | | |

| | | |
|--|-----|------|
| Daily or nearly daily | 10 | 4.4 |
| NIC | | |
| NIC frequency | | |
| (During the past 30 days, on how many days did you smoke cigarettes?) | | |
| 0 days | 132 | 58.7 |
| 1 to 2 days | 14 | 6.2 |
| 3 to 5 days | 6 | 2.7 |
| 6 to 9 days | 10 | 4.4 |
| 10 to 19 days | 16 | 7.1 |
| 20 to 29 days | 15 | 6.7 |
| All 30 days | 30 | 13.3 |
| NIC quantity | | |
| (During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?) | | |
| I did not smoke cigarettes during the past 30 days | 132 | 58.7 |
| Less than 1 cigarette per day | 14 | 6.2 |
| 1 cigarette per day | 10 | 4.4 |
| 2 to 5 cigarettes per day | 45 | 20.0 |
| 6 to 10 cigarettes per day | 10 | 4.4 |
| 11 to 20 cigarettes per day | 11 | 4.9 |
| More than 20 cigarettes per day | 1 | .4 |

Note: AUDIT = Alcohol Use Identification Test; CUDIT-R = Cannabid Use Identification Test-Revised; NIC = Nicotine Use Test

Table 5.2

The frequency of wine and wine type products, beer or cider, spirits

| Days in a week | Wine | | Beer or cider | | Spirits | |
|----------------|-----------|------|---------------|------|-----------|------|
| | Frequency | % | Frequency | % | Frequency | % |
| 0 | 117 | 52.0 | 127 | 57.0 | 117 | 52.0 |
| 1 | 49 | 21.8 | 44 | 19.7 | 62 | 27.6 |
| 2 | 22 | 9.8 | 24 | 10.8 | 24 | 10.7 |
| 3 | 9 | 4.0 | 4 | 1.8 | 9 | 4.0 |
| 4 | 1 | .4 | 3 | 1.3 | 5 | 2.2 |
| 5 | 4 | 1.8 | 5 | 2.2 | 2 | .9 |
| 6 | 6 | 2.7 | 4 | 1.8 | 3 | 1.3 |
| 7 | 14 | 6.2 | 12 | 5.3 | 1 | .4 |

5.7.3. Descriptive statistics and the correlation between the variables

The descriptive statistics and Pearson correlation analysis of this study are shown in tables 5.3 and 5.4. Men were coded as ‘1’ and women were coded as ‘2’ in this study. The correlation analysis between variables showed that mood disorder was statistically

significantly positively correlated with all three scores of ADHD (r (ASRS-IA) = .59, r (ASRS-HI) = .59, r (ASRS-total) = .64, $p < .001$, $n = 223$) and also with alcohol ($r = .58$), cannabis ($r = .50$) and nicotine use ($r = .37$; $p < .001$, $n = 223$). In addition, there was a statistically significant negative correlation between bipolar disorder and the age of participants ($r = -.21$, $p < .05$, $n = 214$). All three scores of ADHD was also correlated significantly with all substance misuse scores.

Table 5.3
Descriptive statistics and demographic information

| | Mean | Std. Deviation | <i>n</i> | Range | Minimum | Maximum |
|------------|-------|----------------|----------|-------|---------|---------|
| Age | 23.29 | 7.10 | 214 | 32 | 18 | 50 |
| BD | 8.42 | 4.16 | 223 | 15 | 1 | 16 |
| ASRS-IA | 17.92 | 7.23 | 223 | 36 | 0 | 36 |
| ASRS-HI | 14.48 | 7.22 | 223 | 36 | 0 | 36 |
| ASRS-total | 32.40 | 13.26 | 223 | 70 | 0 | 70 |
| AUDIT | 9.59 | 9.25 | 223 | 47 | 0 | 47 |
| AUQ | 31.75 | 39.64 | 223 | 206 | 0 | 206 |
| CUDIT-R | 6.17 | 8.57 | 223 | 36 | 0 | 36 |
| CAN | 17.15 | 16.95 | 223 | 80 | 0 | 80 |
| NIC | 21.72 | 18.10 | 223 | 53 | 0 | 53 |

Note: BD= Bipolar Disorder; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

Table 5.4
Pearson correlation between ADHD, bipolar disorder as a Mood Disorder and substance misuse variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|--------------|--------|--------|-------|-------|-------|-------|-------|-------|-------|-------|----|
| 1-Age | 1 | | | | | | | | | | |
| 2-Gender | -.37** | 1 | | | | | | | | | |
| 3-BD | -.21** | -.07 | 1 | | | | | | | | |
| 4-ASRS-IA | -.27** | .01 | .59** | 1 | | | | | | | |
| 5-ASRS-HI | -.08 | -.20** | .59** | .69** | 1 | | | | | | |
| 6-ASRS-total | -.19** | -.10 | .64** | .92** | .92** | 1 | | | | | |
| 7-AUDIT | -.01 | -.18** | .58** | .63** | .66** | .70** | 1 | | | | |
| 8-AUQ | .02 | -.19** | .46** | .52** | .56** | .59** | .82** | 1 | | | |
| 9-CUDIT-R | .00 | -.25** | .51** | .61** | .67** | .70** | .71** | .63** | 1 | | |
| 10-CAN | -.05 | -.19** | .44** | .47** | .47** | .51** | .60** | .59** | .73** | 1 | |
| 11-NIC | .19** | -.22** | .36** | .34** | .36** | .38** | .61** | .61** | .50** | .63** | 1 |

Note: BD= Bipolar Disorder; ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

5.7.4. Does bipolar disorder as a mood disorder mediate the relationship between ASRS-IA and substance misuse?

A) AUDIT:

The results of the multiple regression in the first step of the mediation analysis showed that ASRS-IA predicted bipolar disorder in a significant way ($\beta = .59, p < .001$) and it accounted for 34.4% of the variance ($R^2 = .34, F(1,221) = 116.12, p < .001$). In the second step, bipolar disorder predicted AUDIT scores significantly and accounted for 33.5% of the variance ($R^2 = .33, F(1,221) = 11.22, p < .001$) ($\beta = .58, p < .001$). In the third step, ASRS-IA predicted alcohol use significantly ($\beta = .63, p < .001$). It accounted for 39.2% of the variance in AUDIT ($R^2 = .39, F(1,221) = 142.5, p < .001$).

The last step showed that controlling for the mediator (bipolar disorder), ASRS-IA scores were still a significant predictor of AUDIT scores, ($\beta = .44, t(223) = 7.14, p < .001$). The indirect effect size between ASRS-IA and AUDIT scores was .98 with a 95% confidence interval that did not include zero. The Sobel test was conducted and bipolar disorder could mediate ASRS-IA and AUDIT scores partially ($z = 4.41, p < .001$) (Table 5.5).

B) AUQ

In the first step of the mediation model, independent variable, ASRS-IA, predicted bipolar disorder as the mediator significantly ($R^2 = .34, F(1,221) = 116.12, p < .001$) and accounted for 34.4% of the variance ($\beta = .59, p < .001$). The second step of the multiple regression showed that bipolar disorder is a significant predictor of AUQ ($\beta = .46, p < .001$). it accounted for 20.8% of the variance in AUQ scores ($R^2 = .21, F(1,221) = 58.09, p < .001$). The third step of the mediation model revealed that ASRS-IA predicts AUQ scores

significantly ($R^2 = .27$, $F(1,221) = 81.8$, $p < .001$) and accounted for 27% of the variance in AUQ ($\beta = .52$, $p < .001$). The last step indicated that after controlling bipolar disorder as the mediator, ASRS-IA is still a significant predictor of AUQ score ($\beta = .38$, $t(223) = 5.54$, $p < .001$). The indirect effect size between ASRS-IA and AUQ scores calculated by PROCESS macro was 1.10 with a 95% confidence interval, which did not include zero. Sobel test was conducted and the results showed that bipolar disorder is a significant partial mediator between ASRS-IA and AUQ ($z = 3.07$, $p = .002$) (Table 5.5).

C) CUDIT-R

The first step of the multiple regression was the same as AUDIT and AUQ. ASRS-IA predicted bipolar disorder significantly and accounted for 34.4% of the variance ($\beta = .59$, $p < .001$). The second regression analysis indicated that bipolar disorder is a significant predictor of CUDIT-R scores ($\beta = .51$, $p < .001$) and accounted for 25.6% of the variance in CUDIT-R scores ($R^2 = .26$, $F(1,221) = 75.9$, $p < .001$). The third step of the mediation model revealed that inattention symptom of ADHD predicted CUDIT-R significantly ($\beta = .61$, $p < .001$). It accounted for 37.4% of the variance ($R^2 = .37$, $F(1,221) = 131.95$, $p < .001$). In the fourth step, ASRS-IA was still a significant predictor of CUDIT-R scores after controlling bipolar disorder as the mediator ($\beta = .48$, $t(223) = 7.48$, $p < .001$). The indirect effect size between ASRS-IA and CUDIT-R scores calculated by PROCESS macro was .80 with a 95% confidence interval, which did not include zero. Sobel test results showed that bipolar disorder as a mood disorder is a partial mediator between ASRS-IA and CUDIT-R scores ($z = 3.27$, $p = .001$) (Table 5.5).

D) CAN

In Step 1 of the mediation model, ASRS-IA predicted the mediator, bipolar disorder, in a significant way the same as previous substance misuse scores in this section. Step 2 indicated that bipolar disorder as the mediator predicted CAN significantly ($R^2 = .19$, $F(1,221) = 52.54$, $p < .001$) and accounted for 19.2% of the variance in CAN scores ($\beta = .44$, $p < .001$). The results of the third step demonstrated that ASRS-IA is a significant predictor of CAN ($R^2 = .22$, $F(1,221) = 62.36$, $p < .001$) and it accounted for 22% of the variance ($\beta = .47$, $p < .001$). The last step of the multiple regression showed that after controlling the mediator, inattention symptom of ADHD is still a significant predictor of CAN scores ($\beta = .32$, $t(223) = 4.51$, $p < .001$). The indirect effect size between ASRS-IA and AUQ scores calculated by PROCESS macro was 1.13 with a 95% confidence interval, which did not include zero. The results of the Sobel test showed that bipolar disorder as a mood disorder is a partial mediator between these two variables ($z = 3.15$, $p = .001$) (Table 5.5).

E) NIC

The results of the first step of the mediation model were the same as the previous substances. In the second step, bipolar disorder predicted nicotine use significantly ($\beta = .36$, $p < .001$) and accounted for 12.7% of the variance in NIC scores ($R^2 = .13$, $F(1,221) = 32.02$, $p < .001$). The third step of the mediation analysis indicated that ASRS-IA can predict nicotine use significantly ($\beta = .34$, $p < .001$). It accounted for 11.3% of the variance in nicotine use ($R^2 = .11$, $F(1,221) = 28.09$, $p < .001$). The result of the fourth step revealed that after controlling the mediator, bipolar disorder, the inattention symptom of ADHD is still a

significant predictor ($\beta = .19, t(223) = 2.52, p < .05$). The indirect effect size between ASRS-IA and AUQ scores was 35.59 with a 95% confidence interval, which did not include zero. The Sobel test results showed that bipolar disorder is a partial mediator between ASRS-IA and nicotine use ($z = 3.03, p = .002$) (Table 5.5).

Table 5.5
Four steps mediation analysis of bipolar disorder between ASRS-IA and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|-----------|--------------------|-------|--------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-IA | BD | .34** | 116.12 | .58** | 4.41** | .98* | .54 | 1.43 |
| 2 | BD | AUDIT | .33** | 111.22 | .57** | | | | |
| 3 | ASRS -IA | AUDIT | .39** | 142.49 | .62** | | | | |
| 4 | ASRS -IA | AUDIT | .46** | 93.71 | .43** | | | | |
| | BD | | | | .32** | | | | |
| 1 | ASRS -IA | BD | .33** | 116.12 | .58** | 3.07* | 1.10* | .37 | 1.84 |
| 2 | BD | AUQ | .21** | 58.09 | .45** | | | | |
| 3 | ASRS -IA | AUQ | .27** | 81.77 | .52** | | | | |
| 4 | ASRS -IA | AUQ | .30** | 48.26 | .38** | | | | |
| | BD | | | | .23** | | | | |
| 1 | ASRS -IA | BD | .34** | 116.12 | .58** | 3.27** | .80* | .28 | 1.31 |
| 2 | BD | CUDIT-R | .26** | 75.88 | .50** | | | | |
| 3 | ASRS -IA | CUDIT-R | .37** | 131.95 | .61** | | | | |
| 4 | ASRS -IA | CUDIT-R | .41** | 75.40 | .48** | | | | |
| | BD | | | | .22** | | | | |
| 1 | ASRS -IA | BD | .33** | 116.12 | .58** | 3.15* | 1.13* | .48 | 1.80 |
| 2 | BD | CAN | .19** | 52.54 | .43** | | | | |
| 3 | ASRS -IA | CAN | .22** | 62.36 | .46** | | | | |
| 4 | ASRS -IA | CAN | .26** | 38.76 | .32** | | | | |
| | BD | | | | .24** | | | | |
| 1 | ASRS -IA | BD | .34** | 116.12 | .58** | 3.03* | 35.59* | 11.52 | 59.84 |
| 2 | BD | NIC | .13** | 32.02 | .35** | | | | |
| 3 | ASRS -IA | NIC | .11** | 28.09 | .33** | | | | |
| 4 | ASRS -IA | NIC | .15** | 19.59 | .19** | | | | |
| | BD | | | | .24** | | | | |

Note: BD= Bipolar Disorder; ASRS = ADHD Self-Report Scale; Mood = mood disorder total score; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

5.7.5. Does bipolar disorder as a mood disorder mediate the relationship between ASRS-HI and substance misuse?

A) AUDIT

The first step of the mediation analysis showed that hyperactivity/impulsivity symptom of ADHD is a significant predictor of bipolar disorder ($R^2 = .34$, $F(1,221) = 117.15$, $p < .001$) and accounted for 34.6% of the variance in mood disorder scores ($\beta = .59$, $p < .001$). The second step indicated that the mediator can predict AUDIT scores significantly ($\beta = .58$, $p < .001$). It accounted for 33.5% of the variance ($R^2 = .33$, $F(1,221) = 111.22$, $p < .001$). The third step revealed that ASRS-HI predicts AUDIT in a significant way ($R^2 = .44$, $F(1,221) = 174.76$, $p < .001$) and accounted for 44.2% of the variance ($\beta = .66$, $p < .001$). In the fourth step of the mediation model, both the mediator and ASRS-HI as the independent variable were statistically significant predictors of AUDIT scores (β (ASRS-IA) = .5, $t(223) = 8.37$, $p < .001$). The indirect effect of ASRS-IA on AUDIT scores via bipolar disorder was .88, with a 95% confidence interval, which did not include zero. This means that the effect was significantly greater than zero at $\alpha = .05$, and the Sobel test results indicated that bipolar disorder as a mood disorder is a partial mediator between hyperactivity/impulsivity symptom of ADHD and AUDIT score ($z = 4.69$, $p < .001$) (Table 5.6).

B) AUQ

The first step of the mediation model for the relation between ASRS-HI, bipolar disorder and AUQ scores was the same as the AUDIT results. The second step of it showed that bipolar disorder is a significant predictor of AUQ ($R^2 = .21$, $F(1,221) = 58.09$, $p < .001$) and accounted for 20.8% of the variance ($\beta = .46$, $p < .001$). In the third step, ASRS-HI predicted AUQ significantly ($R^2 = .31$, $F(1,221) = 99.72$, $p < .001$) and accounted for 31.1% of the variance ($\beta = .56$, $p < .001$). The last step of the mediation analysis revealed that after controlling for bipolar disorder as the mediator, ASRS-HI was still a statistically significant predictor of AUQ score ($\beta = .44$, $t(223) = 6.51$, $p < .001$). The indirect effect size between ASRS-HI and AUQ was .93. The Sobel test was conducted and the results showed that bipolar disorder as a mood disorder is a partial mediator between ASRS-HI and AUQ score ($z = 2.71$, $p = .006$) (Table 5.6).

C) CUDIT-R

The results of the first step of the mediation model were the same as previous sections and ASRS-HI was a significant predictor of bipolar disorder as the mediator. In the second step, bipolar disorder predicted CUDIT-R scores significantly ($R^2 = .26$, $F(1,221) = 75.9$, $p < .001$) and accounted for 25.6% of the variance ($\beta = .51$, $p < .001$). In the third step, hyperactivity/impulsivity symptom of ADHD predicted CUDIT-R in a statistically significant way ($R^2 = .44$, $F(1,221) = 176.53$, $p < .001$) and accounted for 44.4% of the variance in CUDIT-R scores ($\beta = .67$, $p < .001$). The last step indicated that in the presence of a mediator, both ASRS-HI as the independent variable and bipolar disorder as the mediator were significant predictors of CUDIT-R scores (β (ASRS-HI) = .56, $t(223) = 9.24$, $p < .001$, β

(bipolar disorder) = .17, $t(223) = 2.84, p < .05$) (Table 5.14). The indirect effect size between ASRS-HI and CUDIT-R calculated by Hayes Process Macro was .63, and a Sobel test was conducted and found partial mediation in the model ($z = 2.89, p = .003$) (Table 5.6).

D) CAN

In the first step of the mediation analysis, ASRS-HI predicted bipolar disorder as a mood disorder significantly like previous sections. Step 2 of the regression model indicated that bipolar disorder can predict CAN significantly ($\beta = .44, p < .001$). It accounted for 19.2% of the variance in CAN ($R^2 = .19, F(1,221) = 52.54, p < .001$). The third step showed that ASRS-HI is a significant predictor of CAN ($R^2 = .22, F(1,221) = 63.8, p < .001$) and the fourth step revealed that the scores of ASRS-HI and bipolar disorder predicted CAN significantly ($\beta = .33, t(223) = 4.60, p < .001$). The indirect effect of ASRS-HI on CAN score via bipolar disorder did not include zero and was 1.11. (Table 5.6)

E) NIC

It has been mentioned in the previous sections that hyperactivity/impulsivity symptom of ADHD is a significant predictor of bipolar disorder. The second step of the mediation analysis indicated that bipolar disorder predicted nicotine use significantly ($\beta = .35, p < .001$). It accounted for 13% of the variance in NIC scores ($R^2 = .13, F(1,221) = 32.02, p < .001$). The third step showed that ASRS-HI is a statistically significant predictor of NIC and accounted for 13.3% of the variance in nicotine use ($R^2 = .13, F(1,221) = 33.8, p < .001$) ($\beta =$

.36, $p < .001$) (Table 5.21). In the last step it has been shown that none of bipolar disorder as the mediator and ASRS-HI as the independent variable predicted nicotine use significantly (β (ASRS-HI) = .24, $t(223) = 3.10$, $p > .05$, β (bipolar disorder) = .22, $t(223) = 2.84$, $p > .05$) (Table 5.22). The indirect effect size between ASRS-HI and NIC scores was 31.95 in PROCESS macro with a 95% confidence interval and the Sobel test results demonstrated that bipolar disorder is not a partial mediation in the model ($z = 2.74$, $p = .06$) (Table 5.6).

Table 5.6
Four steps mediation analysis of bipolar disorder between ASRS-HI and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|-----------|--------------------|-------|--------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-HI | BD | .35** | 117.15 | .58** | 4.69** | .88* | .52 | 1.29 |
| 2 | BD | AUDIT | .33** | 111.22 | .57** | | | | |
| 3 | ASRS -HI | AUDIT | .44** | 174.76 | .66** | | | | |
| 4 | ASRS -HI | AUDIT | .49** | 107.98 | .49** | | | | |
| | | BD | | | .28** | | | | |
| 1 | ASRS -HI | BD | .35** | 117.15 | .58** | 2.71* | .93* | .25 | 1.67 |
| 2 | BD | AUQ | .21** | 58.09 | .45** | | | | |
| 3 | ASRS -HI | AUQ | .31** | 99.72 | .55** | | | | |
| 4 | ASRS -HI | AUQ | .34** | 55.66 | .44** | | | | |
| | | BD | | | .19** | | | | |
| 1 | ASRS -HI | BD | .35** | 117.15 | .58** | 2.89* | .63* | .20 | 1.10 |
| 2 | BD | CUDIT-R | .26** | 75.88 | .50** | | | | |
| 3 | ASRS -HI | CUDIT-R | .44** | 176.53 | .66** | | | | |
| 4 | ASRS -HI | CUDIT-R | .46** | 95.12 | .56** | | | | |
| | | BD | | | .17** | | | | |
| 1 | ASRS -HI | BD | .35** | 117.15 | .58** | 3.15* | 1.11* | .58 | 1.78 |
| 2 | BD | CAN | .19** | 52.54 | .43** | | | | |
| 3 | ASRS -HI | CAN | .22** | 63.78 | .47** | | | | |
| 4 | ASRS -HI | CAN | .26** | 39.25 | .32** | | | | |
| | | BD | | | .24** | | | | |
| 1 | ASRS -HI | BD | .35** | 117.15 | .58** | 2.74 | 31.95 | 9.99 | 55.28 |
| 2 | BD | NIC | .13** | 32.02 | .35** | | | | |
| 3 | ASRS -HI | NIC | .13** | 33.78 | .36** | | | | |
| 4 | ASRS -HI | NIC | .16** | 21.45 | .23** | | | | |
| | | BD | | | .21** | | | | |

Note: BD= Bipolar Disorder; ASRS-HI = Hyperactivity/Impulsivity symptom of ADHD; Mood = mood disorder total score; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

5.8. Discussion

This study set out to investigate whether the symptoms of bipolar disorder, as a mood disorder, would mediate the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine in a sample of typically developing university students. To our knowledge, this is the first research to divide ADHD scores into two categories: inattention and hyperactivity/impulsivity, to explore the role of bipolar disorder between each of them and all substance misuse scores. Bipolar disorder was not a full mediator between the two ADHD symptom clusters and any substance misuse scores in this analysis.

According to the results of the Sobel and PROCESS macro tests, bipolar disorder was a partial mediator (a) between inattention and alcohol, cannabis and nicotine use and (b) between hyperactivity/impulsivity and alcohol and cannabis use. This outcome suggests that bipolar disorder accounted for some of the relationships between ADHD scores and the use of alcohol, cannabis and nicotine. Moreover, it implies that in addition to a significant association between ADHD-bipolar disorder and bipolar disorder-substance misuse separately, there could be a direct relationship between ADHD symptoms and substance misuse. There could also be some other variables that can mediate the relationship between different ADHD symptoms and the use of alcohol, cannabis and nicotine. Impaired emotional regulation, which was presented in Chapter 4, was one factor that mediated the association between the hyperactivity/impulsivity symptom of ADHD and the use of alcohol and cannabis. Sleep quality is another variable and will be discussed in Chapter 6.

A four-step regression analysis was conducted between the variables to test this study's hypothesis. The first step showed that both ADHD scores predicted bipolar disorder significantly. While previous studies have revealed a connection between ADHD and mood disorders (Biederman et al., 2002; Biederman et al., 1993; Kooij et al., 2001; Kooij et al., 2004), this study contributes to the field by demonstrating that inattention and hyperactivity/impulsivity predict bipolar disorder separately. Reviewing the associations between the variables in this analysis shows that each ADHD symptom cluster and bipolar disorder have equal correlations (Table 5.4). Furthermore, both inattention and hyperactivity/impulsivity had the same coefficient of determination (R^2), which presents how much of the variance in bipolar disorder is explained by ADHD symptom clusters in the regression model (Tables 5.5. and 5.6). The current study indicated that the higher an individual's sub-score in the ADHD questionnaire, the more likely they are to develop mood disorders, specifically bipolar disorder. According to the MDQ responses, 15.5% of participants met the questionnaire's criteria for a positive screen for bipolar disorder, which is higher than the rate of bipolar disorder in England's general population (3%) (Mental Health Statistics for England, 2018). This result might be due to the higher incidence of mood disorders among university students, as discussed in the introduction section (please refer to Section 5.4).

Epidemiological studies indicate that 20% of people with ADHD have bipolar disorder symptoms; this figure may reach 50% in those with ADHD who also have considerable bipolar symptoms (Halmoy et al., 2010; Kessler et al., 2006; McGough et al., 2005). McIntyre et al. (2010) found that in 399 adults with mood disorders, 5.4% of those with depression and 17.6% of those with bipolar disorder had been diagnosed with ADHD, lower

quality of life and an onset of mood disorder at an earlier age than those without ADHD (Nierenberg et al., 2005; Perugi et al., 2013; Wingo & Ghaemi, 2007). In another study of 1,000 adults with bipolar disorder, Nierenberg et al. (2005) found that 14.7% of males and 5.8% of females with lifelong ADHD had earlier age-onset of mood disorder, more frequent depressive episodes and a worse course of bipolar disorder.

Comorbid ADHD and bipolar disorder lead to early onset of bipolar disorder, more depressive and mixed episodes, fewer episodes without symptoms, poorer response to treatment and worse outcome (Karaahmet et al., 2013; Nierenberg et al., 2005; Tamam et al., 2008). Furthermore, individuals with both conditions have more psychiatric comorbidities, such as substance misuse (Tamam et al., 2008). Several hypotheses attempt to explain the high rate of association between these two conditions, including impulsivity as an overlapping dimension or shared genetic backgrounds (Youngstrom et al., 2010). Moreover, studies have identified shared brain areas and significant interaction effects of bipolar disorder and ADHD in the anterior and posterior cingulate, left superior and middle frontal gyri and left inferior parietal lobule (Biederman et al., 2008; Townsend et al., 2013). In those with ADHD and bipolar disorder, there are shared impairments, such as response inhibition. A part of the brain that plays a crucial role in response inhibition is the inferior frontal cortical (IFC)-striatal network, a shared neural dysfunction in both groups (Epstein et al., 2007; Townsend et al., 2013).

On the one hand, previous investigations mainly concentrated on the relationship between mood disorders and ADHD as a diagnosed condition in university students or other adult populations. On the other hand, this study aimed to show that the hyperactivity/impulsivity and inattention symptoms of ADHD could significantly predict

bipolar disorder in typically developing university students. Non-clinical investigations that explored bipolar disorder in university students are limited; further studies are needed to assess the symptoms of ADHD, bipolar disorder and substance misuse in typically developing individuals.

In this study's second phase of the mediation analysis, bipolar disorder was found to be a mediator that predicted all substance misuse scores significantly. Mood disorders and substance misuse are co-occurring and highly prevalent disorders in the general population (Farren et al., 2012; Frye & Salloum, 2006; Grant et al., 2004; Hasin et al., 2005; Hasin et al., 2007; Merikangas et al., 2007; Pettinati et al., 2013). The course of both depression and bipolar disorder could be worsened by comorbid substance misuse (Agosti & Levin, 2006; Murray & Lopez, 1997; Tohen et al., 1998). According to the results of previous studies on the individual relationships between bipolar disorder and the misuse of alcohol, cannabis and nicotine, 20% to 67% of people seeking treatment for AUD had depression, while 6% to 8% of them had bipolar disorder (Brady et al., 1998). Furthermore, cannabis is the most commonly misused illegal substance among those with bipolar disorder, with research showing that 38% of them do so (Cerullo & Strakowski, 2007; Etain et al., 2012). Young people with bipolar disorder also misuse cannabis significantly (Dell'Osso et al., 2011, 2013). The severity of the bipolar disorder symptoms and noncompliance to treatment is higher in those with bipolar disorder and comorbid chronic cannabis misuse (van Rossum et al., 2009). In addition, as a nicotinic cholinergic receptor agonist, nicotine releases dopamine and norepinephrine, both of which play a critical role in addiction (Geraciotti et al., 1999; Thomson et al., 2015). These two neurotransmitters are also involved in the pathophysiology of bipolar disorder (Heffner et al., 2011). Thus, individuals with bipolar disorder could be

more vulnerable to the rewarding effects of nicotine (Stockings et al., 2013; Thomson et al., 2015).

In the third step of the mediation analysis, both scores of the ADHD questionnaire predicted substance misuse significantly. Investigators argued that people who misuse substances often suffer from other concurrent psychiatric conditions, particularly internalising disorders, such as mood and anxiety disorders (Chan et al., 2008; Chen et al., 2011; Karam et al., 2015). In recent years, there has been a growing interest in substance misuse and comorbid externalising disorders, such as ADHD, a childhood neuropsychological condition with symptoms persisting into adulthood (Faraone et al., 2006; Groenman et al., 2013). It is one of the most common disorders among individuals who misuse substances and those who seek treatment for SUD (Charach et al., 2011; Groenman et al., 2013; Kessler et al., 2006; Lee et al., 2011; van de Glind et al., 2014; van Emmerik-van Oortmerssen et al., 2014). A meta-analysis on 29 studies by Emmerik-van Oortmerssen et al. (2012) showed that 25% of adolescents and 21% of adults with ADHD symptoms had comorbid substance misuse. Substance misuse with comorbid ADHD has a complex clinical presentation since other psychiatric comorbidities are high; those with ADHD symptoms have more severe substance misuse and poorer SUD treatment outcomes (Carpentier et al., 2011; Kaye et al., 2013; Rounsaville et al., 1991; van Emmerik-van Oortmerssen et al., 2014; Wilens et al., 2005). The current research could demonstrate that each of the ADHD symptoms is a significant predictor of alcohol, cannabis and nicotine use. This case may be due to genetic vulnerabilities or shared brain regions involved in impulsive behaviours or impaired EF in those with ADHD and substance misuse, as discussed in Chapters 2 and 3 (please refer to Sections 2.8 and 3.8).

Bipolar disorder was a partial mediator between the inattention symptom of ADHD and substance misuse scores in the final segment of the mediation model. In previous investigations, patients with mood disorders showed a considerable reduction in attention, EF and memory (Camelo et al., 2013). Studies that focus on cognitive performance deficits should consider the role of impaired attention and working memory in individuals with mood disorders because these two cognitive functions are vital in many neuropsychological tests. For instance, Camelo et al. (2013) found that one of the most compromised functions of people with bipolar disorder is attention. Attention impairments can cause changes in memory, visuospatial abilities and learning (Camelo et al., 2013). In addition, inattention is one of the main symptoms of ADHD (DSM-5, 2013). The present study results indicated that inattention as a symptom of ADHD increases the risk of alcohol, cannabis, and nicotine use through a bipolar disorder pathway. It can be concluded that besides a direct relationship between ADHD and substance misuse, mood disorder (i.e. bipolar disorder in this study) can increase the likelihood of alcohol, cannabis and nicotine use in university students with higher levels of ADHD inattention symptoms. Many studies revealed that people with more inattention symptoms use more nicotine, which may affect the inattention symptom through its positive effect on arousal and attention (Bilgi et al., 2017; Kalil et al., 2008; Levin & Rezvani, 2002; Warburton & Arnall, 1994). According to past research, inattention rather than hyperactivity/impulsivity is an essential factor in alcohol-related problems in university students (Glass & Flory, 2012; Mesman, 2013). Thus, studies revealed that inattention is the most compromised feature in individuals with bipolar disorder and ADHD. This study's findings may be explained by the shared parts of the brain involved in attention (DLPFC) in individuals with the symptoms of ADHD, bipolar disorder and substance misuse (Arnsten 2010; Rajkowska et al., 2001). Additionally, investigators suggested a shared genetic link

between bipolar disorder and ADHD, especially the inattention symptom of ADHD (Joo et al., 2010)

The current study also discovered that bipolar disorder mediated the relationship between the hyperactivity/impulsivity symptom of ADHD and the use of alcohol and cannabis use. Loflin et al. (2014) studied 2,811 current cannabis users and found that cannabis could lessen the hyperactivity/impulsivity symptom of ADHD, but not the inattention symptom. These results indicated that people with higher hyperactivity/impulsivity symptoms of ADHD are more likely to self-medicate with cannabis than those with inattention symptoms, which may explain this study's findings that higher hyperactivity/impulsivity symptoms can predict a higher CUDIT-R score via a bipolar disorder pathway. In this study, bipolar disorder did not mediate the relationship between the hyperactivity/impulsivity symptom of ADHD and nicotine use. This finding may be explained by the fact that nicotine smoking in those with the hyperactivity/impulsivity symptom of ADHD may be mediated by variables other than bipolar disorder, as presented in Chapter 6. There are similarities and overlaps between the symptoms of ADHD and bipolar disorder, which may be attributed to genetic factors, shared brain regions or the self-medication hypothesis, as discussed earlier in Chapter 1 (Section 1.4.4).

This study contributes to a better understanding of ADHD symptoms and their relationship with the use of alcohol, cannabis and nicotine in the context of mood disorders. There may be a variety of explanations for the mediation relationship between the variables in this analysis. For instance, those with more ADHD symptoms and mood disorders may act more impulsively without regard to the adverse effects of substance misuse (De Wit, 2009; Nigg, 2001; Perry & Carroll, 2008). The other reason may be the connection between

response inhibition and the hyperactivity/impulsivity symptom of ADHD, which may increase the risk of substance misuse in those with more ADHD and bipolar disorder symptoms (Lee et al., 2020). Additionally, as previously mentioned, people with ADHD use different substances to cope with their symptoms and daily life problems (Silva et al., 2014). Substance misuse has been shown to decrease the negative affect and craving, suggesting that the usage is motivated by coping mechanisms (Buckner et al., 2015). Self-medication is one of the proposed causes. Still, neurobiologists and genetic investigators have suggested other explanations for the significant association between ADHD, bipolar disorder and the use of alcohol, cannabis and nicotine.

The first familial and identical twin studies' results showed a strong genetic basis for bipolar disorder (Craddock & Sklar, 2013; McMahon et al., 2010). In general, identical twin concordance rates range from 40% to 70%; the heritability of bipolar disorder has been reported to be 90% in most recent studies (Craddock & Sklar, 2013). Hence, in part, this suggests that specific inheritance mechanisms are involved in propagating the condition (Craddock & Sklar, 2013; McMahon et al., 2010). Despite differences in genetic risks between bipolar disorder and other psychiatric disease states, the most notable finding is the high degree of genetic overlap between conditions (Joo et al., 2010; Maletic & Raison, 2014).

In addition, studies in the pathobiology of bipolar disorder proposed the critical role of two interrelated prefrontal limbic networks (Maletic & Raison, 2014, Strakowski et al., 2012). The first is the automatic and internal emotional regulatory network, which is a loop that includes the ventromedial PFC, subgenual ACC, nucleus accumbens, globus pallidus and thalamus. This system controls the amygdala's responses to endogenously generated feeling

states (Maletic & Raison, 2014). The second network includes the ventrolateral PFC, mid- and dorsal-cingulate cortex, ventromedial striatum, globus pallidus and thalamus; it is referred to as the volitional/external regulatory network (Strakowski et al., 2012). The origination point of the volitional/cognitive regulatory arc is the DLPFC, connected with the ventrolateral PFC (Strakowski et al., 2012). The ventrolateral PFC controls external-induced emotions, helps cognitive-emotional regulation and inhibits maladaptive affect (Strakowski et al., 2012). These two networks regulate amygdala responses in complex emotional situations and share components (Strakowski et al., 2012). Compared to healthy controls, elements of these complex systems are impaired in people with bipolar disorder (Maletic & Raison, 2014).

The activity of ventrolateral PFC, which is involved in top-down and volitional regulation of affect and inhibition of maladaptive emotional responses, is reported to be both reduced and elevated in bipolar disorder's depressive state and decreased in its manic state (Altshuler et al., 2005; Strakowski et al., 2012; Townsend & Altshuler, 2012). Impaired activity of DLPFC in bipolar disorder may be linked to working memory deficits, attention problems and poor EF (Gruber et al., 2004; Phillips et al., 2008). Combining the DLPFC, dorsal ACC and parts of the parietal cortex formed the executive/cognitive network, regulating limbic formations (Drevets, 2000). There is an association between the duration of bipolar disorder and decreased thickness of DLPFC (Lyo et al., 2006). Furthermore, based on the neural networks involved in ADHD and substance misuse (Chapter 1, Section 1.3), it can be concluded that impairments in the same parts of the brain in people with ADHD symptoms, substance misuse and bipolar disorder may be the reason for using increasing amounts of alcohol, cannabis and nicotine. According to the mediating relationship between

these three conditions, effective interventions can be developed to decrease the risk of substance misuse in an individual based on their ADHD presentation and mood disorders.

In this study, the percentage of risky alcohol and cannabis use, daily nicotine use and those who are likely to have bipolar disorder or ADHD were measured (please refer to Section 5.7.2). The results found that 32.9% of the participants gained an AUDIT score of 8 or higher, indicating hazardous drinking, while 20% exhibited risky cannabis use. Moreover, 13.3% of the participants smoked nicotine on all 30 days of the last month. The prevalence of substance misuse in the current study's sample was higher than in the Studies 1 and 2, which could be due to the recruitment process occurring immediately after the Christmas and New Year holidays, thereby affecting the participants' responses to the questionnaires. Based on previous investigations, substance misuse is higher during the holidays (Kim et al., 2017; Song et al., 2020). The incidence of bipolar disorder in the sample was 15.5%, higher than the general population's prevalence, which may be due to university students exhibiting more mood disorders than their non-university counterparts (Demery et al., 2012). According to the NHS report (2019), although the exact cause of bipolar disorder is unknown, it could be triggered by extreme stress, overwhelming problems and life-changing events. These elements can be exacerbated by starting university in undergraduate students who were the focus of this study. Genetic and neurological factors discussed earlier in this section may also be reasons behind bipolar disorder.

5.9. Limitations

It is crucial to consider the limitations of this study. The questionnaires, like those in Chapter 4, were completed online and were rather lengthy. Fatigue or boredom may have had an impact on the completion of these questionnaires. As for all self-report studies, the potential for confounds arises from social desirability bias (possibility of providing invalid answers), response bias (individual's tendency to respond in a particular way regardless of the question), and item clarity (highly structured questionnaires that force individuals to choose an answer that may not match their views) (Demetriou et al., 2015).

Since this study takes a dimensional approach when examining ADHD symptoms, substance use and mood disorder, it is impossible to extrapolate these findings to individuals with clinically diagnosed conditions; future research should focus on clinical studies. Additionally, participants were recruited from first-year undergraduate students at a single university, limiting the generalisability of the results to a bigger population (Creswell, 2014). Recruiting participants of various age groups, ethnicities, educational attainments and income levels could help explore each variable's role in a more diverse sample. In the UK, studies with typically developing university students with symptoms of BD, ADHD and substance misuse are scarce. Furthermore, future research will require measuring these variables in several universities around the UK.

Another limitation of this investigation was using a cross-sectional study since a longitudinal study was not feasible during a PhD programme. In this case, the causal relationship between the variables is unclear since only the correlation, association and predictive capacity of each variable is shown; longitudinal studies are recommended in the future. In addition, this study investigated the role of BD, as a mood disorder, in the

relationship between ADHD symptoms and substance misuse, restricting the results. It is suggested that future research should measure different mood disorders.

Finally, testing participants immediately after Christmas and New Year may limit the findings since the rate of misusing substances may vary during holidays and specific occasions in a year (Kim et al., 2017; Song et al., 2020). According to the DSM-5, *major depressive disorder with seasonal pattern* can affect an individual's mood during the fall and winter seasons when there is less sunlight. Depression with seasonal pattern criteria is described as depression that begins and ends during a specific season every year for at least two years, with more seasons of depression than seasons without depression over a lifetime. This condition could co-occur with other depressive, bipolar, attention deficit, alcoholism and eating disorders, making diagnosis difficult (DSM-5, 2013; Lurie et al., 2006). Having major depressive disorder with a seasonal pattern may affect the rates of substance misuse, particularly in the fall and winter. Thus, future investigations in the fall and winter should measure this form of depression and its relationship with ADHD and substance misuse.

5.10. Conclusion

This study has shown that bipolar disorder is a partial mediator (a) between inattention and alcohol, cannabis and nicotine use; and (b) between hyperactivity/impulsivity and alcohol and cannabis use. This research is also the first to explore possible pathways linking ADHD symptom clusters and substance misuse. Moreover, future treatment and intervention programmes should consider (a) the relationship between inattention, bipolar disorder and nicotine use; and (b) the association between inattention and hyperactivity/impulsivity, bipolar disorder and the use of alcohol and cannabis.

Another common condition between adults with ADHD and substance misuse is poor sleep quality, which has been linked to substance misuse in adults with ADHD symptoms. The following chapter examines the role of sleep quality in the relationship between ADHD and substance misuse to determine whether or not it can mediate the relationship between each of the ADHD symptoms and every substance misuse score.

Chapter 6

6. Does sleep quality mediate the relationship between ADHD symptoms and substance misuse?

Overview

This chapter describes a cross-sectional study exploring the role of sleep quality in the relationship between ADHD symptom clusters and substance misuse. A sample of first-year undergraduate students was recruited to complete measures on sleep quality, ADHD symptoms, and the use of alcohol, cannabis and nicotine. This is the first study to divide the two main ADHD symptom clusters (inattention and hyperactivity/impulsivity) to examine each symptom's relationship with sleep quality and substance misuse. A four-step regression model was employed to investigate whether sleep quality mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use. Sleep quality mediated the relationship between both ADHD symptom clusters and alcohol, cannabis and nicotine use. This research was conducted in completion of Studies 3 and 4. The results of the two previous studies of this thesis showed that emotional regulation was a partial mediator between inattention and alcohol and cannabis use. Additionally, bipolar disorder, as a mood disorder, was a partial mediator (a) between inattention and alcohol, cannabis and nicotine use and (b) between hyperactivity/impulsivity and alcohol and cannabis use. Therefore, these partial mediation relationships connote that there might be other variables that mediate the relationship between ADHD symptom clusters and substance misuse. This study found that poor sleep quality is another significant partial mediator between ADHD symptoms and alcohol, cannabis and nicotine use.

6.1. Introduction

In Chapters 4 and 5, emotional regulation and bipolar disorder mediated the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use. In this final empirical chapter, however, this study attempted to explore the role of another variable common to individuals with ADHD and those with substance misuse: sleep quality.

Sleep problems can considerably affect life quality (Garbarino et al., 2016). They can be risk factors in developing other health issues, such as diabetes, cancer, obesity and cardiovascular disease (Depner et al., 2014; Knutson, 2010; Pinheiro et al., 2006). Sleep problems also have significant comorbidity with mental disorders such as ADHD (Walters et al., 2008), which is characterised by inattention, hyperactivity and impulsivity and can have serious impacts on an individual's life (DSM-5, 2013, Karlsdotter et al., 2016). Even though the relationship between these two conditions is still unclear, some studies have shown associations between them. Sleep problems can heighten the risk of ADHD, while ADHD symptoms may increase sleep disturbances (Hvolby, 2015; Yoon et al., 2012).

Previous investigations also found an association between sleep disturbances, such as insomnia, and the misuse of alcohol, nicotine and other illegal drugs. In an epidemiological study involving 3,000 participants, Wetter and Young (1994) discovered that symptoms of insomnia, such as difficulty falling asleep and non-restorative sleep, and hypersomnia or parasomnia, such as excessive daytime sleepiness and nightmares, are more common in current smokers. It is also worth noting the connection between ADHD symptomology and smoking, which may be a confounding variable in Wetter and Young's study (Lambert & Hartsough, 1998). Moreover, significant associations between alcohol use and insomnia have been detected by many researchers (Roehrs et al., 1991; Roehrs et al., 1999; Williams et al., 1983). Several studies show that psychoactive substances, such as nicotine, cannabis and

alcohol, can disturb sleep quality in a dosed manner (Haney et al., 1999; Johanson et al., 1999; Roehrs et al., 1999; Salin-Pascual et al., 1999). Therefore, the next two sections are dedicated to the relationship between ADHD, substance misuse and sleep quality in greater depth.

As mentioned in Chapter 1's Section 1.3, people with ADHD use higher amounts of a substance compared to the general population. The present study may help future investigations have a better understanding of the relationship between inattention-sleep quality, hyperactivity/impulsivity-sleep quality and substance misuse-sleep quality. Given that sleep disturbances may persist for a long time (even after SUD treatment), choosing the best treatment strategies can help those with ADHD have a better sleep architecture and control their substance misuse. Additionally, more advanced pharmacologic agents may aid in developing new strategies for a more successful substance misuse treatment programme and the prevention of relapse in individuals with ADHD symptoms and substance misuse.

6.2. ADHD and sleep quality

Even though the diagnosis of ADHD relies on the observation of an awake individual, children with ADHD symptoms have a prevalence of sleep problems ranging from 25% to 55% (Corkum et al., 1998; Hodgkins et al., 2013; Hvolby, 2015; Owens, 2005; Sung et al., 2008; Yoon et al., 2012). In an Australian investigation, 62% of the children with ADHD had moderate to severe sleep disturbances, with 22% of them taking sleep medication during the week of observation (Efron et al., 2014). Although there are few studies on the association between sleep disturbances and adult ADHD, some investigations show that adults with ADHD have regular sleep onset difficulties (van Veen et al., 2010). Moreover, Yoon et al. (2012) reported that the prevalence of sleep problems is higher in adults with ADHD

symptoms (60%–80%) than children or adolescents with ADHD symptoms (25%–50%) (Yoon et al., 2012).

Nevertheless, the results of various investigations on poor sleep quality in adults with ADHD are mixed. Few studies reveal that, in addition to the main symptoms of ADHD that persist into adulthood, such as hyperactivity, impulsivity and inattention, sleep problems could be chronic during the lifespan of adults with ADHD (Michielsen et al., 2012; Simon et al., 2009). Investigators have also found significant associations between poor sleep quality and persistent ADHD symptoms in young adults (Gregory et al., 2017; Surman et al., 2006).

Although some studies discovered a connection between adult ADHD and sleep onset latency, restless legs syndrome, poor sleep efficiency and high nocturnal motor activity (Bogdan & Reeves, 2016; Fargason et al., 2013; Philipsen et al., 2005), other research could not find a significant difference in sleep latency, the number of sleep awakenings and total time in bed (as crucial objective sleep parameters) in this group of people (Kooij et al., 2001). These inconsistent results from various studies may be due to a variety of factors, including the use of different diagnostic methods for ADHD and the absence of a control group in some investigations (Fisher et al., 2014; Langberg et al., 2016).

Hence, there has been a growing interest in the association between ADHD and sleep quality over the past decade (Cortese et al., 2013). Furthermore, a multifaceted and complex association between sleep and ADHD has been detected (Cortese et al., 2006b; Owens, 2008). Sleep problems could be a fundamental feature of ADHD; they may exacerbate or be aggravated by ADHD symptoms (Hvolby, 2015). In other words, poor sleep quality can cause ADHD or ADHD-like symptoms, which can result in misdiagnosis. (Cortese et al., 2006b; Owens, 2008). This possibility is due to poor or disordered sleep symptoms being remarkably similar to behaviours and functional impairments of ADHD

(Beebe 2011; Corkum et al., 2011; Gruber, 2009; O'Brien, 2009). Therefore, based on the bidirectional relationship between ADHD and poor sleep quality and the limited investigations on the connection between ADHD and sleep in adults, further longitudinal studies are recommended to explore the cause-and-effect association between these variables.

Many theories have been suggested to explain the correlation between sleep quality and ADHD. The overlap between the function of different brain regions involved in attention, arousal and sleep quality may be reflected by the common nature of the relationship between ADHD and sleep (Owens, 2008; Owens et al., 2013; Yoon et al., 2012). Clinical investigations show that besides the association between stimulants and sleep disturbance in individuals with ADHD, stimulants have paradoxical effects, such as reducing ADHD symptoms, which calms the individual and endorses sleep (Jerome, 2001; Kinsbourne, 1973; Kooij et al., 2001; Kratochvil et al., 2005). Moreover, it is recommended to use an additional short-acting stimulant or a stimulant with a longer period of action (due to decreased drug concentration in blood and potential symptom rebound) to prevent sleep problems caused by increased hyperactivity or behavioural problems at bedtime (Cortese et al., 2013a, 2013b; Lecendreux et al., 2000). Thus, according to the European and US guidelines, sleep quality should be assessed during the evaluation of ADHD and before the initiation of medical treatment (Graham et al., 2011; Wolraich et al., 2011).

There are different pharmacological and non-pharmacological interventions to improve sleep quality or lessen sleep disturbances in individuals with ADHD symptoms. One example is having a healthy sleep practice that includes (a) calming and structured bedtime routines; (b) adequate opportunity for sleep; (c) a regular sleep/wake schedule; (d) a particular place specifically designed for sleep; (e) attention to environmental factors, such as bedroom furniture, lighting and temperature; and (f) avoidance of caffeine, large amounts of

liquids, naps, exercise and alerting activities right before bedtime (Cortese et al., 2013a; Owens, 2008; Yoon et al., 2012). These healthy sleep practices can be used in parallel with or without medication (Cortese et al., 2013a).

ADHD psychostimulant medication may complicate interrelationships, impairing sleep in some individuals with ADHD, yet improving sleep in others due to their calming effect (Graham et al., 2011, Jerome, 2001; Kinsbourne, 1973; Kooij et al., 2001; Kratochvil et al., 2005). For instance, the sympathomimetic action of stimulants promotes wakefulness in narcolepsy treatment-seekers (Morgenthaler et al., 2007). Therefore, primary sleep disorders should be diagnosed before initiating ADHD medication (Cortese et al., 2013a; Graham et al., 2011; Lecendreux & Cortese, 2007; Wolraich et al., 2011). Furthermore, pharmacological and behavioural treatments may address sleep impairments in those with ADHD (Cortese et al., 2013a). Thus, individuals with ADHD receiving pharmacotherapy should consider adding behavioural interventions into their multimodal ADHD management plan to enhance sleep quality (Graham et al., 2011; Lecendreux & Cortese, 2007; Wolraich et al., 2011).

Iron deficiency is associated with the aetiology of Restless Legs Syndrome (RLS) and ADHD, which can change dopamine expression and the synthesis and catabolism of monoaminergic neurotransmitters (Allen & Earley 2007; Cortese et al., 2005, 2012). A small study found that iron could substantially reduce the symptoms of ADHD in children (Konofal et al., 2008). Furthermore, individuals with ADHD symptoms exhibit a delayed pattern of melatonin secretion (Hvolby, 2015). Studies showed that melatonin treatment could reduce sleep onset delay in those with ADHD symptoms (Cortese et al., 2013a).

The relationship between ADHD and poor sleep quality, whether subjective or objective, does not show causation. According to Hvolby (2015), four hypothetical scenarios

demonstrate the potential relationship between ADHD or ADHD-like symptoms and sleep problems:

- Scenario 1 ADHD symptoms directly lead to sleep disturbances, which could be due to hyperactivity or nighttime behaviours. This situation is more common in individuals with hyperactivity than inattentive symptoms of ADHD. Stimulants may reduce sleep problems that come with ADHD. However, an insufficient efficacy period may result in symptom rebound during sleep.
- Scenario 2 Poor sleep quality and sleep disturbances may cause ADHD-like symptoms and behavioural and functional impairments. Before diagnosing ADHD, researchers recommend excluding primary sleep disorders (Cortese et al., 2006b, 2013a; Lecendreux & Cortese, 2007). In this scenario, stimulant medications could be ineffective or even worsen sleep problems. According to case studies, treating a sleep disorder could improve ADHD symptoms (Miano et al., 2013; O'Brien, 2009).
- Scenario 3 ADHD and sleep disturbances are coincident occurrences. They can also worsen each other's symptoms. Determining the best treatment strategy is extremely complicated in this case due to the opposing or mixed effects of medications on sleep. Common psychiatric comorbidities in individuals with ADHD symptoms may be associated with sleep disturbances. Furthermore, daytime sleepiness exacerbates internalising symptoms in people with ADHD; besides attentional functioning, poor quality of sleep can also have an adverse impact on emotional regulation (Hansen et al., 2013). Based on Hvolby et al. (2008), ADHD and sleep

disturbances can co-occur independently of psychiatric comorbidities. However, both internalising and externalising comorbid disorders may aggravate the symptoms of sleep disorders and ADHD.

Scenario 4 It is hypothesised that the mechanisms of common and overlapping neurobiological diseases can increase ADHD symptoms and sleep disorders. ADHD and certain sleep disorders, such as circadian-rhythm disorders, delayed sleep-phase disorder and sleep/wake disorders, may share common pathophysiological mechanisms.

Instead of a constant level of impairment, individuals with ADHD have shown intra-individual differences in neuropsychological tasks (Spencer et al., 2009; Tamm et al., 2012). The same picture of instability and unpredictability has been observed in the sleep patterns of individuals with ADHD (Gruber & Sadeh, 2004; Gruber et al., 2000; Lecendreux & Cortese, 2007; Tsai & Huang, 2010). Furthermore, genetic vulnerability to sleep dysregulation has been found in at least a subset of people with ADHD (Owens et al., 2013). Gregory et al. (2017) observed a substantial genetic overlap between ADHD and sleep quality, which was consistent with other studies using the same measures (Gregory et al., 2011; Taylor et al., 2015).

Yoon et al. (2013) studied 284 adults with ADHD symptoms (inattention and combined presentations of ADHD) to measure their subjective parameters of sleep. A large proportion of their adult participants with ADHD symptoms reported excessive daytime sleepiness, fatigue and poor sleep quality. Though they did not collect data on the age and gender of healthy controls, previous investigations have shown that adults with ADHD have worse sleep quality, fatigue level and sleepiness than age-matched healthy controls. Yoon et

al.'s (2013) findings also indicated that adults in the ADHD-inattention group had worse fatigue level and sleep quality than those in the combined group.

Other investigations have determined an association between sleep problems, such as periodic limb movement disorder (PLMD), and hyperactivity (Chervin & Archbold, 2001; Silvestri et al., 2009). Vogel et al. (2017) investigated the association between current overall ADHD and the severity of inattention and hyperactivity symptoms with the current presence or persistent history of adult sleep problems in participants of the Netherlands Sleep Registry. Their findings revealed that hyperactivity severity is significantly associated with current and persistent sleep problems. Inattention symptoms of ADHD may lead to a sense of 'forgetting time', resulting in late bedtimes and being an evening chronotype (Gau & Chiang, 2009).

Sleep disturbances might be connected to the severity of inattention or hyperactivity/impulsivity symptoms in adults with ADHD, a theory that has been rarely studied. Therefore, the relationships between two ADHD symptom clusters and poor sleep quality should be further investigated. Finding a pattern across the severity of the symptoms and their association with different types of sleep problems is critical. This pattern can be a crucial indicator of the type of sleep problem that can co-occur, cause or be a result of the severity of a particular ADHD symptom. Therefore, it could help in assessing or treating sleep problems in those with more severe ADHD symptoms.

6.3. Substance misuse and sleep quality

There is a growing interest in investigating substance misuse in university students due to the negative impact of substance misuse on a student's academic, social, health and psychological life (Fadhel, 2020). Among a wide range of other problems and substance misuse, the most common psychological issues are poor sleep quality and insomnia (Angarita

et al., 2016; Dunn et al., 2018; Khurshid, 2018; Lydon-Staley et al., 2017). The relationship between substance misuse and poor sleep quality is bilateral, and each problem may be a cause or a consequence of the other (Angarita et al., 2016; Valentino & Volkow, 2020), which may be due to the common neurobiology of sleep and substance misuse (Valentino & Volkow, 2020).

In a study based on the effects of alcohol use on the sleep quality in individuals aged 20 and above, a significant correlation between alcohol use scores and sleep quality of male subjects was detected (Park et al., 2015). Investigators found an association between alcohol use patterns and subjective sleep quality, sleep duration and sleep continuation. They discovered that those who consume more alcohol have poorer sleep quality, more difficulty in maintaining sleep and shorter overall sleep duration (Park et al., 2015; Pieters et al., 2010). Alcohol users disclosed more self-reported sleep problems (35% to 70%) compared to the general population (15% to 30%) (Brower et al., 2001).

In a study of 568 university students with one-, three- and five-month follow-ups, weekly alcoholic drinks, sleep quality and perceptions of sleep adequacy were measured to explore the effect of sleep problems on later alcohol consumption (Miller et al., 2016). The results indicated that more weekly drinks and inadequate sleep predicted alcohol outcomes at baseline and one month later. There were significant interactions between the quantity of weekly drinking and adequate sleep, which predicted alcohol-related outcomes at baseline and one-, three- and five-month assessments. The findings also revealed a positive association between weekly drinking quantity and alcohol-related outcomes in individuals who get adequate sleep and those who do not, with the link being greater in people with inadequate sleep.

According to Lydon et al. (2016), alcohol use has been linked to lower sleep quality, but not sleep duration, in laboratory studies. They investigated sleep quality and alcohol use in 150 adults, finding that sleep quality was poorer on nights following alcohol consumption. Their examination of individuals' daily lives revealed that alcohol could not enhance sleep quality and duration.

Although the aetiology of sleep quality is not clear, it has been revealed that it is a heritable trait with a significant genetic dependence (Barclay et al., 2010; Partinen et al., 1983). Another heritable sleep disorder is *insufficient sleep duration*, which is defined as sleeping less than 6 hours per night and is linked to neuropsychiatric disorders, obesity and cardio-metabolic problems (Grandner et al., 2010; Goel, 2017).

There is not much data on the genetic background of sleep duration in individuals with alcohol misuse, but Chakravorty et al. (2018) focused on the familial association of sleep duration in a group of participants with AUD. They attempted to determine whether a first-degree family history of AUD is a risk factor for sleep disturbances, poor sleep quality and complaints about disturbed sleep duration. Moreover, if there is such a link, they wanted to discern whether the connection is moderated by comorbid conditions, such as alcohol consumption and mood disorders with alcohol misuse and sleep disturbances. Their results revealed that participants with a history of alcohol misuse had lower scores on the mean sleep adequacy and the sleep duration scale compared to those without a family history of AUD.

A longitudinal study by Warren et al. (2017) explored whether the initiation of cigarette and alcohol use was linked to sleep duration and impaired sleep-wake patterns in late elementary school and whether sleep-related changes in inhibitory control could mediate these relationships. Their results revealed that average sleep duration at fourth grade is a

significant predictor of sixth-grade cigarette smoking, but not alcohol consumption. Furthermore, indirect effects to cigarette and alcohol usage were found through fifth-grade inhibitory control. A connection between weekend delays in bedtime during fourth grade and the use of cigarette and alcohol was also discovered through inhibitory control. Moreover, Warren et al. (2017) indicated that reduced nightly sleeps increased the risk of cigarette use. Through impaired sleep-related inhibitory control, their investigation suggested a pathway linking reduced sleep duration and changing sleep patterns during weekends to later substance misuse.

Riemerth et al. (2009) indicated that nicotine smoking affects sleep negatively. They found that about 20% of participants woke in the middle of the night and had to smoke a cigarette to fall back asleep. They called this symptom ‘nocturnal sleep-disturbing nicotine craving’, which may also be present for other substances (Gottfredson, 2018; Rieder et al., 2001; Riemerth et al., 2009). Furthermore, the likelihood of sleep problems, such as insomnia, poor sleep quality, shorter sleep duration, daytime sleepiness and increased difficulty maintaining sleep is higher in cigarette-smokers than non-smokers (Cohrs et al., 2014; Deleanu et al., 2016; Gangwisch et al., 2006; Gangwisch et al., 2007; Jaehne et al., 2012; Liu et al., 2013; McNamara et al., 2014, Roane & Taylor, 2008; Wilson, 2005; Yaggi et al., 2005). Therefore, examining the association between poor sleep quality and nicotine use is critical for many chronic health conditions.

Investigations also indicate a link between cannabis use and insomnia (Roane & Taylor, 2008), self-report sleep problems (Johnson & Breslau, 2001) and sleep duration problems (McKnight-Eily et al., 2011). Like alcohol, cannabis might improve subjective sleep complaints, specifically when used for short periods (Haney et al., 1999). For example, participants using self-report questionnaires disclosed greater ease in falling asleep (Chait,

1990). However, chronic cannabis use has been reported to have negative subjective effects on sleep (Haney et al., 1999). Thus, the sleep-promoting effects of cannabis are decreased in chronic users compared to occasional users (Chait, 1990; Chait & Zacny, 1992).

Numerous longitudinal studies have revealed the relationship between substance misuse and poor sleep quality (Mike et al., 2016). For instance, childhood ‘sleeping difficulty’ and ‘overtiredness’ reported by parents can predict boys’ alcohol and cannabis use during 12 to 14 years of age (Wong et al., 2004). One year later, alcohol use was linked with insomnia during 12 to 19 years of age (Hasler et al., 2014). Additionally, a longitudinal study by National Adolescent Health found an association between subsequent illegal substance misuse and decreased sleep duration and difficulty falling asleep (Wong et al., 2015). Two years later, sleep duration was determined to be a predictor of cannabis use (Pasch et al., 2012).

In a more recent study (Ara et al., 2016), researchers reported a complex bidirectional association between substance misuse and sleep problems, such as insomnia, hypersomnia, excessive daytime sleepiness and obstructive sleep apnoea. Substance misuse can affect receptors and neurotransmitters involved in normal sleep regulation. Hence, disruption of these neurotransmitter systems may trigger changes in normal sleep patterns, resulting in sleep disturbances. One important factor that substance misuse treatment programmes fail to recognise is that sleep problems can persist even after abstinence, leading to relapse (Ara et al., 2016). Therefore, choosing appropriate strategies to counter sleep quality problems can help those who misuse substances avoid relapse. Moreover, different pharmacologic and non-pharmacologic strategies can treat sleep problems in individuals who misuse substances. There is a large variety of strategies, but there is limited data on which pharmacologic options are more effective in treating sleep problems in those who misuse substances.

As mentioned in the earlier sections of this chapter, individuals with ADHD symptoms and substance misusers both have sleep disturbances and poor sleep quality. Based on the information provided in this section, people with ADHD have more sleep problems, worsening their symptoms. Moreover, those with poor sleep quality exhibit ADHD-like symptoms. Sleep problems can also heighten the risk of initiation, intoxication and relapse in individuals with substance misuse. Those with SUD have more sleep disturbances than healthy adults. Consequently, studies indicate that untreated sleep problems may exacerbate both ADHD and substance misuse symptoms. Thus, interventions for sleep problems are an essential part of managing ADHD and substance misuse. However, research on sleep quality in adults with ADHD is scarce. None of the previous investigations focused on the role of sleep quality in the relationship between each ADHD symptom cluster and substance misuse, as this study did. In addition, university student's sleep quality is particularly interesting as they are often in environments that make sleep difficult; 60% of university students report poor sleep quality (Foulkes et al., 2019; Lund et al., 2010). In past studies, the key factors that caused poor sleep quality include fundamental aspects of moving to university, such as living with peers and adapting to a novel situation with a new schedule. Other causes were working late at night and spending a long time in the bedroom during the day (Foulkes et al., 2019). Poor sleep quality raises the risk of mental illness (Freeman et al., 2017) and lowers academic achievement (Gomes et al., 2011) in university students. The current study concentrated on the role of sleep quality in the relationship between ADHD symptoms and substance misuse in university students due to their higher rates of substance misuse and poor sleep quality.

6.4. Aim of this study

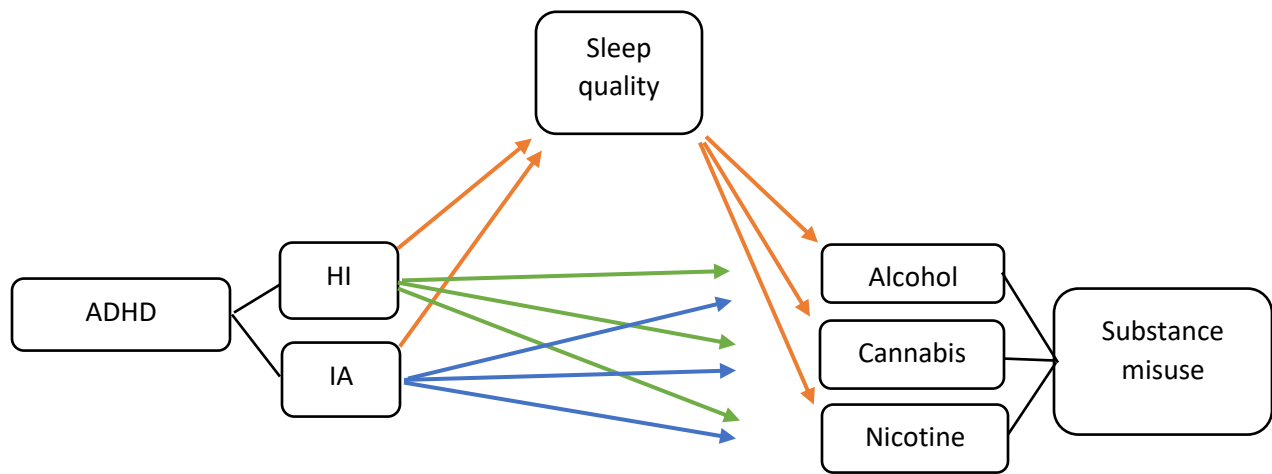
This study aims to test whether sleep quality mediates the relationship between inattention and hyperactivity/impulsivity and the use of alcohol, cannabis and nicotine separately. To reach this goal, a four-step regression model analysed the mediation relationship between the variables in this analysis:

- to determine if each ADHD symptom significantly predicts sleep quality on its own
- to explore whether sleep quality indicates the use of alcohol, cannabis and nicotine
- to investigate how far ADHD symptoms predict the use of alcohol, cannabis and nicotine
- to examine how the predictive capacity of ADHD symptoms changes if sleep quality is considered when predicting the use of alcohol, cannabis and nicotine (Figure 6.1)

If ADHD symptoms are not significant predictors of substance misuse in the presence of sleep quality, there is complete mediation in the model. If both ADHD symptoms and sleep quality are substantial predictors of alcohol, cannabis and nicotine use, there is partial mediation between the variables. This study is the first to explore the role of sleep quality as a mediator between hyperactivity/impulsivity and inattention symptoms and the use of alcohol, cannabis and nicotine separately.

As mentioned in Section 6.2, investigators have revealed associations between inattention and hyperactivity/impulsivity and poor sleep quality, but the evidence in this field

is limited. Hence, it is hypothesised that both ADHD symptom clusters predict poor sleep quality in the first step of the mediation analysis. Based on the outcomes of previous studies presented in Section 6.3, poor sleep quality is assumed to predict alcohol, cannabis and nicotine use in the analysis's second phase. Moreover, since ADHD symptom clusters significantly predicted substance misuse in the third step of this thesis's Studies 3 and 4, it is theorised that both ADHD symptom clusters would also predict alcohol, cannabis and nicotine use in this study's third stage. Furthermore, the key findings of Studies 3 and 4 revealed that emotional regulation and bipolar disorder are partial mediators between ADHD symptoms and certain substance misuse scores. They account for only a part of the relationship between ADHD and substance misuse, implying that other variables could mediate this connection. Therefore, it is hypothesised that sleep quality will be a partial mediator between ADHD symptoms and substance misuse.



- The direct effect of ASRS-HI on alcohol, cannabis and nicotine misuse.
- The direct effect of ASRS-IA on alcohol, cannabis and nicotine misuse.
- The indirect effect of ASRS-HI and IA on the use of alcohol, cannabis and nicotine via sleep quality.

Figure 6.1: The relationship between the different variables of this study. Based on previous literature, ADHD can predict all three, alcohol, nicotine and cannabis use significantly. Some studies showed the relationship between each ADHD symptom and these three substance misuses. The current study is an attempt to find if sleep quality can mediate each ADHD subtype and alcohol, nicotine and cannabis use significantly.

6.5. Methods

6.5.1. Participants

This study was combined with the previous one (Chapter 5), thereby having the same participants. The 270 respondents aged 18 and above (mean = 23.29 years, $SD = 7.10$) were recruited from the Department of Psychology’s Research Participation Scheme at Goldsmiths, University of London. Due to their incomplete answers to the questionnaires (only one or two questionnaires were completed among seven questionnaires), 47 students were excluded. Ultimately, there were 223 participants; 75.1% of them were women ($n = 169$). Individuals with a major psychiatric disorder (e.g. schizophrenia, borderline personality

disorder, obsessive-compulsive disorder) and/or major physical health problems (e.g. brain injury) were removed from the study. Respondents confirmed that they were 18 or older and agreed to participate through an online consent form. All of them completed the measures anonymously.

6.5.2. Procedure

This study was approved by the Department of Psychology's ethics committee at Goldsmiths, University of London (Ethics Reference Number PS130617ZSS– same as the previous study; please see Appendix 15 for the approved Ethical Approval Form).

Participants were recruited from the Department of Psychology's Research Participation Scheme at Goldsmiths, University of London. The study's information and link were on the Research Participation Scheme page, together with the researcher's email address.

Participants could email the researcher and ask any question about the study. On the first page of the online survey, more details about the questionnaires' number and length, ethical issues and the study's overall focus were given. The questionnaires were designed in a way that participants could skip a question that they did not want to answer. There was a debrief page at the end of the survey for participants, with comprehensive information about the study.

As mentioned, this study was combined with Chapter 5 (the role of mood disorders in the relationship between ADHD symptoms and substance misuse). In addition to the Pittsburgh Sleep Quality Index (PSQI), substance misuse and ADHD questionnaires, participants completed the Mood Disorder Questionnaire (MDQ).

6.5.3. Measures

To assess substance misuse and ADHD symptoms, measures (AUDIT, AUQ, CUDIT-R, CAN, NIC, and ASRS) used in Chapters 2, 3, 4 and 5 were also employed in this study (please refer to Section 2.6.3 for full details). For each questionnaire, the Cronbach alpha is as follows: ASRS-IA = .89, ASRS-HI = .89, ASRS-total = .93, AUDIT = .95, AUQ = .74, NIC = .86, CUDIT-R = .99, CAN = .68.

6.5.3.1. The Pittsburgh Sleep Quality Index (PSQI) (Buysse, 1988);

PSQI is a month-long self-report questionnaire that measures sleep quality. It consists of 19 individual items, forming seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime dysfunction. A global score is calculated as a sum of the scores for all seven components. For 18 months, investigators assessed the PSQI's clinical and clinimetric properties in 52 healthy subjects (good sleepers) and 54 patients with depression (poor sleepers). It was evaluated for internal homogeneity, test-retest reliability and validity. In distinguishing good and poor sleepers, a global score of more than 5 had diagnostic sensitivity and specificity of 89.6% and 86.5%, respectively. Buysse et al. (1988) recommended the cutoff score, which was also used in other studies (e.g. Buysse et al., 2008; Grandner et al., 2006; Mezick et al., 2008). In this study, PSQI's Cronbach alpha was .80.

6.5.4. Data analysis strategy

Data were analysed using IBM SPSS version 20. Pearson correlation was conducted to examine the connection between the variables of the study and the results are presented in Table 6.2. Each participant's sleep quality was determined using a global score, the sum of

scores for all seven PSQI components. The scores of ADHD were divided into two categories: inattention (ASRS-IA) and hyperactivity/impulsivity (ASRS-HI), the main ADHD symptoms. A four-step mediation analysis was conducted for each grouping to discern whether sleep quality mediates the relationship between each ADHD score and the use of alcohol, cannabis and nicotine (Figure 6.1). Additionally, to measure the magnitude of the indirect effect between the variables, the Sobel test and PROCESS macro were used.

6.6. Results

6.6.1. Outliers and multi-collinearity

An analysis of standard residuals shows that the data contained seven outliers in alcohol, cannabis and nicotine use scores (Std. Residual Min >-3.29 , Std. Residual Max <3.29), which were deleted from the data. The assumption of collinearity indicated that multi-collinearity was not a concern (VIF value is less than 10 and the Tolerance is more than 0.1). In this study to correct for slight positive skew (1.71) (kurtosis = 2.60) in the AUDIT scores, AUQ scores (skewness = 2.14, kurtosis = 4.56) and CUDIT-R (skewness = 1.63, kurtosis = 1.75) and CAN (skewness = 1.04, kurtosis = 1.54) scores were log transformed (new = LG10 (old + a); a= 1). The new skewness and kurtosis of these scores were as follows: AUDIT scores (skewness = -.27, kurtosis = .08), AUQ scores (skewness = -.45, kurtosis = -.36) and CUDIT-R (skewness = .05, kurtosis = -.74) and CAN (skewness = -.66, kurtosis = -.87).

6.6.2. The percentage of alcohol, cannabis and nicotine frequency, quantity, hazardous use and dependence

Based on the fact that the current study was combined with the study in Chapter 5, and they both had the same participants, the results are also the same. Alcohol, cannabis and nicotine frequency, quantity, hazardous and dependence were asked by the same questions as the previous chapter (Chapter 5, Section 5.6.2). The results revealed that 35.6% of the participants gained a PSQI score of 5 or higher, indicating poor sleep quality.

6.6.3. Descriptive statistics and correlation between the variables

The descriptive statistics and correlation analysis of this study are provided in tables 6.1 and 6.2. Men were coded as '1' (24.21%) and women were coded as '2' (75.78%) in this study. The correlation analysis between variables showed that PSQI was correlated with all three scores of ADHD (r (ASRS-IA) = .59, r (ASRS-HI) = .62, r (ASRS-total) = .66, $p < .001$, $n = 223$) and also with alcohol ($r = .61$), cannabis ($r = .60$) and nicotine use ($r = .38$) significantly ($p < .001$, $n = 223$). Further, there was a significant negative correlation between PSQI and the gender of participants ($p < .05$). All three scores of ADHD was also correlated significantly with all substance misuse scores.

Table 6.1
Descriptive statistics and demographic information

| | Mean | Std. Deviation | <i>n</i> | Range | Minimum | Maximum |
|-------------|-------|----------------|----------|-------|---------|---------|
| Age | 23.29 | 7.10 | 214 | 32 | 18 | 50 |
| PSQI | 4.04 | 4.3 | 223 | 16 | 0 | 16 |
| ASRS-IA | 17.92 | 7.22 | 223 | 36 | 0 | 36 |
| ASRS-HI | 14.48 | 7.21 | 223 | 36 | 0 | 36 |
| ASRS-total | 32.40 | 13.26 | 223 | 70 | 0 | 70 |
| AUDIT_log | .87 | .37 | 223 | 1.68 | .0 | 1.68 |
| AUQ_log | 1.20 | .58 | 223 | 2.32 | .0 | 2.32 |
| CUDIT-R_log | .68 | .44 | 223 | 1.57 | .0 | 1.57 |
| CAN_log | 1.02 | .55 | 223 | 1.91 | .0 | 1.91 |
| NIC | 21.72 | 18.10 | 223 | 53 | 0 | 53 |

Note: ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

Table 6.2
Pearson correlation between ADHD, sleep quality and substance misuse variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 |
|--------------|--------|--------|-------|-------|-------|-------|-------|-------|-------|-----|----|
| 1-Age | 1 | | | | | | | | | | |
| 2-Gender | -.37** | 1 | | | | | | | | | |
| 3- PSQI | -.05 | -.16* | 1 | | | | | | | | |
| 4-ASRS-IA | -.27** | .01 | .59** | 1 | | | | | | | |
| 5-ASRS-HI | -.08 | -.20** | .62** | .69** | 1 | | | | | | |
| 6-ASRS-total | -.19** | -.10 | .66** | .92** | .92** | 1 | | | | | |
| 7-AUDIT | -.01 | -.18** | .60** | .63** | .66** | .70** | 1 | | | | |
| 8-AUQ | .02 | -.19** | .53** | .52** | .56** | .59** | .82** | 1 | | | |
| 9-CUDIT-R | .00 | -.25** | .60** | .61** | .67** | .70** | .71** | .63** | 1 | | |
| 10-CAN | -.05 | -.19** | .45** | .47** | .47** | .51** | .60** | .59** | .73** | 1 | |
| 11-NIC | .19 | -.22 | .38 | .34 | .36 | .38 | .61 | .61 | .50 | .63 | 1 |

Note: ASRS = ADHD Self-Report Scale; IA = Inattentive; HI = Hyperactive; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; NIC = Nicotine Use Questionnaire; CAN = Cannabis Use Frequency Questionnaire.

6.6.4. Does sleep quality mediate the relationship between ASRS-IA and substance misuse?

A) AUDIT:

The results of the multiple regression in the first step of the mediation analysis showed that ASRS-IA predicted PSQI in a significant way ($\beta = .55, p < .001$) and it accounted

for 30% of the variance ($R^2 = .30$, $F(1,221) = 96.08$, $p < .001$). In the second step, PSQI predicted AUDIT scores significantly ($R^2 = .32$, $F(1,221) = 103.77$, $p < .001$) and accounted for 32% of the variance ($\beta = .56$, $p < .001$). In the third step, ASRS-IA predicted AUDIT significantly ($\beta = .62$, $p < .001$). It accounted for 39.2% of the variance in AUDIT ($R^2 = .39$, $F(1,221) = 142.49$, $p < .001$).

The last step showed that controlling for the mediator (sleep quality), ASRS-IA scores were still a significant predictor of AUDIT scores, ($\beta = .45$, $t(223) = 7.62$, $p < .001$). The indirect effect of ASRS-HI on AUDIT scores via PSQI was .91 and was statistically significant ($p < .001$), with a 95% confidence interval, which did not include zero. The Sobel test was conducted and PSQI could mediate ASRS-IA and AUDIT scores partially ($z = 4.98$, $p < .001$).

B) AUQ

In the first step of the mediation model, independent variable, ASRS-IA, significantly predicted PSQI as the mediator ($R^2 = .35$, $F(1,221) = 119.81$, $p < .001$) and accounted for 30% of the variance ($\beta = .55$, $p < .001$). The second step of the multiple regression showed that PSQI was a significant predictor of AUQ ($\beta = .49$, $p < .001$). It accounted for 24.7% of the variance in AUQ scores ($R^2 = .25$, $F(1,221) = 72.62$, $p < .001$). The third step of the mediation model revealed that ASRS-IA predicts AUQ scores significantly ($R^2 = .27$, $F(1,221) = 81.77$, $p < .001$) and accounted for 27% of the variance AUQ ($\beta = .52$, $p < .001$).

The last step indicated that after controlling PSQI as the mediator, ASRS-IA was still a significant predictor of AUQ score ($\beta = .35$, $t(223) = 5.35$, $p < .001$). The indirect effect of ASRS-HI on AUQ scores via PSQI was 1.35, with a 95% confidence interval, which did not

include zero. The Sobel test was conducted and the results showed that sleep quality was a significant partial mediator between ASRS-IA and AUQ ($z = 4.73, p < .001$) (Table 6.3).

C) CUDIT-R

The first step of the multiple regression was the same as AUDIT and AUQ. The second regression analysis indicated that sleep quality was a significant predictor of CUDIT-R scores ($\beta = .57, p < .001$) and accounted for 33.1% of the variance in CUDIT-R ($R^2 = .33, F(1,221) = 109.49, p < .001$). The third step of the mediation model revealed that inattention symptom of ADHD predicted CUDIT-R significantly ($\beta = .61, p < .001$). It accounted for 37.4% of the variance ($R^2 = .37, F(1,221) = 131.95, p < .001$).

In the fourth step, ASRS-IA was still a significant predictor of CUDIT-R scores after controlling PSQI as the mediator ($\beta = .40, t(223) = 6.46, p < .001$). The indirect effect of ASRS-HI on CUDIT-R scores via PSQI was .13, with a 95% confidence interval, which did not include zero. The Sobel tests results showed that PSQI was a partial mediator between ASRS-IA and CUDIT-R scores ($z = 4.90, p < .001$) (Table 6.3).

D) CAN

In step 1 of the mediation model, our IV, ASRS-IA, predicted the mediator, PSQI, in a significant way the same as previous substance misuse scores in this section. Step 2 indicated that PSQI as the mediator predicted CAN significantly ($R^2 = .17, F(1,221) = 45.11, p < .001$) and accounted for 17% of the variance in CAN scores ($\beta = .41, p < .001$). The results of the

third step demonstrated that ASRS-IA was a significant predictor of CAN ($R^2 = .22$, $F(1,221) = 62.36$, $p < .001$) and it accounted for 22% of the variance ($\beta = .47$, $p < .001$).

The last step of the multiple regression showed that after controlling the mediator, inattention symptom of ADHD was still a significant predictor of CAN scores ($\beta = .32$, $t(223) = 4.98$, $p < .001$) and the indirect effect of ASRS-HI on CAN score via PSQI was .94, with a 95% confidence interval, which did not include zero. The results of the Sobel test showed that PSQI was a partial mediator between these two variables ($z = 3.54$, $p < .001$) (Table 6.3).

E) NIC

The results of the first step of the mediation model were the same as the previous substances. In the second step PSQI predicted nicotine use significantly ($\beta = .38$, $p < .001$) and accounted for 15% of the variance in NIC scores ($R^2 = .15$, $F(1,221) = 37.78$, $p < .001$). The third step of the mediation analysis indicated that ASRS-IA can predict nicotine use significantly ($\beta = .34$, $p < .001$). It accounted for 11.3 % of the variance in nicotine use ($R^2 = .113$, $F(1,221) = 28.09$, $p < .001$).

The result of the fourth step revealed that after controlling the mediator, PSQI, the inattention symptom of ADHD was still a significant predictor ($\beta = .17$, $t(223) = 2.20$, $p < .05$). The indirect effect of ASRS-HI on CAN scores via PSQI was 40.18, with a 95% confidence interval, which did not include zero. The Sobel test results show that mood disorder was a partial mediator between ASRS-IA and nicotine use ($z = 3.5$, $p < .001$) (Table 6.3).

Table 6.3

Four steps mediation analysis of sleep quality between ASRS-IA and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|-----------|--------------------|-------|--------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-IA | Sleep | .30** | 96.08 | .55** | 4.98** | .19 * | .52 | 1.29 |
| 2 | Sleep | AUDIT | .32** | 103.77 | .56** | | | | |
| 3 | ASRS -IA | AUDIT | .39** | 142.49 | .62** | | | | |
| 4 | ASRS -IA | AUDIT | .46** | 94.40 | .45** | | | | |
| | | Sleep | | | .32** | | | | |
| 1 | ASRS -IA | Sleep | .30** | 96.08 | .55** | 4.72** | .13* | .69 | 1.98 |
| 2 | Sleep | AUQ | .25** | 72.62 | .49** | | | | |
| 3 | ASRS -IA | AUQ | .27** | 81.77 | .52** | | | | |
| 4 | ASRS -IA | AUQ | .33** | 55.19 | .35** | | | | |
| | | Sleep | | | .30** | | | | |
| 1 | ASRS -IA | Sleep | .30** | 96.08 | .55** | 4.90** | 1.15 * | .62 | 1.59 |
| 2 | Sleep | CUDIT-R | .33** | 109.49 | .57** | | | | |
| 3 | ASRS -IA | CUDIT-R | .37** | 131.95 | .61** | | | | |
| 4 | ASRS -IA | CUDIT-R | .46** | 92.13 | .42** | | | | |
| | | Sleep | | | .34** | | | | |
| 1 | ASRS -IA | Sleep | .30** | 96.08 | .55** | 3.54** | .94* | .41 | 1.48 |
| 2 | Sleep | CAN | .17** | 45.11 | .41** | | | | |
| 3 | ASRS -IA | CAN | .22** | 62.36 | .46** | | | | |
| 4 | ASRS -IA | CAN | .25** | 37.43 | .34** | | | | |
| | | Sleep | | | .22** | | | | |
| 1 | ASRS -IA | Sleep | .30** | 96.08 | .55** | 3.49** | 40.18* | 20.78 | 60.17 |
| 2 | Sleep | NIC | .15** | 39.14 | .38** | | | | |
| 3 | ASRS -IA | NIC | .11** | 28.09 | .33** | | | | |
| 4 | ASRS -IA | NIC | .17** | 22.83 | .17** | | | | |
| | | Sleep | | | .29** | | | | |

Note: ASRS-IA = Inattention symptom of ADHD; Sleep = PSQI total score; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

6.6.5. Does sleep quality mediate the relationship between ASRS-HI and substance misuse?

A) AUDIT

The first step of the mediation analysis showed that hyperactivity/impulsivity symptom of ADHD is a significant predictor of PSQI ($R^2 = .34$, $F(1,221) = 115.37$, $p < .001$) and accounted for 38.7% of the variance in PSQI scores ($\beta = .58$, $p < .001$). The second step indicated that the mediator can predict AUDIT scores significantly ($\beta = .56$, $p < .001$). It accounted for 32% of the variance ($R^2 = .32$, $F(1,221) = 103.77$, $p < .001$). The third step revealed that ASRS-HI predicts AUDIT in a significant way ($R^2 = .44$, $F(1,221) = 174.76$, $p < .001$) and accounted for 44.2% of the variance in AUDIT scores ($\beta = .67$, $p < .001$).

In the fourth step of the mediation model, both the mediator and ASRS-HI as the independent variable were statistically significant predictors of AUDIT scores (β (ASRS-HI) = .51, $t(223) = 8.53$, $p < .001$). The indirect effect of ASRS-HI on AUDIT scores via PSQI was .82, with a 95% confidence interval, which did not include zero. This means that the effect was significantly greater than zero at $\alpha = .05$. The Sobel test results indicated that PSQI was a partial mediator between hyperactivity/impulsivity symptom of ADHD and AUDIT ($z = 4.44$, $p < .001$) (Table 6.4).

B) AUQ

The first step of the mediation model for the relation between ASRS-HI, PSQI and AUQ scores were the same as the AUDIT results. The second step of it showed that PSQI is a significant predictor of AUQ ($R^2 = .25$, $F(1,221) = 72.62$, $p < .001$) and accounted for 24.7%

of the variance ($\beta = .50, p < .001$). In the third step, ASRS-HI predicted AUQ significantly ($R^2 = .31, F(1, 221) = 99.72, p < .001$) and accounted for 31.1% of the variance in AUQ scores ($\beta = .56, p < .001$).

The last step of the mediation analysis revealed that after controlling for PSQI as the mediator, ASRS-HI was still a statistically significant predictor of AUQ score ($\beta = .41, t(223) = 6.07, p < .001$). The indirect effect of ASRS-HI on AUQ scores via PSQI was 1.23, with a 95% confidence interval, which did not include zero. Sobel test was conducted and the results showed that sleep quality was a partial mediator between ASRS-HI and AUQ ($z = 4.2, p < .001$) (Table 6.4).

C) CUDIT-R

The results of the first step of the mediation model were the same as previous sections and ASRS-HI was a significant predictor of PSQI as the mediator. In the second step, PSQI predicted CUDIT-R scores significantly ($R^2 = .33, F(1, 221) = 109.49, p < .001$) and accounted for 33.1% of the variance ($\beta = .57, p < .001$). In the third step, hyperactivity/impulsivity symptom of ADHD predicted CUDIT-R in a statistically significant way ($R^2 = .44, F(1, 221) = 176.53, p < .001$) and accounted for 44.4% of the variance in CUDIT-R scores ($\beta = .67, p < .001$).

The last step indicated that in the presence of the mediator, both ASRS-HI as the independent variable and PSQI as the mediator were significant predictors of CUDIT-R scores (β (ASRS-HI) = .50, $t(223) = 8.49, p < .001$, β (sleep quality) = .28, $t(223) = 4.77, p < .001$) (Table 6.14). The indirect effect of ASRS-HI on CUDIT-R scores via PSQI was

1.01, with a 95% confidence interval, which did not include zero. A Sobel test was conducted and found partial mediation in the model ($z = 4.2, p < .001$) (Table 6.4).

D) CAN

In the first step of the mediation analysis, ASRS-HI predicted PSQI significantly like previous sections. Step 2 of the regression model indicated that PSQI can predict CAN significantly ($\beta = .41, p < .001$). It accounted for 17% of the variance in CAN scores ($R^2 = .17, F(1,221) = 45.11, p < .001$). The third step showed that ASRS-HI is a significant predictor of CAN scores ($R^2 = .22, F(1,221) = 63.78, p < .001$) and the fourth step revealed that after controlling for PSQI, hyperactivity/impulsivity symptom of ADHD can still predict CAN scores significantly ($\beta = .35, t(223) = 4.91, p < .001$). The indirect effect of ASRS-HI on CAN score via PSQI was 1.19, with a 95% confidence interval, which did not include zero. Sobel test results showed that PSQI was a partial mediator between HI and cannabis use frequency ($z = 3.1, p = .002$) (Table 6.4).

E) NIC

It has been mentioned in the previous sections that hyperactivity/impulsivity symptom of ADHD is a significant predictor of PSQI. The second step of the mediation analysis indicated that PSQI can predict nicotine use significantly ($\beta = .38, p < .001$). It accounted for 14.6% of the variance in NIC scores ($R^2 = .15, F(1,221) = 37.78, p < .001$). The third step showed that ASRS-HI was a statistically significant predictor of NIC ($R^2 = .13, F(1,221) = 33.78, p < .001$) and accounted for 13.3% of the variance in nicotine use ($\beta = .36, p < .001$). It has been shown that both of PSQI as the mediator and ASRS-HI as the independent variable predicted

nicotine use significantly (β (ASRS-HI) = .21, t (223) = 2.76, $p > .05$, β (PSQI) = .27, t (223) = 3.52, $p > .05$). The indirect effect of ASRS-HI on NIC scores via PSQI was 39.4, with a 95% confidence interval, which did not include zero. The Sobel test results demonstrated that PSQI was a partial mediation in the model ($z = 3.12$, $p = .001$) (Table 6.4).

Table 6.4
Four steps mediation analysis of sleep quality between ASRS-HI and substance misuse scores

| Step | Predictor | Dependent variable | R^2 | F | β (Standardized Coefficient) | Sobel test | Indirect effect size | 95% CI | |
|------|-----------|--------------------|-------|--------|---------------------------------------|------------|----------------------|--------|-------|
| | | | | | | | | LL | UL |
| 1 | ASRS-HI | Sleep | .34** | 115.37 | .58** | 4.44** | .82* | .43 | 1.21 |
| 2 | Sleep | AUDIT | .32** | 103.77 | .56** | | | | |
| 3 | ASRS-HI | AUDIT | .44** | 174.76 | .66** | | | | |
| 4 | ASRS-HI | AUDIT | .49** | 105.17 | .51** | | | | |
| | | Sleep | | | .27** | | | | |
| 1 | ASRS-HI | Sleep | .34** | 115.37 | .58** | 4.20** | 1.23* | .62 | 1.83 |
| 2 | Sleep | AUQ | .25** | 72.62 | .49** | | | | |
| 3 | ASRS-HI | AUQ | .3** | 99.72 | .55** | | | | |
| 4 | ASRS-HI | AUQ | .35** | 60.62 | .40** | | | | |
| | | Sleep | | | .26** | | | | |
| 1 | ASRS-HI | Sleep | .34** | 115.37 | .58** | 4.20** | 1.01* | .56 | 1.49 |
| 2 | Sleep | CUDIT-R | .33** | 109.49 | .57** | | | | |
| 3 | ASRS-HI | CUDIT-R | .44** | 176.53 | .66** | | | | |
| 4 | ASRS-HI | CUDIT-R | .50** | 108.40 | .50** | | | | |
| | | Sleep | | | .28** | | | | |
| 1 | ASRS-HI | Sleep | .34** | 115.37 | .58** | 3.10* | .93* | .34 | 1.42 |
| 2 | Sleep | CAN | .17** | 45.11 | .41** | | | | |
| 3 | ASRS-HI | CAN | .22** | 63.78 | .47** | | | | |
| 4 | ASRS-HI | CAN | .25** | 36.97 | .35** | | | | |
| | | Sleep | | | .20** | | | | |
| 1 | ASRS-HI | Sleep | .34** | 115.37 | .58** | 3.12* | 39.04* | 16.87 | 59.41 |
| 2 | Sleep | NIC | .15** | 39.14 | .39** | | | | |
| 3 | ASRS-HI | NIC | .13** | 33.78 | .36** | | | | |
| 4 | ASRS-HI | NIC | .18** | 23.98 | .21** | | | | |
| | | Sleep | | | .26** | | | | |

Note: ASRS-HI = Hyperactivity/Impulsivity symptom of ADHD; Sleep = PSQI total score; AUDIT = Alcohol Use Disorder Identification Test; AUQ = Alcohol Use Questionnaire; CUDIT-R = Cannabis Use Disorder Identification Test; CI = Confidence Interval; LL = Lower Limit; UL = Upper Limit
 $p < .05$ *; $p < .001$ **

6.7. Discussion

The results revealed that 35.6% of the participants gained a PSQI score of 5 or higher, indicating poor sleep quality. According to Lund et al. (2010), 60% of university students are poor-quality sleepers, who have more physical and psychological health problems than those with good sleep quality. In their analysis, stress during the academic year accounted for 24% of the variance in sleep quality (as measured by PSQI). In another PSQI study, 74% of the students had insomnia symptoms as described by the DSM-5 (Schlarb et al., 2017). Thus, the main aspects of university life, such as living with peers and adapting to new academic environments and programmes, may heighten the risk of poor sleep quality in university students (Foulkes et al., 2019).

This study investigated whether sleep quality has a mediating role in the relationship between the ADHD symptoms (inattention and hyperactivity/impulsivity) and the use of alcohol, cannabis and nicotine. The findings of this study's four-step regression analysis revealed that there was only a partial mediation between the variables, rather than a complete one, between the two ADHD symptom clusters (inattention and hyperactivity/impulsivity), sleep quality (as the mediator) and all the substance misuse questionnaire scores. This outcome suggests that sleep quality accounts for a part of the connection between ADHD symptoms and substance misuse. Other variables that may account for the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine have been discussed in Chapters 4 and 5.

In the first step of the mediation analysis, both inattention and hyperactivity/impulsivity significantly predicted sleep quality. Previous studies have shown that the severity of ADHD

symptoms is linked to poor sleep quality, both now and in the past (Vogel et al., 2017). Children with ADHD are more likely to have disturbed sleep (Owens, 2009), shorter sleep duration (Touchette et al., 2007) and a higher risk for other sleep problems (Owens et al., 2000), which could persist into adolescence and adulthood (Sobanski et al., 2008). Gregory et al. (2017) indicated that young adults with persistent childhood ADHD had poorer sleep quality. Sleep problems in adults with ADHD were also found to be more prevalent than maternal insomnia and psychiatric comorbidities. Moreover, they discovered that the association between ADHD and poor sleep quality was based on genetic (55%) and environmental (45%) factors. In a study by Gaultney (2014), university students with ADHD symptoms had a higher risk of insomnia, restless legs syndrome and periodic limb movement disorder. Research on sleep quality in adults with ADHD is limited, with most of them measuring ADHD as a full neurodevelopmental disorder. In this study, the ADHD symptoms were divided into two categories, with results showing that both facets of ADHD significantly predicted poor sleep quality. As presented in Section 6.6.4 and 6.6.5, the results specifically revealed that the hyperactivity/impulsivity symptom of ADHD accounted for more variance (38.7%) in poor sleep quality than the inattention symptom (30%) (Tables 6.3 and 6.4).

According to studies of children with ADHD symptoms, those with hyperactivity/impulsivity and combined types of ADHD had more sleep issues than those with the inattention type of ADHD; objective measures confirmed more activity or restlessness in individuals with ADHD (Konofal et al., 2001; Spruyt & Gozal, 2012). Further investigations on different ADHD presentations and sleep problems in adults are required.

In the second step of the regression model, sleep quality predicted the use of alcohol, cannabis and nicotine significantly. Sleep problems and insomnia are significant risk factors in developing substance misuse and later SUD (Breslau et al., 1996; Weissman et al., 1997; Wong et al., 2009; Wong et al., 2010). Furthermore, Chakrovorty et al. (2018) reported a link between insomnia and substance misuse withdrawal. They found that insomnia-focused CBT could help people with alcohol and other drug misuses. They suggested that those with short-term insomnia should stop using substances and consider taking sleep medication during abstinence.

From 2011 to 2014, the American Academy of Sleep Medicine (AASM) conducted a study with 8,683 student-athletes at US universities as part of the American Health Association's National University Health Assessment (American Academy of Sleep Medicine, 2017). Students were asked whether their sleep difficulties were traumatic or extremely difficult to handle and whether they have used specific substances in the past 30 days. After controlling for age, gender and survey year, the data were subjected to regression analysis to determine whether there was an association between any of the substances and sleep quality. The difference between student and typical use and their relationship with sleep were also examined. The results revealed that smoking cigarettes are 151% more likely in student-athletes with sleep problems. They also consumed 36% more alcohol and smoked 66% more cannabis than those without sleep issues. Additionally, sleep problems can predict increased use of controlled, illegal and forbidden substances. For instance, student-athletes with sleep problems were more likely to use methamphetamine at 317%, cocaine at 349% and steroids at 175%. Therefore, AASM's findings indicated that individuals with more sleep problems misuse higher amounts of a substance.

Bhatti et al. (2020), investigated the alcohol and nicotine use and sleep duration of 1,440 university students from three UK universities and found no association between self-reported sleep duration and alcohol use. Their results showed that there is a positive correlation between alcohol and nicotine use in the sample. Further investigation is required to clarify the relationship between substance misuse and sleep quality in university students in the UK.

Although these findings suggest that those with poor sleep quality misuse more substances, other studies indicate the opposite. In other words, they indicate that those who consume more alcohol and other drugs have more sleep problems. In their analysis of 172 adults who received AUD treatment, Brower et al. (2015) found that those with insomnia used more alcohol to sleep, showed longer sleep latency and had lower sleep efficiency than people without AUD. According to a five-month follow-up on 74 of these patients, relapse was more common in people with AUD who had baseline insomnia (Brower et al., 2001). Based on Section 6.3 of this chapter, a bidirectional relationship between substance misuse and sleep quality can be detected, with one causing the symptoms in the other. Even though the current research was a mediation analysis, which indicates a causal relationship between the variables (MacKinnon, 2008), further longitudinal investigations are needed to explore this bidirectional relationship comprehensively.

This study's third regression analysis revealed that ADHD symptoms significantly predict substance misuse, which is consistent with the results of other investigations. For instance, Wilens et al. (2011) established that ADHD heightens the risk of substance misuse and that persistent ADHD was linked to subsequent SUD. They also demonstrated that after controlling for conduct disorder, the association between ADHD and substance misuse

remained significant. However, none of the previous studies investigated the relationship between each ADHD symptom cluster and substance misuse. The results of the current study were consistent with the findings of Studies 3 and 4 of this thesis, finding inattention and hyperactivity/impulsivity to both predict alcohol, cannabis and nicotine use.

The fourth stage of the regression model showed that after adding the mediator, the ADHD symptom clusters remained significant predictors of substance misuse, indicating no full mediation in the regression model. Instead, sleep quality was found to partially mediate the relationship between ADHD symptoms and substance misuse. This result suggests that sleep quality accounts for some of the relationship between ADHD and the use of alcohol, cannabis and nicotine. It also implies that, besides a significant relationship between sleep quality and substance misuse, there is also a direct connection between these variables. Moreover, other variables, such as impaired emotional regulation or bipolar disorder, might mediate this relationship, as discussed in Chapters 4 and 5 of this thesis.

The factors that could explain this relationship is unclear. Several suggestions could explain the relationship between ADHD symptoms, poor sleep quality and the use of alcohol, cannabis and nicotine. First, people with more ADHD symptoms may have more sleep problems and use more substances to cope with their poor sleep quality. Based on the National Resource Centre on ADHD, a programme for Children and Adults with ADHD (CHADD, 2019), alcohol helps people fall asleep quickly (but causes sleep disruptions). Furthermore, using alcohol as a 'sleep aid' may be common among insomniacs (Ancoli-Israel & Roth, 1999; Schweizer et al., 2019). In a five-year longitudinal study of 20,000 UK Biobank participants, there was an association between adequate to inadequate sleep duration and greater odds of heightened daily smoking rate and increased nicotine misuse (Patterson

et al., 2018). Thus, the transition to insufficient sleep could be a predictor of worsening smoking habits, as supported by previous cross-sectional studies (Patterson et al., 2018; Warner & Burns, 2003). In another research, individuals with sleep problems preferred cannabis to other over-the-counter sleep aids (Doremus et al., 2019). Alcohol, cannabis and nicotine use may help those with poor sleep quality and more ADHD symptoms sleep better, but this is not the only option.

Second, studies show that sleep problems may adversely influence affect control and behaviour. Sleep deficits can affect EF, working memory and certain cognitive abilities, such as flexible thinking and multitasking (Durmer & Dinges, 2005; Hvorby, 2015; Pilcher & Huffcutt, 1996). Wajszilber et al. (2018) determined that sleep problems in individuals with ADHD symptoms cause functional impairments that affect mood, attention, behaviour, quality of life and school or work performance. Additionally, Talbot et al. (2010) found that during a task, participants experienced more anxiety and less positive affect, particularly if they had poor sleep. A link between affect problems and sleep issues may denote that behaviour regulation might be more difficult for some people; this poor behaviour control may heighten the risk of substance misuse in an individual. In this thesis's preceding studies, EF deficits, impulsivity facets and mood disorder significantly predicted substance misuse. These indicators may be aggravated by poor sleep quality, leading to increased substance misuse. Thus, focusing on improving one's sleep quality may enhance their EF performance, mood, self-control and cognitive abilities lowering the likelihood of substance misuse.

Third, investigations also demonstrated that poor sleep quality in adults adversely affects response inhibition, which is the ability to inhibit a response (Chuah et al., 2006), detect task errors (Tsai et al., 2005) and make sound decisions (Harrison & Horne, 2000). Wong et al.'s

(2010) longitudinal investigation found that poor response inhibition in adolescence is predicted poor quality of sleep in early childhood. The negative impact of poor sleep quality on EF in general and inhibitory control, in particular, may heighten risky behaviours (Blume et al., 2000; Giancola & Parker, 2001; Nigg et al., 2006). This probability may explain why individuals with more ADHD symptoms show poor response inhibition, EF impairments and cognitive deficits. In addition, lower sleep quality and sleep problems may worsen ADHD symptoms, increasing the likelihood of impulsive actions and decisions or more alcohol, nicotine and cannabis use, which may progress to SUD.

Gregory et al. (2017) examined the ADHD presentation and sleep quality of 2,232 twins and found that only children with persistent ADHD symptoms had poorer sleep quality in young adulthood. They also discovered a significant genetic overlap between ADHD and poor sleep quality (55%) and environmental causes (45%) in those with ADHD symptoms. To further understand the genetic overlap of these two disorders, polygenic risk score analyses could be used.

However, there is a direct path linking ADHD to substance misuse, which could be due to shared neurological brain regions or common genetic factors, as discussed in earlier chapters (please refer to Section 1.3). Previous studies in this thesis revealed that different variables could mediate the relationship between ADHD symptoms and substance misuse (please refer to Chapters 4 and 5), explaining the role of sleep quality as a partial mediator in this relationship. The current study's results showed that sleep quality is another factor that may increase the risk of developing alcohol, cannabis and nicotine use in adults with inattention and/or hyperactivity/impulsivity; a suitable intervention programme is needed to decrease this probability. Thus, interventions to improve sleep quality may help to reduce the

risk of developing substance misuse in those with inattention and hyperactivity/impulsivity symptoms.

6.8. Limitations

While this study has further elucidated the nature of the relationship between ADHD symptoms, sleep quality and the use of alcohol, cannabis and nicotine, separately, some limitations should be taken into consideration. For some parts, the limitations of this study are similar to those outlined in Chapter 5 due to their shared sample and design. For instance, in the current study, a dimensional approach was used; it is unclear whether or not the findings would be similar to the results of a categorical approach. Therefore, measuring the ADHD symptoms and substance misuse categorically may help us explore the relationship between the variables at a deeper level. Investigating sleep problems in university students, such as RLS, Periodic Limb Movement in Sleep (PLMS), Sleep Disordered Breathing (SDB), etc., may reveal a more detailed relationship between each ADHD symptom and sleep disorders.

Moreover, participants were undergraduate students of Goldsmiths, University of London, which limits the generalisability of the findings to the general or clinical populations (Creswell, 2014). Furthermore, instead of a longitudinal study, a cross-sectional study measuring the different variables in a population at a specific point in time was used due to financial and time constraints. This form of research cannot demonstrate a cause-and-effect relationship between the variables. Cross-sectional studies often do not include data on other variables that might affect the relationship between the hypothesised cause and effect (Mann,

2003). Thus, longitudinal investigations on this topic are recommended to test hypotheses about causal mechanisms between these variables.

There are some specific limitations in this study that should be considered in future investigations. For instance, the current study's participants were mostly women; further research should include an equal number of men and women. In addition, past investigations proposed that various aspects of university life might contribute to the students' poor sleep quality; these facets include noise problems, socialisation among peers, unstructured academic life and its costs and poor sleep quality's effect on academic performance and university life (Foulkes et al., 2019). These factors were not considered in the current study, which may affect the student's sleep quality and substance misuse. Furthermore, although some students live on campus, others do not, which can also affect their sleep quality and use of alcohol, cannabis and nicotine. Therefore, future investigations should take into account the unique aspects of the campus environment and their impact on sleep quality and substance misuse among university students.

Another limitation of this research was using a subjective measure of sleep quality. While it is a valid way of measuring sleep quality, using objective measures of sleep quality, such as actigraphy, is recommended (Ancoli-Israel et al., 2003; Benca, 2005).

The results of the current study showed that both symptoms of ADHD predict poor sleep quality, but hyperactivity/impulsivity accounts for more variance in sleep quality than inattention. Furthermore, sleep quality mediates the relationship between both symptoms of ADHD and the use of alcohol, cannabis and nicotine.

6.9. Conclusion

This study has found support for the theory that the relationship between ADHD symptom clusters and substance misuse may be partially explained by poor sleep quality. This is the first study to separate the ADHD symptom clusters of inattention and hyperactivity/impulsivity to explore the relationship of each symptom cluster with sleep quality and alcohol, cannabis and nicotine use. Studies 3 and 4 of this thesis revealed that emotional regulation and bipolar disorder explain a part of the relationship between ADHD symptom clusters and substance misuse. Moreover, in this study, poor sleep quality was found to be a partial mediator between ADHD symptom clusters and the use of alcohol, cannabis and nicotine.

Chapter 7

Overview

This chapter will review the present thesis's main findings and consider their implications for the theory of ADHD and substance misuse and the development of future prevention and intervention programmes. It will also acknowledge the research's limitations before offering suggestions for prospective investigations.

7.1. Key findings

The investigations in this thesis explored the relationship between each ADHD symptom cluster and the use of alcohol, cannabis and nicotine in typically developing university students by using a dimensional approach to measure the variables. As mentioned in Chapter 1, substance misuse, an important comorbid condition among individuals with ADHD symptoms, could result in SUD (please refer to Section 1.3); moreover, university students are at a higher risk of hazardous alcohol, cannabis, and nicotine misuse (Section 1.2.2). Due to the significant overlap between these disorders, researchers proposed investigating ADHD using a dimensional rather than a categorical approach (Heidbreder, 2015; Katzman et al., 2017). An important objective of dimensional approaches in mental health investigations is to better understand the basic dimensions of the underlying functions in the full range of human behaviour, from normal to abnormal (RDoC, 2016). Therefore, given the dimensional structure of the ADHD symptom clusters and substance misuse, a dimensional approach was used for all the studies in this thesis. It was found that 'subthreshold' individuals who do not meet the full criteria for disorders, ADHD symptoms or substance misuse are associated with possible functional impairments; this result provided

useful information about the relationship between inattention and hyperactivity/impulsivity symptoms of ADHD and the use of alcohol, cannabis and nicotine.

Regarding the relationship between ADHD and substance misuse, only a few studies have examined this in adults, ultimately generating mixed findings. None of the previous studies divided the ADHD symptom clusters and explored the connection between each symptom and substance misuse in healthy adults. Some studies have attempted to suggest some possible reasons for developing substance misuse in those with ADHD symptoms. For instance, in individuals with ADHD and those who take alcohol, nicotine, cannabis and other drugs, changes in synaptic dopamine concentration have been introduced as a critical neurotransmitter in the relationship between ADHD and substance misuse (Wang, 2013). Various scenarios could explain this link. Some people with ADHD self-medicate and misuse substances to raise their synaptic dopamine concentration (Arias et al., 2008; Crunelle et al., 2017; Wilens et al., 2007), while those with higher synaptic dopamine concentration exhibit more impulsive behaviours towards alcohol or drug-taking and are less able to inhibit their response to legal or illegal substances. Additionally, common genetic backgrounds have been identified. For instance, the common genetic connection between the hyperactivity symptom of ADHD and alcohol misuse were pointed out by Edwards and Kendler (2012).

In fMRI studies, both conditions shared the PFC region of the brain (Arnsten 2010; Bush, 2010; Goldstein & Volkow 2011; Korponay et al., 2017; Qin et al., 2016). Inattention and distraction symptoms are thought to be related to the impairment of the DLPFC, which regulates attention (Arnsten 2010; Chao & Knight, 1995; Woods & Knight, 1986), whereas impulsivity and hyperactivity symptoms are linked to the impairment of the right inferior PFC (Arnsten, 2010; Aron et al., 2004). Moreover, emotional responses are regulated by the

ventromedial PFC (Arnsten, 2010). Furthermore, general PFC activation that leads to drug-taking and presentation of drug-related cues is replaced by extensive hypoactivity of the PFC in individuals that misuse substances while being exposed to higher-order cognitive or emotional challenges or during extended withdrawal when there is no stimulation (Goldstein & Volkow, 2011).

Previous investigations have identified the functions that are most often involved in specific areas of the PFC, such as the combination of various dimensions of cognition and behaviour (Lezak et al., 2004), ability to continue and set shift, response inhibition, working memory, planning, reasoning, problem-solving, organisational skills and abstract thinking (Alvarez et al., 2006; Clark et al., 2008). It also estimates the affective value and the subjective emotional experience of sensory stimuli (Rolls & Grabenhorst, 2008). There are common impairments and personality traits associated with ADHD and substance misuse, such as impaired EF, impulsivity, emotional dysregulation, bipolar disorder as a mood disorder and poor sleep quality. These commonalities share the same brain regions, neurotransmitters and they are heritable. The aforementioned brain regions are engaged in EF, impulsive behaviours, emotional regulation, mood disorders and quality of sleep, all of which focused on the studies in this thesis and were thoroughly discussed in each chapter. This thesis's main findings are presented below, with references to the three overall aims outlined in Chapter 1.

Aim 1: To investigate which facet of EF predicts alcohol, cannabis, and nicotine use and examine whether inattention and hyperactivity/impulsivity symptoms explain additional variance in developing substance misuse after accounting for EF facets.

The first aim sought to reframe existing literature on the role of EF in the relationship between each ADHD symptom cluster and substance misuse in terms of multi-trait conceptualisation of EF. Separating EF into a number of narrower facets has helped in better understanding the association between EF facets and ADHD and substance misuse (Jansari et al., 2012; Stautz & Cooper, 2013; Marceau et al., 2017; Montgomery et al., 2010, 2012). However, it has not been widely employed to explain alcohol, cannabis and nicotine use in adults with inattention and hyperactivity/impulsivity. Most previous investigations of adults with ADHD have excluded any substance misuse, which prompted this thesis to conduct its studies and explore the risk factors in the connection between ADHD symptom clusters and substance misuse. It was proposed that understanding the complexities of impaired EF in relation to substance misuse and ADHD could aid the development of screening and prevention methods and inform cessation treatment for those with ADHD symptoms, non-substance misusers and escalating misusers.

The study presented in Chapter 2 was the first to use a new virtual reality task (JEF[®]; Jansari et al., 2014) to measure the role of each EF facet in the relationship between ADHD symptom cluster and substance misuse, demonstrating that distinct EF facets do show differences in patterns of association with alcohol, cannabis and nicotine use in adults. Response inhibition predicted adults' alcohol, cannabis and nicotine use, indicating that substance misuse in adults is related to a reduced ability to inhibit or interrupt the expression of cognitive, emotional, or behavioural responses, such as misusing substances. Moreover,

adaptive thinking predicted alcohol and cannabis use but not nicotine use, implying that the inability to respond to environmental changes by shifting attention between unrelated tasks or thinking outside the box is linked to alcohol and cannabis use cues. Furthermore, time-based prospective memory predicted alcohol use, denoting an association between remembering to perform a task at a specific time in the future and alcohol use. Creative thinking, another facet of EF, predicted cannabis use, indicating that those with lower creative thinking scores used more cannabis in their lives.

Hyperactivity/impulsivity predicted alcohol use over and above the facets of EF in the second step of Study 1's hierarchical regression analysis. Moreover, since accounting for EF facets, cannabis use was not predicted by any of the ADHD symptoms, showing that various aspects of EF predict cannabis use based on ADHD symptom pathways. Furthermore, over and beyond the facets of EF, the hyperactivity/impulsivity symptom of ADHD predicted nicotine and lifetime cannabis use. These results indicated that response inhibition, adaptive thinking, time-based prospective memory and creative thinking from an inattention pathway predicted alcohol, cannabis and nicotine use. According to Weafer et al. (2010), there is an association between response inhibition and attention inhibition. Additionally, Boot et al. (2017) found that participants with higher hyperactivity/impulsivity symptoms of ADHD reported being more creative in daily life. The inattention symptom of ADHD also did not predict any substance misuse score over and above the facets of EF. It can, therefore, be postulated that people with higher hyperactivity/impulsivity symptoms may misuse different substances for reasons other than EF impairments.

Such findings add to existing literature by introducing a new virtual reality task to measure EF facets and by investigating the unique predictive capacity of each ADHD

symptom cluster in predicting alcohol, cannabis and nicotine use after accounting EF facets. However, the JEF[®] does not assess different aspects of impulsivity as an accepted shared personality trait in individuals with ADHD and in those with substance misuse. In addition, although previous investigations have provided useful information on how specific impulsivity facets contribute to the relationship between ADHD and substance misuse, their impulsivity evaluations have limitations. None of them used a comprehensive model of impulsivity to detect which aspects of personality relate to the connection between ADHD and substance misuse. Thus, Study 2 addressed this gap by investigating the role of impulsivity's various facets and their association with ADHD symptoms and substance misuse in typically developing individuals.

Aim 2: To investigate which facet of impulsivity predicts alcohol, cannabis and nicotine use and to examine whether inattention and hyperactivity/impulsivity symptoms separately explain additional variance in developing substance misuse after accounting for impulsivity facets.

Chapter 3 of this thesis was the first to comprehensively investigate the association between each impulsivity facet and substance misuse. The UPPS-P model (Cyders & Smith, 2008; Whiteside & Lynam, 2001) and BIS-11 (Patton et al., 1995) were selected as methods of operationalisation for trait impulsivity due to their growing acceptance in previous studies showing that the separable impulsivity-related traits of UPPS-P and BIS-11 can be linked to different aspects of substance misuse through separate pathways (Moreno et al., 2012; VanderVeen et al., 2016). In adults, BIS-motor, negative urgency and sensation seeking predicted alcohol, cannabis and nicotine use significantly. Thus, misusing these substances is related to the propensity to act impulsively without considering their negative consequences

and a difficulty to regulate impulsive behaviour when in a negative emotional state.

Substance misuse is also associated with enjoying and following exciting, new, novel and complex experiences, even if these are physically or socially risky or dangerous.

Previous studies have found that the BIS-motor is a significant predictor of alcohol, cannabis, and nicotine misuse (Balodis et al., 2010; Bjork et al., 2004; Chivers et al., 2016; García-Montes et al., 2009; Goudriaan et al., 2007; Lawrence et al., 2009; Mitchell et al., 2005; Moreno, 2012). The relation between the BIS-motor and substance misuse could be explained by the fact that motor impulsiveness is correlated significantly with inhibition control in previous investigations (Enticott et al., 2006), and the underlying mechanisms of impulsivity's motor dimension are engaged in inhibitory control (Caswell et al., 2013). In those that misuse substances, investigators have demonstrated a more functional connection between the cortical and subcortical PFC (substantia nigra and subthalamic nucleus) of the brain, resulting in more effort to stop an ongoing response (Filbey & Yezhuvath, 2013). Substance misuse also reduces the neural efficacy of the motor adaptation part of the brain, which is located in the PFC and basal ganglia (Gentili et al., 2015). According to Churchwell et al. (2010), BIS-non-planning predicted occasional cannabis use. They likewise found that a volume reduction in the medio-orbital PFC has been seen in individuals who misuse cannabis and those with higher scores on BIS-non-planning, indicating that both groups use the same parts of the brain.

Several studies show that negative urgency is a significant predictor of alcohol misuse, AUD (Adams et al., 2012; Fischer et al., 2007; Jones et al., 2014; Smith et al., 2007; Stautz & Cooper, 2013; Verdejo-García et al., 2007), nicotine misuse and other drug abuses (Gutman et al., 2011; Kaiser et al., 2012). Some investigations have proposed reasons for the

association between negative urgency and alcohol misuse. To illustrate, a connection between negative urgency and more inhibitory brain activity during negative emotion has been seen in those with alcohol misuse (Cyders et al., 2014b); moreover, the ventromedial PFC's activity can mediate the relationship between urgency and alcohol misuse during alcohol-related cues (Cyders et al., 2014a). Therefore, those with a higher negative urgency score have more impaired response inhibition during negative emotions, predicting their increased alcohol consumption. In addition, previous investigations have suggested that people with more favourable expectations about the consequences of alcohol or nicotine use in negative emotional situations use more of them (Anthenien et al., 2017; Gutman et al., 2011; Hu et al., 2008). There may be different reasons for the association between alcohol, cannabis and nicotine use and negative urgency, including the use of substances to cope with the negative emotions, or negative affect interfering with cognitive processes, leading to poor risk-evaluation (Shields et al., 2016).

People with higher levels of sensation seeking misuse more substances than those with lower sensation seeking levels (Martins et al., 2008). It has been suggested that, like other facets of impulsivity and EF, sensation seeking is controlled by the frontal regions of the brain (Joseph et al., 2009; Santesso et al., 2008), which could be attributed to higher levels of physiological reward responses due to immature cognitive control (located in the PFC) of substance misuse behaviours (Bardo et al., 1996; Kaynak et al., 2013).

The hierarchical regression analysis's second step revealed that the inattention symptom of ADHD predicted the use of alcohol, cannabis and nicotine over and above the facets of impulsivity in the sample. This score in the ADHD questionnaire (ASRS) indicated a significant improvement in the proportion of the explained variance in the use of alcohol,

cannabis and nicotine. This could denote that in people with higher ADHD symptoms, higher inattention scores increase the risk of alcohol, cannabis and nicotine use in ways other than impulsivity. Hence, those with higher inattention scores use more alcohol, cannabis and nicotine for reasons other than high BIS-motor, sensation seeking and negative urgency. Those with higher hyperactivity/impulsivity symptoms use more alcohol, cannabis and nicotine through higher BIS-motor, sensation seeking and negative urgency score pathways.

These two studies found a significant link (a) between the hyperactivity/impulsivity symptom of ADHD and various facets of impulsivity and (b) between the inattention symptom of ADHD and the facets of EF. This outcome provided insight into the role of impulsivity and EF in the relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine. Finding the best intervention for those with ADHD symptoms based on their EF or impulsivity facet impairments may help decrease their risk of substance misuse. After accounting for the facets of UPPS-P, ADHD symptoms could not explain the additional variance in AUQ and NIC scores; they only added to the variance of the scores of these two questionnaires over and above the scores of BIS-11. This result needs further investigation to explore the relationship between these two substance use questionnaires and UPPS-P. Ultimately, this thesis's second goal was achieved, and a novel and comprehensive contribution to the literature was made.

Previous studies have proposed an association between impulsivity and poor EF performance in individuals with ADHD symptoms and substance misuse, which could be linked to emotional dysregulation. Emotional impulsiveness is another term for impulsivity; it is described as behaving impulsively and rashly under the pressure of positive or negative emotions (Shapiro, 1965). An individual with these behaviours reacts to a stimulus based on

an immediate emotional reaction (desire or anger), regardless of the consequences (Wingrove & Bond, 1997). Past investigations suggest that emotional impulsiveness has been included in ADHD for a long time, dating from 1902 to 1976 (Barkley, 2010; Skirrow et al., 2009). The role of emotional regulation in the association between ADHD symptom clusters and the use of alcohol, cannabis and nicotine was the focus of Study 3.

Aim 3: To explore whether emotional regulation mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.

Past research has shown a significant association between emotional regulation-ADHD and emotional regulation-substance misuse. Study 3 sought to address the gap in the literature by examining the mediation relationship between ADHD, emotional regulation and substance misuse. The third study's main objective was to explore whether emotional regulation is a major mediator between ADHD symptoms and the use of alcohol, cannabis and nicotine. Thus, a four-step regression analysis was conducted to reach this goal. The results established that emotional regulation was not a full mediator of inattention and alcohol, cannabis, and nicotine use; however, it partly mediated the relationship between hyperactivity/impulsivity and alcohol and cannabis, but not nicotine use. This finding denoted that people with hyperactivity/impulsivity use alcohol and cannabis through emotional regulation. Being a partial mediator implies that, besides a significant relationship between emotional regulation-ADHD symptoms and emotional regulation-substance misuse, there is either a direct relationship between ADHD symptom clusters and substance misuse. There might also be other variables that mediate this relationship, as explored in Studies 4 and 5.

Several points may explain the relationship between hyperactivity/impulsivity and emotional dysregulation. For instance, Walcott and Landau (2010) demonstrated that greater response disinhibition is associated with poorer emotional regulation, revealing the connection between hyperactivity/impulsivity symptoms of ADHD and emotional dysregulation (Berlin & Bohlin, 2002). Furthermore, in previous studies, hyperactivity/impulsivity symptoms of ADHD were stronger predictors of emotional regulation problems and substance misuse. In one research, while both hyperactivity/impulsivity and inattention are predictors of depression, hyperactivity/impulsivity are stronger predictors of emotional regulation issues and depressive symptoms than inattention (Seymour et al., 2014). They are also significant indicators of emotional lability (rapid mood shifts) in individuals with ADHD symptoms (Skirrow & Asherson, 2013), which could be due to ADHD and impaired emotional regulation sharing the same parts of the brain, as presented in Section 4.7.

The results of Study 3 have revealed a novel association between emotional dysregulation as a risk factor and the development of substance misuse in those with hyperactivity/impulsivity. Separating the ADHD symptom clusters and investigating the unique relationships of inattention-emotional regulation-substance misuse and hyperactivity/impulsivity-emotional regulation-substance misuse made a novel contribution to the literature.

Aim 4: To determine whether bipolar disorder mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.

Findings from Chapter 5's cross-sectional research suggest that other variables mediate the relationship between ADHD symptoms and substance misuse due to emotional regulation being a partial mediator between hyperactivity/impulsivity and alcohol and cannabis use. Previous studies have shown that mood disorders are common to both ADHD and substance misuse (Chapter 5). It is worth noting that the prevalence of ADHD symptoms in those with bipolar disorder is higher (21.2%) than those with depression (9.4%) (Kessler, 2006). Furthermore, both ADHD and bipolar disorder have similar characteristic conditions and diagnostic criteria; their symptoms also overlap, making diagnosis more complicated (Kent & Craddock, 2003; Wingo & Ghaemi, 2007). The two conditions' similarities, such as increased energy, high distractibility, physical restlessness and impaired response inhibition, led some researchers to find statistically significant associations between bipolar disorder and ADHD but not depression (Kessler et al., 2010; Kim et al., 2019). Therefore, Study 4's main aim was to explore whether bipolar disorder, as a mood disorder, can mediate the relationship between each ADHD symptom and the use of alcohol, cannabis and nicotine. A four-step regression analysis was performed to realise this goal. Results showed that bipolar disorder partially mediates the relationship between inattention and alcohol, cannabis, and nicotine use. Therefore, the inattention symptom of ADHD can raise the risk of using alcohol, cannabis and nicotine due to a mood disorder. In their review, Camelo et al. (2013) discovered that poor attention is one of the most compromised functions in people with bipolar disorder. Attention impairments can cause changes in memory, visuospatial abilities and learning. Moreover, inattention is one of the main ADHD symptoms. Many studies show that patients with more inattention symptoms use more nicotine, which may affect such symptoms due to its positive effect on arousal and attention (Bilgi et al., 2017; Kalil et al., 2008; Levin & Rezvani, 2002; Warburton & Arnall, 1994). In addition, previous studies have

determined that inattention, rather than hyperactivity/impulsivity, is an important factor in alcohol-related problems in university students (Glass & Flory, 2012; Mesman, 2013).

There were also partial mediations between hyperactivity/impulsivity and alcohol and cannabis, but not nicotine use, via a bipolar disorder pathway. Partial mediation indicates that bipolar disorder accounts for some, but not all, of the relationship between inattention and substance misuse and between hyperactivity/impulsivity and the use of alcohol and cannabis. This finding could be explained by the results of other studies in this thesis, which point to different pathways, such as emotional regulation impairment.

The relationship between ADHD symptoms, bipolar disorder and substance misuse may be due to various factors. First, those with ADHD symptoms use different substances to cope with their symptoms and problems in everyday life. Substance misuse has been shown to decrease their negative affect and craving, suggesting that it could be related to coping motives (Buckner et al., 2015). Second, those with more ADHD symptoms and bipolar disorder may act more impulsively without considering the negative consequences of substance misuse. The association between response inhibition and hyperactivity/impulsivity may also explain the link between ADHD, bipolar disorder and substance misuse (Lee et al., 2020). Third, bipolar disorder and other psychiatric conditions, such as ADHD, have a high degree of genetic overlap, explaining why they develop the same problems, such as SUD (Joo et al., 2010; Maletic & Raison, 2014). Fourth, sections of the brain shared by bipolar disorder, ADHD and substance misuse that include different parts of PFC (please refer to Section 5.8) may describe their connections (Gruber et al., 2004; Phillips et al., 2008; Strakowski et al., 2012).

Aim 5: To discern whether sleep quality mediates the relationship between each ADHD symptom cluster and alcohol, cannabis and nicotine use separately.

There are risk factors that may increase the likelihood of substance misuse in individuals with ADHD symptoms. Based on the findings of Studies 3 and 4, other factors could mediate the relationship between ADHD and substance misuse. Poor sleep quality is typical in people with ADHD symptoms and those who misuse substances. In Study 5, ADHD symptoms were split into two clusters: inattention and hyperactivity/impulsivity, with each group having its relationship with sleep quality and substance misuse. The findings showed that poor sleep quality partially mediates the relationship between ADHD symptom clusters and alcohol, cannabis and nicotine use. This outcome suggests that sleep quality accounts for some of the connection between ADHD and the use of alcohol, cannabis and nicotine. It also implies that, in addition to the significant relationship between sleep quality-ADHD and sleep quality-substance misuse, there is a direct relationship between ADHD symptoms and the use of alcohol, cannabis and nicotine, as stated in Section 6.7.

Studies 3, 4 and 5 reveal possible variables that partially mediate the relationship between inattention, hyperactivity/impulsivity and substance misuse. Emotional dysregulation, bipolar disorder and poor sleep quality have shown an indirect effect of ADHD symptom clusters on substance misuse. Therefore, the third, fourth and fifth goals of this thesis was attained, and the results contributed to the field by showing the unique relationship between inattention and hyperactivity/impulsivity, each of the mediators and increased usage of alcohol, cannabis and nicotine. This is a novel and potentially important finding that adds to how each ADHD symptom cluster is linked to alcohol, cannabis and nicotine through an indirect mediating pathway.

These results offered valuable information about the deficits and problems in individuals with ADHD symptoms, the use of alcohol, cannabis and nicotine and the relationship between the variables. Appropriate interventions should be chosen for each person based on their impairment and substance misuse to reduce the likelihood of developing alcohol, cannabis and nicotine misuse, or SUD at a later stage. Every chapter of this thesis recommends such interventions.

Previous research has found that university students are at a higher risk of hazardous alcohol and other illegal drugs misuse (Bajwa et al., 2013; Drosican, 2009; Gupta et al., 2013; Maher, 2008; Maier et al., 2013; Mohammadpoorasl et al., 2014; Sommet et al., 2012; Suerken et al., 2014). For instance, Gill (2002) measured undergraduate drinking and discovered that 52% of men and 43% of women drank more than the prescribed weekly limits of 21 units for men and 14 units for women. However, the figures for the general population were 37% for 16-year-olds and 33% for 24-year-olds (Rickards et al., 2004). As mentioned in Chapter 1 (Section 1.2.2), Davoren et al. (2016) reported in their systematic review that about two-thirds of university students in the UK and Ireland scored 8 or above after completing the AUDIT. Moreover, Trevor et al. (2014) assessed the illegal substance misuse of 304 university students and 975 non-university students in the UK aged 20 to 22. They determined that 20.7% of students and 16.9% of non-university students used one or more illegal drugs in the last year. The most common drug amongst students was cannabis (19%). Other studies have evaluated the alcohol, cannabis and nicotine use in students at specific UK universities. For instance, Ralph-Nearman et al. (2020) examined the alcohol consumption of 183 students at Nottingham University in the UK and found that 47% were at-risk drinkers. Furthermore, 58% of 119 students from Leeds Metropolitan University in

the UK showed hazardous alcohol misuse (Craigs et al., 2011). Other research on substance misuse among university students has been presented in previous chapters.

Each study's percentage of risky alcohol use amongst participants was reported in every chapter of this thesis: 27% in the first, 28.4% in the second and 50% in the third. The fourth and fifth studies were combined, with 33% of participants yielding an AUDIT score of 8 or higher, indicating risky alcohol use. In addition, based on NHS Digital, cannabis use in 2018–2019 in England and Wales was 17% for people aged 16–24, the highest in a decade. The results of the current thesis determined that 6% of participants in the first study, 8% in the second study, 25.5% in the third study and 20% in the fourth and fifth studies scored eight or above on the CUDIT-R, implying hazardous cannabis use.

The first study's lower levels of alcohol and cannabis use could be due to a smaller sample size relative to the other studies in this thesis. Moreover, research shows that men report higher rates of hazardous alcohol and cannabis use than women (Beenstock et al., 2010; Faulkner et al., 2006; NHS, 2018). The participants in this thesis's studies were mainly women (75% to 81.5%), which may explain the lower level of alcohol and cannabis use in these studies than in NHS reports or previous investigations. Higher rates of alcohol and cannabis use in Studies 3, 4 and 5 could be due to the time of year they were performed. Time can affect participants' responses to questionnaires, leading to different percentages of participants with risky alcohol or cannabis use (Kim et al., 2017; Song et al., 2020). The last three studies were conducted immediately after Christmas and New Year. Participants were asked about their alcohol, cannabis and nicotine use in the previous month or last six months, which may have influenced their answers.

In the current studies, nicotine frequency was calculated by asking participants how many days they smoked cigarettes in the past 30 days. They had the option of choosing 0 day, 1 to 2 days, 3 to 5 days, 6 to 9 days, 10 to 19 days, 20 to 29 days and 30 days. The percentages of participants who smoked nicotine for 30 days of the previous month are as follows: 9.4%, 7.6%, 11.4%, 13.3% and 13.3% in the first, second, third, fourth and fifth studies, respectively. Respondents were also asked how many cigarettes they smoked per day in the previous month. They had several answers from which to choose: (a) did not smoke cigarettes during the past 30 days, (b) less than one cigarette per day, (c) one cigarette per day, (d) two to five cigarettes per day, (e) six to 10 cigarettes per day, (f) 11 to 20 cigarettes per day, and (g) more than 20 cigarettes per day. The percentages of participants who smoked more than 20 cigarettes per day are as follows: 1.4%, 0.6%, 0.9%, 0.4%, 0.4% in the first, second, third, fourth and fifth studies, respectively. Since the Nicotine Use Questionnaire only measured nicotine frequency and quantity over the previous month, future research should include measuring hazardous nicotine use and dependence.

7.2. Implications

The few investigations that focused on the link between ADHD symptoms and substance misuse have measured ADHD as one whole neurodevelopmental condition (Banducci et al., 2016; Bilinski et al., 2012; Crockett et al., 2006; Kendler et al., 2003; Keyzers et al., 2020; Merikangas et al., 2000; Olsen, 2011; Overholser et al., 1997; Ruffle, 2014; Volkow, 2010). Although previous research has shown that the manifestation of the symptoms of this condition varies by person, some investigations have revealed more

hyperactivity and impulsive behaviours. In contrast, others found more attention problems, which can have different impacts on people's personal and social lives (Reimherr et al., 2015). Additionally, the majority of previous investigations have tested clinical samples with diagnosed ADHD and SUD. Thus, the present thesis was motivated by the gap in the literature in terms of measuring different symptoms of ADHD and exploring the unique role of risk factors in the relationship between each ADHD symptom cluster in typically developing samples.

Although substance misuse is one of the most common comorbidities of adult ADHD, it has been excluded as a criterion in many studies with adults with ADHD symptoms (clinical and non-clinical). In this thesis' studies, the ADHD symptoms were divided into two categories (inattention and hyperactivity/impulsivity) to investigate the relationship of each symptom with alcohol, cannabis and nicotine use separately. The main focus of the current studies was the association between these two conditions to determine whether there were pathways linking each symptom category and the use of alcohol, cannabis and nicotine.

In the UK, studies with adults with ADHD and comorbid substance misuse, especially among university students, are scarce; this thesis is an attempt to provide more information about this demographic, which has higher rates of substance misuse, mental health problems and poorer quality of sleep than other groups (Bewick et al., 2010; Cooke et al., 2006; Foulkes et al., 2019; Mohammadpoorasl et al., 2014; Sommet et al., 2012; Suerken et al., 2014). Since none of the previous studies used a comprehensive assessment of shared variables in the relationship between ADHD symptoms and substance misuse, the current studies employed more rigorous and ecologically valid measures. This thesis provides data on the most significant shared variables in the relationship between ADHD symptoms and

substance misuse, making it a valuable resource for future research and treatment interventions.

The following sections summarise each study's achievement, elucidating their contributions to the field. First, the study on the role of EF in the relationship between ADHD and substance misuse advanced the field by introducing a new virtual reality task that can measure different facets of EF and has ecological validity, unlike other traditional EF tests. Regarding adult ADHD and the use of alcohol, cannabis and nicotine, this research was the first to measure EF through the JEF[®]. This test helped in assessing various aspects of participants' EF performance with a virtual task that was highly realistic.

The second study focused on the role of impulsivity in the use of alcohol, cannabis and nicotine in individuals with ADHD symptoms. Impulsivity has been shown to be a multi-faceted construct, with impulsive behaviour being a product of cognitive impairment and personality process dysfunctions. Even though previous investigations indicate that certain facets of impulsivity contribute to substance misuse in adults with ADHD, none used a comprehensive assessment of impulsivity to discern which facets of personality traits contribute to the relationship between ADHD symptoms and substance misuse. Therefore, in the second study, combining the UPPS-P and BIS-11 provided a more in-depth measurement of impulsivity as a personality trait. In addition, using the UPPS-P instead of the UPPS helped to measure positive urgency as a significant predictor of substance misuse in previous investigations, which have shown that a positive mood may increase risky behaviours (Yuen & Lee, 2003) and university students drink more during celebrations than they do during the academic days (Del Boca et al., 2004; Robinson et al., 2016). Although this study did not find a significant association between positive urgency and substance misuse, testing it as a

variable that could cause or be affected by different substance misuse could add to the current knowledge of ADHD symptoms and alcohol, cannabis and nicotine use. Various characteristics may lead to impulsive action; hence, this study attempted to identify the facets of impulsivity that increase the risk of substance misuse and determine whether ADHD symptoms can add to the variance in predicting alcohol, cannabis and nicotine use after accounting for the facets of impulsivity.

Past research has established that emotional regulation is impaired in people with ADHD symptoms and those with substance misuse. However, Study 3 of this thesis provided information on the mediating capacity of emotional regulation in the relationship between each ADHD symptom and the use of alcohol, cannabis and nicotine separately. This study discovered an indirect connection between hyperactivity/impulsivity and the use of alcohol and cannabis.

In Study 4, bipolar disorder did not mediate the relationship between hyperactivity/impulsivity and nicotine use, indicating that other variables may mediate this relationship. Thus, Study 5 explored this probability and discovered that sleep quality mediated the ADHD symptoms and alcohol, nicotine and cannabis use. Investigating the unique relationship between each symptom cluster and sleep quality and substance misuse revealed that treating sleep disturbances and improving sleep quality may decrease the likelihood of substance misuse by university students with inattention and hyperactivity/impulsivity symptoms. This study showed the unique indirect relationship between hyperactivity/impulsivity and nicotine use through poor sleep quality (Figure 7.1).

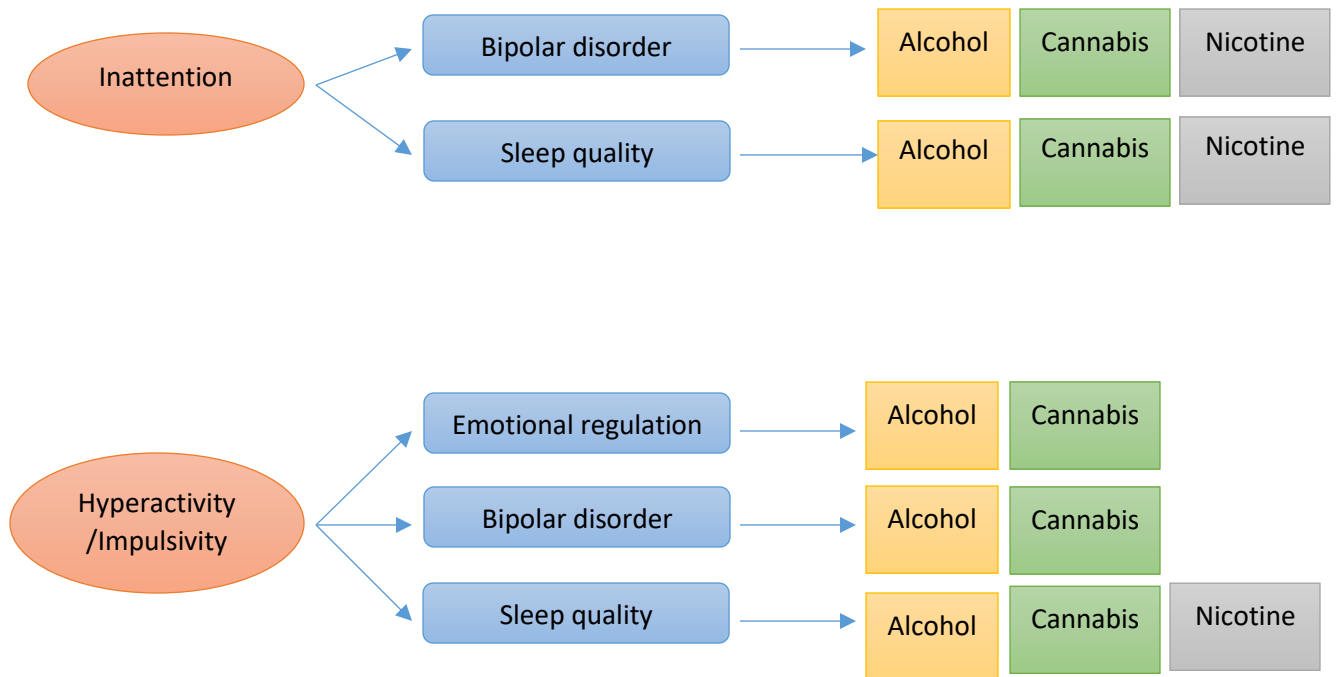


Figure 7.1: the mediating relationship between ADHD symptom clusters as independent variables, risk factors as the mediators and substance misuse as the dependent variables.

Studies 3, 4 and 5 have discovered a mediating relationship between the two ADHD symptom clusters, possible risk factors, such as emotional dysregulation, bipolar disorder and poor sleep quality, and the use of alcohol, cannabis and nicotine. By considering the unique relationship between the variables, useful interventions or treatment programmes based on each individual's impairment and problem could be developed.

7.3. Limitations

Each study's limitations have been underlined in the respective chapters. Still, some of the overall thesis's limitations will be discussed here, emphasising the samples and measures used.

Samples

The first limitation to note in all empirical studies reported in this thesis was the use of samples with a female bias. The over-representation of women is acknowledged as a cause of generalisation difficulties. Additionally, previous investigations and the NHS reports have revealed different levels of substance misuse in men and in women and testing women as the majority of a study's sample may affect the results of the percentage and frequency of substance misuse (Beenstock et al., 2010; Faulkner et al., 2006; NHS, 2018). Thus, future research is recommended to make greater efforts to use samples with more balanced gender ratios.

Second, there were issues about sample age range restriction. Even though the studies in this thesis investigated issues relevant to first-year undergraduates across all stages, older participants were included. The average age range was also higher than in previous studies. Studies 1 and 2 involved adults over the age of 18, but the remaining three studies tested first-year undergraduates. Thus, any interpretation of the findings presented must acknowledge the precise age range of the samples in each study, avoiding generalising findings to younger or older age groups.

Third, the studies in this thesis sampled university students, which may not represent the wider population (Henrich et al., 2010). University students are more likely to misuse substances or have poorer sleep quality than non-student peers (Slutske, 2005), indicating that the results of the studies involving only university students could not be generalised to the rest of society.

The small number of participants reporting cannabis use was also another limitation of the samples. Future adult substance misuse research may benefit from strategies that

oversample those with cannabis misuse or dependence. Additionally, the empirical studies samples were from a non-clinical population. In this thesis's studies, the ADHD symptom dimensions were measured rather than diagnostically analysed. While this approach aided in investigating the unique association between ADHD symptoms and various substance misuse scores, it is unclear whether the findings of a categorical diagnostic approach would be similar. The studies show that the more ADHD symptoms, the greater the likelihood of developing substance misuse and SUD. Therefore, substance misuse is more common in those diagnosed with ADHD. Moreover, there are limited studies on the role of different variables, such as EF facets, aspects of impulsivity and emotional regulation, mood disorders and sleep quality, in the relationship between each ADHD symptom cluster and substance misuse in adults diagnosed with ADHD. Consequently, in future studies, it is recommended to use clinical approaches since they may explain the nature of these relations at higher levels of symptomatology and substance misuse.

The online form of participant recruitment was the final limitation of the samples used. The majority of participants were recruited online through a platform comprised of university students interested in participating in research surveys in exchange for course credit. Although online recruitment is a convenient and quick way to recruit a large sample, it has some limitations, such as racial and ethnic differences in internet accessibility and use (Dutton & Blank, 2011; Lane et al., 2015).

Measure

This thesis's empirical studies relied on self-reported information. This approach is affordable and faster, with quantitative data that can be analysed. However, there are disadvantages to this method, such as response bias. In the last study for this thesis, sleep

quality was measured through a self-report questionnaire; however, investigations show that self-reported sleep quality is more biased than objective sleep measures. In a study by Jackson et al. (2018), all races overestimated their self-reported sleep duration. Furthermore, greater bias was seen in reports of the significant association between sleep duration and health.

Additionally, young individuals' self-reports of ADHD symptoms are less severe than parents' reports, resulting in lower rates of ADHD persistence into adulthood (Barkley et al., 2002; Du Rietz et al., 2016; Kooij et al., 2008; Pierrehumbert et al., 2006). This finding shows that when self-reports are used in follow-up investigations, the estimated persistence of adult ADHD is lower (Barkley et al., 2002; Wolraich et al., 2005). Moffitt et al. (2015) reported that the persistence rate of adult ADHD was 5% when self-reports are used, which is considerably lower than the persistence rates recorded in investigations that used both self- and parent-reports, which ranged from 15% to 35% (Biederman et al., 2010; Faraone et al., 2006). Furthermore, clinical ADHD investigations revealed a weaker association between self-reported ADHD symptoms, poor school performance in adolescents (Pierrehumbert et al., 2006) and important life events in young adulthood (Barkley et al., 2002) as compared to adult reports. Moreover, the heritability of adolescent and adult ADHD estimated through self-reports is lower (38% to 48%) than when measured by parent-reports (64% to 82%) and clinically diagnosed ADHD (88%).

Students with high levels of ADHD symptoms but had never been diagnosed with ADHD were included in the study samples for this thesis. Finding individuals at high risk of ADHD with a comprehensive clinical assessment is important. Using ADHD treatment for those diagnosed with ADHD might decrease the negative consequences of untreated ADHD later in life, including substance misuse and subsequent SUD (Arria et al., 2008; Fayyad et

al., 2007), antisocial behaviours (Biederman et al., 2006; Manuzza & Klein, 2000) and poor academic and occupational achievements (De Graaf et al., 2008; Manuzza & Klein, 2000; Weyandt & DuPaul, 2006). Therefore, a combination of different methods, such as diagnostic interviews and measurements, in addition to self-report questionnaires, may be useful in obtaining more detailed and realistic data from the sample.

These investigations were cross-sectional, demonstrating the correlation, connection and predictive capacity of each variable; however, future longitudinal studies are recommended and suggested to determine their cause-and-effect relationship. On the one hand, there are several advantages to using cross-sectional studies:

- Since follow-up sessions are not required, the testing process is more affordable.
- Good control over the testing process with no long-term consideration denotes a single testing session with considerations for that session.
- The completeness of the key data points is maximised.
- The investigator considers the whole sample at once by taking into account every local influence when the data is gathered.
- The data can be analysed by other researchers for different purposes, providing access to multiple results, exposures and variables simultaneously and a foundation for future investigations. This research type does not look at the reasons for a case but instead lays the underpinning for future studies.

On the other hand, there are some disadvantages. Cross-sectional studies need larger sample sizes for greater accuracy because studying a group of people at once requires minimising the risk of error due to chance and coincidence in the results. Bias can also influence results, and non-response can lead to a bias when the outcome is measured. Furthermore, cross-sectional studies do not provide causal relationships between the

variables. However, they do offer information about the correlation and reveal the relationship between the variables. Consequently, using longitudinal investigations are recommended for future investigations to augment the information provided by the studies in this thesis.

Conducting longitudinal studies with a more diverse sample or clinical investigations was not possible for a PhD investigation with limited time and budget. Therefore, for a post-doctoral study, longitudinal clinical studies with more diverse populations and various data-gathering methods, such as interviews and increased face-to-face activities, are the main priority. Moreover, these studies included a large number of tests. Thus, issues related to multiple testing require consideration in future investigations.

Furthermore, it should be considered that the studies described in this thesis did not use a nicotine test with a cutoff score to assess those with risky nicotine use or nicotine dependence. The analyses in this thesis measured lifetime and last month nicotine use. Thus, future research should focus on evaluating risky nicotine use or nicotine dependence.

As mentioned earlier, there have been few studies on the use of alcohol, cannabis and nicotine by UK university students, with diverse findings. Therefore, more investigations are required in this area to determine the prevalence of these three substances among typically developing UK university students. Moreover, although there are three types of ADHD, each with its own set of symptoms and problems in daily life, previous studies measured ADHD as one neurodevelopmental condition, focusing on those diagnosed with childhood ADHD. Additionally, there are university students who have not been diagnosed with ADHD but scored higher than the cutoff score on ADHD self-report tests, indicating a high probability of adult ADHD and a number of ADHD-related problems in their lives. Thus, further non-clinical research on the relationship between undiagnosed adult ADHD, with its different

subtypes and symptoms, and substance misuse amongst UK university students, is needed. In addition, previous literature on some of the variables used in this thesis's studies, such as the prevalence of bipolar disorder, different ADHD symptoms, different types of substance misuse and emotional regulation deficits in UK university students, was limited, necessitating further investigation. The limitations of each study in this thesis are pointed out in the relevant section of each chapter.

7.4. Future direction

This section will offer suggestions for future research based on the findings and limitations of this study. The ideas here will focus on broader research themes as potential directions in line with each study of this thesis.

The JEF[®] used in Study 1 to examine different facets of EF is considered a reliable and valid virtual reality task (Jansari et al., 2014). However, younger participants who were more familiar with computer-based technology performed better on this task. Thus, older adults are more likely to perform poorly on this task, which may or may not be due to impaired EF. As a result, prospective studies should consider the individual's age and capacity to use computerised tasks.

The UPPS-P, which was used to measure different facets of trait impulsivity in participants in the study presented in Chapter 3, was also a valid and reliable self-report measure (Smith et al., 2007). However, the causal relationship between the various aspects of impulsivity measured by this test and the development of substance misuse is not well established. It is unclear whether sensation seeking or negative urgency leads to increased substance misuse or whether misusing more alcohol, cannabis and nicotine leads to increased sensation seeking or higher levels of negative urgency. Some researchers have suggested that

impulsivity and EF facets and substance misuse have bidirectional relationships, indicating that one may be a consequence or determinant of the other (De Wit, 2009). In the future, researchers must examine the causal association between alcohol, cannabis and nicotine use and impulsivity and EF facets.

More studies are required to determine whether the impulsivity-related traits, impaired EF facets, emotional dysregulation, bipolar disorder and poor sleep quality linked to alcohol, cannabis and nicotine use are useful in designing intervention programmes to reduce the development of substance misuse and help those with substance dependence. Furthermore, it should be clarified whether the current substance dependence therapies, especially for those with ADHD symptoms, are effective in helping impulsive substance misusers quit misusing. The efficacy of interventions targeting the specific facet of EF or impulsivity-related traits most associated with substance misuse in those with inattention or hyperactivity/impulsivity symptoms, such as negative urgency and response inhibition, should also be examined to reduce the number of substance misusers.

Additionally, future studies should explore whether identifying effective coping strategies and medical and non-medical treatments for those with more hyperactivity/impulsivity symptoms in emotional situations will help them reduce their risk of developing alcohol and cannabis use. These treatment suggestions include the use of mindfulness to regulate one's emotions (Herwig et al., 2010) and third-wave CBT therapies, such as dialectical behaviour therapy (DBT) (Linehan, 1993), acceptance and commitment therapy (ACT) (Hayes et al., 2011), Mindfulness-based cognitive therapy (MBCT) (Segal et al., 2012) and Compassion-focused CBT (Gilbert 2009).

Study 4 revealed that bipolar disorder mediated the inattention symptom of ADHD and the use of alcohol, cannabis and nicotine. It also mediates hyperactivity/impulsivity and alcohol and cannabis use. Treating bipolar disorder in individuals with inattention or hyperactivity/impulsivity symptoms of ADHD may decrease their risk of substance misuse. Study 5 showed that poor sleep quality mediates the relationship between both ADHD symptom clusters and alcohol, cannabis and nicotine use. Thus, it should be established whether the information provided by Study 4 and 5 can aid future investigations and treatment providers in reducing the likelihood of alcohol, cannabis and nicotine use by decreasing attention problems in different ways, such as neurofeedback (Deilami et al., 2016), concentration practice and mindfulness (Britton et al., 2010; Semple, 2010), as well as whether controlling hyperactivity/impulsivity symptoms of ADHD could decrease the probability of developing alcohol and cannabis use.

The use of alcohol, cannabis and nicotine in young adults, especially university students, is a major cause of concern (Conner et al., 2018; Trevor et al., 2014). In the UK, no previous research has investigated whether inattention and hyperactivity/impulsivity could increase the likelihood of alcohol, cannabis and nicotine use in university student samples. Therefore, well-conducted and well-powered longitudinal studies are needed to discern the cause and effects of these issues.

Additionally, the patterns of alcohol, cannabis and nicotine use could affect the mode of action and the effect of the substance, which has been mentioned through this thesis. For instance, different ways of cannabis use may have different effects on the body and mind. The most common route of cannabis self-administration is the inhalation of smoked cannabis, which has immediate effects (Borodovsky et al., 2016; Knapp et al., 2018).

Furthermore, the type of alcohol and the pattern of use can affect sleep quality (Park et al., 2015; Pieters et al., 2010) or cognitive performance (Bernardin et al., 2014). Moreover, nicotine smokers develop regular smoking patterns, ensuring a steady release of dopamine (Markou, 2008; Prochaska, 2011; Ratschen et al., 2011). Thus, further investigations are required on the type of substance and the pattern of use and their bidirectional relationship with EF performance, impulsive traits, emotional regulation, mood disorders and sleep quality in those with ADHD symptoms.

7.5. Conclusion

This thesis has delineated the effects of certain risk factors on developing alcohol, cannabis and nicotine use, such as impaired EF facets, impulsivity facets, emotional dysregulation, bipolar disorder and poor sleep quality, consistently showing that response inhibition, adaptive thinking and time-based prospective memory are the aspects of EF associated with alcohol use. Response inhibition is the EF facet most associated with nicotine use, while those most connected to cannabis use are response inhibition, adaptive thinking and creative thinking. In addition, after accounting for EF facets, hyperactivity/impulsivity explains the unique variance in alcohol, cannabis and nicotine use, indicating that those with hyperactivity/impulsivity symptoms use more of these substances through pathways other than EF facets.

This paper also considered the effects of separable impulsivity-related personality traits on substance misuse, finding that motor impulsiveness, negative urgency and sensation seeking are linked to alcohol, cannabis and nicotine use in adults. It was also discovered that nonplanning predicted cannabis use in participants but not alcohol or nicotine use.

Furthermore, inattention added to the explained variance in alcohol, cannabis and nicotine

use in Study 2, implying that those with inattention symptoms consume more alcohol, cannabis and nicotine by paths other than inattention.

Therefore, Study 2 and 3 suggested the association between inattention and EF facets and between hyperactivity/impulsivity and the facets of impulsivity measured by UPPS-P and BIS-11. However, the facets of EF and impulsivity were not the only predictors of substance misuse in those with inattention and hyperactivity/impulsivity.

Finally, the remaining studies in this thesis analysed whether other risk factors mediate the relationship between each ADHD symptom cluster and the use of alcohol, cannabis and nicotine. There has been an indirect association between inattention and alcohol and cannabis use through emotional dysregulation. The partial mediating capacity of emotional regulation has also shown that other variables, such as bipolar disorder and poor sleep quality, could mediate the relationship between inattention and substance misuse. Moreover, the connection between hyperactivity/impulsivity and the use of alcohol, cannabis and nicotine was partially mediated by bipolar disorder and poor sleep quality. These findings demonstrate the involvement of different risk factors in the unique relations between each ADHD symptom cluster and substance misuse in adults. The results also revealed the necessity of including treatment programmes for emotional dysregulation, poor sleep quality and bipolar disorder in intervention campaigns for individuals based on their impairments and problems. It is hoped that these modest contributions to the literature can inform theoretical development of complex models of substance misuse risk and inspire further research into risk factor interplay.

To summarise, the work presented in this thesis aims to investigate the role of EF and impulsivity facets, emotional regulation, bipolar disorder as a mood disorder and sleep quality in the relationship between ADHD symptoms and the use of alcohol, nicotine and

cannabis. The results showed that certain facets of EF, such as adaptive thinking, time-based prospective memory and response inhibition, predict alcohol use, and the ADHD total score is a significant predictor of alcohol use, over and above the facets of EF. Response inhibition predicted nicotine use, and hyperactivity/impulsivity added to the variance of nicotine use after accounting for EF facets. The CUDIT-R score was significantly predicted by adaptive thinking and response inhibition, but ADHD symptoms could not predict it over and above the facets of EF. Moreover, the CAN score was predicted by creative thinking deficits, and hyperactivity/impulsivity explained a statistically significant variance in the CAN score after accounting for the EF facets.

In the second study, the AUDIT and AUQ scores were predicted using BIS-motor, negative urgency and sensation seeking, and the ADHD total score added to the variance in predicting the AUDIT score. The same facets of impulsivity significantly predicted nicotine use, and ASRS-IA explained a considerable amount of variance in nicotine use after accounting for BIS subscales. Additionally, the CUDIT-R score was significant predicted by BIS-motor, BIS-non-planning and negative urgency and sensation seeking. However, both ADHD symptoms explained the additional variance in the CUDIT-R score after accounting for impulsivity facets. BIS-motor was the only significant predictor of the CAN score, but ADHD symptoms did not predict additional variance in the CAN score over and above BIS-motor.

In the third study, emotional dysregulation was a partial mediator between ASRS-HI and alcohol and cannabis, but not nicotine use. Based on the fourth study, bipolar disorder is a partial mediator (a) between ASRS-IA and the use of alcohol, cannabis and nicotine and (b) between ASRS-HI and alcohol and cannabis use. Lastly, sleep quality was found to be a

significant partial mediator between ASRS-HI and ASRS-IA and all of the final study's substance misuse scores.

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Appendices

Appendix 1: Online consent form

CONSENT FORM

I confirm that I have read and understand information sheet. I have had the opportunity to consider information, ask questions and have had these answered with satisfaction.

I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being affected.

I understand that only the research team will have access to identifiable data.

I understand that the data will be stored on the computer or on paper and may contribute to scientific papers and presentations. I agree that the data can be made available anonymously to other researchers. These data will not be linked to me as an individual and my name will not be passed on to data is collected.

I confirm that I am over 18 years old and I agree to take part in the above study.

Agree

Disagree

Participant Information

You are being asked to take part in a study on how our decisions impact our daily behaviours in life. The main purpose of this study is to find out how having problems in this can explain the relation between ADHD symptoms and heightened rates of substance misuse. To take part in this study you DO NOT need to be a substance misuser nor have ADHD symptoms because we are going to look at the whole spectrum to see why the people with ADHD symptoms misuse a higher rate of substances.

Any major psychiatric disorder such as conduct disorder, bipolar affective disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder and major physical health problems such as brain injury are excluded.

This is the study of Zahra Safaryazdi, at Goldsmiths and under the supervision of Dr. Ashok Jansari and Dr. Andrew Cooper.

We will evaluate questionnaires and the possible relation among them.

In this study, you will be asked to complete some online questionnaires with multiple answers and you should choose the answer, which describes you the best.

These questionnaires have been used widely in previous investigations and they are highly unlikely to cause any distress. However, you can stop answering the questions and withdraw the study at any time without explanation.

All information that is collected from you during the course of the research will be kept strictly confidential. Any information about you that leaves the department will have your name and address removed so that you cannot be recognized from it. Only members of the research team will have access to the data. Your name, however, will not be disclosed outside of the study and you will be assigned a code number so that your data will be linked to this code rather than your actual name, to ensure confidentiality. Only members of the study team and regulatory authorities (who monitor the quality of the research) will have access to this identifiable data.

If you have any questions after reading this information sheet, you should ask the researcher before the study begins. After completing the questionnaires, you will be able to ask more questions about the study and your participation.

For any further information, please don't hesitate to contact:

Zahra Safaryazdi at zsafa001@gold.ac.uk

Dr. Ashok Jansari at a.jansari@gold.ac.uk

Dr. Andrew Cooper at a.cooper@gold.ac.uk

Debriefing of the study:

Attention Deficit Hyperactivity disorder (ADHD) is a neuropsychological disorder that starts in early childhood and the symptoms can continue into adulthood. Three main symptoms of child and adult ADHD are inattention, hyperactivity and impulsivity.

The prevalence of ADHD in people who misuse substances is high. In a review investigators argued that this prevalence is 23.3% to 31%. Individuals with ADHD misuse alcohol and other drugs, more than their healthy peers.

Previous investigations indicate that there are common endophenotypes between individuals with ADHD and substance misusers without ADHD such as cognitive and trait impairments that have been studied in previous investigations such as Emotional Regulation deficits. It might help to be better understood by taking an emotional regulation angle.

Impaired Emotion Regulation (ER) is common to both people with ADHD and substance misusers. Regulation of emotion is the ability to react to the emotional experiences in a way that is socially acceptable and appropriately flexible to permit unplanned reactions as well as the ability to delay impulsive reactions if needed (Cole, Michel & Teti, 1994).

Humans use various strategies such as repression and suppression to control their emotional experiences and expressions under distress (Gross, 2002). These control strategies demand psychological effort. During stress, pleasurable or immediate goals, a conflict may occur in various regulatory goals, which can endanger volitional behaviour that will lead to loss of impulse control (Tice, Bratslavsky & Baumeister 2001, Kuhl & Koole 1994).

The current investigation is an attempt to explore if there is an indirect relationship between ADHD symptoms and high rates of alcohol, cannabis and nicotine use from an emotional dysregulation way in a group of students using a dimensional assessment of symptoms.

It is anticipated that impaired emotional regulation can mediate the relationship between hyperactivity/impulsivity and inattention symptoms of ADHD and the use of alcohol, cannabis and nicotine.

For any further information, please don't hesitate to contact:

Zahra Safaryazdi at zsafa001@gold.ac.uk

Dr. Ashok Jansari at a.jansari@gold.ac.uk

Dr. Andrew Cooper at a.cooper@gold.ac.uk

Dr. Alice Jones at a.jones@gold.ac.uk

Thank you for your participation

Appendix 4: Alcohol Use Identification Test (AUDIT)

AUDIT questionnaire: screen for alcohol misuse

Please circle the answer that is correct for you

1. How often do you have a drink containing alcohol?

- Never
- Monthly or less
- 2,4 times a month
- 2,3 times a week
- 4 or more times a week

2. How many standard drinks containing alcohol do you have on a typical day when drinking?

- 1 or 2
- 3 or 4
- 5 or 6
- 7 to 9
- 10 or more

3. How often do you have six or more drinks on one occasion?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

4. During the past year, how often have you found that you were not able to stop drinking once you had started?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

5. During the past year, how often have you failed to do what was normally expected of you because of drinking?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

6. During the past year, how often have you needed a drink in the morning to get yourself going after a heavy drinking session?

2

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

7. During the past year, how often have you had a feeling of guilt or remorse after drinking?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

8. During the past year, have you been unable to remember what happened the night before because you had been drinking?

- Never
- Less than monthly
- Monthly
- Weekly
- Daily or almost daily

9. Have you or someone else been injured as a result of your drinking?

- No
- Yes, but not in the past year
- Yes, during the past year

10. Has a relative or friend, doctor or other health worker been concerned about your drinking or suggested you cut down?

- No
- Yes, but not in the past year
- Yes, during the past year

Appendix 5: Alcohol Use Questionnaire (AUQ)

The following questions ask you about your use of various types of alcoholic drinks.

Please consider your drinking for the last **six months** in answering the questions and take your time to give an accurate answer to each question.

1. On how many days per week do you drink wine, or any wine type product e.g. sherry, port martini (at least one small glass)? _____
Please state your usual brand(s) _____
2. On those days you do drink wine (or similar), about how many glasses (pub measure) do you drink? _____ If you are unsure, please estimate the number of bottles or parts of a bottle _____
3. How many glasses (pub measure) of wine do you have in a week in total?

4. On how many days per week do you drink beer or cider (at least half a pint)? Please state usual brand (e.g. Carlsberg, Stella Artois etc _____
5. On those days you do drink beer/ cider, about how many pints do you typically have?

6. How many pints of beer/cider do you drink in a week, in total? _____
7. On how many days per week do you drink spirits (Whisky, vodka, gin, rum etc-but not beer or wine)? _____ Please state usual brand (eg Smirnoff Blue Label _____
8. On those days do you drink spirits, about how many shots (pub measure) do you typically have? _____
If unsure, please estimate number of bottles or parts of a bottle _____
9. How many drinks of spirits do you have in a week, in total? _____
10. When you drink, how fast do you drink? (a drink is a glass of wine, a pint of beer or a shot of spirits, straight or mixed). Please circle the correct response.

Drinks per hour: 7+ 6 5 4 3 2 1
1 drink in 2 hours
1 drink in 3 or more hours
11. How many times have you been drunk in the last 6 months? By 'drunk' we mean loss of co-ordination, nausea, and/or inability to speak clearly. _____
12. What percentage of the times that you drink do you get drunk? _____

Appendix 6: Cannabis Use Identification Test (CUDIT-R)

The Cannabis Use Disorder Identification Test - Revised (CUDIT-R)

Have you used any cannabis over the past six months? YES / NO

If YES, please answer the following questions about your cannabis use. Circle the response that is most correct for you in relation to your cannabis use *over the past six months*

| | | | | | | |
|----|---|------------------|--|------------------------|------------------------------------|-----------------------------|
| 1. | How often do you use cannabis? | Never 0 | Monthly or less 1 | 2-4 times a month 2 | 2-3 times a week 3 | 4 or more times a week 4 |
| 2. | How many hours were you “stoned” on a typical day when you had been using cannabis? | Less than 1 0 | 1 or 2 1 | 3 or 4 2 | 5 or 6 3 | 7 or more 4 |
| 3. | How often during the past 6 months did you find that you were not able to stop using cannabis once you had started? | Never 0 | Less than monthly 1 | Monthly 2 | Weekly 3 | Daily or almost daily 4 |
| 4. | How often during the past 6 months did you fail to do what was normally expected from you because of using cannabis? | Never 0 | Less than monthly 1 | Monthly 2 | Weekly 3 | Daily or almost daily 4 |
| 5. | How often in the past 6 months have you devoted a great deal of your time to getting, using, or recovering from cannabis? | Never 0 | Less than monthly 1 | Monthly 2 | Weekly 3 | Daily or almost daily 4 |
| 6. | How often in the past 6 months have you had a problem with your memory or concentration after using cannabis? | Never 0 | Less than monthly 1 | Monthly 2 | Weekly 3 | Daily or almost daily 4 |
| 7. | How often do you use cannabis in situations that could be physically hazardous, such as driving, operating machinery, or caring for children: | Never 0 | Less than monthly 1 | Monthly 2 | Weekly 3 | Daily or almost daily 4 |
| 8. | Have you ever thought about cutting down, or stopping, your use of cannabis? | Never 0 | Yes, but not in the past 6 months 2 | | Yes, during the past 6 months 4 | |

Appendix 7: Cannabis Use Questionnaire

Cannabis Use Questionnaire

Cannabis or Marijuana is also referred to as: pot, grass, dagga, weed, mull, hash, cones, reefer, ganja, mary jane (MJ) and wacky baccy. Cannabis is usually smoked via a pipe, bong (water-filtered pipe) or in a cannabis cigarette (joint or spliff), or baked into food and eaten or made into a tea and drunk. When answering the following questions regarding cannabis use, please consider any of the above behaviours applicable.

Age: _____ years old

Gender: Male Female

1) If you have ever used cannabis before, at what age did you first use it? (If you have never used cannabis please go to question seven)

_____ years old

2) On how many separate days in the last month have you used cannabis?

3) Approximately how many times in the last month did you use cannabis until you were intoxicated or ‘stoned’?

4) In the last year, on how many separate days have you used cannabis?

| | | |
|----------------------|--------------------------|----------------------|
| Never | Once a week | 2 or 3 times a week |
| 4 to 5 times a week | Daily or nearly daily | Once a month |
| 2 to 3 times a month | Once every 2 to 3 months | Once every 12 months |

5) How would you describe your current cannabis use?

| | | |
|--------------|---------------------|-----------------------|
| Non-User | Heavy User | Previous light User |
| Light User | Previous heavy User | Previous average User |
| Average User | | |

6) In the last month, on approximately how many days have you consumed alcohol?

7) In the last month on approximately how many occasions have you used other illegal drugs?

Please Specify Drugs Used:

8) Have you ever received treatment for depression?

Yes No

9) Have you ever received treatment for any anxiety disorders?

Yes No

Smoking questionnaire

National Youth Tobacco Survey & Global Youth Tobacco Survey

1. Have you ever tried cigarette smoking, even one or two puffs?
 - a. Yes
 - b. No

2. How old were you when you smoked a whole cigarette for the first time?
 - a. I have never smoked a whole cigarette
 - b. 8 years old or younger
 - c. 9
 - d. 10
 - e. 11
 - f. 12
 - g. 13
 - h. 14
 - i. 15
 - j. 16
 - k. 17 years or older

3. About how many cigarettes have you smoked in your entire life?
 - a. None
 - b. One or more puffs, but never a whole cigarette
 - c. 1 cigarette
 - d. 2 to 5 cigarettes
 - e. 6 to 15 cigarettes (about ½ a pack)
 - f. 16 to 25 cigarettes (about 1 pack)
 - g. 26 to 99 cigarettes (more than 1 pack, but less than 5 packs)
 - h. 100 or more cigarettes (5 or more packs)

4. Have you ever smoked cigarettes daily, that is, at least one cigarette every day for 30 days?
 - a. Yes
 - b. No

5. During the past 30 days, on how many days did you smoke cigarettes?
 - a. 0 days
 - b. 1 to 2 days
 - c. 3 to 5 days
 - d. 6 to 9 days
 - e. 10 to 19 days
 - f. 20 to 29 days
 - g. All 30 days

6. During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?
 - a. I did not smoke cigarettes during the past 30 days
 - b. Less than 1 cigarette per day
 - c. 1 cigarette per day
 - d. 2 to 5 cigarettes per day
 - e. 6 to 10 cigarettes per day
 - f. 11 to 20 cigarettes per day
 - g. More than 20 cigarettes per day

7. When was the last time you smoked a cigarette, even one or two puffs?
 - a. I have never smoked even one or two puffs
 - b. Earlier today
 - c. Not today, but sometime in the past 7 days
 - d. Not during the past 7 days, but sometime during the past 30 days
 - e. Not during the past 30 days, but sometime during the past 6 months
 - f. Not during the past 6 months, but sometime during the last year
 - g. 1 to 4 years ago
 - h. 5 or more years ago

8. Where do you smoke cigarettes? (choose one or more answers)
 - a. I do not smoke now
 - b. At home
 - c. At school
 - d. At work
 - e. In the car
 - f. At a friend's house
 - g. At sports events, parties, clubs or other social events
 - h. Outdoors (pavement, parks, other places)

9. During the past 30 days, how did you usually get your own cigarettes? (choose only one answer)
 - a. I did not smoke cigarettes during the past 30 days
 - b. I bought them in a shop such as a convenience store, supermarket or petrol station
 - c. I bought them from a vending machine
 - d. I gave someone else money to buy them for me
 - e. I borrowed them from someone else
 - f. I stole them
 - g. A person 18 years old or older gave them to me
 - h. I got them another way

10. When you bought or tried to buy cigarettes in a shop during the past 30 days, were you ever asked to show proof of age?
 - a. I did not try to buy cigarettes in a shop during the past 30 days
 - b. Yes, I was asked to show proof of age
 - c. No, I was not asked to show proof of age

11. During the past 30 days, did anyone ever refuse to sell you cigarettes because of your age?
 - a. I did not try to buy cigarettes in a shop during the past 30 days
 - b. Yes, someone refused to sell me cigarettes because of my age
 - c. No, no one refused to sell me cigarettes because of my age

12. During the past 7 days, on how many days were you in the same room with someone who was smoking cigarettes?
 - a. 0 days
 - b. 1 or 2 days
 - c. 3 or 4 days
 - d. 5 or 6 days
 - e. 7 days

13. During the past 7 days, on how many days have people smoked in your presence, in places other than your home?
 - a. 0 days
 - b. 1 to 2 days
 - c. 3 to 4 days
 - d. 5 to 6 days
 - e. 7 days

14. Does someone who lives with you smoke cigarettes?
 - a. Yes
 - b. No

15. Which statement best describes the rules about smoking inside your home?
 - a. Smoking is not allowed anywhere inside my home
 - b. Smoking is allowed in some places or at some times
 - c. Smoking is allowed anywhere in my home
 - d. There are no rules about smoking in my home

16. Do your parents smoke?
 - a. None
 - b. Both
 - c. Father only
 - d. Mother only
 - e. Don't know

17. How many of your close friends, smoke cigarettes?
 - a. None
 - b. 1 to 2 close friends
 - c. 3 to 4 close friends
 - d. 5 to 6 close friends
 - e. 7 or more close friends

Appendix 9: ADHD Self-Report Scale (ASRS)

| Adult Self-Report Scale (ASRS) Symptom Checklist | | | | | | |
|--|--------------|--------|-----------|-------|------------|-------|
| Patient Name | Today's Date | | | | | |
| <p>Please answer the questions below, rating yourself on each of the criteria shown using the scale on the right side of the page. As you answer each question, circle the correct number that best describes how you have felt and conducted yourself over the past 6 months. Please give this completed checklist to your healthcare professional to discuss during today's appointment.</p> | | | | | | |
| | Never | Rarely | Sometimes | Often | Very Often | Score |
| 1. How often do you make careless mistakes when you have to work on a boring or difficult project? | 0 | 1 | 2 | 3 | 4 | |
| 2. How often do you have difficulty keeping your attention when you are doing boring or repetitive work? | 0 | 1 | 2 | 3 | 4 | |
| 3. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly? | 0 | 1 | 2 | 3 | 4 | |
| 4. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done? | 0 | 1 | 2 | 3 | 4 | |
| 5. How often do you have difficulty getting things in order when you have to do a task that requires organization? | 0 | 1 | 2 | 3 | 4 | |
| 6. When you have a task that requires a lot of thought, how often do you avoid or delay getting started? | 0 | 1 | 2 | 3 | 4 | |
| 7. How often do you misplace or have difficulty finding things at home or at work? | 0 | 1 | 2 | 3 | 4 | |
| 8. How often are you distracted by activity or noise around you? | 0 | 1 | 2 | 3 | 4 | |
| 9. How often do you have problems remembering appointments or obligations? | 0 | 1 | 2 | 3 | 4 | |
| Part A – Total | | | | | | |
| 10. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time? | 0 | 1 | 2 | 3 | 4 | |
| 11. How often do you leave your seat in meetings or other situations in which you are expected to remain seated? | 0 | 1 | 2 | 3 | 4 | |
| 12. How often do you feel restless or fidgety? | 0 | 1 | 2 | 3 | 4 | |
| 13. How often do you have difficulty unwinding and relaxing when you have time to yourself? | 0 | 1 | 2 | 3 | 4 | |
| 14. How often do you feel overly active and compelled to do things, like you were driven by a motor? | 0 | 1 | 2 | 3 | 4 | |
| 15. How often do you find yourself talking too much when you are in social situations? | 0 | 1 | 2 | 3 | 4 | |
| 16. When you're in a conversation, how often do you find yourself finishing the sentences of the people you are talking to, before they can finish them themselves? | 0 | 1 | 2 | 3 | 4 | |
| 17. How often do you have difficulty waiting your turn in situations when turn taking is required? | 0 | 1 | 2 | 3 | 4 | |
| 18. How often do you interrupt others when they are busy? | 0 | 1 | 2 | 3 | 4 | |
| Part B – Total | | | | | | |

Appendix10: Barratt Impulsiveness Scale (BIS-11)

| | Rarely/Never | Occasionally | Often | Almost Always/Always |
|----|--|--------------|-------|----------------------|
| 1 | I plan tasks carefully. | | | ① ② ③ ④ |
| 2 | I do things without thinking. | | | ① ② ③ ④ |
| 3 | I make-up my mind quickly. | | | ① ② ③ ④ |
| 4 | I am happy-go-lucky. | | | ① ② ③ ④ |
| 5 | I don't "pay attention." | | | ① ② ③ ④ |
| 6 | I have "racing" thoughts. | | | ① ② ③ ④ |
| 7 | I plan trips well ahead of time. | | | ① ② ③ ④ |
| 8 | I am self controlled. | | | ① ② ③ ④ |
| 9 | I concentrate easily. | | | ① ② ③ ④ |
| 10 | I save regularly. | | | ① ② ③ ④ |
| 11 | I "squirm" at plays or lectures. | | | ① ② ③ ④ |
| 12 | I am a careful thinker. | | | ① ② ③ ④ |
| 13 | I plan for job security. | | | ① ② ③ ④ |
| 14 | I say things without thinking. | | | ① ② ③ ④ |
| 15 | I like to think about complex problems. | | | ① ② ③ ④ |
| 16 | I change jobs. | | | ① ② ③ ④ |
| 17 | I act "on impulse." | | | ① ② ③ ④ |
| 18 | I get easily bored when solving thought problems. | | | ① ② ③ ④ |
| 19 | I act on the spur of the moment. | | | ① ② ③ ④ |
| 20 | I am a steady thinker. | | | ① ② ③ ④ |
| 21 | I change residences. | | | ① ② ③ ④ |
| 22 | I buy things on impulse. | | | ① ② ③ ④ |
| 23 | I can only think about one thing at a time. | | | ① ② ③ ④ |
| 24 | I change hobbies. | | | ① ② ③ ④ |
| 25 | I spend or charge more than I earn. | | | ① ② ③ ④ |
| 26 | I often have extraneous thoughts when thinking. | | | ① ② ③ ④ |
| 27 | I am more interested in the present than the future. | | | ① ② ③ ④ |
| 28 | I am restless at the theater or lectures. | | | ① ② ③ ④ |
| 29 | I like puzzles. | | | ① ② ③ ④ |
| 30 | I am future oriented. | | | ① ② ③ ④ |

Appendix 11: Mood Disorder Questionnaire

| Instructions: Please answer each question to the best of your ability. | | YES | NO |
|--|-----------------------|-----------------------|-----------|
| 1. Has there ever been a period of time when you were not your usual self and... | | | |
| ...you felt so good or so hyper that other people thought you were not your normal self or you were so hyper that you got into trouble? | <input type="radio"/> | <input type="radio"/> | |
| ...you were so irritable that you shouted at people or started fights or arguments? | <input type="radio"/> | <input type="radio"/> | |
| ...you felt much more self-confident than usual? | <input type="radio"/> | <input type="radio"/> | |
| ...you got much less sleep than usual and found you didn't really miss it? | <input type="radio"/> | <input type="radio"/> | |
| ...you were much more talkative or spoke much faster than usual? | <input type="radio"/> | <input type="radio"/> | |
| ...thoughts raced through your head or you couldn't slow your mind down? | <input type="radio"/> | <input type="radio"/> | |
| ...you were so easily distracted by things around you that you had trouble concentrating or staying on track? | <input type="radio"/> | <input type="radio"/> | |
| ...you had much more energy than usual? | <input type="radio"/> | <input type="radio"/> | |
| ...you were much more active or did many more things than usual? | <input type="radio"/> | <input type="radio"/> | |
| ...you were much more social or outgoing than usual, for example, you telephoned friends in the middle of the night? | <input type="radio"/> | <input type="radio"/> | |
| ...you were much more interested in sex than usual? | <input type="radio"/> | <input type="radio"/> | |
| ...you did things that were unusual for you or that other people might have thought were excessive, foolish, or risky? | <input type="radio"/> | <input type="radio"/> | |
| ...spending money got you or your family into trouble? | <input type="radio"/> | <input type="radio"/> | |
| 2. If you checked YES to more than one of the above, have several of these ever happened during the same period of time? | <input type="radio"/> | <input type="radio"/> | |
| 3. How much of a problem did any of these cause you – like being unable to work; having family, money or legal troubles; getting into arguments or fights? <i>Please circle one response only.</i> | | | |
| No Problem Minor Problem Moderate Problem Serious Problem | | | |
| 4. Have any of your blood relatives (i.e. children, siblings, parents, grandparents, aunts, uncles) had manic-depressive illness or bipolar disorder? | <input type="radio"/> | <input type="radio"/> | |
| 5. Has a health professional ever told you that you have manic-depressive illness or bipolar disorder? | <input type="radio"/> | <input type="radio"/> | |

Appendix 12: Study 1 Ethics Approval Form

EAF2: Ethical Approval Form for Goldsmiths Students

**DEPARTMENT OF PSYCHOLOGY
GOLDSMITHS, UNIVERSITY OF LONDON**

Tick one box:

2nd Year UNDERGRADUATE project (GROUP)

2nd Year UNDERGRADUATE project (MINI)

3rd Year UNDERGRADUATE project (FINAL)

POSTGRADUATE project - CCN, FCPHS, MMB, RMIP, SPGE, **MPhil/PHD** (please circle)

Title of project
Executive function in non-clinical ADHD individuals with and without substance use

Name of student(s)
Zahra Safaryazdi _____ Email _____ zsafa001@gold.ac.uk _____

Name of student(s) Add additional names for group projects
_____ Email _____

Name of supervisor(s)
Dr. Ashok Jansari
Dr. Andrew Cooper _____

Date _____

BEFORE FILLING IN THIS FORM, PLEASE READ THE BPS Code and Practice Document AND ALL RELEVANT ETHICAL GUIDELINES DOCUMENTS PROVIDED ON THE PSYCHOLOGY DEPARTMENT ETHICS COMMITTEE WEBPAGE: <https://learn.gold.ac.uk/course/view.php?id=288>

IT IS RESPONSIBILITY OF ALL PSYCHOLOGY STUDENTS TO BE FAMILIAR WITH AND FOLLOW ESTABLISHED ETHICAL GUIDELINES AND PROCEDURES.

| | | YES | NO | N/A |
|---|---|-----|----|-----|
| 1 | Will you describe the main experimental procedures to participants in advance, so that they are informed about what to expect? | X | | |
| 2 | Will you make it clear to participants that this is a student project? | X | | |
| 3 | Will you tell participants that their participation is voluntary? | X | | |
| 4 | Will you obtain written consent for participation? | X | | |
| 5 | If the research is observational, will you ask participants for their consent to being observed? | | | X |
| 6 | Will you tell participants that they may withdraw from the research at any time and for any reason? | X | | |
| 7 | With questionnaires, will you give participants the option of omitting questions they do not want to answer? | X | | |
| 8 | Will you tell participants that their data will be treated with full confidentiality and that, if published, it will not be identifiable as theirs? | X | | |
| 9 | Will you debrief participants at the end of their participation (i.e. give them a brief explanation of the study)? | X | | |

| | | | | |
|----|---|---|---|---|
| 10 | Will your project involve deliberately misleading participants in any way? | | X | |
| 11 | Are you asking any questions of sensitive or potentially upsetting content? | | X | |
| 12 | Is there any realistic risk of any participants experiencing either physical or psychological distress or discomfort? If Yes, give details on a separate sheet and state what you will tell participants to do if they should experience any problems (e.g. who they can contact for help). | | X | |
| 13 | Does your project involve work with animals? | | X | |
| 14 | Do participants fall into any of the following special groups? If they do, please refer to BPS guidelines and other relevant documents. Note that you may also need to obtain satisfactory CRB clearance (or equivalent for overseas students). | Children (under 18 years of age) | | X |
| | | People with learning or communication difficulties | | X |
| | | Patients | | X |
| | | People in custody | | X |
| | | People engaged in illegal activities (e.g. drug-taking) | X | |

If you have ticked **Yes** to any of the questions Q10 to Q14 (10,11,12,13,14) you should normally tick **box B** overleaf; if not (e.g., in the case of secondary data analysis), please give a full explanation on a separate sheet.

On-line Data collection

If you are planning collecting data on-line, please read the document [Conducting research on the internet: guidelines for ethical practice in psychological research online \(PDF\)](#).

Are you collecting data on-line? YES X NO

If you answered **YES**, please provide the following information:

1. Provide the link to the planned on-line survey/test and any log-in information.
2. For questions 1 to 9 above, describe how you will meet these criteria on-line. For example, for question 4, instead of written consent, participants must confirm that they are 18 or over and tick on-line to agree to participate, or agree to participate by proceeding to the next page; for question 7, there should be an option of skipping individual questions.

The study is in two parts. Participants will be recruited from the Research Participation Scheme at the Department of Psychology at Goldsmiths, University of London. They will be asked to complete 6 questionnaires online and based on their scores they will be asked to come and take the computerised tasks. The information about the study and the link of it will be on the Research Participation Scheme page with the researcher's email address. Participants can email the researcher and ask any questions about the study. More detailed information is on the first page of the online survey.

There is an online consent form that participants confirm that they are 18 or over and they agree to take part in the study and they are happy to be contacted for the second part of the study otherwise, they can't go through the questionnaires.

The questionnaires are designed in a way that participants can skip a question that they do not want to answer.

This is the link of the online study:

https://goldpsych.eu.qualtrics.com/SE/?SID=SV_eJQbpGS2UHZK0ux

ALL APPLICANTS: PLEASE TICK EITHER BOX A OR BOX B BELOW AND PROVIDE THE DETAILS REQUIRED IN SUPPORT OF YOUR APPLICATION. THEN SIGN THE FORM.

A. I consider that this project has no significant ethical implications to be brought before the Departmental Ethics Committee. PLEASE TICK:

Give a brief description of participants and procedure (methods, assessments used etc) in up to 150 words.

This form (without any attachments) should be submitted in hard copy to the Chair of the Department Ethics Committee, Dr Yulia Kovas, via her pigeon-hole, AND electronically to Val West (v.west@gold.ac.uk).

B. I consider that this project may have ethical implications that should be brought before the Departmental Ethics Committee, and/or it will be carried out with children or other vulnerable populations. PLEASE TICK:

Please provide all the information listed below in a separate attachment.

1. Title of project.
 2. Purpose of project and its academic rationale.
 3. Brief description of methods and measurements.
 4. Participants: recruitment methods, number, age, gender, exclusion/inclusion criteria.
 5. Consent and participant information arrangements, debriefing.
- Please attach intended information and consent forms, and any debriefing information.**
6. A clear but concise statement of the ethical considerations raised by the project and how you intend to deal with them.
 7. Estimated start date and duration of project.

*This form with attachments should be submitted electronically to the Departmental Ethics Committee (v.west@gold.ac.uk) for consideration. A hard copy (without attachments) should be placed in the pigeon-hole of the Chair of the Department Ethics Committee, Dr Yulia Kovas.
If any of the above information is missing, your application will be returned to you.*


Supervisors:

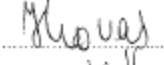
There is an obligation on the supervisor to bring to the attention of the Departmental Ethics Committee any issues with ethical implications not clearly covered by the above checklist. By signing this form, supervisors confirm that they have seen ALL PLANNED ASSESSMENT INSTRUMENTS, INCLUDING THOSE ADMINISTERED ON-LINE (BY FOLLOWING THE ON-LINE LINKS).

Students and Supervisors:

We are familiar with the BPS Guidelines for ethical practices in psychological research, and have discussed them.

Signed  Date 5/5/2016
Name Zahra Safaryazdi
(Student)

Signed  Date 27/4/16
Name Dr. Ashok Jansari
(Supervisor)

Signed  Date 20.05.16
Name Dr. Andrew Cooper
(Supervisor)

Chair, Ethics Committee

Appendix 13: Study 2 Ethics Approval Form

26/1/16

EAF2: Ethical Approval Form for Goldsmiths Students

DEPARTMENT OF PSYCHOLOGY
GOLDSMITHS, UNIVERSITY OF LONDON

Tick one box:

2nd Year UNDERGRADUATE project (GROUP)

2nd Year UNDERGRADUATE project (MINI)

3rd Year UNDERGRADUATE project (FINAL)

POSTGRADUATE project - CCN, FCPHS, MMB, RMIP, SPGE, MPhil/PhD (please circle)

Title of project
Impulsivity in non-clinical ADHD individuals with and without substance use

Name of student(s)
Zahra Safaryazdi Email zsafa001@gold.ac.uk

Name of student(s) Add additional names for group projects
Email

Name of supervisor(s)
Dr. Ashok Jansari
Dr. Andrew Cooper

Date 11/1/2016

BEFORE FILLING IN THIS FORM, PLEASE READ THE BPS Code and Practice Document AND ALL RELEVANT ETHICAL GUIDELINES DOCUMENTS PROVIDED ON THE PSYCHOLOGY DEPARTMENT ETHICS COMMITTEE WEBSITE: <https://learn.gold.ac.uk/course/view.php?id=288>.

IT IS RESPONSIBILITY OF ALL PSYCHOLOGY STUDENTS TO BE FAMILIAR WITH AND FOLLOW ESTABLISHED ETHICAL GUIDELINES AND PROCEDURES.

| | | YES | NO | N/A |
|----|---|-----|----|-----|
| 1 | Will you describe the main experimental procedures to participants in advance, so that they are informed about what to expect? | X | | |
| 2 | Will you make it clear to participants that this is a student project? | X | | |
| 3 | Will you tell participants that their participation is voluntary? | X | | |
| 4 | Will you obtain written consent for participation? | X | | |
| 5 | If the research is observational, will you ask participants for their consent to being observed? | | | X |
| 6 | Will you tell participants that they may withdraw from the research at any time and for any reason? | X | | |
| 7 | With questionnaires, will you give participants the option of omitting questions they do not want to answer? | X | | |
| 8 | Will you tell participants that their data will be treated with full confidentiality and that, if published, it will not be identifiable as theirs? | X | | |
| 9 | Will you debrief participants at the end of their participation (i.e. give them a brief explanation of the study)? | X | | |
| 10 | Will your project involve deliberately misleading participants in any way? | | X | |

| | | | | |
|----|---|---|---|--|
| 11 | Are you asking any questions of sensitive or potentially upsetting content? | | X | |
| 12 | Is there any realistic risk of any participants experiencing either physical or psychological distress or discomfort? If Yes, give details on a separate sheet and state what you will tell participants to do if they should experience any problems (e.g. who they can contact for help). | | X | |
| 13 | Does your project involve work with animals? | | X | |
| 14 | Do participants fall into any of the following special groups? If they do, please refer to BPS guidelines and other relevant documents. Note that you may also need to obtain satisfactory CRB clearance (or equivalent for overseas students). | Children (under 18 years of age) | X | |
| | | People with learning or communication difficulties | X | |
| | | Patients | X | |
| | | People in custody | X | |
| | | People engaged in illegal activities (e.g. drug-taking) | X | |

If you have ticked Yes to any of the questions Q10 to Q14 (10,11,12,13,14) you should normally tick box B overleaf; if not (e.g., in the case of secondary data analysis), please give a full explanation on a separate sheet.

On-line Data collection

If you are planning collecting data on-line, please read the document **Conducting research on the internet: guidelines for ethical practice in psychological research online (PDF)**.

Are you collecting data on-line? YES X NO

If you answered YES, please provide the following information:

1. Provide the link to the planned on-line survey/test and any log-in information.
2. For questions 1 to 9 above, describe how you will meet these criteria on-line. For example, for question 4, instead of written consent, participants must confirm that they are 18 or over and tick on-line to agree to participate, or agree to participate by proceeding to the next page; for question 7, there should be an option of skipping individual questions.

Participants will be recruited from the Research Participation Scheme at the Department of Psychology at Goldsmiths, University of London. The information about the study and the link of it will be on the Research Participation Scheme page with researcher's email address. Participants can email the researcher and ask any question about the study. More detailed information is on the first page of the online survey.

There is an online consent form that participants confirm that they are 18 or over and they agree to take part in the study otherwise, they can't go through the questionnaires.

The questionnaires are designed in a way that participants can skip a question that they do not want to answer.

This is the link of the online study:

https://goldpsych.eu.qualtrics.com/jfe2/preview/SV_cutChGi3sb3Qx0N

ALL APPLICANTS: PLEASE TICK EITHER BOX A OR BOX B BELOW AND PROVIDE THE DETAILS REQUIRED IN SUPPORT OF YOUR APPLICATION. THEN SIGN THE FORM.

| | | |
|---|--------------|---|
| A. I consider that this project has no significant ethical implications to be brought before the Departmental Ethics Committee. | PLEASE TICK: | X |
|---|--------------|---|

Give a brief description of participants and procedure (methods, assessments used etc) in up to 150 words.

This study will examine the relation among ADHD symptoms (attention, hyperactivity/impulsivity), substance use rates (alcohol, nicotine and cannabis) and different facets of impulsivity in a group of university students aged above 18 years old. There is one self report ADHD symptoms questionnaire (ASRS), two questionnaires that measure different facets of impulsivity (UPPS and BIS-11) and substance use questionnaires (two measures for alcohol consumption, two measures for cannabis use and a smoking questionnaire). These questionnaires are online and the link would be emailed to the participants.

This form (without any attachments) should be submitted in hard copy to the Chair of the Department Ethics Committee, Dr Yulia Kovas, via her pigeon-hole, AND electronically to Val West (v.west@gold.ac.uk).

B. I consider that this project **may** have ethical implications that should be brought before the Departmental Ethics Committee, and/or it will be carried out with children or other vulnerable populations. PLEASE TICK:

Please provide all the information listed below in a separate attachment.

1. Title of project.
2. Purpose of project and its academic rationale.
3. Brief description of methods and measurements.
4. Participants: recruitment methods, number, age, gender, exclusion/inclusion criteria.
5. Consent and participant information arrangements, debriefing.

Please attach intended information and consent forms, and any debriefing information.

6. A clear but concise statement of the ethical considerations raised by the project and how you intend to deal with them.
7. Estimated start date and duration of project.

This form with attachments should be submitted electronically to the Departmental Ethics Committee (v.west@gold.ac.uk) for consideration. A hard copy (without attachments) should be placed in the pigeon-hole of the Chair of the Department Ethics Committee, Dr Yulia Kovas.

If any of the above information is missing, your application will be returned to you.

Supervisors:

There is an obligation on the supervisor to bring to the attention of the Departmental Ethics Committee any issues with ethical implications not clearly covered by the above checklist. By signing this form, supervisors confirm that they have seen ALL PLANNED ASSESSMENT INSTRUMENTS, INCLUDING THOSE ADMINISTERED ON-LINE (BY FOLLOWING THE ON-LINE LINKS).

Students and Supervisors:

We are familiar with the BPS Guidelines for ethical practices in psychological research, and have discussed them.

Signed  Date 11/1/2016

Name Zahra Safaryzdi
(Student)

Signed  Date 19/1/2016

Name Ashok Jansari
(Supervisor)

Signed.....*[Signature]*.....Date 15/1/16
Name Andrew Cooper
(Supervisor)

STATEMENT OF ETHICAL APPROVAL

This project has been considered using agreed Departmental procedures and is now approved. This approval is valid for a maximum period of five years.

Signed.....*[Signature]*.....Date 25/1/16

Name.....*[Signature]*.....
(Chair, Departmental Ethics Committee)

(ETHICSform June 2013)

Appendix 14: Study 3 Ethics Approval Form

EAF2: Ethical Approval Form for Goldsmiths Students
8.2.17

DEPARTMENT OF PSYCHOLOGY
GOLDSMITHS, UNIVERSITY OF LONDON

Tick one box:

2nd Year UNDERGRADUATE project (GROUP)

2nd Year UNDERGRADUATE project (MINI)

3rd Year UNDERGRADUATE project (FINAL)

POSTGRADUATE project - CCN, FCPHS, MMB, RMIP, SPGE, MPhil/PhD (please circle)

Title of project

The role of emotion regulation in the relation between ADHD symptoms and substance use

Name of student(s) Zahra Safaryazdi _____ Email zsafa001@gold.ac.uk_

Name of student(s) Add additional names for group projects _____

_____ Email _____

Name of supervisor(s)

Dr Ashok Jansari, Dr. Andrew Cooper, Dr. Alice Jones

Date _____

BEFORE FILLING IN THIS FORM, PLEASE READ THE BPS Code and Practice Document AND ALL RELEVANT ETHICAL GUIDELINES DOCUMENTS PROVIDED ON THE PSYCHOLOGY DEPARTMENT ETHICS COMMITTEE WEBSITE: <https://learn.gold.ac.uk/course/view.php?id=288>

IT IS RESPONSIBILITY OF ALL PSYCHOLOGY STUDENTS TO BE FAMILIAR WITH AND FOLLOW ESTABLISHED ETHICAL GUIDELINES AND PROCEDURES.

| | | YES | NO | N/A |
|----|--|-----|----|-----|
| 1 | Will you describe the main experimental procedures to participants in advance, so that they are informed about what to expect? | ● | | |
| 2 | Will you make it clear to participants that this is a student project? | ● | | |
| 3 | Will you tell participants that their participation is voluntary? | ● | | |
| 4 | Will you obtain written consent for participation? | ● | | |
| 5 | If the research is observational, will you ask participants for their consent to being observed? | ● | | |
| 6 | Will you tell participants that they may withdraw from the research at any time and for any reason? | ● | | |
| 7 | With questionnaires, will you give participants the option of omitting questions they do not want to answer? | ● | | |
| 8 | Will you tell participants that their data will be treated with full confidentiality and that, if published, it will not be identifiable as theirs? | ● | | |
| 9 | Will you debrief participants at the end of their participation (i.e. give them a brief explanation of the study)? | ● | | |
| 10 | Will your project involve deliberately misleading participants in any way? | | ● | |
| 11 | Are you asking any questions of sensitive or potentially upsetting content? | | ● | |
| 12 | Is there any realistic risk of any participants experiencing either physical or psychological distress or discomfort? If Yes, give details on a separate sheet and state what you will tell participants to do if they should experience any | | ● | |

Give a brief description of participants and procedure (methods, assessments used etc) in up to 150 words.

This form (without any attachments) should be submitted in hard copy to the Chair of the Department Ethics Committee, Professor Yulia Kovas, via her pigeon-hole.

B. I consider that this project may have ethical implications that should be brought before the Departmental Ethics Committee, and/or it will be carried out with children or other vulnerable populations. PLEASE TICK.

Please provide all the information listed below in a separate attachment.

1. Title of project.
2. Purpose of project and its academic rationale.
3. Brief description of methods and measurements.
4. Participants: recruitment methods, number, age, gender, exclusion/inclusion criteria.
5. Consent and participant information arrangements, debriefing.

Please attach intended information and consent forms, and any debriefing information.

6. A clear but concise statement of the ethical considerations raised by the project and how you intend to deal with them.
7. Estimated start date and duration of project.

This form with attachments should be submitted electronically to the Departmental Ethics Committee (v.west@gold.ac.uk) for consideration. A hard copy (without attachments) should be placed in the pigeon-hole of the Chair of the Department Ethics Committee, Professor Yulia Kovas.

If any of the above information is missing, your application will be returned to you.

Supervisors:

There is an obligation on the supervisor to bring to the attention of the Departmental Ethics Committee any issues with ethical implications not clearly covered by the above checklist. By signing this form, supervisors confirm that they have seen ALL PLANNED ASSESSMENT INSTRUMENTS, INCLUDING THOSE ADMINISTERED ON-LINE (BY FOLLOWING THE ON-LINE LINKS).

Students and Supervisors:

We are familiar with the BPS Guidelines for ethical practices in psychological research, and have discussed them.

Signed...

Date 26/01/2017

Name Zahra Safaryazdi

Signed.....

Date

26/1/17

Name Ashley Jansons
(Supervisor)

STATEMENT OF ETHICAL APPROVAL

This project has been considered using agreed Departmental procedures and is now approved. This approval is valid for a maximum period of five years.

Signed C Rix Date _____

Name C Rix
(Chair, Departmental Ethics Committee)

(ETHICSform June 2013)

Appendix 15: Study 4 and 5 Ethics Approval Form

22/6

EAF2: Ethical Approval Form for Goldsmiths Students

DEPARTMENT OF PSYCHOLOGY
GOLDSMITHS, UNIVERSITY OF LONDON

Tick one box:

2nd Year UNDERGRADUATE project (GROUP)

2nd Year UNDERGRADUATE project (MINI)

3rd Year UNDERGRADUATE project (FINAL)

POSTGRADUATE project - CCN, FCPHS, MMB, RMIP, SPGE, MPhil/PhD (please circle)

Title of project

The role of mood and sleep disorder in the relation between ADHD symptoms and substance use

Name of student(s) Zahra Safaryazdi _____ Email zsafa001@gold.ac.uk_

Name of student(s) Add additional names for group projects

_____ Email _____

Name of supervisor(s)

Dr Ashok Jansari, Dr. Andrew Cooper, Dr. Alice Jones

Date _____

BEFORE FILLING IN THIS FORM, PLEASE READ THE BPS Code and Practice Document AND ALL RELEVANT ETHICAL GUIDELINES DOCUMENTS PROVIDED ON THE PSYCHOLOGY DEPARTMENT ETHICS COMMITTEE WEBPAGE: <https://learn.gold.ac.uk/course/view.php?id=288>

IT IS RESPONSIBILITY OF ALL PSYCHOLOGY STUDENTS TO BE FAMILIAR WITH AND FOLLOW ESTABLISHED ETHICAL GUIDELINES AND PROCEDURES.

| | | YES | NO | N/A |
|----|--|-----|----|-----|
| 1 | Will you describe the main experimental procedures to participants in advance, so that they are informed about what to expect? | ● | | |
| 2 | Will you make it clear to participants that this is a student project? | ● | | |
| 3 | Will you tell participants that their participation is voluntary? | ● | | |
| 4 | Will you obtain written consent for participation? | ● | | |
| 5 | If the research is observational, will you ask participants for their consent to being observed? | ● | | |
| 6 | Will you tell participants that they may withdraw from the research at any time and for any reason? | ● | | |
| 7 | With questionnaires, will you give participants the option of omitting questions they do not want to answer? | ● | | |
| 8 | Will you tell participants that their data will be treated with full confidentiality and that, if published, it will not be identifiable as theirs? | ● | | |
| 9 | Will you debrief participants at the end of their participation (i.e. give them a brief explanation of the study)? | ● | | |
| 10 | Will your project involve deliberately misleading participants in any way? | | ● | |
| 11 | Are you asking any questions of sensitive or potentially upsetting content? | | ● | |
| 12 | Is there any realistic risk of any participants experiencing either physical or psychological distress or discomfort? If Yes, give details on a separate sheet | | ● | |

| | | | | |
|----|--|---|----------------------------------|--|
| | and state what you will tell participants to do if they should experience any problems (e.g. who they can contact for help). | | | |
| 13 | Does your project involve work with animals? | | <input checked="" type="radio"/> | |
| 14 | Do participants fall into any of the following special groups? If they do, please refer to BPS guidelines and other relevant documents. Note that you may also need to obtain satisfactory CRB clearance (or equivalent for overseas students). | Children (under 18 years of age) | <input checked="" type="radio"/> | |
| | | People with learning or communication difficulties | <input checked="" type="radio"/> | |
| | | Patients | <input checked="" type="radio"/> | |
| | | People in custody | <input checked="" type="radio"/> | |
| | | People engaged in illegal activities (e.g. drug-taking) | <input checked="" type="radio"/> | |

If you have ticked **Yes** to any of the questions Q10 to Q14 (10,11,12,13,14) you should normally tick **box B** overleaf; if not (e.g., in the case of secondary data analysis), please give a full explanation on a separate sheet.

On-line Data collection

If you are planning collecting data on-line, please read the document **Conducting research on the internet: guidelines for ethical practice in psychological research online (PDF)**.

Are you collecting data on-line? YES NO

If you answered **YES**, please provide the following information:

1. Provide the link to the planned on-line survey/test and any log-in information.
2. For questions 1 to 9 above, describe how you will meet these criteria on-line. For example, for question 4, instead of written consent, participants must confirm that they are 18 or over and tick on-line to agree to participate, or agree to participate by proceeding to the next page; for question 7, there should be an option of skipping individual questions.

Participants will be recruited from the Research Participation Scheme at the Department of Psychology at Goldsmiths, University of London and from my friends and relatives via facebook or Instagram pages. The information about the study and the link of it will be on the Research Participation Scheme page with researcher's email address. Participants can email the researcher and ask any question about the study. More detailed information is on the first page of the online survey.

There is an online consent form that participants confirm that they are 18 or over and they agree to take part in the study otherwise, they can't go through the questionnaires.

The questionnaires are designed in a way that participants can skip a question that they do not want to answer.

This is the link of the online study:

https://goldpsych.eu.qualtrics.com/jfe/form/SV_57ujsheQxtgXqux

ALL APPLICANTS: PLEASE TICK EITHER BOX A OR BOX B BELOW AND PROVIDE THE DETAILS REQUIRED IN SUPPORT OF YOUR APPLICATION. THEN SIGN THE FORM.

| | |
|---|--------------------------|
| A. I consider that this project has no significant ethical implications to be brought before the Departmental Ethics Committee. | <input type="checkbox"/> |
| | PLEASE TICK: |

Name..... ASHOK JANSAKI
(Supervisor)

STATEMENT OF ETHICAL APPROVAL

This project has been considered using agreed Departmental procedures and is now approved. This approval is valid for a maximum period of five years.

Signed..... C.Rix Date..... 26/06/17

Name..... C.Rix
(Chair, Departmental Ethics Committee)

(ETHC Form June 2013)

Appendix 16: Advertisement poster of Study 1

| | | |
|---|---|--|
|  <p>Department of Psychology</p> | <h1 style="margin: 0;">Volunteers Wanted</h1> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
|  | <p>Are you healthy (without any psychiatric disorder like conduct disorder, bipolar affective disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder and major physical health problems like brain injury) and aged above 18?</p> <p>Would you be prepared to spare up to 35 mins to perform some simple online Questionnaires, and 1 hour to play two face to face computer games?</p> <p style="text-align: center;">If you are interested, please contact Zahra Safaryazdi Goldsmiths, University of London email: zsafa001@gold.ac.uk</p> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |

Appendix 17: Advertisement poster of Study 2

| | | |
|---|--|--|
|  <p>Goldsmiths UNIVERSITY OF LONDON</p> <p>Department of Psychology</p> | <h1><u>Volunteers Wanted</u></h1> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
|  | <p>Are you healthy (without any psychiatric disorder like conduct disorder, bipolar affective disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder and major physical health problems like brain injury) and aged above 18?</p> <p>Would you be prepared to spare 35 minutes to perform some simple <i>online</i> questionnaires? You will receive 2 credits for your time</p> <p>If you are interested, please contact Zahra Safaryazdi Goldsmiths, University of London email: zsafa001@gold.ac.uk</p> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
| | | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |

Appendix 18: Advertisement poster of Study 3

| | | |
|---|---|--|
|  <p>Department of Psychology</p> | <h1>Volunteers Wanted</h1> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
|  | <p>Are you healthy (without any psychiatric disorder like conduct disorder, bipolar affective disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder and major physical health problems like brain injury) and aged above 18?</p> <p>Would you be prepared to spare 35 minutes to perform some simple <i>online</i> questionnaires? You will receive 2 credits for your time</p> <p>If you are interested, please contact Zahra Safaryazdi Goldsmiths, University of London email: zsafa001@gold.ac.uk</p> | <p>PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk</p> |
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Appendix 19: Advertisement poster of Study 4 and 5

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|---|--|---|---|
|  | <h1>Volunteers Wanted</h1> | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
| Department of Psychology | | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
|  | <p>Are you healthy (without any psychiatric disorder like conduct disorder, schizophrenia, borderline personality disorder, obsessive-compulsive disorder and major physical health problems like brain injury) and aged above 18?</p> | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
| | <p>Would you be prepared to spare 30 to 40 minutes to perform some simple <u>online</u> questionnaires? You will receive 2 credits for your time</p> | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
| | <p>If you are interested, please contact Zahra Safaryazdi Goldsmiths, University of London</p> | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
| | <p>email: zsafa001@gold.ac.uk</p> | PSYCHOLOGY RESEARCH Zahra Safaryazdi Email: zsafa001@gold.ac.uk | |
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