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**Smells like teen spirits: Investigating impulsivity and alcohol-related
cognition as modifiable risk factors for adolescent alcohol use intervention**

Kiri Anne Patton

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Centre for Youth Substance Abuse Research

Abstract

Alcohol use remains a significant cause of the personal and societal burden of disease worldwide. Prevention interventions for adolescents have been identified as a cost-effective way to prevent or reduce harm. Despite this, there remains mixed evidence about what factors and psychological processes contribute to effective prevention interventions. Utilising current theories of alcohol use risk may enable the identification and application of evidence-based risk factors for intervention to improve efficacy. This thesis focuses on the biosocial Cognitive Theory (bSCT) of substance abuse risk as a promising framework, which theorises that impulsivity imparts risk for alcohol misuse through the cognitive factors of drinking refusal self-efficacy and alcohol expectancies. The time-efficient measurement of core concepts in research, clinical, and prevention contexts is desirable to reduce participant burden. Chapters 4 and 5 are dedicated to reducing full-length scales of drinking refusal self-efficacy and alcohol expectancies to two 9-item scales (53% and 63% reduction, respectively). The interplay between impulsivity and these cognitive factors was then explored in the first prospective test of the bSCT model in adolescents (Chapter 6). It was found that the impulsivity factor of Rash Impulsiveness increased alcohol use up to two years in the future for adolescents aged 13 and 15 years. This impact arose through a mediation where Rash Impulsiveness predicted reduced drinking refusal self-efficacy, which in turn predicted increased alcohol consumption. The impulsivity factor of Reward Drive indirectly predicted alcohol use through increasing positive alcohol expectancies, which was related to decreased drinking refusal self-efficacy. This finding was especially pronounced for older adolescents. Based on strong theoretical findings concerning these modifiable factors, a developmentally appropriate school-based alcohol use prevention intervention was developed. This Randomised Controlled Trial (RCT) included 404 adolescents and examined the effectiveness of a brief Cognitive Behavioural Therapy (CBT)-based prevention intervention targeting key psychological mechanisms (Chapters 7 and 8). The RCT was the first evaluation of Mindfulness Meditation (MM) as an addition to brief CBT for adolescent substance use with an active control (Progressive Muscle Relaxation; PMR). Both active interventions (CBT+MM and CBT+PMR) reduced the rate of alcohol use increase over 6-months compared to an assessment-only control. However, the hypothesised mechanisms of impact relating to drinking refusal self-efficacy, alcohol expectancies, and impulsivity showed mixed outcomes. MM did not show additive effects over PMR. Overall, the thesis supported the use of psychological theories to inform risk and interventions for adolescent

alcohol use. Impulsivity, drinking refusal self-efficacy, and alcohol expectancies were shown to explain a large component of risk of adolescent alcohol use. Despite the effectiveness of targeting these risk factors in the present alcohol use intervention, determining their precise mechanisms of action requires further research.

Declaration by author

This thesis is composed of my original work, and contains no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

I have clearly stated the contribution of others to my thesis as a whole, including statistical assistance, survey design, data analysis, significant technical procedures, professional editorial advice, financial support and any other original research work used or reported in my thesis. The content of my thesis is the result of work I have carried out since the commencement of my higher degree by research candidature and does not include a substantial part of work that has been submitted to qualify for the award of any other degree or diploma in any university or other tertiary institution. I have clearly stated which parts of my thesis, if any, have been submitted to qualify for another award.

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Publications during candidature

Peer-reviewed papers

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Patton, Kiri	Conception and design (50%) Analysis and interpretation (55%) Drafting and production (50%)
Connor, Jason P	Conception and design (10%) Analysis and interpretation (5%) Drafting and production (10%)
Rundle-Thiele, Sharyn	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (10%)
Dietrich, Timo	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (10%)
Young, Ross McD	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)
Gullo, Matthew J	Conception and design (25%) Analysis and interpretation (25%) Drafting and production (15%)

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Rundle-Thiele, Sharyn	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (10%)
Dietrich, Timo	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (10%)
Young, Ross McD	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)
Gullo, Matthew J	Conception and design (25%) Analysis and interpretation (25%) Drafting and production (15%)

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Gullo, Matthew J	Conception and design (20%) Analysis and interpretation (25%) Drafting and production (20%)
Connor, Jason P	Conception and design (10%) Analysis and interpretation (5%) Drafting and production (10%)
Chan, Gary C K	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)
Kelly, Adrian B	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)
Catalano, Richard F	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)
Toumbourou, John W	Conception and design (5%) Analysis and interpretation (5%) Drafting and production (5%)

Contributions by others to the thesis

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Statement of parts of the thesis submitted to qualify for the award of another degree

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Research Involving Human or Animal Subjects

For the studies included in Chapters 4 and 5, UQ human ethics approval was granted to utilise data from Griffith University HREC dated 24/02/2014 (Approval #2014001623). For the study included in Chapter 6, UQ human ethics approval was granted to utilise data from the International Youth Development Study (Approval #2015000294). For the study included in Chapter 7, UQ human ethics approval was granted to conduct a randomised controlled trial (Approval #2015000875). Brisbane Catholic Education research ethics approval was also granted (Approval #196).

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List of Abbreviations used in the thesis

- RCT: Randomised Control Trial
- VTA: Ventral Tegmental Area
- NAc: Nucleus Accumbens
- DSM-5: Diagnostic and Statistical Manual of Mental Disorders, fifth edition
- ICD-10: International Statistical Classification of Diseases and Related Health Problems, 10th revision
- DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (Text Revision)
- USA: United States of America
- CBT: Cognitive Behavioural Therapy
- MET: Motivational Enhancement Therapy
- NHMRC: National Health and Medical Research Council
- DARE: Drug and Alcohol Resistance Education
- REAL: Refuse, Explain, Avoid, Leave
- SURPS: Substance Use Risk Profile Scale
- MBSR: Mindfulness-Based Stress-Reduction
- MBCT: Mindfulness-Based Cognitive Therapy
- DBT: Dialectical Behaviour Therapy
- ACT: Acceptance Commitment Therapy
- STIC: Stop, Take a breath, Imagine the future consequences, and Choose
- MBT: Mindfulness-Based Therapy
- MI: Motivational Interviewing
- ADHD: Attention Deficit Hyperactivity Disorder
- BAS: Behavioural Approach System
- SPSRQ: Sensitivity to Punishment and Reward Questionnaire
- 2-CARS: 2-Component Approach to Reinforcing Substances
- UPPS: Urgency, Premeditation, Perseverance, Sensation seeking
- UPPS+P: Urgency, Premeditation, Perseverance, Sensation seeking, Positive urgency
- SCT: Social Cognitive Theory
- SLT: Social Learning Theory
- bSCT: bioSocial Cognitive Theory
- POAE: Positive Alcohol Outcome Expectancies
- COEA: Comprehensive Effects of Alcohol
- DEQ-A: Drinking Expectancy Questionnaire – Adolescent version
- DEQ-SA: Drinking Expectancy Questionnaire – Shortened Adolescent version
- AUDIT-C: Alcohol Use Disorders Identification Test – Consumption
- AUD: Alcohol Use Disorder
- AEQ-A: Alcohol Expectancy Questionnaire-Adolescent
- AEQ: Alcohol Expectancy Questionnaire
- DEQ: Drinking Expectancy Questionnaire
- AEQ-AB: Alcohol Expectancy Questionnaire-Adolescent Brief
- NAEQ: Negative Alcohol Expectancy Questionnaire
- GOKA: Game On: Know Alcohol
- AUDIT: Alcohol Use Disorders Identification Test
- EFA: Exploratory Factor Analysis

- CFA: Confirmatory Factor Analysis
- CFI: Comparative Fit Index
- RMSEA: Root Mean-Square Error of Approximation
- SRMR: Standardized Root Mean-square Residual
- AIC: Akaike Information Criterion
- ML: Maximum Likelihood
- MANOVA: Multivariate ANalysis Of VAriance
- ANOVA: ANalysis Of VAriance
- SD: Standard Deviation
- SE: Standard Error
- s^2 : squared semi-partial coefficient
- DRSEQ-RA: Drinking Refusal Self-Efficacy Questionnaire – Revised Adolescent version
- NIRT: Non-parametric Item Response Theory
- DRSEQ-SRA: Drinking Refusal Self-Efficacy Questionnaire – Shortened Revised Adolescent version
- DRSEQ-R: Drinking Refusal Self-Efficacy Questionnaire-Revised
- MCAR: Missing Completely at Random
- IRT: Item Response Theory
- MMH: monotone homogeneity
- OCC: Option Characteristic Curves
- ICC: Item Characteristic Curves
- DRSE: Drinking Refusal Self-Efficacy
- IYDS: International Youth Development Study
- SEM: Structural Equation Modelling
- CI: Confidence Interval
- MM: Mindfulness Meditation
- PMR: Progressive Muscle Relaxation
- BIS-B: Barratt Impulsiveness Scale – Brief
- FAS-II: Family Affluence Scale – II
- RD: Reward Drive
- SR-S: Sensitivity to Reward Scale
- WHO: World Health Organisation
- MAAS-A: Mindful Attention Awareness Scale-Adolescent
- MLM: Multilevel Modelling
- VPC: Variance Partition Coefficient

Chapter 1: Thesis Aims and Background

Brief introduction and thesis aims

Alcohol use is a highly pervasive and prevalent legal intoxicating substance. The consumption of beverages containing alcohol is a common cross-cultural phenomenon. Alcohol is central to many rituals, religions, and social interactions. Low doses produce effects of relaxation, euphoria, and reduced inhibition (Feola, de Wit, & Richards, 2000; Koob & Bloom, 1988). While alcohol can be used without harm at low doses, both consistent moderate levels of alcohol consumption over time (3+ standard drinks per day) and high dosages of alcohol (5+ standard drink) in a single drinking session can lead to significant harm (National Health and Medical Research Council, 2009).

When searching for pithy quotes to use in this section, I found many more cultural and political leaders endorsing alcohol use rather than cautioning against it. And indeed, Western societies appear to be leaning towards legalising drugs rather than restricting them. So, the question is as it always was, if alcohol is to remain a common component of cultural and social contexts, how can we increase the safe use of this substance?

In this thesis, I make the argument that prevention of problem alcohol use is more effective than treatment and that measurement and targeting of risk factors is central to prevention of alcohol misuse in adolescents. In Chapters 2 and 3, I explore the effects of alcohol use in adolescence and identify several risk factors for use. A focus was made on risk factors that could be hypothetically modifiable with psychological intervention. The identified risk factors were the cognitive factors of drinking refusal self-efficacy and alcohol expectancies, as well as impulsivity. I delve into the appropriate measurement of the two cognitive factors in chapter 4 and 5 before presenting research investigating the prospective mediational relationships between these risk factors and adolescent alcohol use (Chapter 6). The results of a Randomised Control Trial (RCT) aimed at targeting these risk factors are presented in Chapter 7.

The aim of this thesis then is to determine whether targeting theoretically supported risk-factors for future adolescent alcohol use can increase the safe use of alcohol. Further, I aim to investigate the mechanisms by which these factors relate to alcohol use and predict prevention outcomes. By identifying mechanisms of intervention effects, future work can refine interventions to be more effective. To evaluate intervention outcomes, accurate and brief measurement of concepts is required, so a secondary aim of the thesis is to develop brief

versions of scales to measure the cognitive risk factors of drinking refusal self-efficacy and alcohol expectancies in adolescents.

Introduction to Alcohol and Alcohol Misuse

What is alcohol?

When discussing alcohol use, it is useful to look at the neurological responses that occur when alcohol is consumed. Alcohol (ethanol) is a psychoactive drug, which acts as a Central Nervous System depressant. Recreational drugs, including ethanol, are hypothesised to act on the reward pathways in the brain, specifically the ventral tegmental area (VTA) and the nucleus accumbens (NAc), indirectly increasing dopamine levels (Nestler, 2005). Alcohol alters the brain through a complex array of neuronal effects within several brain structures. These include the prefrontal cortex, the VTA, the NAc, the hippocampus, and the amygdala (Feduccia, Chatterjee, & Bartlett, 2012; Lovinger, White, & Weight, 1989; Yoshimoto, McBride, Lumeng, & Li, 1992). Acute alcohol use is hypothesised to be reinforced through its actions on the GABA_A receptor system, glycine, dopaminergic mechanisms within the mesolimbic and mesocorticolimbic dopamine systems, neuronal nicotinic acetylcholine receptors, opioid peptide interactions, serotonin systems, and glutamate systems (Feduccia et al., 2012; Koob et al., 1998; Lovinger et al., 1989; Mihic et al., 1997; Nestler, 2005; Yoshimoto et al., 1992). In light of these complex mechanisms and actions of alcohol use, especially those implicated in neural reward systems, addiction and dependence can be understood as a biological reaction rather than solely a weakness of character as it has previously been characterised.

Diagnostic criteria for alcohol use disorders.

There are two primary classification systems of substance abuse. The Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5; American Psychiatric Association, 2013) and the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10; World Health Organization, 1992) both outline clinical disorders associated with alcohol. The DSM-5 and the ICD-10 include craving, tolerance, withdrawal, salient drug priority compared to other life demands, and continued use despite harmful health or social consequences as key criteria to Alcohol Use Disorder (DSM-5) and Alcohol Dependence Syndrome (ICD-10). Alcohol misuse in the DSM-5 is scored on a continuum depending on the number of symptoms present where moderate and severe Alcohol Use Disorder in the DSM-5 aligns with Alcohol Dependence Syndrome in the

ICD-10 and alcohol dependence in DSM-IV. Mild Alcohol Use Disorder in the DSM-5 aligns with Harmful Use of alcohol in the ICD-10, which is characterised by use that is physically or mentally harmful but may not have yet caused more severe symptoms, such as tolerance or withdrawal. The parallels between alcohol misuse diagnoses were improved in the revisions to the previous DSM (DSM-IV-TR) based on recommendations by the research and clinical community (Hasin et al., 2013; Regier, Kuhl, & Kupfer, 2013). For example, substance abuse and substance dependence disorders, which were previously separate criteria, were combined into the diagnosis of Substance Use Disorder and craving was added to the dependence criteria (Hasin et al., 2013). The two systems show initial signs of compatibility, with the greatest differences occurring between the Mild Alcohol Use Disorder and Harmful Use categorisations (Hoffmann & Kopak, 2015). The DSM-5 is more commonly used in Australia and the United States of America. Therefore, I will be referring to prevalence using this diagnostic criterion.

Prevalence of alcohol misuse and associated harms.

Alcohol Use Disorders are widely prevalent in the United States of America (USA) and Australia. According to a recent summary of alcohol use disorders in *The Lancet*, more than 1 in 3 male and 1 in 5 female adults in the USA have met DSM-5 criteria for an Alcohol Use Disorder in their lifetime (Connor, Haber, & Hall, 2016). Further, 17.6% of males and 10.4% of females had experienced Alcohol Use Disorder within the last year (surveyed 2012-13). When the DSM-5 Alcohol Use Disorder criteria were applied to a large Australian dataset collected in 1997 ($N = 10,641$), the prevalence of Alcohol Use Disorders was calculated to be 9.7% (Mewton, Slade, McBride, Grove, & Teesson, 2011). While this is lower than the prevalence of Alcohol Use Disorders in America, it still indicates that almost 1 in 10 Australian adults struggle with clinical levels of alcohol use.

The findings of a high prevalence of disordered alcohol use are supported by the most recent national Australian data, which showed that Australians are placing themselves at elevated risk for harm caused by alcohol use (Australian Institute of Health and Welfare, 2017). Current Australian guidelines recommend that both males and females above the age of 18 years drink no more than two standard drinks on average per day to decrease risk of lifetime harm. Secondly, the guidelines state that drinking more than four standard drinks per sitting puts the drinker at increased risk of single occasion harm, e.g., unintentional injury (National Health and Medical Research Council, 2009). The guidelines recommend abstinence from alcohol under the age of 18 years old, which is congruent with the legal

drinking age in Australia (National Health and Medical Research Council, 2009). The Australian Institute of Health and Welfare found that 17.1% of people in Australia consumed in excess of guidelines for lifetime risk of alcohol-related harm. Additionally, 25% drank 5 or more standard drinks on a single occasion at least monthly, placing them at elevated risk of single-occasion harm (Australian Institute of Health and Welfare, 2017). In 2010, the World Health Organization calculated that Australians above 15 years of age drank within the category of 10.0-12.4 litres of pure alcohol per person per year (World Health Organization, 2014). This consumption places Australia in the second highest category of consumption, behind only Russia and several Eastern European countries. Encouragingly, from 2004 to 2016 daily alcohol use by Australians has been declining across most age groups (Australian Institute of Health and Welfare, 2014, 2017). Despite the declining trend of use, a large proportion of Australians are still drinking at levels above the recommendations, indicating that research into prevention and reduction in associated harms may provide enormous individual and societal benefits.

Individual benefits from reduced alcohol use could include a reduction in rates of chronic health conditions caused by harmful alcohol use. Harmful consumption of alcohol is associated with a number of long-term health conditions, including neuropsychiatric conditions such as depression and anxiety, diabetes mellitus, cardiovascular diseases, increasing risk of hypertension, atrial fibrillation, and haemorrhagic stroke, the gastrointestinal diseases liver cirrhosis and pancreatitis, and cancers of the digestive system such as mouth, oesophageal, colon and rectum, and liver cancers as well as breast cancer in women (World Health Organization, 2014). Acute alcohol use also increases occurrences of intentional and unintentional injury and risky sexual behaviour, the latter of which increases the risk of sexually transmitted diseases (World Health Organization, 2014). Further, heavy drinking is detrimental to immune functioning and this has been linked to greater risk of pneumonia and tuberculosis (World Health Organization, 2014).

These health effects have personal and societal implications. The global burden of disease attributable to alcohol use disorders has been estimated at 9.6% (7.7 – 11.8) of disability-adjusted life years (DALYs), 44.4% (29.1-60.0) years of life lost to premature mortality (YLLs), 7.9% (6.0 – 10.0) of years lived with disability (YDLs; Whiteford et al., 2013), and 5.9% of all global deaths (World Health Organization, 2014). Alcohol use is associated with a high economic cost. It was estimated that the economic cost related to alcohol in 2004/2005 in Australia was \$15.3 billion (Collins & Lapsley, 2008). In 2010, a

similar estimate of \$14.352 billion was calculated by the Australian Institute of Criminology, with the most substantial costs associated with the criminal justice system, the health system, productivity, and traffic accidents (Manning, Smith, & Mazerolle, 2013). Further, it is not only the drinking individual who experiences the harm. An estimated \$14.2 billion was spent by not-at-fault others in one year due to costs arising from alcohol-related car accidents, assaults, child protection, out-of-pocket expenses, time spent caring for or covering drinkers' work, and diverted household finances to alcohol (Laslett et al., 2014). Due to the large cost associated with alcohol use, efforts have been made to develop effective treatments to reduce disordered alcohol use.

Current treatments

Brief interventions are commonly used to treat Alcohol Use Disorders as they are less resource and time intensive, which is beneficial for both the patient and practitioner, and they demonstrate moderate efficacy. Brief interventions usually range from a single 5-20 minute session to 3 sessions (Connor et al., 2016). Brief interventions for hazardous and harmful alcohol use in primary health care settings are moderately effective and usually consist of feedback, advice and goal-setting (Connor et al., 2016; O'Donnell et al., 2014). Pharmacotherapy is often used to aid treatment and reduce relapse (Connor et al., 2016). However, there are conflicting findings regarding the optimum duration and frequency of treatment (O'Donnell et al., 2014).

In their review of Alcohol Use Disorders in *The Lancet*, Connor and colleagues (2016) considered Cognitive Behavioural Therapy (CBT) and Motivational Enhancement Therapy (MET) to have high levels of evidence regarding their effectiveness as brief alcohol use interventions. Table 1.1 below describes common non-pharmacological behavioural treatments and classifies their evidence base.

Table 1.1.

Description of non-pharmacological behavioural treatments reproduced from Connor and colleagues (2016).

	Description	Level of evidence*
Cognitive behaviour therapy	This approach addresses cognitive, affective, and interpersonal triggers for alcohol use. It enhances drinking refusal self-efficacy† skills; identifies and modifies alcohol expectancies‡; improves problem-solving skills; and develops more effective coping strategies, including relaxation approaches.	High ²⁰⁻²⁴
Motivational enhancement therapy	This therapy is a patient-centred approach that enhances motivation to change behaviour. It uses a collaborative therapeutic approach to assist patients to recognise and resolve ambivalence, and develop their own reasons to reduce or abstain from drinking. Key strategies include collaborative identification of the gap between the patient's present and desired health (ie, goal-status discrepancy), recognition of their resistance to change, avoidance of confrontational communication, and guided assessment of the pros and cons for change.	High ²⁰⁻²²
Behavioural therapies based on conditioning	Cue exposure: repeated exposure to conditioned cues (eg, image or smell of alcohol, or associated emotion) can induce habituation or craving. Exposure to cues during treatment in the absence of drinking (with or without coping skill practice) is thought to reduce habituation. It is often combined with other cognitive therapy or skills. Contingency management: this approach introduces a tangible reinforcer, such as money or vouchers, to increase session attendance or abstinence. It is more suitable for inpatient and residential settings, and needs more translatable evidence.	Low; ²³ Moderate ^{25,26}
12-step facilitation	This approach offers continuous mutual peer support, usually in the form of self-help groups run by Alcoholics Anonymous. Participation is free of charge. Participants need to "surrender to a higher power" to facilitate change. Some groups use a buddy system (a sponsor) to provide support between group meetings.	Mixed ^{21,22,27}

*Summary based on authors' narrative review of the highest level of evidence for each treatment. †Individuals' beliefs about their ability to refrain from drinking. ‡Individuals' expectations about the effects of alcohol consumption.

Cognitive Behavioural Therapy (CBT) aims to identify and alter maladaptive alcohol-related cognitions and cues. The mechanisms of action are considered to be reducing positive expectations of the outcomes of alcohol use, building self-efficacy in refusing and resisting alcohol use, improving problem-solving, and increasing non-alcohol coping mechanisms, which can include relaxation strategies (Connor et al., 2016). Motivational enhancement therapy can be used as a stand-alone brief intervention or can be used to motivate patients to engage in other brief interventions (usually CBT). Motivational enhancement therapy aims to collaboratively work with the patient to identify reasons for changing their behaviour through highlighting the discrepancies between their current actions and their ideal behaviour and values and through problem-solving the pros and cons of changing their behaviour. A key component of motivational enhancement therapy is acceptance of the patient's actions and opinions and a non-confrontational approach (Connor et al., 2016). In sum, Motivational Enhancement Therapy and CBT have a high evidence base for their use as moderately effective Alcohol Use Disorder brief interventions.

Despite there being a strong evidence base for several Alcohol Use Disorder brief interventions, Alcohol Use Disorder treatment is underutilised. One possibility for this lack of engagement in treatment is perceived stigma and denial (Cohen, Feinn, Arias, & Kranzler, 2007). For those persons who do seek assistance, abstinence rates post-treatment are low, ranging from 25% to 43% (Connor et al., 2016). Due to the high individual and societal harm of problematic alcohol misuse and the relatively low treatment success, prevention and early intervention have been proposed to ameliorate harms. A typical age for intervention efforts is adolescence, as this period is associated with low or no alcohol use for the most part

(Australian Institute of Health and Welfare, 2017; World Health Organization, 2014). Further, increased consumption during adolescence predicts poor social and health outcomes later in life (Boden, Fergusson, & Horwood, 2013; Bonomo, Bowes, Coffey, Carlin, & Patton, 2004; Rossow & Kuntsche, 2013). Therefore, adolescence presents as a risk period for future alcohol use where effective prevention interventions may reduce the future harmful use of alcohol or prevent it altogether.

Chapter 2: Impact of Adolescent Alcohol Use and Overview of Current Prevention Programs

Adolescence as a risk period

Adolescence is a unique period where increasing responsibilities and freedoms are combined with physical and cognitive development. Neurological studies show that white matter continues to develop and grey matter is pruned during and after adolescence suggesting that executive functioning is not fully realised during this period (Asato, Terwilliger, Woo, & Luna, 2010; Giedd et al., 1999; Pfefferbaum et al., 2015). The prefrontal cortex, in particular, is a work in progress during adolescence although emerging research is highlighting a complex network of neuronal risk for alcohol use during adolescence (Robert & Schumann, 2017). Commonly researched mechanisms of risk also include the dopaminergic systems, which are related to reward sensitivity. The dopaminergic system increases in activity during adolescence, creating a potent combination of immature cognitive abilities, reduced impulse control, and increased orientation to risk and reward, which can alter decision making (Hartley & Somerville, 2015; Jaworska & MacQueen, 2015). Adolescents' underdeveloped prefrontal cortex, immature brain matter, and increased orientation to reward contribute to a behavioural pattern of risk for reinforcing behaviours.

It is for these reasons that adolescence is considered a key risk period for addiction (Chambers, Taylor, & Potenza, 2003). Adolescents are prone to higher thresholds for risk when peers are present and in high-arousal situations and show higher discounting of delayed rewards compared to immediate rewards (Hartley & Somerville, 2015). There is variation within adolescents as to the extent they display these characteristics. Those with increased trait impulsivity and reduced inhibitory control are particularly at risk of problematic alcohol use (Squeglia, Jacobus, Nguyen-Louie, & Tapert, 2014; Stautz & Cooper, 2013). The variation in expression of impulsivity and sensation seeking provides a possible area for intervention.

Animal studies and prospective human studies have investigated the impact of alcohol use during adolescence on functional and structural outcomes. Higher intensity frequency and intensity of drinking during middle adolescence are associated with decreased verbal memory, visuospatial ability, and psychomotor speed in late adolescence (Nguyen-Louie et al., 2015). Squeglia and colleagues (2014) found that 20 out of 40 adolescents followed over 3-years developed habitual heavy alcohol use during the study. These adolescents had

reduced brain matter volume in frontal brain regions prior to alcohol use initiation. After continued heavy use they showed volume reduction in the temporal and subcortical brain regions. These brain areas are implicated in object and language comprehension as well as motor control, sensory integration, habit learning, and feedback processing (Squeglia, Rinker, et al., 2014). In a sample of adolescents who did not differ at baseline, alcohol initiation also resulted in decreased brain volume (Luciana, Collins, Muetzel, & Lim, 2014). The brain volume decreases were observed in the middle frontal gyrus, which may result in reduced executive processing, including memory and inhibitory control (Luciana et al., 2014). Heavy drinking adolescents followed over 8-years showed accelerated grey matter volume decline in the temporal and frontal brain regions as well as impaired white matter growth in the corpus callosum and pons (Squeglia et al., 2015). These findings suggest that adolescence is a particular development period of risk for alcohol-related neurological change (Spear, 2014; Squeglia, Boissoneault, Van Skike, Nixon, & Matthews, 2014).

Numerous neurological changes occur as a result of alcohol use during adolescence. However, the behavioural consequences of these changes have been less investigated with few studies following adolescents into adulthood (Feldstein Ewing, Sakhardande, & Blakemore, 2014). Animal studies have found that alcohol consumption during adolescence attenuates neuronal responses in the orbitofrontal cortex, which was associated with increased risk tolerance (McMurray, Amodeo, & Roitman, 2015). Additionally, adolescent exposure to alcohol in rats alters adult dopamine and GABAergic transmission, which are implicated in alcohol effects as well as behavioural control and decision-making within the Pre-Frontal Cortex (Trantham-Davidson et al., 2016). Shnitko et al. (2015) also found that high levels of alcohol consumption during adolescence altered dopamine production in the nucleus accumbens of adult rats, an area which is implicated in impulse regulation (Shnitko, Spear, & Robinson, 2015). These findings indicate that neuronal changes from adolescent alcohol use may affect behaviour related to risk, decision making and self-regulation.

Another cause for the interest in adolescence as a period of risk is due to the long-term behavioural effects of alcohol use at this age. A 30-year longitudinal study in New Zealand (the Christchurch Health and Development Study) found that increasing misuse of alcohol was associated with higher rates of committing the “impulsive” crimes of assault and property damage (vandalism, arson; Boden et al., 2013). The increased crime rates remained after controlling for history of previous anxiety and depression, stressful life events, cannabis and other illicit drug use, unemployment, peer and partner substance use and offending, and

conduct/anti-social personality disorder (Boden et al., 2013). This association between alcohol use and problem behaviour can be found across the lifespan, with adult adjustment issues such as offending, personal life issues and employment difficulties persisting from young adulthood into middle age with continued problem use (Jennings, Piquero, Rocque, & Farrington, 2015). A recent review found evidence that adolescent alcohol exposure was related to increased social anxiety, disrupted fear conditioning retention, and increased incentive salience in adulthood (Spear, 2014). Early alcohol use is also associated with increased odds of driving while under the influence and riding with a drinking driver while in high school (Ewing et al., 2015) and lower rates of high school non-completion (Kelly et al., 2015). Thus, the misuse of alcohol during adolescence can have long-term social and emotional effects.

Risky drinking habits and outcomes in adolescence, such as high-frequency use, alcohol-related injuries and heavy episodic drinking have been strongly linked to future alcohol dependence, the consequences of which have been discussed above (Bonomo et al., 2004; Rossow & Kuntsche, 2013). The relationship between early use and adverse outcomes may be attenuated by covariate factors such as social-economic background, individual personality and behavioural differences, and parental behaviour and family functioning (Newton-Howes & Boden, 2015). However, Rossow and colleagues (2013) did control for several of these covariates, and the systematic review by McCambridge and colleagues (2011) concluded that the evidence was consistent enough to support the link between adolescent alcohol use and continued alcohol problems and dependence. Despite their conclusion regarding alcohol use, they did caution that the links between adolescent alcohol use and other outcomes such as social and psychological consequences needed further investigations which included these potential confounds (McCambridge, McAlaney, & Rowe, 2011). Rose and colleagues (2014) addressed potential covariates by evaluating outcomes associated with problem alcohol consumption in late adolescence (approximately 18 years of age) using a sample of drinking-discordant twins. Their results indicated support for the influence of these factors, as the effect sizes for alcohol use on adult outcomes were reduced in monozygotic twin pairs. However, there was support for the association between late adolescent drinking problems and continued problem drinking, risky sexual behaviour, and increases psychiatric symptoms as measured by the General Health Questionnaire at age 25 (Rose, Winter, Viken, & Kaprio, 2014). The strong link between adolescent and adult alcohol

use is one of the reasons prevention interventions are thought to provide high impact if successful.

Prevalence of adolescent alcohol use

I have detailed many consequences of adolescent alcohol use, including neuronal, behavioural, social, and health outcomes, but how much do Australian adolescents drink? Among 15-19-year-olds in the Western Pacific Region (Australia included), 51.3% reported they were current or former drinkers and were almost twice as likely to engage in patterns of heavy episodic drinking than adults 15 years and older (12.5% versus 7.7% prevalence; World Health Organization, 2014). This pattern of drinking is stratified by sex, with male Western Pacific Region adolescents engaging in heavy episodic drinking at a much higher frequency (18.3%) than female adolescents (6.1%; World Health Organization, 2014). In relation to the National Health and Medical Research Council (NHMRC) guidelines, 7.3% of Australians aged 12 – 17 years in 2013 reported drinking more than two standard drinks on average per day putting them at an increased risk of lifetime harm (Australian Institute of Health and Welfare, 2014). Approximately 13.2% reported drinking 5 standard drinks or more in one sitting at least yearly, increasing their risk for single occasion harm (Australian Institute of Health and Welfare, 2014). Despite the guidelines recommending abstinence under the age of 18 years, 14.3% of 12-15 year olds and 54% of 16 – 17 year olds reported that they had consumed a full alcoholic beverage previously (Australian Institute of Health and Welfare, 2014). The average age of first alcohol consumption for 14 – 24-year-olds was 15.7 years (Australian Institute of Health and Welfare, 2014). These prevalence rates confirm the utility of prevention interventions in this age group.

Adolescent alcohol use prevention interventions

Alcohol misuse prevention programs fall into three broad categories; school-based interventions; family-based interventions; and community-based interventions. Prevention programs can be delivered in a universal format, that is, they are delivered to all adolescents within the chosen group, or they can be delivered in a targeted manner to adolescents determined to be at risk of developing future alcohol problems. Both universal and targeted programs involve unique benefits and limitations. For example, targeted interventions may be more cost effective as they are only delivered to those at most risk, but universal interventions may be more practical to implement as they do not involve pre-screening and finding the time and resources to separate adolescents from their peers.

The three main intervention themes of school-based interventions are social resistance skills training, normative education, and competence-enhancement (Griffin & Botvin, 2010). Social resistance skills training involves increasing awareness of social influences and teaching resistance skills. Normative education provides students with information regarding the effects of alcohol use with the aim to challenge beliefs around alcohol. Competence-enhancement involves increasing skills that may assist students in resisting or reducing alcohol use, such as cognitive training, social skills training, problem-solving skills, and coping training. There is evidence to show that interventions including drinking refusal training, social-skills training and self-management skills training resulted in better outcomes for students aged 10-15 years than normative education interventions (Lemstra et al., 2010). Within school-based interventions, improving psychosocial functioning and general life-skills rather than delivering substance-specific information shows greater evidence for reducing alcohol consumption but not harms (Stockings et al., 2016). The finding that outcome is dependent on intervention targets further emphasises the need for investigation into components of interventions and their mechanisms of action, with the balance of evidence being weighted towards skills development rather than information delivery in school-based substance use interventions.

The ongoing investigations into what intervention components are effective for adolescents could explain some of the inconsistent findings of intervention efficacy. A recent Cochrane review found low to mixed evidence for the effectiveness of brief school-based interventions on substance use outcomes (Carney, Myers, Louw, & Okwundu, 2016). However, the authors did conclude that they were unwilling to make a definitive judgment on overall efficacy due to the current lack of high-quality studies, especially in low-middle income countries (Carney et al., 2016). A 2011 Cochrane review, however, found small but consistent effectiveness of universal family-based prevention programs (Foxcroft & Tsertsvadze, 2011). The systematic review and meta-analysis by Onrust and colleagues (2016) proposed that the discrepancies in intervention outcomes may be due to failure to use a developmental approach in interpreting study results. They found that intervention characteristics of universal and targeted interventions, such as problem-solving, or social skills training, had differing impacts in early, mid, and late adolescence (Onrust, Otten, Lammers, & Smit, 2016). Standardized effect sizes for universal program characteristics ranged from .01 (social influence) to -.17 (using cognitive behavioural therapy (CBT) techniques) for early adolescents, .00 (social norm) to .14 (refusal skills) for middle

adolescents, and .00 (transtheoretical model) to -.57 (social influence approach) for late adolescents (Onrust et al., 2016). Significant predictors of program effectiveness for each adolescent developmental group are listed in Table 2.1 below.

Table 2.1.

Significant predictors of program effectiveness by developmental group (Onrust et al., 2016).

Developmental period	Target/strategy	Effect size (<i>B</i>)	<i>p</i>
Early adolescence (Grade 6 and 7)	Self-control	-.14	.01
	Problem-solving	-.09	.03
	Healthy alternatives	-.14	.02
	Cognitive Behavioural Therapy techniques	-.17	.01
	Including parents or teachers in behavioural management	-.12	.01
	Including parents in the intervention	-.09	.02
	Middle adolescence (Grade 8 and 9)	Refusal skills training	.14 ⁺
Late adolescence (Grade 10-12)	Self-control	-.20	.02
	Refusal skills training	-.41	.01
	Social norms alterations	-.23	.02
	Using a social influence approach	-.57	.01
	Cognitive Behavioural Therapy techniques	-.32	.01
	Including parents in the intervention	-.29	.02

⁺Positive value indicates adverse effect.

These findings may help to explain the discrepant results between studies as the same intervention targets can have vastly different effects depending on age. For example, refusal skills training reduced alcohol use with a medium effect size in late adolescence but had a

small adverse effect (i.e., significantly increased alcohol use) in middle adolescence. Further, intervening in middle adolescence at all did not seem to produce significant effects whereas intervening in late adolescence had the largest effect sizes (Onrust et al., 2016). These outcomes make apparent the need for consistent and rigorous testing of drug prevention programs including the mechanisms of action. This sentiment is commonly shared, with calls for registration of trials, inclusion of active control groups, investigation of mechanisms of change and active treatment components, reporting of all outcomes to reduce reporting bias, and publishing of study protocols (Carney et al., 2016; Gorman, 2015; Magill & Longabaugh, 2013). By following these recommendations, a credible evidence base may be built and we can gain a better understanding of intervention and specific intervention component efficacy.

One such existing prevention intervention attempting to change strategies based on underwhelming alcohol use outcomes is the Drug and Alcohol Resistance Education (DARE) program. Created in 1983 by the Los Angeles Police Department and the Los Angeles Unified School District, DARE became the most widely disseminated drug and alcohol prevention program across America (Ennett, Tobler, Ringwalt, & Flewelling, 1994). The 17-session program was delivered to fifth-and-sixth-grade students by Police Officers and contained drug education, resistance and decision-making training, identification of drug alternatives, and self-esteem building. While theoretically convincing, long-term analyses of the DARE program have shown minimal effects on tobacco use and no or small effects for alcohol and drugs (Clayton, Cattarello, & Walden, 1991; Cuijpers, 2002; Ennett et al., 1994; Hansen & McNeal, 1997). Hansen and McNeal (1997) recommended that the entire DARE program should be replaced to better target theoretically important mediators of drug and alcohol use, such as normative beliefs.

A program overhaul seems to be exactly what the DARE program has done. Their new program '*keepin' it REAL*' was developed by the Drug Resistance Strategies Project to change cultural norms on drug use using culturally based narrative and performance framework. Despite this change the focus is similar to the original DARE program, as the main components are communication and life-skills training, such as drug resistance (REAL = Refuse, Explain, Avoid, Leave) and decision making. However, the skills taught are based on validated theories and strategies and an effort was made to incorporate values and narrative from differing cultures into the curriculum (Hecht et al., 2003). While this approach was initially promising (Hecht et al., 2003), subsequent studies found little evidence for efficacy for the program in 5th or 7th grade (Elek, Wagstaff, & Hecht, 2010; Marsiglia, Kulis,

Yabiku, Nieri, & Coleman, 2011). With the new findings of the efficacy of different approaches at different ages, it is unsurprising that the intervention may be less effective for different age groups. However, due to the widespread use of this program, they are in a privileged position where they can continue to provide care to thousands of adolescents and therefore should continue to alter their program to reflect the best evidence-based practice.

While the universal program, DARE, may have had varying levels of success, the *Prevention* program provides an example of one of the more successful targeted interventions. This program is a two 90-minute session intervention delivered by masters level psychologists. There is also a version of the intervention that is delivered by trained school staff: the *Adventure* program. Initially developed for adult female treatment-seeking drug and alcohol users (Conrod et al., 2000), the program was then revised for adolescents using focus groups to devise age-relevant scenarios and content (Conrod, Stewart, Comeau, & Maclean, 2006). The *Prevention* and *Adventure* programs target the personality risk factors of impulsivity and neurotic personality traits, which have been linked to separate pathways of adolescent alcohol use (Conrod et al., 2006). To identify those at risk, adolescents are screened for four personality traits, identified as lower-order factors of impulsivity and neuroticism: Anxiety Sensitivity, Negative Thinking, Sensation Seeking, and Impulsivity (Conrod, Castellanos, & Mackie, 2008). Screening is conducted using the *Prevention* group's Substance Use Risk Profile Scale (SURPS). Adolescents scoring one standard deviation or higher on one or more of these personality risk traits are invited to participate in the program. In this sense, only those deemed most at risk are targeted for the intervention. Adolescents are delivered a manualised intervention consisting of psychoeducation, motivational interviewing and cognitive-behavioural skills training specifically targeting cognitions relevant to their risk factor (e.g., boredom-susceptibility and reward-seeking for the Sensation Seeking condition; Conrod et al., 2008). Several studies have been conducted showing the small to moderately sized effectiveness of the intervention on reducing the likelihood and growth of adolescents drinking and binge drinking during the follow-up periods (up to 24 months; Conrod et al., 2013; Conrod, Castellanos-Ryan, & Mackie, 2011; Conrod et al., 2008, 2006; O'Leary-Barrett, Castellanos-Ryan, Pihl, & Conrod, 2016). Interestingly, there was also possible evidence of herd effects with adolescents identified as low risk and who did not receive an intervention also benefiting in schools where their high-risk peers had received an intervention (Conrod et al., 2013). While there has been evidence that the interventions can reduce adolescent mental health problems (Castellanos & Conrod, 2006; O'Leary-Barrett

et al., 2013), a recent study specifically investigating this question found no evidence of *Prevention*'s effectiveness for mental health outcomes (Goossens et al., 2016).

Despite encouraging findings for *Prevention*, there remains some question over the nature of the targeted effects. Despite all studies delivering four differing interventions, it is not clear that they are equally effective in reducing alcohol use. Indeed, some recent studies have combined these four groups into one single intervention group for analyses (e.g., Goossens et al., 2016; O'Leary-Barrett et al., 2016). While this certainly improves parsimony, it does not answer the question of whether the targeted cognitions are differentially affecting persons with different risk factors. Indeed, earlier studies which did analyse condition differences showed that only the Sensation Seeking group demonstrated significant reductions in binge-drinking from the intervention (Conrod et al., 2008, 2006). More recent work has shown similar findings, with the largest effects of the program on reducing binge drinking outcomes being found for the Sensation Seeking and Anxiety Sensitivity groups (Lammers et al., 2017). However, delayed growth in future drinking was only found within the Sensation Seeking group (Lammers et al., 2017). While Anxiety Sensitivity and Hopelessness (a precursor to Negative Thinking) interventions have separately shown increased abstinence, and reduced consumption and alcohol problems (Conrod et al., 2006; Lammers et al., 2017) these results were not found in a other investigations (Conrod et al., 2008). Additionally, when those identified as having a specific risk factor were delivered an incongruent program, there was only a small reduction in outcomes (Conrod et al., 2000). These inconsistent results show the appropriateness of evaluating the interventions separately to provide useful and interesting information as to the differing treatment effects based on personality risk factors.

Mindfulness-based interventions for alcohol use

One particular personality risk factor for alcohol use, which will be discussed in the next chapter, is impulsivity. Impulsivity is considered particularly pertinent for adolescents due to the neurological immaturity discussed above, that is, that the neurological processes involved in self-regulation and reward saliency are still developing at this age. Mindfulness Meditation has been proposed as a strategy to reduce alcohol consumption through the mechanism of reducing impulsive behaviours.

Mindfulness derived from Buddhist practices and was adapted for Western therapy use by Jon Kabat-Zinn as a strategy for stress-reduction (Mindfulness-Based Stress-

Reduction; MBSR; Kabat-Zinn, 1990). It has since been incorporated into Cognitive and behavioural therapies, including Mindfulness-Based Cognitive Therapy (MBCT; Segal, Williams, & Teasdale, 2002), Dialectical Behaviour Therapy (DBT; Linehan, 1987, 1993) and Acceptance Commitment Therapy (ACT; Hayes, Strosahl, & Wilson, 1999). Theoretical components of mindfulness include nonjudgmental, deliberate attention to one's experiences. Practice usually involves formal meditation exercises and informal attention practice, e.g., concentrating on eating or daily activities. This deliberate attention has been seen as a possible moderator of rash, thoughtless behaviour.

Mindfulness has been proposed as a strategy to reduce impulsive substance use choices even in high-risk populations. When Mindfulness was taught to a small group ($N = 10$) of incarcerated male adolescents who were partaking in court-mandated substance-abuse treatment, one remarked that Mindfulness had assisted him with his decision making: *"Instead of reacting I can just stop, think about it, feel me, you know I can actually choose instead of reacting to it"* (Himmelstein, Saul, Garcia-Romeu, & Pinedo, 2014). Another adolescent indicated Mindfulness had helped him abstain from substance use:

"I aint gonna lie. I was supposed to not come back to camp, and I was supposed to hit the blunt [marijuana], when I was in the house. 'Cause my boy, when we got back to the house, he was out there rolling a blunt. I ain't gonna lie, once I seen him in the wheelchair, I already knew I was gonna do something; drink, or something ... I used STIC [a Mindfulness technique from the intervention - Stop, Take a breath, Imagine the future consequences, and Choose]. I kinda looked at him (takes a deep breath while talking), and I took a deep breath, and just calmed down, sat down, and I was like, "damn man, it's good to see you." But at the same time I was really thinkin' about the blunt. He was like, "you gonna smoke?" I was like, "nah, I'm good." he was like, "fool, what the fuck? Since when do you say no?" I felt more me, doing me. I'm like, "nah I'm good" ... you feel me?" (Himmelstein et al., 2014)

The ability to self-regulate in the face of temptation is the exact goal of these interventions for substance use, and Mindfulness may be uniquely placed to facilitate this outcome as it has been shown to improve emotion regulation in adolescents (Deplus, Billieux, Scharff, & Philippot, 2016).

The mechanism of effect of Mindfulness has been hypothesised as greater executive control, including inhibitory control, attention, and emotion regulation (Chambers, Gullone,

& Allen, 2009; Mrazek, Franklin, Phillips, Baird, & Schooler, 2013; Oberle, Schonert-Reichl, Lawlor, & Thomson, 2012; Semple & Burke, 2012; Teper, Segal, & Inzlicht, 2013). Increased mindful attention predicts inhibitory control abilities in young adolescents (Oberle et al., 2012). There is also evidence in adult populations that consistent mindfulness practice increases grey matter in the prefrontal cortex and insula over time, which are implicated in executive control and emotional awareness (Witkiewitz, Lustyk, & Bowen, 2013). It is hypothesised that these changes may cause functional improvements in “top-down” control of emotions and impulses through improved present-moment awareness.

One could see Mindfulness as an antipode of impulsivity (i.e., more mindful = less impulsive). Similarities include concepts of present, where mindfulness encourages present-moment awareness and impulsivity is associated with a preoccupation with moment-to-moment experiences with decreased ability to limit behaviour based on future consequences (Dawe, Gullo, & Loxton, 2004; Murphy & MacKillop, 2012). Indeed, in several domains, mindfulness and impulsivity are negatively correlated (Lattimore, Fisher, & Malinowski, 2011; Murphy & MacKillop, 2012; Robinson, Ladd, & Anderson, 2014). However, several studies have found areas where mindfulness and impulsivity do not overlap. Murphy and MacKillop (2012) found that mindfulness did not correlate with sensation seeking or discounting of delayed rewards (both central components of impulsivity) and Lattimore and colleagues (2011) found that mindfulness was associated with disinhibition but not restraint. Further studies have found varying strengths and of relationships between mindfulness and impulsivity (Peters, Erisman, Upton, Baer, & Roemer, 2011; Robinson et al., 2014). These findings are consistent with the multifaceted nature of impulsivity, with current theories proposing it comprises between two (Dawe & Loxton, 2004) and five factors (Cyders et al., 2007), which will be discussed further in Chapter 3. This in itself supports the hypothesis that impulsivity is a separate construct to mindfulness. Taken together, the evidence indicates that Mindfulness and Impulsivity are related but not the same trait.

Mindfulness has demonstrated mixed evidence of efficacy. In their systematic review of the literature, Zgierska and colleagues (2009) concluded that there is preliminary evidence for the efficacy of Mindfulness or mediation-based interventions to assist substance abuse treatment outcomes and reduce relapse. The review by Goyal and colleagues (2014) also concluded that further research needed to be conducted in this area as they consider there to be insufficient evidence for Mindfulness on substance abuse treatments at present (Goyal et al., 2014). Khoury and colleagues conducted a meta-analysis of Mindfulness-based therapy

(MBT) across mental and physical health outcomes and included 8 pre-post design studies where alcohol/substance was the primary outcome. They found that the effects of MBT were larger for psychological conditions overall and Hedge's g for the 8 substance use trials ranged from .07 to 1.6 with an average of .48 (medium effect size; Khoury et al., 2013). Despite this, the authors conclude that the weight of evidence and the effect sizes of the interventions favour existing Cognitive Behavioural Therapy treatments. Thus, the gold standard evidence currently shows that mindfulness interventions range in effect according to the chosen outcome and further studies are required to determine specific effects and strengthen the evidence base for this treatment.

In adolescent populations, Mindfulness interventions show evidence for improving mental health outcomes, including depression, anxiety, stress, self-esteem, sleep quality, attention, and behavioural problems (Biegel, Brown, Shapiro, & Schubert, 2009; Bögels, Hoogstad, van Dun, de Schutter, & Restifo, 2008; Khoury et al., 2013; Raes, Griffith, Gucht, & Williams, 2014). Greater Mindfulness abilities have also been associated with reduced alcohol use (Robinson et al., 2014). While there have been few studies investigating the effects of Mindfulness alone on substance use in adolescent populations, there has been preliminary support for Mindfulness as a prevention intervention for adolescent alcohol misuse and as a supplementary addition to existing interventions.

Mindfulness techniques have been shown to reduce alcohol consumption and frequency of use compared to a wait-list control when added to CBT and MI school-based intervention as an "after-care" strategy (total $N = 67$; Harris, Stewart, & Stanton, 2017). When Mindfulness-Based Stress Reduction (MBSR) was included within a cognitive therapy and psychoeducation-based sleep intervention for 55 substance-abusing adolescents who reported sleep or daytime sleepiness problems, the authors found that program completers had lower drug problems than non-completers at the 12-month follow-up (Bootzin & Stevens, 2005). However, it is difficult to determine the cause of these effects due to the comprehensive intervention and lack of control group. Indeed, a later uncontrolled trial on a separate sample of adolescents ($N = 55$) by the same research group concluded that the improved sleep may have been the cause of the reduced substance use-related problems (Britton et al., 2010). However, this latter study did include an active control (sleep intervention + cognitive therapy versus sleep intervention + MBSR) and the Mindfulness condition reported enhanced sleep outcomes with dosage effects (self-reported frequency of Mindfulness meditation significantly predicted improved sleep outcomes; Britton et al.,

2010). Therefore Mindfulness meditation possibly indirectly improved substance-use outcomes (Britton et al., 2010). As with the adult studies, mindfulness shows promising initial findings but further investigation and more robust studies are required to determine the effects of mindfulness on adolescent substance use.

The lack of an appropriate control is widespread throughout the mindfulness efficacy literature. The meta-analysis of mindfulness-based therapy by Khoury and colleagues (2013) found that only 35 (approximately 17%) of their 209 included studies included an active psychological control condition, with the remaining using a pre-post design or comparing the mindfulness-based therapy to a waitlist control. However, there was little effect of Mindfulness-Based Therapies when compared to psychological treatments at the final follow-up. The psychological interventions included relaxation, psychoeducation, and behavioural therapies including Cognitive Behaviour Therapy. That is to say, Mindfulness-Based Therapy was effective in targeting psychological disorders, particularly anxiety and depression. It was not more effective than active controls (Khoury et al., 2013). This provides further support for the need for active controls as mindfulness may be effective but perhaps not more effective than existing treatments.

The content of active controls should be equivalent to the intervention condition. Zoogman and colleagues (2015) conducted a meta-analysis on mindfulness interventions for youths. While they categorised 60% of their sample as having active control comparisons, this appeared to be including treatment as usual. For example, other school classes and health education classes were considered active controls. They found a small overall effect size of mindfulness interventions when compared to active controls. The small effect size increased to a moderate effect size when only including clinical samples. However, it could be argued that treatment as usual is not an active control as it would be the same as not receiving the intervention, i.e., status quo. Therefore, it is uncertain what effects mindfulness has for adolescents when compared to other psychological intervention or active controls.

Both of the reviews by Zoogman and colleagues (2015) and Khoury and colleagues (2013) collapsed effects across treatment outcomes, which reduces interpretability of effectiveness for specific outcomes. For adults, there is good evidence that Mindfulness delivered in aftercare assists in relapse prevention post substance use treatment compared to existing relapse prevention strategies, including cognitive behavioural therapy (Bowen et al., 2014). Further, Mindfulness-based interventions show small-to-large effect sizes for adult

alcohol outcomes (quantity and frequency of use), with evidence of reductions in craving and withdrawal symptoms (Li, Howard, Garland, McGovern, & Lazar, 2017). However, is little recent evidence regarding the effect of mindfulness on substance use outcomes specifically for adolescents.

In sum, methodological limitations persist in investigations of Mindfulness efficacy (Farias et al., 2016; Zgierska et al., 2009). For adolescent substance use especially, there is a dearth of large-scale, well-controlled trials. Enthusiasm over the treatment has led to its application to a wide range of cohorts and disorders. Current recommendations include using rigorous scientific methods including active controls to narrow this scope and determine in what situations and for whom Mindfulness is effective and the mechanisms of its success (Farias et al., 2016; Li et al., 2017). Further, alternative explanations for effects on outcomes need to be explored, particularly in adolescents, including whether Mindfulness has effects above and beyond other psychological treatments and active controls such as relaxation (Sedlmeier et al., 2012). This thesis aims to contribute to this line of knowledge by investigating possible mechanisms by which mindfulness may impart benefits (i.e., reduced impulsivity), and by testing the use of mindfulness in a well-controlled, population-specific clinical trial.

Chapter 3: Cognitive and Personality Risk Factors for Adolescent Alcohol Use

Cognitive and personality risk factors

Over the past few decades, there has been increasing interest in mechanisms of risk pertaining to alcohol use in adolescence. Modifiable risk factors may provide targets for prevention of early onset of alcohol use, even within groups where there are disadvantageous environmental or genetic factors (Conrod, 2016; Stockings et al., 2016). As discussed previously, these modifiable risk factors may form the basis of targeted interventions or may be used to explain the mechanisms of change in universal interventions. This chapter will discuss impulsivity, alcohol-expectancies, and drinking-refusal self-efficacy as potential mechanisms of risk, which may be able to be targeted with early intervention to ameliorate future alcohol-related harm.

Impulsivity

Impulsivity is a robust predictor of concurrent and future alcohol use. Longitudinal analyses have found that impulsivity traits in childhood and early adolescence are predictive of concurrent adolescent alcohol consumption and problematic alcohol use (Stautz & Cooper, 2013) as well as future alcohol and drug misuse (Moffitt et al., 2011; Nigg et al., 2006; Tarter et al., 2003). This relationship was found to be independent of IQ, social class, parental alcohol use disorder, antisocial personality disorder, attention deficit hyperactivity disorder (ADHD), age, and conduct symptoms (Moffitt et al., 2011; Nigg et al., 2006). While trait impulsivity decreases during the transition from adolescence into adulthood, there are individual differences in the rate of decline. Slower decreases in impulsivity predict higher rates of growth in alcohol, cannabis, and cigarette use (Quinn & Harden, 2013). Individual differences in adolescent impulsivity may alter trajectories of use, and therefore this construct provides an area of interest for prevention and risk. To model this risk, we require a consistent definition and operationalisation of impulsivity.

The factors comprising impulsivity have been debated. In 2004, Dawe and Loxton noted that, while there was a consensus that impulsivity was a multi-dimensional construct, there was little consistency in the definition of these factors (Dawe & Loxton, 2004). They proposed that existing measures of impulsivity such as the Impulsiveness Questionnaire of the Eysenck Personality Scales, Cloninger's Novelty-Seeking scale, and the Barratt Impulsivity Scale were all converging on a similar impulsivity construct, that of *rash-*

spontaneous impulsiveness, or rash impulsivity. Rash Impulsivity is characterised by a tendency to act without considering the consequences of one's actions, e.g., "I often follow my instincts, hunches or intuition without thinking through all the details"(Dawe & Loxton, 2004). Similarly, they proposed that Gray's Behavioural Approach System (BAS) Funseeking, Drive, and Reward Responsiveness subscales, as well as the Sensitivity to Reward subscale of the Sensitivity to Punishment and Reward Questionnaire (SPSRQ) were measuring impulsivity related to reward sensitivity or drive (Dawe & Loxton, 2004). *Reward Drive* is characterised by an increased salience of, and stimulation by, reward, e.g., "If I see a chance to get something I want, I move on it right away". These two facets of impulsivity, Rash Impulsivity and Reward Drive, were proposed as a two-factor impulsivity model.

It was hypothesised that the two-factor model of impulsivity, comprising of Rash Impulsivity and Reward Drive, created distinct pathways of risk for substance use and other addictive behaviours. Dawe and colleagues (2004) proposed that individuals high in Reward Drive are more likely to be drawn to the perceived rewarding aspects of drug use and would be more responsive to drug-related cues, thus increasing the risk for drug use onset and maintenance. Individuals high in Rash Impulsiveness are less able to inhibit their behaviour and thus may struggle to curb approach behaviour or cease drug use despite negative consequences (Dawe et al., 2004). They further supported the two-factor model with neurological research. It was argued that Reward Drive was related to hyperactivity in the dopaminergic pathways of reward, particularly those extending from the ventral tegmental area (VTA) to the nucleus accumbens, creating heightened reinforcement for positive drug cues and subsequent craving. Rash Impulsiveness, on the other hand, was thought to be related to less efficient functioning of the orbitofrontal cortex, which is involved in regulating approach behaviour and the ability to inhibit responses (Dawe et al., 2004). This model of substance use was named the 2-Component Approach to Reinforcing Substances (2-CARS) model where each impulsivity factor is analogous to a car. Reward Drive can be seen as a powerful car speeding towards a salient goal. The driver of the car could stop but they are likely to reach their appetitive goal with greater velocity and find it more difficult to stop quickly due to the power of the engine. The second car representing Rash Impulsiveness also speeds towards an appetitive goal, however, this car has a fault in the braking system such that the driver finds it difficult to stop when hazards (representative of negative behavioural consequences) are apparent. The 2-CARS model is represented in Figure 1.1 (Gullo & Dawe, 2008).

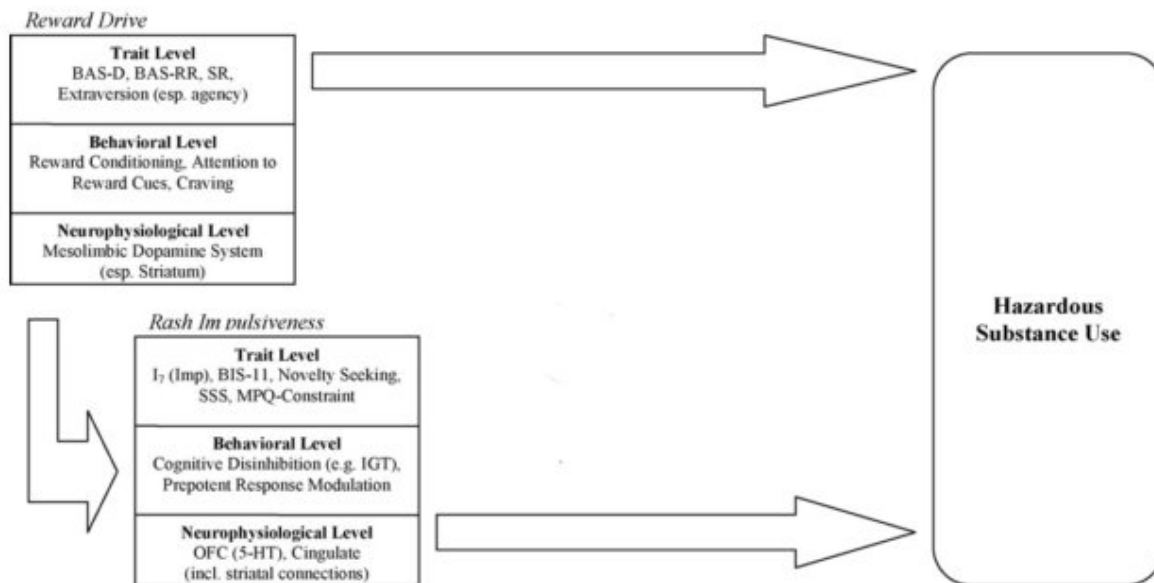


Figure 3.1 The 2-CARS model. Modified from Gullo and Dawe (2008).

Once the concept of impulsivity was recognised as multi-factorial, a discussion arose as to which and how many factors it comprised (Gullo, Loxton, & Dawe, 2014). One theory of impulsivity included four factors: negative Urgency, (lack of) Premeditation, (lack of) Perseverance, and Sensation seeking (UPPS; Whiteside & Lynam, 2001), which was later expanded to a five-factor model with the inclusion of positive urgency (UPPS+P; Cyders et al., 2007) among others. In a critical review of impulsivity and addiction, Gullo et al. (2014) that these additional impulsivity factors did not uniquely contribute to substance use beyond the general categories of disinhibition (rash impulsiveness) and approach behaviour (reward drive). Thus, a two-factor model was more parsimonious, more evidence-based and more consistent with current neuroscientific findings (Gullo, Loxton, et al., 2014).

The 2-CARS model has been proposed for use in adolescent populations. In their review, Gullo and Dawe (2008) linked the 2-CARS model to the adolescent period, stating that this developmental period is associated with increased reward saliency and inhibitory impairment due to hyperactivity in the limbic system and immature prefrontal cortex neurodevelopment. This research group later supported the predictions of the 2-CARS in two populations with developing alcohol use (Gullo, Ward, Dawe, Powell, & Jackson, 2011). A large sample of young adults ($N = 213$) from the United Kingdom (mean age = 19.02 years, $SD = .99$ years) and 286 Australian young adults (mean age = 21.01 years, $SD = 5.66$ years) participated in cross-sectional surveys. The authors found that rash impulsivity was a robust predictor of hazardous alcohol use. Reward Drive predicted hazardous alcohol use for the UK

sample but not the Australian sample. It is noteworthy, though, that the differences in path effect sizes between UK and Australia were not significantly different. They concluded that Rash Impulsiveness is the more robust direct predictor. However, Reward Drive may be affecting alcohol use via indirect pathways (Gullo et al., 2011). Further evidence for the differing influences of the two factors can be found in the paper by Wood and colleagues (2013). The authors found that the rash impulsivity and reward drive had differential effects on adolescent substance use through mediating relationships on risk-taking, prosocial behaviour, and family environment (Wood, Dawe, & Gullo, 2013). Thus, the 2-CARS shows appropriateness and utility for use in adolescent populations, with each factor providing separate pathways of risk for adolescent substance use.

The 2-CARS model has been supported in a range of contexts. It has shown predictive and aetiological utility in young adult populations (Gullo, Loxton, et al., 2017; Gullo et al., 2011) and for various addictive behaviours, including alcohol, heroin, ecstasy, and cannabis use, disordered eating, and pathological gambling (Dawe & Loxton, 2004; Dissabandara et al., 2014; Egan, Kambouropoulos, & Staiger, 2010; MacLaren, Fugelsang, Harrigan, & Dixon, 2012). Despite these strengths, a limitation of the 2-CARS model is the focus on biological and behavioural processes without considering cognitive factors that could explain the differential pathways of risk. It has been proposed that alcohol-related cognitions derived from Bandura's Social Learning and Social Cognitive Theories play a mediating role. That is, impulsivity alters cognitions regarding alcohol and these altered cognitions impact on alcohol consumption (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010a). Before these relationships are discussed further, the background of these cognitions and their existing applications to alcohol risk will be explained.

Alcohol-related cognitions

Social Learning Theory and Social Cognitive Theory

Social Learning Theory (Albert Bandura, 1977) arose in an era of behaviourism and psychodynamics. Behaviourists advocated for a view of behaviour as learned through experience and conditioning and Freudian theories proposed that subconscious needs, impulses, and drives underlie motivation. For Bandura (1977), these explanations were unhelpful in predicting psychological change or failed to explain behaviour learned without evidence of rehearsal. They also did not explain different changes in behaviour depending on context.

Bandura (1977) expanded on behaviourist theories, agreeing that learning was achieved through response consequences (positive and negative reinforcement or punishment). However, he argued that learning occurred within an environmental and social context and that “*psychological functioning is a continuous reciprocal interaction between personal, behavioural, and environmental determinants*” (Bandura, 1977). Thus, we can learn by observing others (modelling) as well as through direct experience. Response consequences can also be vicariously processed, e.g., learning the outcomes of an action through observation of someone’s experience in one’s social environment. Bandura completed experiments to show this vicarious social learning, including the famous Bobo doll experiment (Bandura, Ross, & Ross, 1961), which showed children observing the aggressive actions of adults towards a ‘Bobo doll’ toy and later mimicking this behaviour without prompting. This process of gathering information from which one can make hypotheses regarding the outcomes of one’s actions and one’s efficacy in carrying out behaviour was proposed as a cognitive mechanism that mediated the effects of environmental input on behaviour. These concepts were the basis for outcome expectancies and self-efficacy.

Social Cognitive Theory (SCT), which Bandura espoused in his seminal work ‘Social Foundations of Thought and Action’ (1986), expanded on SLT. Bandura proposed that “*human functioning is explained in terms of a model of triadic reciprocity in which behaviour, cognitive and other personal factors, and environmental events all operate as interacting determinants of each other*”. Bandura postulated that individuals act according to the perceived reinforcing outcomes the action would afford, i.e., their expectancies regarding the outcomes of their actions. Additionally, he proposed that individuals develop expectancies surrounding their abilities to carry out certain actions, which is termed self-efficacy. SCT identifies the cognitive factors of outcome expectancies and self-efficacy as central to behaviour initiation and maintenance (Bandura, 1986). When seen within the context of triadic reciprocity, vicarious and direct environmental influences and behavioural consequences may help to inform and reinforce these cognitive factors, just as expectancies and self-efficacy may influence actions and shape one’s chosen environment.

Alcohol Expectancies

Outcome expectancies are pertinent to alcohol use and have been applied to both adult and adolescent drinking. Alcohol expectancies are one’s beliefs of the rewarding and harmful outcomes of drinking alcohol (Bandura, 1977; Brown, Christiansen, & Goldman, 1987). One might believe that drinking alcohol will make them more able to socialise competently (a

positive alcohol expectancy). They may also believe that drinking alcohol will cause them increased risk of injury (a negative alcohol expectancy; Jones & McMahon, 1993; Leigh, 1989). These expectancies may alter their intentions to consume alcohol and therefore their alcohol use behaviour.

A second, related mechanism of risk for alcohol use which is gaining popularity and support is drinking motives (Kuntsche, Knibbe, Gmel, & Engels, 2005). Drinking motives extend on alcohol expectancies and ask not only what people expect from alcohol use but what do people wish to gain from drinking alcohol. Thus, drinking motives have been shown to mediate the effects of alcohol expectancies on alcohol use (Diep, Schelleman-Offermans, Kuntsche, De Vries, & Knibbe, 2016; Kuntsche, Knibbe, Engels, & Gmel, 2007). However, drinking motives are only applicable to existing drinkers whereas alcohol expectancies need not arise from direct personal experience or desires. As proposed in SLT, expectations may be learned from personal experimentation or social contexts (Albert Bandura, 1977, 1986). Alcohol expectancies can be formed through observation of friends and family and even broader social influences such as media or advertising. Indeed, alcohol expectancies have been reported in children as young as four years of age (Jester, Wong, et al., 2015; Kuntsche, 2017). Therefore, alcohol expectancies provide greater scope in investigating alcohol outcomes in non-drinking adults and alcohol-naïve populations such as adolescents.

One of the first papers to investigate alcohol use and patterns of alcohol expectancies was conducted in 1980 by Brown, Goldman, Inn and Anderson. Experiments had previously been conducted linking single alcohol expectations with the relevant behavioural response, but this was the first study to identify a range of adult alcohol expectancies and associate them with consumption (Brown, Goldman, Inn, & Anderson, 1980). They found that heavier consumption of alcohol was associated with positive expectancies of sexual enhancement (e.g., “after a few drinks, I am more sexually responsive”), and arousal/aggressive behaviour (e.g., “drinking makes me feel flushed” and “after a few drinks it is easier to pick a fight”). The same research group built on these results in future papers, finding that as positive alcohol expectancies increased, so did alcohol consumption, e.g., occasional drinking college students had the lowest positive alcohol expectancies, followed by non-excessive drinking medical students, non-excessive drinking medical patients, moderate drinking college students, heavy drinking college students, excessive drinking medical patients and finally alcoholics who had the highest positive alcohol expectancies (Brown, Goldman, & Christiansen, 1985). This pattern was replicated by other research groups who also found that

alcohol expectancies were associated with drinking patterns and identified their utility in alcohol use onset, maintenance, and treatment outcomes (Knight & Godfrey, 1993; Lee & Oei, 1993).

Initial expectancy research exclusively focused on positive alcohol expectancies before focus began to shift to include negative expectancies as important predictors of alcohol use and treatment outcomes (Fromme, Stroot, & Kaplan, 1993; Jones & McMahon, 1994; Young & Oei, 1993). It is the combination of positive and negative beliefs that influence drinking behaviour. It is easy to imagine that if someone has high expectations of the rewarding aspects of alcohol use and very few expectations of the undesirable aspects of alcohol use, then they will be at greater risk of drinking if given the opportunity. Lee, Greely, and Oei (1999) investigated the relative importance of positive and negative alcohol expectancies on alcohol consumption on a community sample of 193 adults. They measured participants expectations around whether drinking alcohol will have a detrimental effect on their affect or cause them to become dependent or lose control of their alcohol use (negative expectancies) as well as positive alcohol expectancies surrounding improved sexual, social, and cognitive functioning and reduction in tension. Alcohol outcomes were weekly consumption, frequency of consumption, and quantity per drinking session. While negative expectancies accounted for a larger percentage of variance in frequency of consumption (i.e., higher negative expectancies predicted less frequent consumption), positive expectancies explained more unique variance in quantity per session (i.e., higher positive expectancies were associated with increased consumption per session). Further, both positive and negative alcohol expectancies predicted unique variance in each alcohol consumption outcome and the authors concluded that both positive and negative alcohol expectancies should be included when predicting alcohol use outcomes (Lee, Greely, & Oei, 1999).

Leigh and Stacy (2004) also found evidence to support separate mechanisms of effect of positive and negative expectancies. They found that positive alcohol expectancies predicted consumption amongst existing drinkers, and that negative expectancies predicted abstinence from drinking (Leigh & Stacy, 2004). Despite this, commonly used measurement tools only assess positive alcohol expectancies, so information about negative expectancies is missing in much of the existing literature. Subsequently alcohol expectancies have been identified as a risk factor for initiation and maintenance of Alcohol Use Disorders (Connor et al., 2016) and have been frequent targets for alcohol use interventions (Scott-Sheldon, Terry, Carey, Garey, & Carey, 2012; Young, Connor, & Feeney, 2011). Although cautions about

inconsistent results have been made along with calls for greater clarification of treatment mechanisms (Jones, Will, & Fromme, 2001)

Alcohol Expectancies Across the Lifespan

Alcohol expectancies change with age and experience with alcohol. Children as young as 5 years old have thoughts about the effects of alcohol, usually learned from their parents or persons within their sphere of influence (Jayne & Valentine, 2015). While alcohol expectancies are predominantly negative during childhood (Jayne & Valentine, 2015; Leigh & Stacy, 2004), positive expectancies increase into adolescence and early adulthood. Copeland and colleagues found that the positive alcohol expectancies of children change as they progress to adolescence. They assessed the alcohol expectancies of 76 second-graders, 59 third-graders, 65 fourth-graders, 63 fifth-graders, and 50 sixth-graders (total $N = 313$) and then followed them up over 6, 12, and 18 months. There was evidence that positive alcohol expectancies increased with age, as older cohorts had higher alcohol expectancies than younger cohorts cross-sectionally, and positive alcohol expectancies increased over time but especially between third and fourth grade, and fourth and fifth grade (Copeland, Proctor, Terlecki, Kulesza, & Williamson, 2014).

This increase in positive expectancies continues into mid-adolescence and predicts alcohol consumption. Findings from the Millennium Cohort Study indicate that positive expectancies predict drinking at age 11, and negative expectancies predict abstinence (Kelly Yvonne et al., 2015). For 12-17-year-olds, Leigh and Stacy (2004) found that both positive and negative expectancies predicted alcohol outcomes, but amongst existing drinkers negative expectancies were not a significant predictor of use, indicating the increasing importance of positive expectancies and their impact on consumption (Leigh & Stacy, 2004). In a sample of 750 Norwegian adolescents surveyed and then followed-up annually for 2 years ($M = 13.3$ years, $SD = .3$ at Time 1) positive alcohol expectancies predicted alcohol use at each subsequent year (Aas, Leigh, Anderssen, & Jakobsen, 1998). They also found that for adolescents who had not yet consumed alcohol at Time 1, having positive alcohol expectancies at Time 1 was related to alcohol initiation at Time 2. Further, the relationship between alcohol use and positive alcohol expectancies was reciprocal as increased alcohol use also predicted subsequent increases in positive alcohol expectancies (Aas et al., 1998).

Age also appears to impact alcohol expectancies in terms of strength and the areas of expectancies present. Adults endorse a range of expectancies (Gullo et al., 2010a) and

negative expectancies are a stronger predictor of alcohol outcomes for existing drinkers 36-60 years of age compared with those older or younger than this (Leigh & Stacy, 2004).

Comparatively, adolescent drinking is more influenced by expectancies regarding the positive social outcomes of alcohol consumption (Jester, Wong, et al., 2015; Smith, Goldman, Greenbaum, & Christiansen, 1995; Tomlinson & Brown, 2012; Young-Wolff et al., 2015), which is unsurprising due to the influence of peers on appraisals and norms during this developmental period (Colder et al., 2017).

Jester, Wong and colleagues (2015) investigated changes in alcohol expectancies in childhood using a sample of 614 children and adolescents assessed at 3-year intervals (ages 6-8 at Time 1; ages 15-17 at Time 4). Additionally, 460 of the children had one or both parents with a current or previous alcohol use disorder. A bidirectional effect of social alcohol expectancies was found. Social/relaxation expectancies (e.g., “drinking beer or wine would help me make friends/relax me”) predicted earlier onset of binge drinking and drinking to intoxication and onset of alcohol use increased social/relaxation expectancies. Social/relaxation expectancies were higher for children of alcoholics from Time 2 (aged 9-11 years) onwards (Jester, Wong, et al., 2015) indicating possible vicarious learning as predicted by SLT and SCT.

This reciprocal relationship was initially found by Smith and colleagues (1995) who showed that increased adolescent expectancies regarding social facilitation were predictive of increased future alcohol consumption and high consumption was associated with increased endorsement of social facilitation expectancies (Smith et al., 1995). Tomlinson and Brown (2012) also supported the relationship between social alcohol expectancies and alcohol outcomes in adolescents but found that being socially anxious reduced this relationship. They hypothesised that socially anxious youths had been less exposed to situations where drinking may have been an option. Thus they were consuming less alcohol and not experiencing the reciprocal additive effects of alcohol consumption on social alcohol expectancies (Tomlinson & Brown, 2012). It is also possible that genetic influences increase after the onset of alcohol use. Young-Wolff and colleagues concluded in their analysis of 1,292 adolescent twins that environmental factors can account for much of the difference in alcohol-related expectancies prior to the onset of alcohol use, but that genetic factors are increasingly predictive of expectancies after onset (Young-Wolff et al., 2015). They speculated that this may have been due to genetic differences in physiological responses becoming apparent once alcohol had been consumed. Prior to this, environmental factors played a larger role.

SCT predicts that context is important to expectancies and behaviours and yet few studies take this into account. Indeed, social expectancies should and are influenced by social contexts, such as peer drinking norms (Colder et al., 2017). Alcohol expectancies were found to be associated with alcohol use modality in a large sample (N = 1,580) of adolescents. Those who were social drinkers reported social-related expectancies and those who experienced alcohol-related problems expected alcohol to improve the cognitive and motor functioning (Christiansen & Goldman, 1983). These factors were as predictive of alcohol use as demographic variables.

Drinking Refusal Self-Efficacy.

As discussed previously, SCT identifies expectations regarding control over one's actions (self-efficacy) as crucial in explaining behavioural onset and continuance (Bandura, 1986). Self-efficacy within the context of alcohol research typically focuses on an individual's perceived ability to refrain from drinking in various situations. Self-efficacy is a robust predictor of alcohol use disorder (AUD) treatment outcomes, where decreased self-efficacy predicts poorer outcomes (Adamson, Sellman, & Frampton, 2009; Kadden & Litt, 2011; Oei & Baldwin, 1993; Young et al., 2011; Young, Oei, & Crook, 1991).

Drinking refusal self-efficacy was conceptualised after alcohol expectancies. Young and colleagues (1991) proposed that self-efficacy may contribute to treatment and relapse outcomes in conjunction to alcohol expectancies. In developing the Drinking Self-Efficacy Questionnaire (later modified as the Drinking Refusal Self-Efficacy Questionnaire), they demonstrated that drinking refusal self-efficacy combined with personality factors were able to distinguish between problem and non-problem drinkers in two community samples (Young et al., 1991). Factor analyses revealed three separate domains of self-efficacy relevant to drinking refusal. These were 1) Opportunistic, i.e., self-efficacy when the opportunity to drink is presented, 2) Emotional Relief, i.e., self-efficacy when feeling tense or experiencing negative emotions, 3) Social Pressure, i.e., self-efficacy when in a social context with drinking present (Young et al., 1991). Drinking refusal self-efficacy moderates the effect of protective behavioural strategies on alcohol consumption (Ehret, Ghaidarov, & LaBrie, 2014) and has been proposed as a mechanism for change and stages of change in alcohol interventions (Black & Chung, 2014; Cho, 2005). However, much of the research into drinking refusal self-efficacy does not examine this construct alone but as a co-predictor with alcohol expectancies, due to their shared theoretical background as alcohol-related cognitions derived from Social Cognitive Theory.

Relationship between alcohol expectancies and drinking refusal self-efficacy

Oei and Baldwin (1993) identified drinking refusal self-efficacy and alcohol expectancies as crucial in the acquisition and maintenance of alcohol use and abuse. They proposed that the acquisition phase was characterised by a cognitive consideration of alcohol behaviour, which was influenced by one's beliefs of the outcomes of alcohol use (expectancies; Oei & Baldwin, 1993). Self-efficacy is then hypothesised to mediate the path between consideration and behavioural outcome, e.g., positive beliefs about alcohol use outcomes may not impact behaviour if drinking refusal self-efficacy is high. The pairing of alcohol opportunity and behaviour (influenced by expectancies and drinking refusal self-efficacy) then creates a conditioned response, where alcohol-related cues may prompt automatic processing and craving. Further, beliefs about drinking and self-efficacy outcomes are also reinforced. These conditioned associations and strengthened beliefs are proposed as a mechanism behind the maintenance of alcohol use and abuse (Oei & Baldwin, 1993). These relationships are represented in Figure 3.2 below.

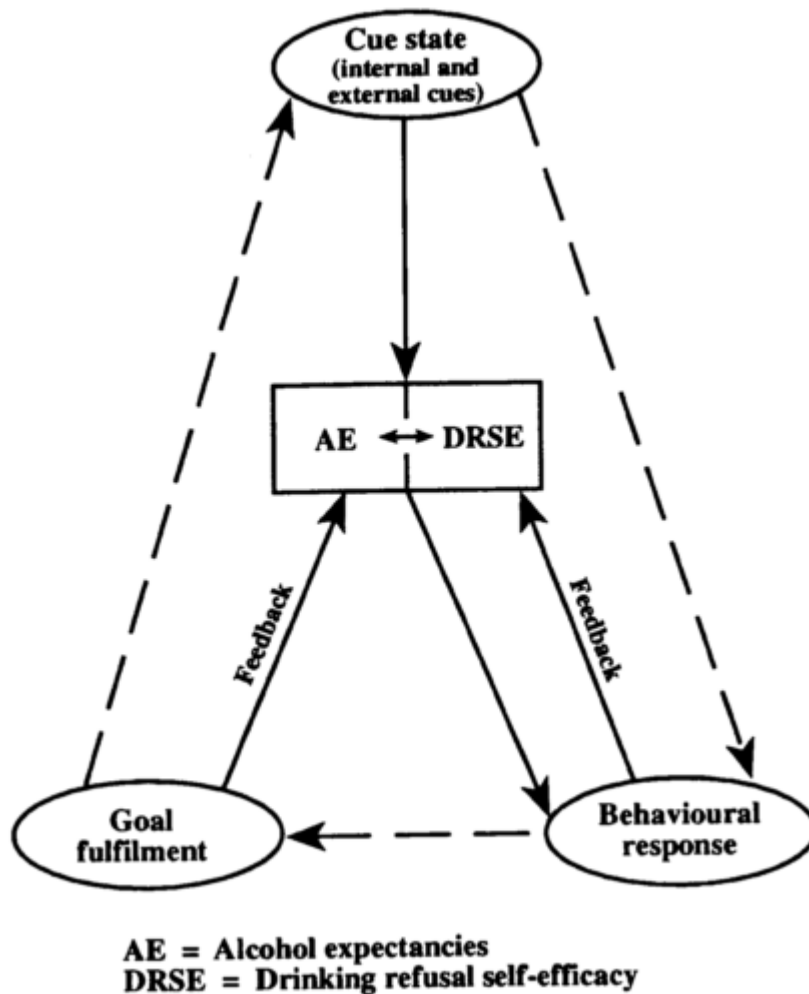


Figure 3.2 “A conceptual two-process model of alcohol use and abuse.” Replicated from Oei and Baldwin (1993).

Note. “Solid lines denote links between cognitive constructs and the stages of drinking behaviours; dashed lines indicate the temporal sequence of drinking behaviours. For practical purposes, cognitive constructs are hypothesised to intervene between awareness of the need state and performance of the behavioural response.” (Oei & Baldwin, 1993)

Initial investigations of the relationship between alcohol expectancies and drinking refusal self-efficacy supported their related but differential effects on alcohol outcomes. Where alcohol expectancies predicted the quantity of alcohol use, drinking refusal self-efficacy predicted the frequency of use and maximum consumption per single drinking occasion in a community sample (Lee & Oei, 1993). The differential effects of alcohol expectancies and drinking refusal self-efficacy on frequency and quantity of alcohol consumption were supported using structural equation modelling by Baldwin, Oei, and Young (1993). In young adults, both drinking refusal self-efficacy and positive alcohol

expectancies explain unique variance in levels of alcohol dependence as well as frequency and quantity of use (Young, Connor, Ricciardelli, & Saunders, 2006). These beliefs are specific to alcohol use and do not reflect general beliefs about addictive substances, as shown by Oei and Burrow (2000) who found that alcohol expectancies and drinking refusal self-efficacy did not predict tobacco or caffeine consumption (Oei & Burrow, 2000).

The clinical utility of the interactive effects of these constructs was shown by Brown and colleagues (1998). They found that decreases in expectancies regarding the effect of alcohol were greater over alcohol and drug treatment if initial drinking refusal self-efficacy was low. This may mean that decreased drinking refusal self-efficacy can lead to the willingness to re-examine other alcohol-related cognitions. Connor and colleagues (2007) found that increased alcohol expectancies and decreased drinking refusal self-efficacy were associated with increased severity of dependence in a sample of alcohol-dependent treatment seekers (Connor, Gudgeon, Young, & Saunders, 2007). Further, it is proposed that different profiles of alcohol expectancies and drinking refusal self-efficacy can distinguish between social drinkers, binge drinkers, and heavy drinkers (Morawska & Oei, 2005; Oei & Morawska, 2004).

Connor and colleagues (2011) tested the effect of the SCT variables on adolescent alcohol use. They investigated the prospective effects of alcohol expectancies and drinking refusal self-efficacy on alcohol use cross-sectionally and then 1 year later using an adolescent sample (Time 1 $N = 192$, Time 1 mean age = 13.8 years, $SD = 0.5$ years). They found that the effect of alcohol expectancies on Time 2 alcohol use was mediated by drinking refusal self-efficacy, that is increasing alcohol expectancies significantly decreased drinking refusal self-efficacy, which in-turn significantly increased alcohol use 1 year later (see Figure 3.3 below). This mediational pathway is predicted by the two-process model of acquisition and maintenance of alcohol use and abuse (Oei & Baldwin, 1993). These mediational relationships have also been found in adult and alcohol-dependent populations as well as in cannabis users (Baldwin et al., 1993; Connor et al., 2008; Connor, Gullo, Feeney, Kavanagh, & Young, 2014).

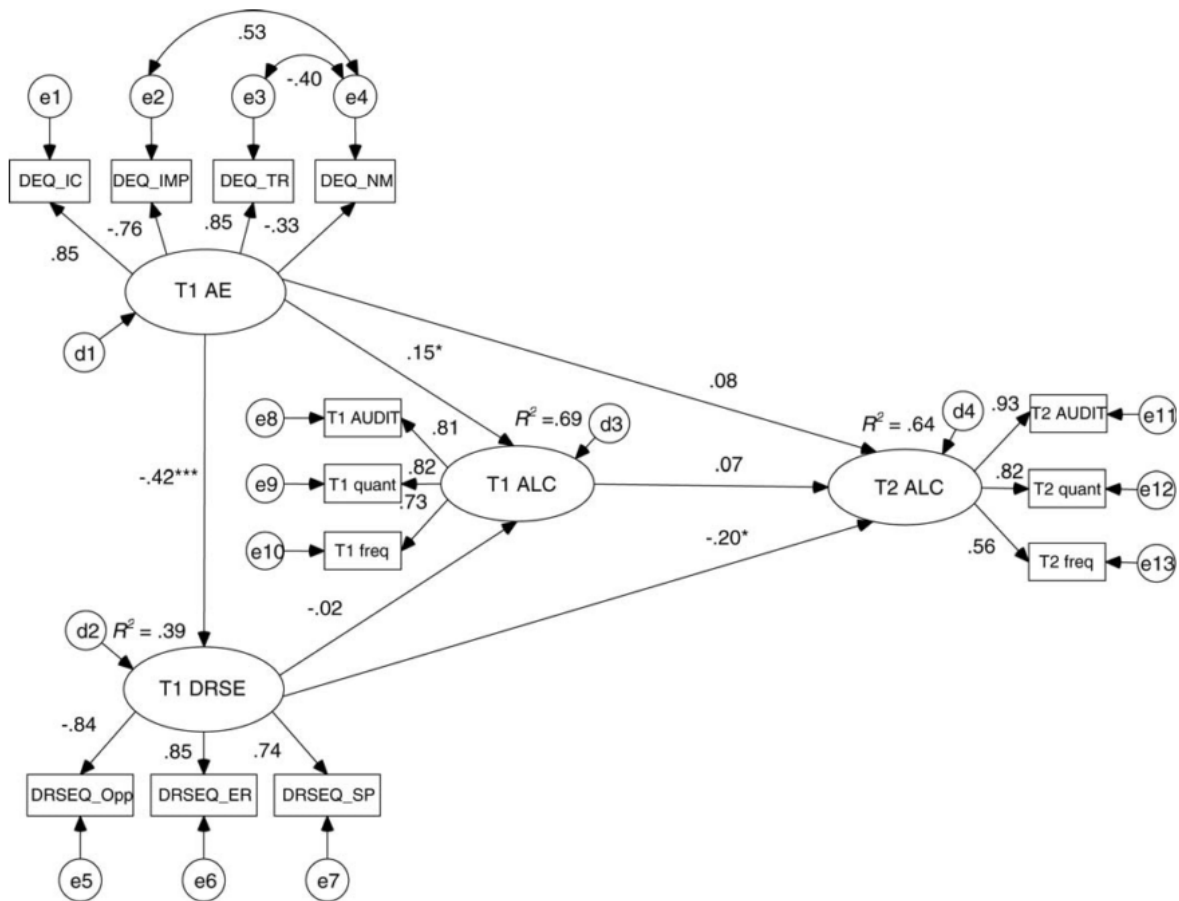


Figure 3.3. “Prospective structural model of AEs [Alcohol Expectancies] and DRSE [Drinking Refusal Self-Efficacy] predicting future drinking (N= 192).” Replicated from Connor et al. (2011).

Note. “Model includes gender, age, peer drinking, past-year cigarette use, SDQ [Strengths and Difficulties Questionnaire] Difficulties and SDQ Strengths score as covariates, each with direct effects on all latent variables. However, for clarity, these variables are not depicted.” (Connor, George, Gullo, Kelly, & Young, 2011)

Reduced drinking refusal self-efficacy is a risk factor for future alcohol use in adolescents and mediates the relationship between alcohol use and other risk factors such as alcohol expectancies and impulsivity (Connor et al., 2011; Gullo et al., 2010a; Harnett, Lynch, Gullo, Dawe, & Loxton, 2013). While both cognitions predict alcohol use, drinking refusal self-efficacy appears to be the more robust predictor, explaining larger amounts of variance and mediating the effects of alcohol expectancies and other risk factors on alcohol use (Connor et al., 2008, 2011; Ehret et al., 2014).

Associations between Impulsivity and Alcohol-Related Cognitions

Building on the 2-CARS model, Gullo and colleagues (2010) proposed that the SCT factors of Alcohol Expectancies and Drinking Refusal Self-Efficacy may represent specific cognitive mechanisms by which Rash Impulsiveness and Reward Drive impact hazardous alcohol use (see Figure 3.4 below for a visual representation of the proposed model and hypothesised relationships). They argued that individuals higher in Reward Drive would be predisposed (by virtue of their hyperactive dopaminergic reward system) to focus on the positive aspects of alcohol consumption and that the saliency of the learned rewards of alcohol use would be heightened. Thus they proposed that Alcohol Expectancies would mediate the relationship between Reward Drive and hazardous alcohol use (Gullo et al., 2010; hypothesis 1). Further, due to the conceptualisation of Rash Impulsiveness as reflecting individual difference in inhibitory control, the authors proposed that individuals high in Rash Impulsiveness would have lower Drinking Refusal Self-Efficacy (i.e., reduced belief in their ability to inhibit their impulse to drink) and that this cognitive factor would mediate the relationship between Rash Impulsiveness and hazardous alcohol use (hypothesis 2). That is, those people with a tendency to act without thinking may believe they would be less able to refuse alcohol. This lowered self-efficacy increases the likelihood of them drinking, which in turn reinforces beliefs of low drinking refusal self-efficacy. Finally, they hypothesised that the cognitive mediators would also be interdependent, with increased positive alcohol expectancies decreasing drinking refusal self-efficacy (hypothesis 3).

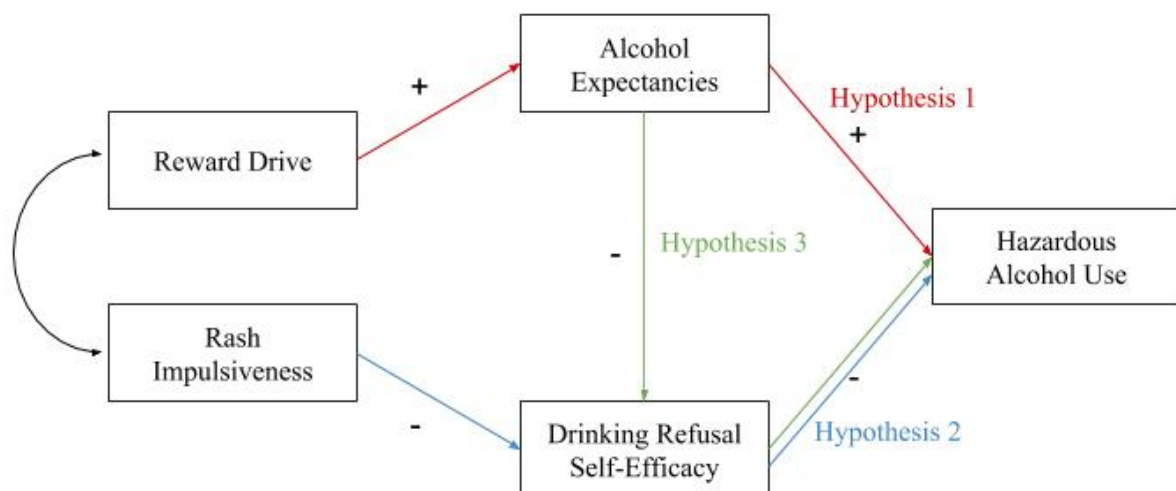


Figure 3.4. A simplified diagrammatic illustration of the hypothesised mediating relationships proposed by Gullo and colleagues (2010).

Gullo and colleagues (2010) investigated these hypotheses using Structural Equation Modelling in a sample of young adults and a sample of treatment-seeking substance users. In the young adult sample ($N = 342$, Mean age = 21.16 years, $SD = 5.16$ years) hypothesis 2 was supported (partial mediation effect). While Reward Drive did predict alcohol expectancies, no direct relationship was found between alcohol expectancies and hazardous alcohol use when controlling for socially-desirable responding. Instead, the effect of expectancies on alcohol use was mediated through drinking refusal self-efficacy (thus supporting and expanding on hypothesis 3). The same outcomes were found in the treatment-seeking substance users sample ($N = 178$, Mean age = 34.1 years, $SD = 7.7$ years); however, full, rather than partial, mediation was found for hypothesis 2. Thus, the authors were successful in identifying distinct cognitive mediators of Rash Impulsiveness and Reward Drive on hazardous alcohol use. From this research, novel mechanisms of change can be targeted in intervention efforts. This model has since been referred to as the bioSocial Cognitive Theory (bSCT; Papinczak, Connor, Harnett, & Gullo, 2018) and will be referred to as such in the proceeding chapters.

The bSCT has also been shown to provide a good fit to data from adults and young adults. Kabbani and Kambouropoulos (2013) utilised a sample of 132 adults ranging from 18 – 70 years of age (mean age, = 33.96 years, $SD = 14.91$ years) and tested the bSCT model compared to hypotheses derived from the *Acquired Preparedness Model* (APM). The APM predicts that disinhibition (analogous to Rash Impulsiveness) impacts alcohol expectancies, rather than Reward Drive as theorised in the bSCT model (Smith & Anderson, 2001). They found that the bSCT model provided a better fit to the data than the alternative model. As predicted Reward Drive was directly associated with increased Alcohol Expectancies, which increased alcohol use and Rash Impulsiveness increased perceived impaired control of alcohol use (a different but theoretically consistent measure of DRSE), which in turn increased alcohol use (Kabbani & Kambouropoulos, 2013). Harnett and colleagues (2013) replicated the bSCT model in a sample of 378 undergraduate psychology students (Mean age = 20.32 years, $SD = 4.45$ years). They found support for the mediating impact of positive alcohol expectancies on the relationship between Reward Drive and Hazardous Drinking (see Figure 3.5). They also found that Drinking-Refusal Self-Efficacy partially mediated the effect of Rash Impulsivity on Hazardous Drinking, as predicted. However, they did not find that Drinking Refusal Self-Efficacy mediated the relationship between Reward Drive and Hazardous alcohol use through the effect of Positive Alcohol Expectancies. Further, they

found statistical support for an additional path in the model between Rash Impulsivity and Positive Alcohol Expectancies. They hypothesised that participants high in Rash Impulsiveness may be cognitively biased towards discounting negative outcomes of drinking and therefore would have elevated Positive Alcohol Expectancies.

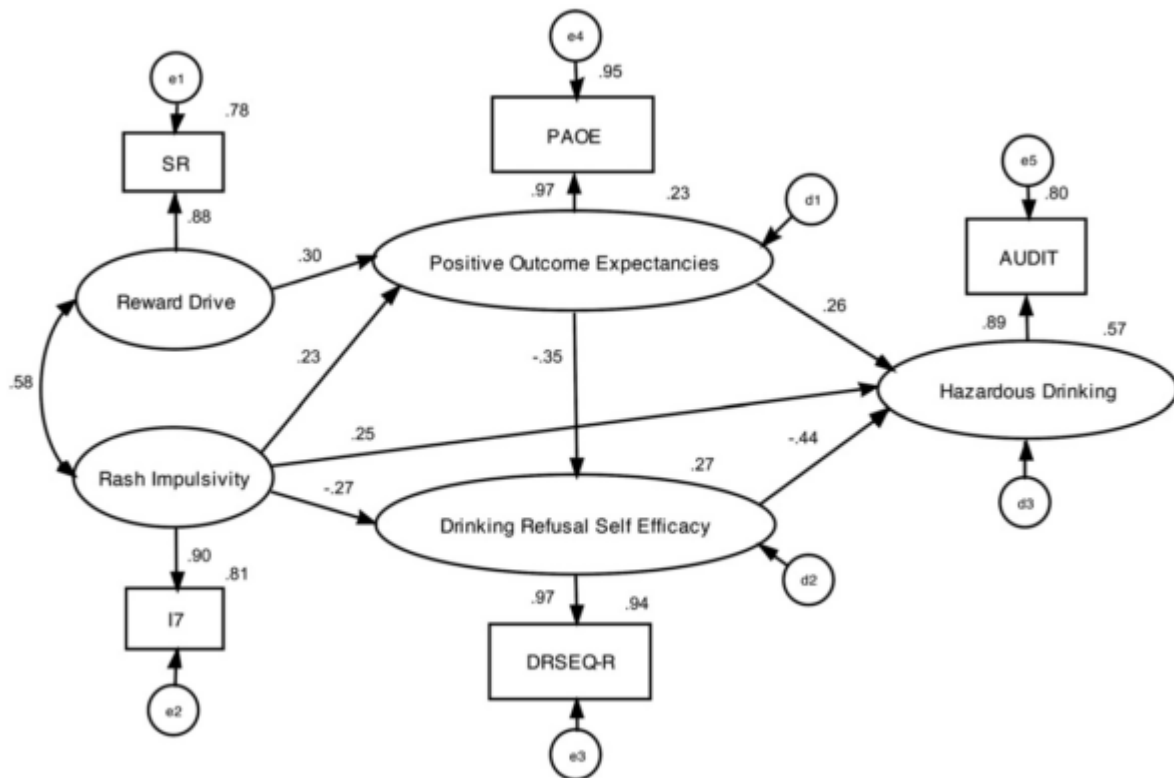


Figure 3.5. “Final structural model of the relationship between impulsivity, alcohol-related cognition, and hazardous alcohol use.” Replicated from Harnett et al. (2013).

Note. “Standardised parameter estimates are presented. Estimates appearing above-right of endogenous variables indicate the amount of variance explained (R²). All estimates are statistically significant at $p < .05$. Note: SR, Sensitivity to Reward; PAOE, Positive Alcohol Outcome Expectancies; I7, Impulsiveness scale; DRSEQ-R, Drinking Refusal Self-Efficacy Questionnaire-Revised; AUDIT, Alcohol Use Disorders Identification Test.” (Harnett et al., 2013)

As mentioned previously, genetic factors appear to influence the development of disordered alcohol use (Young-Wolff et al., 2015). Several papers have investigated the impact of genes on the components of the bSCT model. Gullo and colleagues (2014) found that the relationship between rash impulsiveness and alcohol consumption in an alcohol dependent population was not moderated by genes commonly implicated in risky alcohol use (the DRD2/ANKK1 gene). The lack of gene moderation suggests that the effect of rash

impulsiveness on alcohol consumption may be more driven by psychological rather than biological mechanisms (Gullo, St. John, et al., 2014). However, the A1 allele of the DRD2 gene is associated with earlier onset of use and subsequently lowered drinking-refusal self-efficacy (Connor et al., 2008), and binding of the C allele of the DRD2 gene is associated with increased alcohol dependence for males (Swagell et al., 2012). Leamy and colleagues (2016) hypothesised that polymorphisms implicated in alcohol use within the dopamine (DRD2/ANKK1 gene) and serotonin (HTR2A gene) systems would impact relationships within the bSCT model. The authors did not find evidence for the polymorphisms moderating the relationships between impulsivity, cognitions and alcohol use. However, there was a novel finding of the direct impact of the HTR2A gene, as the presence of this gene was associated with lower positive alcohol expectancies, higher drinking refusal self-efficacy and reduced alcohol misuse (Leamy, Connor, Voisey, Young, & Gullo, 2016). Together these findings suggest that there may be some impact of genes implicated in alcohol risk on alcohol use onset and dependence and this may indirectly affect factors within the bSCT model. However, the relationships within the bSCT model are not fully explained by genetic factors.

Alternative theories to bSCT, such as the Acquired Preparedness Model, have been proposed for the cognitive mechanisms by which impulsivity imparts risk for alcohol use. The theoretical mechanism behind the link between disinhibition and alcohol expectancies theorised in the Acquired Preparedness Model relationship is that there is a learning bias created by trait disinhibition (Smith & Anderson, 2001). That is, disinhibited individuals are more likely to encode positive alcohol outcomes and less likely to encode adverse consequences of alcohol use into their memory, biasing them towards increased positive and decrease negative alcohol expectancies (Smith & Anderson, 2001). These altered expectancies then place them at greater risk of alcohol misuse. The Acquired Preparedness Model has accumulated promising but mixed support in the literature (Anderson, Smith, & Fischer, 2003; Corbin, Iwamoto, & Fromme, 2011; Lopez-Vergara et al., 2012; McCarthy, Kroll, & Smith, 2001; Settles, Cyders, & Smith, 2010).

Despite some support for the Acquired Preparedness Model, it includes some limitations and conceptual gaps. The original Acquired Preparedness Model only included one factor of impulsivity, disinhibition, whereas the current consensus is that at least two factors should be considered in explaining substance use (Stautz, Dinc, & Cooper, 2017). Only a few studies have altered the Acquired Preparedness Model to include a second factor of impulsivity and there is inconsistency with how these factors are conceptualized, e.g.,

positive and negative urgency (Settles et al., 2010) versus behavioural approach system and behavioural inhibition sensitivity (Lopez-Vergara et al., 2012) versus sensation seeking, impulsivity, and novelty seeking (Corbin et al., 2011). Secondly, the causal pathways within the model may be better explained by other mechanisms. Rash Impulsiveness has been related to alcohol expectancies even within the bSCT literature (e.g., Harnett et al., 2013). However, the evidence shows that the effects of Rash Impulsiveness are better explained by drinking refusal self-efficacy (Connor et al., 2011; Gullo et al., 2010a; Harnett et al., 2013). Further, while alcohol expectancies are an important risk factor, previous research has shown that drinking-refusal self-efficacy is a larger and more robust predictor of alcohol expectancies (Connor et al., 2008, 2011; Ehret et al., 2014). Drinking refusal self-efficacy is also central to predicted mechanisms of action for cognitive behavioural therapy alcohol use interventions (Black & Chung, 2014; Cho, 2005; Connor et al., 2016). Therefore, the bSCT model is more consistent with the current impulsivity literature, social cognitive theory, and may provide increased utility in predicting risk and providing targets for treatment through the inclusion of drinking refusal self-efficacy.

As well as providing utility in predicting risk for substance use behaviour, the bSCT may provide useful information for treatments for drug-dependent individuals. Gullo and colleagues (2014) investigated mechanisms of effect of rash impulsiveness on alcohol-dependence severity in 143 adult alcohol-dependent inpatients. They found that perceived impaired control (a proxy measure of drinking refusal self-efficacy) partially mediated this relationship, suggesting that cognitive factors are an important mediating outcome for impulsivity factors (Gullo, St. John, et al., 2014). Further, negative cannabis expectancies and low coping self-efficacy predict treatment-seeking behaviour in cannabis-dependent users (Papinczak, Connor, Feeney, Young, & Gullo, 2017). These factors, in turn, are predictive of response to cognitive-behavioural therapy in those who seek treatment (Gullo, Matveeva, Feeney, Young, & Connor, 2017). Additionally, positive cannabis expectancies partially mediate the relationship between Reward Drive and cannabis outcomes and cannabis refusal self-efficacy fully mediate the relationship between Rash Impulsiveness and cannabis outcomes (Papinczak et al., 2018). This information gives possible targets for increasing treatment seeking and improving treatment outcomes. Therefore, investigating these relationships in adolescents may provide information into the most effective areas to target in prevention interventions.

Measuring Alcohol Expectancies and Drinking Refusal Self-Efficacy

To identify whether affecting alcohol expectancies and drinking refusal self-efficacy cause change within prevention interventions, robust measurement of the constructs for adolescents is required. Scales developed in non-adolescent populations have been applied to this younger population to good effect, e.g., the Comprehensive Effects of Alcohol (COEA; 76-items; Fromme et al., 1993) questionnaire and the Effects of Drinking Alcohol Scale (EDA; 27-items; Fromme & D'Amico, 2000; Leigh, 1987; Leigh & Stacy, 1993, 2004). However, adolescent and adult behaviour differ in the relative importance of driving factors, as adolescents place higher value on social outcomes (Aas, Klepp, Laberg, & Aarø, 1995; Jones et al., 2001). Therefore, specificity to the population is vital for validity. Accordingly, the focus will be on adolescent-specific measures in the present review.

Two common measures of adolescent alcohol expectancies are the 90-item Alcohol Expectancy Questionnaire-Adolescent version (AEQ-A; Brown et al., 1987; Christiansen, Goldman, & Inn, 1982) and the 24-item Drinking Expectancy Questionnaire-Adolescent version (DEQ-A; Connor, George, et al., 2011; Young & Knight, 1989; Young & Oei, 1996). Only one adolescent-specific measure of drinking refusal self-efficacy has been published, the adolescent version of the Drinking Refusal Self-Efficacy Questionnaire-Revised (DRSEQ-R; Oei, Hasking, & Young, 2005), the DRSEQ-RA (Connor et al., 2011; Young, Hasking, Oei, & Loveday, 2007). Both the DRSEQ-R (for adults) and the DRSEQ-RA (for adolescents) comprise 19-items and assess drinking refusal self-efficacy in three contexts; under social pressure, when one feels in need of emotional relief, and when the opportunity arises (Oei et al., 2005; Young et al., 2007). This three-factor structure has been confirmed in both scales, where they each load onto a higher factor of Drinking Refusal Self-Efficacy (Connor et al., 2011; Young et al., 2007). Both the DRSEQ-R and the DRSEQ-RA have been shown to predict alcohol use outcomes (Connor et al., 2011; Young et al., 2007).

The AEQ-A is the adolescent version of the Alcohol Expectancy Questionnaire (AEQ; Brown et al., 1987) and the DEQ-A is the adolescent version of the Drinking Expectancy Questionnaire (DEQ; Young & Knight, 1989). Both the AEQ-A and the DEQ-A assess expectancies of social facilitation and impaired cognitive and motor functioning. The AEQ-A assesses the positive expectancies of *global positive changes, changes in social behaviour, improved cognitive and motor abilities, sexual enhancement, increased arousal, and relaxation and tension reduction*, and the negative expectancies of *cognitive and motor impairment*. The DEQ-A includes the positive subscales of *increased confidence* and *tension*

reduction and the negative subscales of *cognitive and motor impairment* and *negative mood*. But as is reflective of the initial literature less emphasis is placed on negative expectancies in the AEQ-A. Further, the adult measure, the AEQ does not measure negative expectancies, although a separate 60-item questionnaire assessing adult negative expectancies has been proposed as an adjunctive measure (the Negative Alcohol Expectancy Questionnaire; NAEQ; McMahon & Jones, 1993). The lack of inclusion of negative expectancies limits the ability to monitor changes in expectancies over time and with exposure to alcohol from adolescence to adulthood, which previous research has shown is a common phenomenon (see ‘alcohol expectancies across the lifespan’ section above). Comparatively, the DEQ-A includes two negative subscales and is more easily compared to its adult counterpart due to having a similar (but not identical) factor structure. Thus, the ability to map the growth of alcohol expectancies over the life-span and compare adolescent and adult expectancies is more easily facilitated by the DEQ and the DEQ-A, and these scales offer a more comprehensive assessment of negative alcohol expectancies.

Another factor to consider in interventions is scale length to reduce participant burden in clinical and in research contexts. The DEQ-A and the DRSEQ-RA are already relatively short at 24 and 19 items, respectively. The AEQ-A comprises 90 items. One approach that has been used to minimise test length when measuring expectancies is to administer a single subscale of the AEQ-A, such as the *changes in social behaviour measure* (Young-Wolff et al., 2015). However, using the subscales as stand-alone measures has not been independently validated and assessing only positive expectancies may reduce construct validity and comprehensiveness. A shortened version of the AEQ-A was created using 124 (predominately male) detainees at a juvenile correctional facility with a background of alcohol and polysubstance use (Stein et al., 2007). However, this scale would need further validation to determine appropriateness for use in a non-incarcerated adolescent population with normative substance use.

By comparison, the DEQ-A is nearly one-quarter the length of the AEQ-A. The factor structure and the reliability of the DEQ-A have gained preliminary validation using confirmatory factor analysis in a sample of 192 adolescents ($M = 13.8$ years, $SD = 0.5$; Connor, George, et al., 2011). It is recognised that the DEQ-A also requires further validation, but at present it is the shortest adolescent-specific measure of adolescent alcohol expectancies, and the DRSEQ-RA is the only adolescent-specific measure of drinking refusal self-efficacy. As both alcohol-related cognitions are robust predictors of future alcohol use,

shortened scales to be delivered in early intervention and research contexts would be beneficial to reduce the burden on participants and researcher resources.

Summary

In summary, several cognitive and personality risk factors for alcohol use have been reviewed in this chapter, separately and in combination with other constructs. Specifically, this chapter examined firstly Impulsivity and secondly alcohol-related cognitions, each of which have demonstrated predictive utility for alcohol outcomes. The bioSocial Cognitive Theory (bSCT) describes the important connections between two impulsivity risk factors (rash impulsiveness and reward drive) and two social cognitive risk factors (drinking refusal self-efficacy and alcohol expectancies). This model is highlighted and summarised in Figure 3.4. The bSCT has been validated in adolescent populations as well as adult population, in alcohol and cannabis, and is therefore considered a promising framework with which to identify modifiable risk factors for alcohol use in adolescents. Each of the factors within the model may be targeted, and the model provides guidance as to the pathways of risk where interventions may be most effective. For these factors to be investigated, however, short and reliable measures of the cognitive constructs need to be utilised.

Chapter 4: Measuring Adolescent Alcohol Expectancies

Overview

This chapter provides a background to the measurement of adolescent alcohol expectancies and proposes a reduced scale for measurement of the construct. It comprises a version of a manuscript published in *Drug and Alcohol Review* (citation below). The measurement of expectancies across the lifespan with complementary adolescent and adult scales can facilitate research into the development of these cognitions across age groups. Further, the chapter evaluates the alcohol-related cognitions of abstinent and drinking adolescents. Therefore, the chapter is central to the thesis as it begins to explore the measurement and differing profiles of adolescent alcohol expectancies, which is a key concept in later chapters.

Patton, K. A., Connor, J. P., Rundle-Thiele, S., Dietrich, T., Young, R. M., & Gullo, M. J. (2018). Validation of the Adolescent Drinking Expectancy Questionnaire and development of a short form. *Drug and Alcohol Review*, 37(3), 396–405.
doi:10.1111/dar.12567

Abstract

Aims This study aimed to validate the Drinking Expectancy Questionnaire – Adolescent version (DEQ-A) in a large adolescent sample and to develop and validate a brief measure, the Drinking Expectancy Questionnaire – Shortened Adolescent version (DEQ-SA).

Design and Methods Cross-sectional survey of secondary school students ($N = 2,357$, aged 13-16, $M = 14.66$ years, $SD = 0.60$). Students completed the DEQ-A in school, and measures of alcohol consumption including the Alcohol Use Disorders Identification Test – Consumption (AUDIT-C). The data were randomly split and Exploratory Factor Analysis was performed using subsample 1 and Confirmatory Factor Analysis and reliability and validity testing was performed using subsample 2.

Results The 24-item DEQ-A was successfully reduced to 12 items (DEQ-SA) without compromising psychometric properties. The DEQ-A and the DEQ-SA both demonstrated adequate-to-good fit to the data and very good internal reliability. The DEQ-A and DEQ-SA explained 20% and 18% of the variance in alcohol consumption. Adolescents who drank endorsed more positive alcohol expectancies, whereas alcohol-naïve adolescents scored higher on negative alcohol expectancies. As the DEQ-SA comprises two subscales of the DEQ-A the endorsement rates are applicable to both scales.

Discussion and Conclusions The DEQ-A and the short form of this scale developed in this study (DEQ-SA) show good reliability, internal structure, and account for a large proportion of variance in alcohol consumption. Both scales can assist in targeting cognitive change processes within tailored alcohol prevention and treatment approaches and investigating hypothesised mechanisms of change. The DEQ-SA is recommended for more time-limited environments.

Introduction

Hazardous alcohol use in adolescence is one of the leading causes of adolescent morbidity and mortality (Australian Institute of Health and Welfare, 2011, 2014; World Health Organization, 2014) and is predictive of future alcohol-related problems and dependence (Connor et al., 2016; C. Gao, Ogeil, & Lloyd, 2014; Grant & Dawson, 1997; Grant, Stinson, & Harford, 2001). A comprehensive understanding of the mechanisms contributing to hazardous alcohol use in adolescents could enhance the effectiveness of early interventions and Alcohol Use Disorder (AUD) treatments. A recent *Lancet* review on AUDs highlighted outcome expectancies as a central psychological mechanism supporting initiation and maintenance of alcohol problems (Connor et al., 2016). Drawn from Social Cognitive Theory (Bandura, 1986), alcohol expectancies are the perceived outcomes of drinking (Bandura, 1977; Brown et al., 1987). Another mechanism of adolescent alcohol use is drinking motives (Kuntsche et al., 2005), which can mediate the effect of alcohol expectancies on alcohol use (Diep et al., 2016; Kuntsche et al., 2007). However, alcohol expectancies are developed from both vicarious and experiential learning (Bandura, 1977, 1986) whereas drinking motives apply exclusively to pre-existing drinkers. This makes alcohol expectancies particularly pertinent for adolescent populations who may not have engaged in alcohol use previously. Alcohol expectancies are robust predictors of consumption and problem use in adult and adolescent populations (Brown et al., 1985; Christiansen & Goldman, 1983; Connor, George, et al., 2011; Jones et al., 2001; Young & Oei, 1993) and are consequently a common target for prevention and treatment (Scott-Sheldon et al., 2012; Young et al., 2011).

Alcohol expectancies have been broadly classified into positive and negative expectancies. Positive expectancies reflect beliefs that alcohol consumption will result in rewarding outcomes; negative expectancies in undesirable outcomes (Jones & McMahon, 1993; Leigh, 1989). Socially-related positive alcohol expectancies (e.g., “Drinking makes me feel more outgoing”) are strongly associated with the onset and maintenance of alcohol use in an adolescent population (Jester, Steinberg, Heitzeg, & Zucker, 2015; Smith et al., 1995; Tomlinson & Brown, 2012; Young-Wolff et al., 2015). By comparison, adults show elevated positive expectancies across several domains (Gullo et al., 2010a). These findings suggest that specific alcohol expectancies may evolve over time and differentially based on direct and indirect exposure to alcohol (Aas et al., 1998; Bekman, Goldman, Worley, & Anderson,

2011), and that the influence of alcohol expectancies may differ between prevention and treatment contexts due to differences in age and alcohol exposure between these populations.

Due to the strong association between early drinking onset and future alcohol problems, alcohol prevention programs are usually targeted at adolescents (Grant & Dawson, 1997). A robust measure of adolescent alcohol expectancies is critical to assess and target this hypothesised mechanism of change within interventions. While a number of alcohol expectancy questionnaires can be used in an adolescent population (see Fromme & D'Amico, 2000; Schafer & Leigh, 1996), these scales were not created specifically for adolescents and a full review of their suitability is beyond the scope of the present study. The proceeding discussion will focus on adolescent-specific measures.

Scale length requires consideration to avoid respondent fatigue. Importantly, succinct scales have demonstrated comparable psychometric properties and predictive power (Fromme & D'Amico, 2000). Several adolescent alcohol expectancy instruments have been developed, including the 90-item Alcohol Expectancy Questionnaire-Adolescent version (AEQ-A; Brown et al., 1987; Christiansen, Goldman, & Inn, 1982) and the 24-item Drinking Expectancy Questionnaire-Adolescent version (DEQ-A; Connor et al., 2011; Young & Knight, 1989; Young & Oei, 1996). These are both adolescent versions of established adult expectancy questionnaires, the Alcohol Expectancy Questionnaire (AEQ; Brown et al., 1987) and the Drinking Expectancy Questionnaire (DEQ; Young & Knight, 1989). The AEQ-A comprises 7 subscales (*global positive changes, changes in social behaviour, improved cognitive and motor abilities, sexual enhancement, cognitive and motor impairment, increased arousal, and relaxation and tension reduction*) and the DEQ-A comprises 4 subscales (*increased confidence, tension reduction, cognitive and motor impairment, and negative mood*).

The DEQ-A is one of the shortest expectancy measures. While a brief version of the AEQ-A exists, measuring the 7 original AEQ-A subscale domains (the AEQ-AB; 7-items; Stein et al., 2007), it was developed using 124 (predominately male) detainees at a juvenile correctional facility. The sample had a history of regular alcohol and polysubstance use and may not be representative of the general adolescent population. The 'changes in social behaviour' positive expectancy subscale of the AEQ-A has been used as a stand-alone measure (Young-Wolff et al., 2015), but it has not been independently validated. Given evidence that negative alcohol expectancies also predict adolescent alcohol consumption

(Leigh & Stacy, 2004) a brief measure that includes positive and negative expectancies would provide a more comprehensive approach. A 60-item Negative Alcohol Expectancy Questionnaire (NAEQ; McMahon & Jones, 1993) has been published, but was developed for adult populations. While a negative expectancy scale was added to the AEQ-A, the adult AEQ assesses only positive expectancies. This does not allow for a single measure comparison of both positive and negative expectancies over time and at different ages. By contrast, the DEQ and DEQ-A have a similar factor structure and include two negative expectancy subscales (*cognitive and motor impairment* and *negative mood/affective change*). Therefore, the DEQ and DEQ-A offer a comprehensive assessment of negative expectancies and can be used to assess changes in positive and negative alcohol expectancies over the life-span.

Connor et al. (2011) employed confirmatory factor analysis to conduct preliminary psychometric validation on the DEQ-A in a sample of 192 adolescents ($M = 13.8$ years, $SD = 0.5$). They reported support for the hypothesised four-factor structure (*increased confidence, tension reduction, cognitive and motor impairment, and negative mood*) as well as high internal consistency for each subscale. The DEQ-A prospectively predicted alcohol use at 12-month follow-up (Connor et al., 2011), however requires validation in a larger, independent sample with exploratory as well as confirmatory factor analytic techniques (Hopwood & Donnellan, 2010). Additionally, a short form of the DEQ-A would be desirable for prevention applications where brevity of measurement is a central concern.

This study utilises a large sample of adolescents to evaluate the psychometric properties and factor structure of the DEQ-A. Further, we aimed to develop and evaluate a brief version of the DEQ-A that retained the psychometric properties and predictive power of the longer scale. This scale is proposed for use in prevention contexts where short administration time is desired.

Materials and methods

Participants

Participants were Queensland Grade 10 Catholic high school students from 24 schools across Queensland, Australia ($N = 2,609$), recruited in a randomised control trial for the *Game On: Know Alcohol* (GOKA) project (see Dietrich et al., 2015; Rundle-Thiele, Russell-Bennett, Leo, & Dietrich, 2013). Students from both regional and metropolitan schools were sampled. Pre-intervention baseline data were analysed, which were restricted to a sample

aged 13-16, ($M = 14.66$ years, $SD = 0.60$; $N = 2,357$, females = 1,161 (49.3%), gender not reported = 1). Parental ethnic background of the sample was primarily “White” Australian (59.3% fathers; 59.2% mothers) or European (19.6% fathers; 19.9% mothers) and 89.1% of students were born in Australia.

Measures

Drinking Expectancy Questionnaire-Adolescent Version (DEQ-A).

The DEQ-A is a 24-item scale modified from the DEQ, which was developed based on interviews with a diverse sample of alcohol drinkers (Young & Knight, 1989). The items are 5-point Likert-style questions (1 = Strongly Disagree; 5 = Strongly Agree). A four-factor structure was expected following previous findings by Connor et al. (Connor et al., 2011); two relating to positive alcohol expectancies (*Increased confidence*, e.g., “I feel less shy when I am drinking” and *Tension Reduction*, e.g., “Drinking alcohol helps when I am anxious”) and two relating to negative alcohol expectancies (*Cognitive and motor impairment*, e.g., “I am clumsier when drinking alcohol” and *Negative Mood*, e.g., “I feel gloomy when drinking alcohol”; Connor et al., 2011).

Drinking status and consumption.

Participants were asked if they had ever consumed a full alcoholic drink and were categorised into drinking adolescents and non-drinking adolescents. The AUDIT was developed for the World Health Organisation and comprises 10-items. The first 3 items pertaining to frequency and typical quantity of use as well as binge frequency can be combined for use as a measure of alcohol consumption, the AUDIT-C, and uses a 5-point Likert type response style (e.g., 0 = *Never*; 4 = *Daily or almost daily*; Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). Cronbach’s α was 0.86 for the AUDIT-C.

Procedure

This study uses baseline data prior to delivery of a school-based intervention (see Dietrich et al., 2015; Rundle-Thiele et al., 2013). Institutional human ethics committee’s approval was obtained (Griffith University: MKT/26/10/HREC; The University of Queensland: 2014001623).

Data Analysis.

To provide rigorous evaluation of the DEQ-A, exploratory and confirmatory factor analysis was conducted (EFA/CFA), which helps to protect against the adoption of a factor structure that may be influenced by statistical artefacts arising from a particular analytic approach (Hopwood & Donnellan, 2010). Data were randomly split into two groups for these two studies using the 'random select' data function in SPSS. Study 1 included EFA and examination of the subscales. Based on the results of Study 1, several plausible models, including a shortened version of the DEQ-A comprising the highest endorsed positive and negative expectancy subscale, were estimated and compared using CFA in Study 2. Finally, the psychometric properties of the DEQ-A and the shortened scale created in Study 2 were investigated using Cronbach's alpha to assess internal consistency, a one-way MANOVA to test the association between drinking status and expectancy endorsement, and regression analyses to examine scale and subscale association with alcohol consumption. Study 2 analyses utilised the second randomly split dataset. The randomly split groups (Study 1 $N = 1,179$; Study 2 $N = 1,178$) did not differ significantly in age, gender, drinking status, AUDIT risk level, or endorsement of expectancies ($ps = .310-.770$).

Model estimation and evaluation.

In Study 1, EFA was conducted in SPSS (version 22) using Principal Axis Factoring extraction due to the non-normal data distribution (Fabrigar, Wegener, MacCallum, & Strahan, 1999), with a direct oblimin (oblique) rotation given the documented moderate correlations among DEQ factors. Item loadings lower than .30 were suppressed. In Study 2, the χ^2 test statistic was used to examine CFA model fit. Comparative fit index (CFI), root mean-square error of approximation (RMSEA), and the standardized root mean-square residual (SRMR) were also examined. The cut-off criteria for good fit were $CFI \geq .95$, $RMSEA \leq .06$, and $SRMR \leq .08$ (Tabachnick & Fidell, 2007). It should be noted that these "cut-offs" are generally regarded only as guidelines, and models approaching these values were interpreted as having acceptable fit (Marsh, Hau, & Wen, 2004). Additionally, the Akaike Information Criterion (AIC; Akaike, 1987) was also used to compare non-nested models and assess model parsimony, with smaller values being associated with better-fitting models. The data were found to deviate significantly from multivariate normality. This precluded the use of standard Maximum Likelihood (ML) estimation. To reduce the effect of multivariate non-normality, the Satorra-Bentler scaled χ^2 test and robust standard errors were interpreted (Satorra & Bentler, 1994, 2001). The CFAs were run in *R* (version 3.2.1) using

the *lavaan* package (version 18; “R: A language and environment for statistical computing,” 2014; Rosseel, 2012).

Results

Alcohol use (N = 2,357)

Approximately 40% (N = 930) of participants indicated they had consumed an alcoholic drink previously. For these adolescents, AUDIT-C scores ranged from 1–12 (M = 2.47, SD = 2.42) and 23.4% were drinking at “risky” levels according to the AUDIT. 143 adolescents (15.4%) reported drinking 3 or 4 standard drinks on a typical drinking occasion. A further 164 adolescents (17.6%) reported drinking 5 or more standard drinks on a typical drinking occasion.

Study 1: Exploratory factor analysis (N = 1,179)

The EFA on the DEQ-A items extracted four factors accounting for 67.55% variance, but examination of the scree plot and the eigenvalues (> 1.0) suggested a 2-factor solution was more appropriate. Further, while the item loadings approximated the 4 subscales of the DEQ-A, there were high (e.g., -.539) and frequent item cross-loadings (all items of the 3rd and 4th factor also loaded onto either factor 1 or 2; see Table 4.6 in supplementary materials).

A second EFA was conducted on the DEQ-A items restricting extraction to two factors, as suggested by the scree plot. The 2-factor model explained 60.16% of the variance and produced strong factor loadings (.55 - .95), fewer cross-loadings and more theoretically-consistent item groupings. The two factors were labelled *positive expectancies* and *negative expectancies* (see Table 4.1).

Table 4.1

Factor loadings for the exploratory factor analysis with extraction restricted to two factors.

Items	Positive Expectancies	Negative Expectancies
20. Drinking makes me get along with people better	0.95	
9. Drinking makes me feel more outgoing and friendly	0.90	
23. If I'm drinking it's easier to express my feelings	0.90	

17. Drinking makes it easier to talk to strangers	0.83	
5. Drinking makes it easier to openly express love and affection	0.82	
1. I feel less shy when I am drinking	0.78	
13. Drinking alcohol makes me more responsive to the opposite sex	0.76	
19. Drinking makes me feel hopeful about the future	0.75	
7. Drinking alcohol helps when I'm anxious	0.70	
15. Drinking makes the future brighter	0.69	
22. I drink alcohol to relieve tension	0.63	
11. Drinking alcohol helps calm me down when I'm upset	0.63	
3. I drink alcohol to unwind	0.61	
4. I am more sullen and depressed when I'm drinking alcohol		0.83
8. Drinking alcohol makes me feel negative about the future		0.80
16. Drinking alcohol makes me feel sad		0.78
24. I become confused when drinking alcohol		0.73
12. I feel gloomy when drinking alcohol		0.73
10. When I am drinking it is harder to make mental connections		0.70
2. I am more forgetful when I am drinking		0.60
14. When I drink alcohol I accidentally break and destroy things		0.60
18. I think less clearly when drinking alcohol		0.60
6. I am likely to fall down when drinking		0.59
21. I am clumsier when drinking alcohol	0.32	0.55

Note. Item loadings below .30 were omitted for clarity of exposition.

The low endorsement (where high endorsement is defined as average scores > 3 on the 5-point scale) and restriction of range in *negative mood* and *tension reduction* subscales (10.6% and 6.4% high endorsement, respectively) compared with the endorsement of the

increased confidence and *cognitive and motor impairment* subscales (23.5% and 17.4%, respectively) may have impacted the factor reduction. A third EFA was conducted using the positive and negative subscales with the highest endorsement rates (*increased confidence* and *cognitive and motor impairment*; 12-items). Examination of the eigenvalues and the scree plot both indicated extraction of two factors. The two factors explained 67.12% of the variance in the model and were highly correlated, $r = .65$ ($p < .001$). The item loadings were consistent with the pre-determined subscales, i.e., *increased confidence* items all loaded onto factor one and *cognitive and motor impairment* items all loaded onto factor two (see Table 4.2). The proposed shortened version of the DEQ-A therefore comprised these two highly endorsed subscales (12 of 24 items when items 18 and 23 removed per DEQ-RA).

Table 4.2

Item loadings from exploratory factor analysis of the Drinking Expectancy Questionnaire – Shortened Adolescent version (N=1,179).

Subscale of origin	Item	Positive expectancy	Negative expectancy
Increased confidence	20. Drinking makes me get along with people better	.93	
	9. Drinking makes me feel more outgoing and friendly	.91	
	23. If I'm drinking it's easier to express my feelings	.83	
	1. I feel less shy when I am drinking	.81	
	5. Drinking makes it easier to openly express love and affection	.81	
	17. Drinking makes it easier to talk to strangers	.74	
	13. Drinking alcohol makes me more responsive to the opposite sex	.67	
Cognitive and Motor Impairment	24. I become confused when drinking alcohol		.91
	18. I think less clearly when drinking alcohol		.82
	21. I am clumsier when drinking alcohol		.81
	10. When I am drinking it is harder to make mental connections		.78

14. When I drink alcohol I accidentally break and destroy things	.73
6. I am likely to fall down when drinking	.71
2. I am more forgetful when I am drinking	.70

Note. Item loadings lower than .30 suppressed.

Study 2: Confirmatory factor analysis (N = 1,178)

CFAs were conducted on the 4-factor model specified by the 24-item DEQ-A, with each latent factor allowed to covary (Model 1 in Table 4.3). The revised 21-item 4-factor model reported by Connor et al (Connor et al., 2011), in which items 18, 19, and 23 were removed in post hoc model modifications to improve fit and a higher-order Drinking Expectancy factor was added, was also examined (Model 2). An alternative, higher-order 2-factor model separating the four DEQ-A subscales into covarying positive and negative expectancy factors (Model 3) was also tested to further examine results obtained in EFA. These models were compared to a shortened 2-factor model (*increased confidence* and *cognitive and motor impairment* subscales with items 18 and 23 removed as per Connor et al., 2011; Model 4).

Confirmatory factor analysis.

The CFAs showed that the DEQ-A (Model 1; see Table 4.3) and the Connor et al. (2011) revised DEQ-A model (Model 2) had adequate-to-good fit on all indices, as did the shortened 2-factor model (highest endorsed positive and negative subscales; Model 4). As the EFA suggested a two-factor solution (positive and negative expectancies), a CFA was run testing this structure. The two-factor solution (Model 3) showed significantly reduced fit compared with the four-factor structure. The shortened 2-factor model showed high loadings of the measured variables onto the latent factors (Figure 4.1). However, *cognitive and motor impairment* and *increased confidence* were highly correlated with one other, leading to questions about the appropriateness of separating the factors. A subsequent CFA was run where *cognitive and motor impairment* and *increased confidence* items were combined to load onto a single latent factor (Model 5 in Table 4.3). This model showed significantly poorer fit to the data indicating that these subscales are separate factors, despite their correlation. Therefore, Model 4 was retained and labelled the short-form DEQ-A (DEQ-SA). Models 6 and 7 tested the factor structure of the revised DEQ-A and the DEQ-SA across gender groups. The good fit of these models indicates that the structure holds across gender.

Table 4.3

Model fit indices for the DEQ-A and DEQ-SA confirmatory factor analyses (N = 1,178).

Model	χ^2 (df)	CFI	RMSEA	SRMR	AIC
1. 24-item 4-factor DEQ-A model	1375.77* (246)	.95	.06	.05	75949.83
2. Connor et al. (2011) revised 19-item 4-factor DEQ-A model	1069.18* (184)	.95	.06	.05	67800.38
3. 24-item 2-factor DEQ-A model (positive and negative expectancies)	2191.13* (251)	.91	.08	.06	77342.23
4. 12-item DEQ-SA: Shortened 2-factor model (Inc. confidence + Cog/Motor Impairment)	379.18* (53)	.97	.07	.04	40243.71
5. Alternative 12-item 1-factor DEQ-SA model (Inc. confidence + Cog/Motor Impairment combined)	1493.26* (77)	.89	.13	.09	47750.75
6. Connor et al. (2011) revised 19-item 4-factor DEQ-A model grouped by gender	1272.68* (368)	.95	.07	.06	67724.06
7. 12-item DEQ-SA: Shortened 2-factor model (Inc. confidence + Cog/Motor Impairment) grouped by gender	436.86* (106)	.97	.07	.04	40179.30
1. vs. 3. $\chi^2_{diff}(df_{diff})$	815.36(5)*				
4. vs. 5. $\chi^2_{diff}(df_{diff})$	1114.08(24)*				

Note. CFI, comparative fit index; RMSEA, root-mean-square error of approximation; SRMR, standardised root mean-square residual; AIC, Akaike Information Criterion. * $p < .001$.

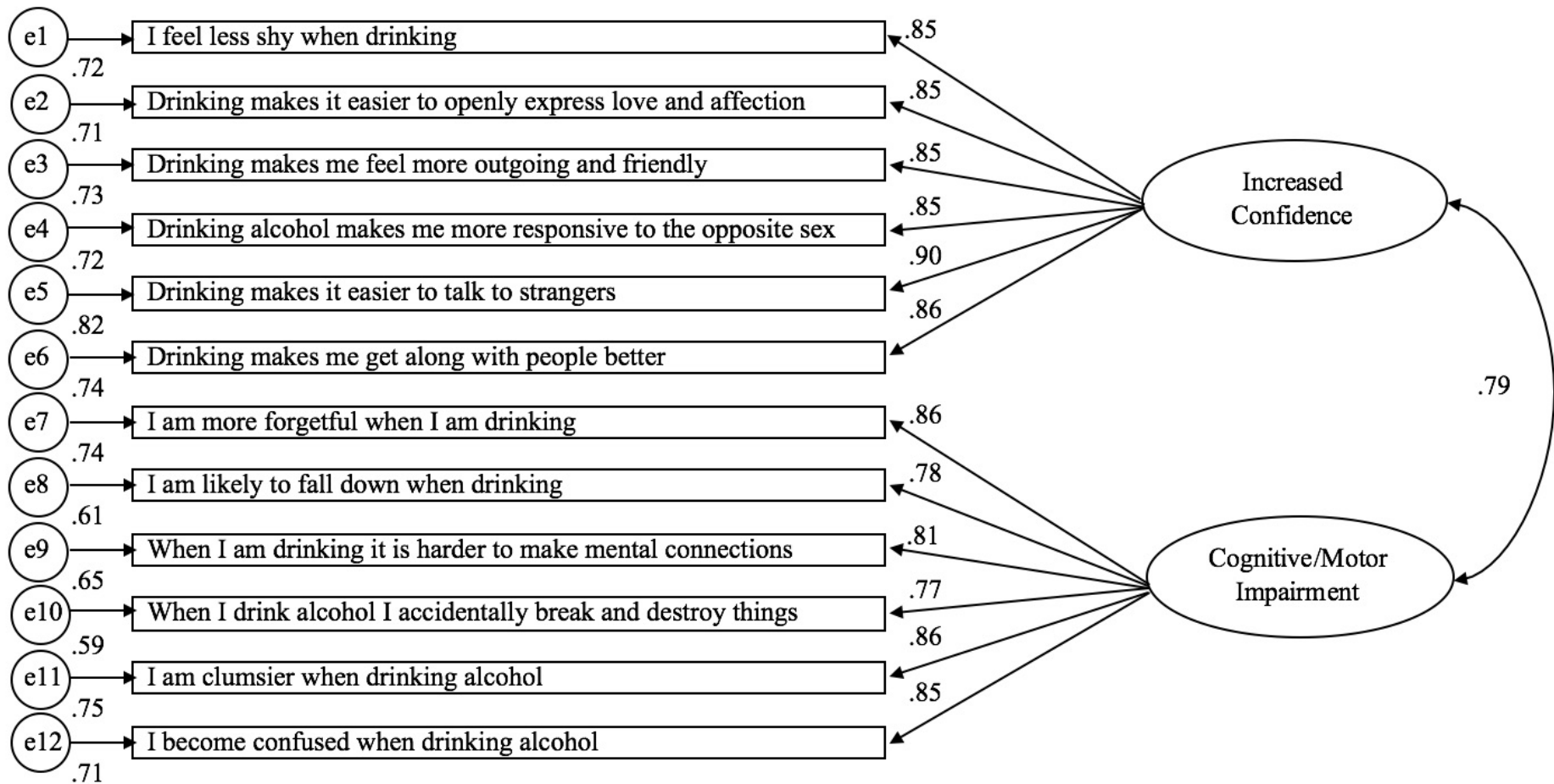


Figure 4.1. Two-factor shortened drinking expectancy measurement model.

Note. Ellipses represent latent constructs, rectangles indicate measured variables, and circles reflect residuals. All parameters are significant at $p < .05$.

Investigation of psychometric properties of scales

Reliability.

The internal reliability of the DEQ-A (Model 1, Table 4.3) and the revised DEQ-A (Connor et al., 2011; Model 2, Table 4.3) and subscales was good to excellent; (*increased confidence* $\alpha = .94$ and $.94$; *tension reduction* $\alpha = .91$ and $.90$; *cognitive and motor impairment* $\alpha = .92$ and $.92$; *negative mood* $\alpha = .86$ and $.86$.) For the total DEQ-A and revised DEQ-A scale $\alpha = .96$ and $.96$. For the DEQ-SA scale (Model 4, Table 4.3; *increased confidence and cognitive and motor impairment*) $\alpha = .95$.

Association between drinking status and expectancy endorsement.

To assess concurrent validity a one-way MANOVA was conducted with adolescents who drank vs. those who did not drink as the categorical independent variable and the DEQ-A subscales as the dependent variables. Drinkers had higher scores on positive expectancy scales and scored lower than the non-drinking adolescents on negative expectancies (see Table 4.4). The difference between drinking and non-drinking adolescents on the combined expectancies total was significant, $F(4, 1174) = 82.24, p < .001$, Pillai's Trace = $.22$, partial $\eta^2 = .22$. A series of follow-up one-way ANOVAs revealed that each contrast between expectancies of drinking and non-drinking adolescents was significant, even after Bonferroni adjustments (see Table 4.4).

Table 4.4.

Comparison of expectancies between drinking and non-drinking adolescents (Study 2).

	<u>Mean(SD)</u>			<u>Comparison</u>	
	Drinking students (N = 448)	Non-drinking students (N = 730)	Total (N = 1,178)	F(df)	Partial η^2
Increased confidence, Mean(SD)	18.11(7.30)	14.26(6.92)	15.72(7.31)	82.65(1, 1176)**	.07
Tension Reduction, Mean(SD)	11.37(5.26)	10.14(5.02)	10.60(5.14)	16.06(1, 1176)**	.01

Cognitive and Motor Impairment, Mean(SD)	14.00(6.28)	15.08(7.32)	14.67(6.96)	6.80(1, 1176)*	.01
Negative Mood, Mean(SD)	7.08(3.47)	8.61(4.26)	8.03(4.05)	40.81(1, 1176)**	.03

** $p < .001$, * $p < .05$

Association with alcohol consumption.

Regressions between the DEQ-A, the revised DEQ-A (Connor et al., 2011), the DEQ-SA and alcohol consumption were run using the Study 2 dataset (see Table 4.5). Gender did not alter the significance or direction of effects when included in the regression model as a moderator (see Table 4.7 in supplementary materials) so we have reported results of both genders combined. The DEQ-A, the revised DEQ-A, and the DEQ-SA each explained significant variance in concurrent alcohol consumption, accounting for 20% (DEQ-A and revised DEQ-A) and 18% (DEQ-SA) of the variance in consumption. To explore prospective prediction data from Connor et al.'s (2011) study of 192 adolescents was reanalysed using only DEQ-SA items. Controlling for Time 1 AUDIT-C scores, Increased Confidence ($B = .10$, $SE = .04$, $p = .033$, $sr^2 = .02$) predicted unique variance in Time 2 AUDIT-C scores at 12 months, but Cognitive and Motor Impairment ($B = .02$, $SE = .04$, $p = .599$, $sr^2 = .001$) did not, $\Delta F(3, 186) = 39.52$, $p < .001$.

Table 4.5.

Associations between DEQ-A and DEQ-SA subscales and AUDIT-C scores (Study 2; $N = 1,178$).

Regression model	Predictors	B	β	sr^2	t	R	$adjR^2$	F -test (df)
1. DEQ-A	Increased confidence	.12	.43	.06	9.43**	.45	.20	74.74 (4, 1173)**
	Tension	.07	.17	.01	3.84**			
	Reduction							

2. Revised DEQ-A (Connor et al., 2011)	Cognitive and Motor Impairment	-.04	-.13	.01	-2.96*	.46	.20	74.74 (4, 1173)**
	Negative Mood	-.10	-.20	.02	-5.07**			
	Increased confidence	.12	.43	.06	9.43**			
	Tension Reduction	.07	.17	.01	3.84**			
3. DEQ-SA	Cognitive and Motor Impairment	-.04	-.13	.01	-2.96*	.43	.18	130.44 (2, 1175)**
	Negative Mood	-.10	-.20	.02	-5.07**			
	Increased confidence	.15	.55	.17	15.64**			
	Cognitive and Motor Impairment	-.07	-.25	.04	-7.25**			

** $p < .001$, * $p < .05$.

Discussion

This study drew on a large sample of adolescents ($N = 2,357$) to validate the adolescent version of the Drinking Expectancy Questionnaire (DEQ-A) and develop a brief measure of adolescent alcohol expectancies (DEQ-SA). The 24-item four-factor DEQ-A was found to have strong psychometric properties and is considered suitable for use in both adolescent alcohol prevention and treatment settings. Similarly, the 12-item DEQ-SA had good reliability and validity and is proposed for screening or research purposes in an adolescent alcohol prevention setting when a shorter assessment timeframe is required.

While there have been no previous EFAs reported on the DEQ-A, CFAs supported a 4-factor structure (2 positive, 2 negative expectancy factors) in a similarly aged sample (Connor et al., 2011). The preliminary DEQ-A scale validation (Connor et al., 2011) resulted in four subscales [*increased confidence*, *tension reduction*, *negative mood*, and *cognitive and motor impairment*] with a higher-order factor. While a two-factor model was found using an

exploratory approach in the current study, confirmatory analyses revealed that the more theoretically consistent four-factor models were found to better fit the data compared to a two-factor model. Differing results from EFA and CFA are not uncommon in psychological measurement due to inherent differences in underlying assumptions and specified parameters of each analysis (Hopwood & Donnellan, 2010).

The purpose of developing the DEQ-SA was twofold: 1) to provide a brief version of the DEQ-A that would retain the psychometric properties and predictive power of the original measure, 2) to be a measure for use in a prevention context which would necessarily involve inclusion of adolescents yet to experience alcohol consumption but who have developed expectancies vicariously. We also see value in using this shortened measure in longitudinal research projects with youth, which will allow researchers to understand changes in expectancies over time (both through repeated use of the DEQ-SA and compatibility with the DEQ-A if adolescents are followed into adulthood) while helping to minimise participant burden. This could assist researchers to map the age-related and alcohol experience-related changes in alcohol expectancies, which have been documented in previous research (H. N. Aas et al., 1998; Bekman et al., 2011). The DEQ-SA comprises the most endorsed subscales of the DEQ-A, which were the positive expectancy subscale of *increased confidence* and the negative expectancy subscale of *cognitive and motor impairment*. These two subscales accounted for almost identical amounts of variance in alcohol consumption as all four subscales combined, indicating that the DEQ-SA has comparable predictive power to the DEQ-A as a stand-alone measure in a young adolescent sample. The factor structure of the DEQ-SA was supported by both exploratory factor analysis and, confirmatory factor analysis and model comparisons.

The current findings suggest that drinking and non-drinking adolescents have differing expectancy profiles, with drinkers reporting high positive alcohol expectancies and low negative alcohol expectancies than their non-drinking counterparts. Overall, this pattern of results is consistent with theoretical models emphasising the greater role of positive expectancies in drinking initiation and early consumption (Jones et al., 2001; Oei & Baldwin, 1993). This study found that adolescent drinking was particularly driven by the socially-rewarding aspects of alcohol consumption.

The finding that non-drinking adolescents had higher negative alcohol expectancies than drinking adolescents (rather than just lower positive expectancies) has received little

emphasis in previous research. This is potentially due to poor measurement of negative alcohol expectancies (e.g., (Christiansen & Goldman, 1983)), decisions not to include negative expectancies in analyses (e.g., (Jones et al., 2001; Scott-Sheldon et al., 2012; Young & Oei, 1993)) or report negative expectancy findings when included (e.g., (Christiansen et al., 1982; Young et al., 2011)). Additionally, studies including negative expectancies have typically investigated predictive power rather than investigating differing endorsement rates between drinking and non-drinking participants. The small but significant effect size for negative expectancies observed in the present study indicate that a large sample (such as in the current study) may be required to find significant results in an adolescent population. This may partially explain the inconsistent reporting on negative expectancies in the literature.

The study has some limitations. While the effect of the *negative expectancy* subscale on alcohol consumption was small, the squared semi-partial correlations showed that the *negative expectancy* subscale did have an additive effect to the model. The decision was made to retain the subscale in the DEQ-SA due to the possibility that its role in drinking increases over the adolescent period. Further research into subscale endorsement and measure applicability and appropriateness should be also pursued in populations with AUDs, older adolescents with higher rates of alcohol experience, and adolescents from diverse backgrounds.

In summary, this study validated an existing adolescent alcohol expectancy measure (the DEQ-A) and developed a brief version (the DEQ-SA) in a large sample of young adolescents. Both the DEQ-A and the DEQ-SA show strong psychometric properties. The scales were associated with alcohol consumption and drinking and non-drinking adolescents had significantly different alcohol expectancy profiles. The DEQ-A can be used in contexts where the influence of various expectancies would be valuable, such as treatment programs, whereas the DEQ-SA could be used for screening, with adolescents who report non-drinking, or where a brief measure is preferable.

Appendix

The Drinking Expectancy Questionnaire – Revised adolescent version (DEQ-RA)

The purpose of these questions is to find out about YOUR thoughts, feelings and beliefs about drinking alcohol. If you have never drunk alcohol, respond with what you think would happen IF you drank alcohol. Please circle the most appropriate response using the following scale:

	1	2	3	4	5			
	Strongly Disagree	Disagree	Neither agree nor Disagree	Agree	Strongly Agree			
1.				1	2	3	4	5
2.				1	2	3	4	5
3.				1	2	3	4	5
4.				1	2	3	4	5
5.				1	2	3	4	5
6.				1	2	3	4	5
7.				1	2	3	4	5
8.				1	2	3	4	5
9.				1	2	3	4	5
10.				1	2	3	4	5
11.				1	2	3	4	5
12.				1	2	3	4	5
13.				1	2	3	4	5
14.				1	2	3	4	5
15.				1	2	3	4	5
16.				1	2	3	4	5
17.				1	2	3	4	5
18.				1	2	3	4	5
19.				1	2	3	4	5
20.				1	2	3	4	5
21.				1	2	3	4	5

The Drinking Expectancy Questionnaire – Shortened Revised Adolescent
version (DEQ-SRA)

The purpose of these questions is to find out about YOUR thoughts, feelings and beliefs about drinking alcohol. If you have never drunk alcohol, respond with what you think would happen IF you drank alcohol. Please circle the most appropriate response using the following scale:

	1	2	3	4	5			
	Strongly	Disagree	Neither agree	Agree	Strongly			
	Disagree		nor Disagree		Agree			
1.	I feel less shy when drinking			1	2	3	4	5
2.	I am more forgetful when I am drinking			1	2	3	4	5
3.	Drinking makes it easier to openly express love and affection			1	2	3	4	5
4.	I am likely to fall down when drinking			1	2	3	4	5
5.	Drinking makes me feel more outgoing and friendly			1	2	3	4	5
6.	When I am drinking it is harder to make mental connections			1	2	3	4	5
7.	Drinking alcohol makes me more responsive to the opposite sex			1	2	3	4	5
8.	When I drink alcohol I accidentally break and destroy things			1	2	3	4	5
9.	Drinking makes it easier to talk to strangers			1	2	3	4	5
10.	Drinking makes me get along with people better			1	2	3	4	5
11.	I am clumsier when drinking alcohol			1	2	3	4	5
12.	I become confused when drinking alcohol			1	2	3	4	5

Supplementary Materials

Table 4.6

Factor loadings for the initial exploratory factor analysis using Principal Axis Factoring (PAF) extraction with a direct oblimin (oblique) rotation.

Subscale of origin	Item	Factor 1	Factor 2	Factor 3	Factor 4
Increased confidence	20. Drinking makes me get along with people better	.85			
	9. Drinking makes me feel more outgoing and friendly	.82			
	23. If I'm drinking it's easier to express my feelings	.81			
	5. Drinking makes it easier to openly express love and affection	.75			
	17. Drinking makes it easier to talk to strangers	.75		-.32	
	1. I feel less shy when I am drinking	.70			-.34
	13. Drinking alcohol makes me more responsive to the opposite sex	.68		-.31	
	19. Drinking makes me feel hopeful about the future	.82			
	15. Drinking makes the future brighter	.79			.31
	7. Drinking alcohol helps when I am anxious	.75			
Tension Reduction	11. Drinking alcohol helps calm me down when I'm upset	.71			
	22. I drink alcohol to relieve tension	.69			
	3. I drink alcohol to unwind	.69			

Cognitive and Motor Impairment	10. When I am drinking it is harder to make mental connections	.70	
	2. I am more forgetful when I am drinking	.61	
	24. I become confused when drinking alcohol	.61	-.42
	6. I am likely to fall down when drinking	.59	
	14. When I drink alcohol I accidentally break and destroy things	.48	-.30
	21. I am clumsier when drinking alcohol	.44	-.54
	18. I think less clearly when drinking alcohol	.48	-.50
	Negative Mood	4. I am more sullen and depressed when I'm drinking alcohol	.85
8. Drinking alcohol makes me feel negative about the future		.82	
12. I feel gloomy when drinking alcohol		.75	
16. Drinking alcohol makes me feel sad		.67	

Note. Item loadings lower than .30 omitted for clarity of exposition

Table 4.7

Associations between DEQ-A and DEQ-SA subscales and AUDIT-C scores (Study 2; N = 1,178) with gender included in the model.

Regression model	Model	Predictors	β	R	adjR ²	R ² change	F-change (df)
1 DEQ-A	Model 1	Increased confidence	0.43**	0.46	0.2	0.21	61.49** (5, 1172)
		Tension Reduction	0.16**				
		Cognitive and Motor Impairment	-0.13*				
		Negative Mood	-0.21**				
		Gender	0.07*				
	Model 2	Interaction: Increased confidence*Gender	-0.11*	0.46	0.21	0.003	1.16 (4, 1168)
		Interaction: Tension Reduction*Gender	-0.03				
		Interaction: Cognitive and Motor Impairment*Gender	-0.02				
		Interaction: Negative Mood*Gender	0.01				
2. Revised DEQ-A [16]	Model 1	Increased confidence	0.43**	0.46	0.2	0.21	61.49** (5, 1172)
		Tension Reduction	0.16**				
		Cognitive and Motor Impairment	-0.13*				
		Negative Mood	-0.21**				

		Gender	0.07*				
		Interaction: Increased confidence*Gender	0.11				
		Interaction: Tension Reduction*Gender	-0.03				
	Model 2			0.46	0.21	0.003	1.16 (4, 1168)
		Interaction: Cognitive and Motor Impairment*Gender	-0.02				
		Interaction: Negative Mood*Gender	0.01				
		Increased confidence	0.55**				
	Model 1	Cognitive and Motor Impairment	-0.26**	0.43	0.18	0.19	89.43** (3, 1174)
		Gender	0.07*				
3 DEQ-SA		Interaction: Increased confidence*Gender	0.10				
	Model 2			0.44	0.19	0.003	2.3 (2, 1172)
		Interaction: Cognitive and Motor Impairment*Gender	-0.03				

** $p < .001$, * $p < .05$.

Missing data analyses

Missing data were approximately 5% or less on all variables and Little's Missing Completely at Random (MCAR; Little, 1988) test was non-significant, $\chi^2(165, N = 2,747) = 163.41, p = .520$. The 138 cases (5%) with missing data were excluded from analyses as is appropriate for MCAR data that is a small proportion of the dataset (Schafer, 1999).

Chapter 5. Measuring Adolescent Drinking Refusal Self-Efficacy

Overview

This chapter comprises a version of a manuscript published in *Addictive Behaviors* (citation below). Similar to the previous chapter, this chapter introduces a key alcohol-related cognition, central to the following studies in the present thesis. The measurement of drinking refusal self-efficacy is explored and the only adolescent scale measuring this concept is reduced to a 9-item scale. The scale developed in the present chapter and the one developed in the previous chapter are available for use in the broader research community to measure these high impact cognitive factors. Both chapters are central to the aim of the present thesis, which is to investigate evidence-based risk factors for adolescent alcohol use that could be modified through psychological intervention.

Patton, K. A., Connor, J. P., Rundle-Thiele, S., Dietrich, T., Young, R. M., & Gullo, M. J. (2018). Measuring adolescent drinking-refusal self-efficacy: Development and validation of the Drinking Refusal Self-Efficacy Questionnaire-Shortened Adolescent version (DRSEQ-SRA). *Addictive Behaviors*, *81*, 70–77.
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Abstract

Background This study aimed to develop and validate a shortened version of the Drinking Refusal Self-Efficacy Questionnaire – Revised Adolescent version (DRSEQ-RA) using a large sample of adolescents. **Methods** Secondary school students ($N = 2,609$, $M = 14.52$ years, $SD = 0.94$) completed the DRSEQ-RA (consisting of subscales: Social Pressure; Opportunistic; Emotional Relief) and the Alcohol Use Disorders Identification Test (AUDIT). These data were analysed using non-parametric item response theory (NIRT) including Mokken scalability coefficients, and confirmatory factor analysis. **Results** Social Pressure subscale items were better able to distinguish between adolescents with lower or higher levels of drinking refusal self-efficacy, while the Opportunistic and Emotional Relief subscale items were able to distinguish adolescents with low drinking-refusal self-efficacy. The DRSEQ-RA was reduced from 19-items to a 9-item scale and retained the original three-factor structure. The reduced scale was named the Drinking Refusal Self-Efficacy Questionnaire – Shortened Revised Adolescent version (DRSEQ-SRA). The DRSEQ-RA and the DRSEQ-SRA have almost identical psychometric properties. They both demonstrated good fit to the data, each explained 18% of the variance in alcohol consumption, $Adj. R^2 = 0.18$, $p < .001$ respectively. The DRSEQ-RA and the DRSEQ-SRA also have excellent scale and subscale internal reliability ($\alpha s = .92 - .99$). **Conclusions** The DRSEQ-SRA is a short, 9-item, measure of adolescent drinking-refusal self-efficacy which demonstrates both reliability and validity. A significant advantage is brevity. The DRSEQ-SRA may be a valuable tool for identifying risk of adolescent drinking and prevention/treatment planning in settings where survey administration time is critical.

Introduction

Adolescent alcohol misuse is a public health problem, contributing to a large proportion of youth morbidity and mortality (Australian Institute of Health and Welfare, 2011; World Health Organization, 2014). Early adolescent drinking is associated with later problem drinking (Connor et al., 2016; Odgers et al., 2008; Warner & White, 2003), use and abuse of other substances, criminal activity, and increased academic problems including dropping out of school (Ellickson, Tucker, & Klein, 2003), conduct problems (Rossow & Kuntsche, 2013), and early (unplanned) parenthood (Odgers et al., 2008).

Prevention and early intervention is recommended as key to reducing the risk of detrimental outcomes from alcohol use (Stockings et al., 2016). In order to design and implement effective interventions, the mechanisms that underpin drinking behaviour need to be understood. One such mechanism of action in adolescents is drinking-refusal self-efficacy, which is the confidence in one's ability to resist drinking alcohol in different contexts. Self-efficacy is one of the most consistent predictors of alcohol dependence treatment outcomes and may contribute to onset and maintenance of alcohol use through direct or vicarious paired associations between alcohol use and outcomes (Adamson et al., 2009; Connor et al., 2016; Kadden & Litt, 2011; Young et al., 2011). Drinking refusal self-efficacy has been shown to mediate the association of other established risk factors with harmful alcohol use such as impulsivity and positive alcohol expectancy in both adolescent and adult populations (Connor et al., 2011; Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010b; Harnett et al., 2013).

Adolescents and adults demonstrate differing patterns of perceived self-efficacy; compared to adult alcohol use, adolescent alcohol use is more likely to be driven by social contexts and expectations of social pressure and social outcomes (H. Aas et al., 1995; Jester, Wong, et al., 2015; Jones et al., 2001; Tomlinson & Brown, 2012; Young-Wolff et al., 2015). An adolescent-specific, reliable and valid drinking refusal self-efficacy measure would assist in both establishing prevalence to inform prevention program design, in addition to evaluating alcohol harm reduction interventions targeting adolescents.

As far as the authors are aware, the adolescent version of the Drinking Refusal Self-Efficacy Questionnaire-Revised (DRSEQ-R) is the only adolescent-specific measure of drinking-refusal self-efficacy. The DRSEQ-R is a self-rated scale that measures the perceived ability to resist drinking (Oei et al., 2005). The DRSEQ-R has been comprehensively

validated in community (Oei et al., 2005), university (Young et al., 2006) and alcohol dependent populations (Young et al., 2011) with similar measures developed for other substance misuse refusal self-efficacy for example cannabis (Young, Gullo, Feeney, & Connor, 2012). The adolescent version of the measure (DRSEQ-RA), has good-to-excellent reliability and promising validity, as scores on the DRSEQ-RA were shown to be negatively related to alcohol consumption (Connor et al., 2011; Young et al., 2007). The DRSEQ-RA and the DRSEQ-R comprise three factors; Social Pressure (e.g., perceived ability to desist drinking “When I am at a party”), Emotional Relief (e.g., perceived ability to resist drinking to regulate mood “When I feel frustrated”), and Opportunistic (e.g., perceived ability to resist drinking when the opportunity arises “When I first arrive home”) (Oei et al., 2005; Young et al., 2007). These three factors load onto a single higher-order refusal self-efficacy factor (Connor et al., 2011; Young et al., 2007).

Drinking refusal self-efficacy is a strong predictor of adolescent alcohol use and has been the focus of large-scale prevention and intervention efforts (Cuijpers, 2002). The importance of drinking refusal self-efficacy in clinical interventions, prevention programs and research indicate that a psychometrically valid and robust measure would prove valuable. A shortened scale capturing comparable information to full-length scales (Fromme & D’Amico, 2000) that is reliable and valid would be of benefit to early intervention efforts and prevention program settings where time, user fatigue and cognitive capacity as well as other resources can be limited.

The current study has two aims: 1) Build on previous psychometric evidence for the DRSEQ-RA by examining the reliability and predictive validity of the measure in a large independent adolescent sample, as well as to confirm the factor structure of the DRSEQ-RA; 2) propose and psychometrically evaluate a shorter version of the DRSEQ-RA, with the purpose of facilitating more efficient data collection for future research involving this construct.

Method

Participants

Pre-intervention baseline data from the Game On: Know Alcohol (GOKA) project see (Rundle-Thiele et al., 2013, 2015) were utilised. Participants were 2,747 Australian Grade 10 students from 24 Queensland schools. Five percent or less of the data were missing on all

variables. As Little's Missing Completely at Random (MCAR; (Little, 1988) was non-significant, $\chi^2(165, N = 2,747) = 163.41, p = .520$, missing cases ($N = 138$; 5%) were excluded from analyses as is appropriate for MCAR data that is a small proportion of the dataset (Schafer, 1999). The average age of the remaining students was 14.52 years, $SD = 0.94, N = 2,609$, males = 1,298 (49.8%), gender missing = 77 (3%).

Measures

Drinking Refusal Self-Efficacy Questionnaire – Revised Adolescent version (DRSEQ-RA)

The DRSEQ-RA is an adolescent-appropriate adaptation of the adult DRSEQ-R, both of which comprise 19-items assessing three areas of belief in one's ability to refuse alcohol: when there is social pressure, when the opportunity arises, or for emotional relief (Oei et al., 2005; Young et al., 2007). The DRSEQ-RA utilises a 6-point Likert scale (1 = "I am very sure I could NOT resist drinking; 6 = "I am very sure I could resist drinking").

Drinking status and alcohol use.

The Alcohol Use Disorders Identification Test (AUDIT) is a 10-item scale developed by the World Health Organization (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993a) and has been used as a valid tool to assess adolescent alcohol problems (Toumbourou et al, 2014). The AUDIT includes three consumption items in addition to seven items assessing dependence and alcohol-related problems. Item 1-8 are assessed using a 5-point Likert type response style (e.g., 0 = Never; 4 = Daily or almost daily) and items 9 and 10 utilise a 3-point Likert scale (0 = 'No'; 2 = 'Yes, but not in the last year', 4 = 'Yes, during the last year'). The first three items comprise the AUDIT-C, which assesses frequency of typical and binge use and typical quantity of use (Bush et al., 1998). Both the AUDIT and the AUDIT-C were analysed in the present study. Cronbach's α for the AUDIT and AUDIT-C was 0.89 and 0.86, respectively. Participants were also categorised into those who did and did not drink based on their response to whether they had ever consumed a full alcoholic beverage.

Statistical method.

Item Response Theory (IRT). Item Response Theory (IRT) infers individuals' scores on psychological latent traits through modelling person parameters, item parameters and item responses (Embretson & Diehl, 2000). IRT differs from classical test theory which infers trait (true) scores by measuring observed scores and accounting for error. The inclusion of item parameters makes this method advantageous for scale reduction, as items can be individually

evaluated for their ability to discriminate differing trait levels of the construct. Nonparametric Item Response Theory (NIRT) was employed in this study as it gives greater allowance for non-monotonic and non-logistic functions, which are assumptions of parametric IRT (Khan, Lewis, & Lindenmayer, 2011; Meijer & Baneke, 2004; L. Peters, Sunderland, Andrews, Rapee, & Mattick, 2012).

Mokken's (1971) model of monotone homogeneity (MMH) was used in the current study to investigate scale and item strength. Originally calculated to estimate the extent to which pairs of items or the scale approximates an ideal Guttman scalogram, Mokken's scalability coefficient (H) is meaningful in that it gives an indication of item commonalities and therefore whether they can be explained by the same underlying trait (Sijtsma & Molenaar, 2002). The scalability coefficients are calculated using covariances between individuals' scores on items. For more information see Meijer and Baneke (2004), Sijtsma and Molenaar (2002) and van der Ark (2012). H_i is the scalability coefficient for item i and is the normalised covariance for that item. If the item is related to other items in the scale H_i will be positive (i.e., we can infer the items measure similar construct and therefore belong to the same scale). H is the scalability coefficient (normalised covariance) for the total scale. Guidelines for interpretation suggest that scales can be classified as weak ($0.3 \leq H < 0.4$), medium ($0.4 \leq H < 0.5$), or strong ($0.5 \geq H$) (Mokken, 1971).

Option Characteristic Curves (OCCs) and Item Characteristic Curves (ICCs) were displayed using nonparametric (Gaussian) Kernel Smoothing (Ramsay, 2000). Individuals are assigned a value based on their scale score and are ranked according to these values. The distribution is broken into quantiles according to a standard normal distribution and ranked values are converted into quantile scores. The probability of choosing certain responses at various quantile locations is estimated by assigning individuals a dichotomous value on an indicator variable based on the options they chose for each item and smoothing (local averaging) the relationship between these indicator variables and the standard normal quantiles. See Ramsay (2000) for further reading.

OCCs detail the probability of individuals selecting each option (probability represented on the y-axis) for the item according to their standardized normal latent trait score (x-axis) and overall quantile position. An ideal OCC would show individuals with low DRSE having a greater probability of selecting the lower item options, individuals with average DRSE having a greater probability of selecting the middle item options, and

individuals with higher DRSE having a greater probability of selecting higher item options. ICCs map the probability of individuals selecting item options (item options are represented on the y-axis; for DRSEQ-RA the options are 1 – 6) according to their standardized normal latent trait score (x-axis) and overall quantile position. In an ideal ICC the probability of selecting an option should increase with increases in the latent trait score.

Procedure and analysis.

A bimodal distribution was observed. Closer inspection of the data revealed that 58 (3.3%) of the participants who reported on the AUDIT that they had never had a drink containing alcohol scored 19 on the total DRSEQ-RA, indicated that they were “very sure [they] could NOT resist drinking” on all of the items. This was interpreted as a misunderstanding of the scale anchors and these answers were reverse coded. The total data were split into two datasets using the ‘Random select’ function in SPSS. There were no significant demographic or outcome differences between the two datasets.

Item analysis was conducted on dataset 1 ($N = 1,324$; M age = 14.58, $SD = .83$; N females = 614(46%), missing = 37(3%)). OCCs and ICCs for individual items were produced in Testgraf (Ramsay, 2000) and the Mokken R package (van der Ark, 2007, 2012) was used to calculate Mokken scalability coefficients. Using this information, the scale was reduced. Dataset 2 ($N = 1,285$; M age = 14.57, $SD = .79$); N females = 620(48%), missing = 40(3%)) was used to examine the psychometric properties of the shortened scale, including regression analyses, reliability calculations, and confirmatory factor analysis.

The CFAs were conducted using the lavaan package (version 18; (Rosseel, 2012) in R (version 3.2.1) using Weighted Least Squares estimation due to data non-normality. Model fit was examined using the χ^2 test and the Akaike Information Criterion (AIC) (Akaike, 1987), comparative fit index (CFI), root mean-square error of approximation (RMSEA), and the standardized root mean-square residual (SRMR). Guidelines to indicate good fit were $CFI \geq .95$, $RMSEA \leq .06$, $SRMR \leq .08$, and smaller AIC values (Akaike, 1987; Marsh et al., 2004; Tabachnick & Fidell, 2007).

Results

Descriptives

See Table 5.1 for descriptive statistics for drinking refusal self-efficacy and alcohol use. Overall, drinking refusal self-efficacy was high and alcohol use was low.

Table 5.1

Descriptive statistics for the AUDIT and DRSEQ-RA scale and subscales (Combined dataset; N = 2,609)

	Minimum	Maximum	Mean	Standard Deviation	Variance
AUDIT	0	40	2.16	4.80	23.08
AUDIT-C	0	12	1.05	2.07	4.28
Social Pressure DRSE	5	30	21.74	7.75	60.04
Emotional Relief DRSE	7	42	36.15	9.13	83.30
Opportunistic DRSE	7	42	37.35	8.10	65.65
Total DRSE	19	114	95.24	22.30	497.36

Item Analysis (Dataset 1; N = 1,324)

Two CFAs were conducted to assess unidimensionality, which is a core assumption of IRT (Khan et al., 2011; Meijer & Baneke, 2004; L. Peters et al., 2012). The theorised structure of the DRSEQ-RA (3 factors with a higher order factor) was compared to a single factor model (see Table 5.2). The theorised model showed mostly good fit to the data (only CFI lower than recommended cut-off). As the theorised model was a better fit to the data ($\chi^2_{\text{diff}}(df_{\text{diff}}) = 182.28(3), p < .001$), IRT analyses were conducted on subscales rather than the whole scale.

Table 5.2

Model fit indices for the DRSEQ-RA confirmatory factor analyses (Dataset 1; N = 1,324)

Model	χ^2 (df)	CFI	RMSEA	SRMR
1. DRSEQ-RA – 3- factors with total score	384.28* (149)	.85	.04	.04

2. The 19 DRSEQ-RA items as a single scale	566.56* (152)	.70	.05	.06
1. vs. 2. $\chi^2_{diff}(df_{diff})$	182.28(3)*			

Note. CFI, comparative fit index; RMSEA, root-mean-square error of approximation; SRMR, standardised root mean-square residual.

* $p < .001$

Smoothing parameters of 0.62, 0.85, and 0.79 were used for the Social Pressure, Emotional Relief, and Opportunistic analyses respectively due to non-monotonicity. As items in each subscale showed similar OCCs and ICCs, examples are shown in Figures 5.1 and 5.2 and all figures are presented in the supplementary materials. The OCCs for the Social Pressure subscale items showed promising results with students in the lowest 5% of DRSE choosing option 1 (very sure could not resist alcohol) with greater probability and students in the highest 50% or 75% of DRSE more likely to choose option 6 (very sure could resist alcohol). The OCCs for the Opportunistic and Emotional Relief subscale items showed that most students were confident in their ability to resist alcohol related to opportunistic and emotional triggers, as indicated by the tendency of students with lower DRSE choosing option 6 (very confident can resist). The population is relatively alcohol naïve (67.60% report frequency of use as ‘never’), therefore this confidence in drinking refusal may be partly explained by the adolescents having few opportunities where their self-efficacy has been tested. However, the choice of option 6 was less likely to be chosen for students in the lower 25% of DRSE ability, and those with very low DRSE were more likely to choose option 1 (very sure could not resist alcohol), showing that these subscale items were distinguishing individuals with low DRSE.

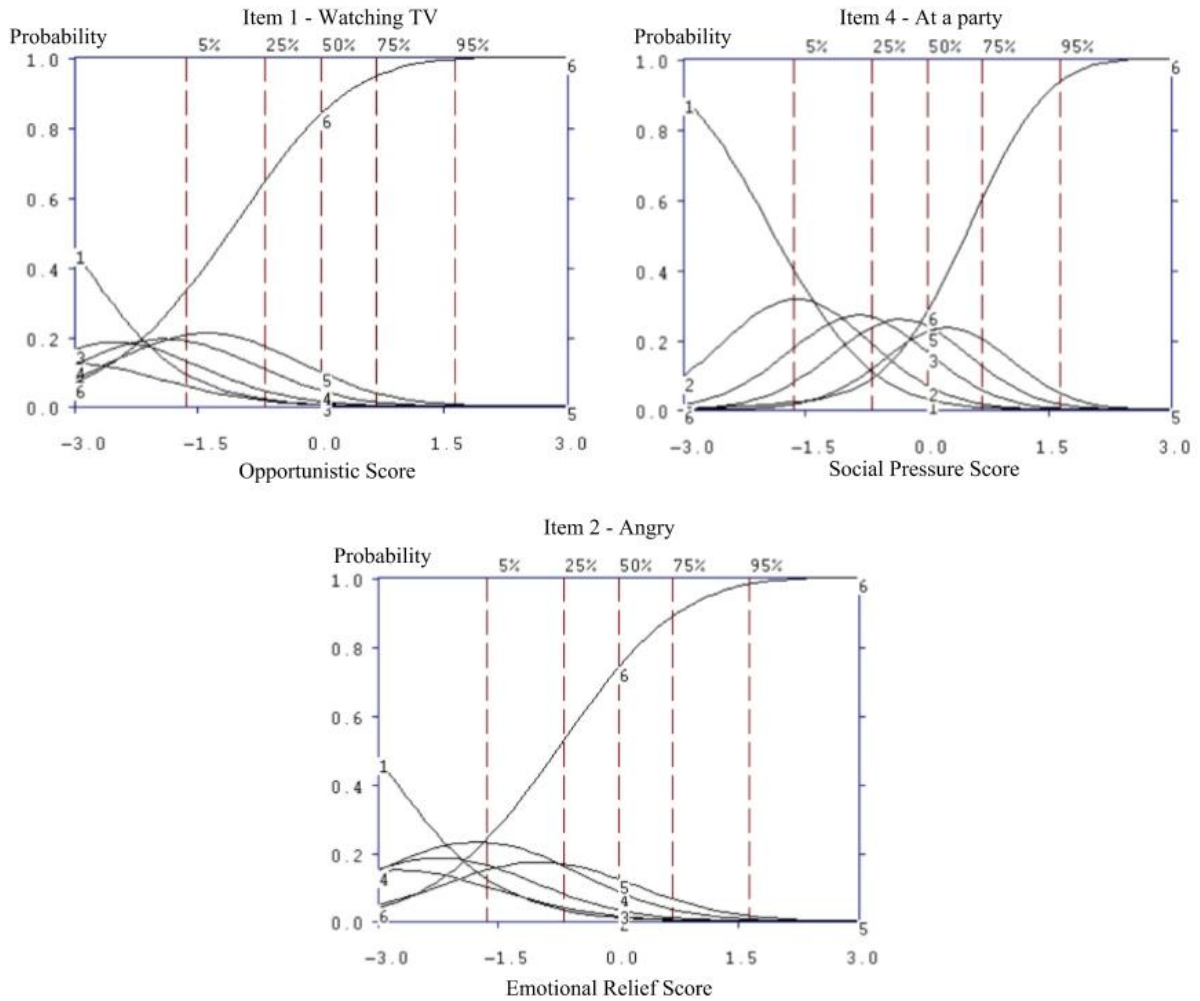


Figure 5.1. Example OCCs for each subscale.

Examination of the ICCs revealed similar findings (see Figure 5.2 for example and supplementary materials for all graphs). While there was some loss of clarity due to the high smoothing parameters, the ICCs showed that the Social Pressure subscale items show consistent increasing slopes, indicating that the items were able to discriminate across quartiles of DRSE up to the 75th percentile. The ICCs for the Opportunistic and Emotional Relief subscale items indicated these scales had less discrimination power compared to the Social Pressure subscale but could discriminate between individuals with DRSE in the lowest 25th to 50th percentiles. Above the 50th percentile there was evidence of a ceiling effect.

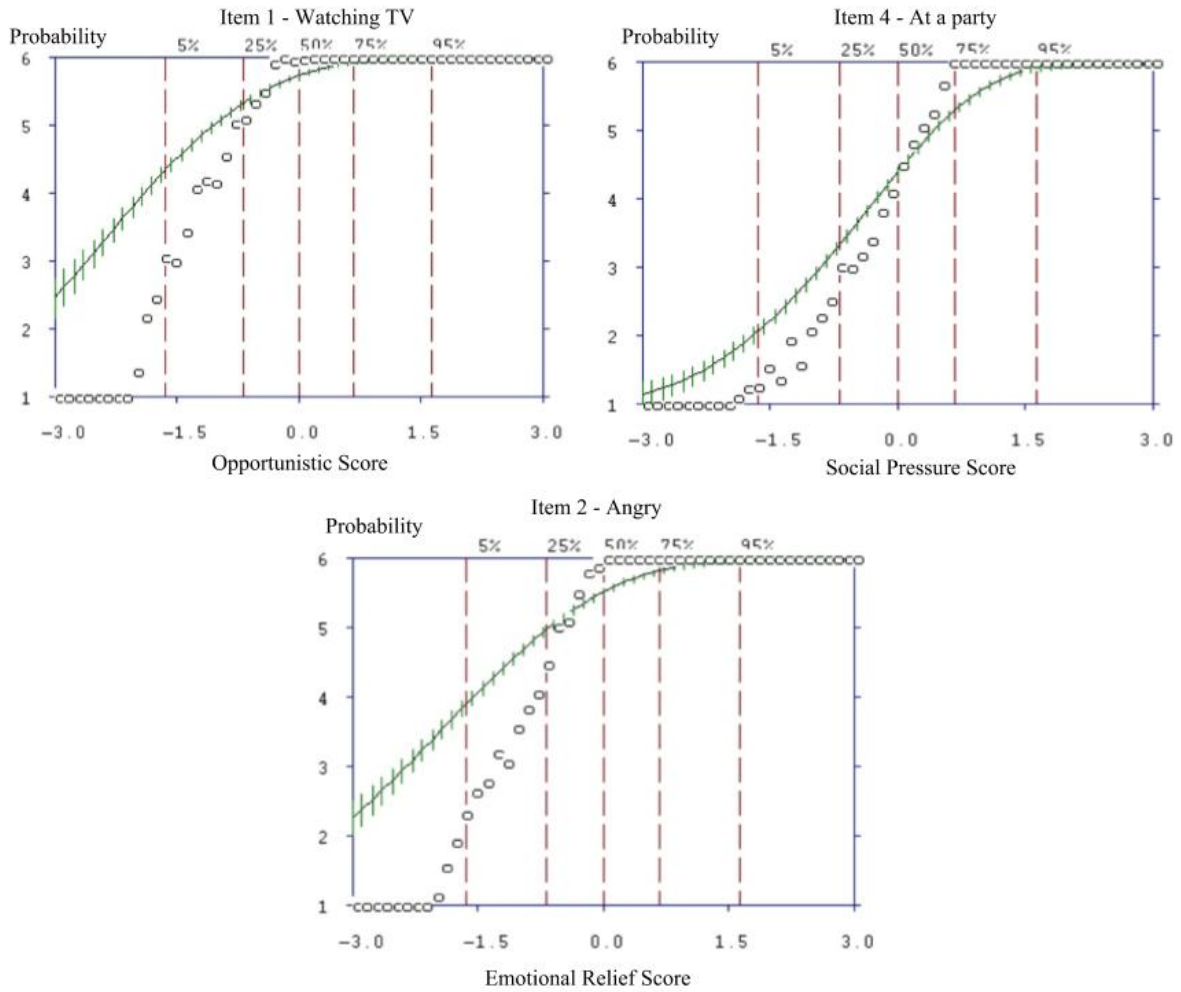


Figure 5.2. Example ICCs for each subscale.

As there were similar graph distributions for all items within the three subscales, the decision was made to reduce the scale based on item conformance. Mokken scalability coefficients indicated that all items and total subscales were strong (range = .685 - .856; see Table 5.3). Items with the greatest scalability coefficients within each subscale were chosen as potential items for the reduced scale. These were items 5, 8, and 17 for the Opportunistic subscale, items 13, 16, and 19 for the Emotional Relief subscale, and items 4, 9, and 12 for the Social Pressure subscale (see supplementary materials for full scale).

Table 5.3

Mokken normalised covariance scores (Hi) for the DRSEQ-RA subscale items and Mokken scalability coefficient (H) for the DRSEQ-RA subscales (Dataset 1; N = 1,324)

	Opportunistic	Emotional Relief	Social Pressure
H ₁ Watching TV	.758		
H ₂ Angry		.826	
H ₃ Having lunch	.685		
H ₄ At a party			.819
H ₅ Way home from school	.786 ⁺		
H ₆ Offered drink			.793
H ₇ Frustrated		.825	
H ₈ Listening to music or reading	.765		
H ₉ Boy/girlfriend is drinking			.799
H ₁₀ Worried		.837	
H ₁₁ By myself	.749		
H ₁₂ Friends drinking			.829
H ₁₃ Upset		.856	
H ₁₄ Just finished playing sport	.760		
H ₁₅ At nightclub/concert			.797
H ₁₆ Feeling down		.845	
H ₁₇ First arrive home	.786		
H ₁₈ Nervous		.813	
H ₁₉ Feel sad		.846	
H	.755	.835	.808

Note. Items in bold selected for shortened scale. ⁺ item initially selected for inspection for shortened scale but not included in final scale.

Psychometric Analysis (Dataset 2; $N = 1,285$)

When the shortened scale was analysed using CFA, analyses feedback indicated the presence of negative error variances. Examination of the CFA on the total scale revealed that item 5 of the Opportunistic subscale (“When I am on my way home from school”) had the least item variance (.08). Item 5 was replaced with item 14 (“When I have just finished playing sport”). Item 14 had the next greatest Mokken H index score and had greater variance (.14).

The CFA on the new shortened scale (item 5 replaced with item 14), herein called the Drinking Refusal Self-Efficacy Questionnaire – Shortened Revised Adolescent version (DRSEQ-SRA) showed mostly good fit to the data (see Table 5.4 and Figure 5.3; only SRMR higher than cut off).

Table 5.4

Model fit indices for the DRSEQ-SRA confirmatory factor analyses (Dataset 2; $N = 1,285$)

Model	χ^2 (df)	CFI	RMSEA	SRMR
Shortened 9-item DRSEQ-RA (DRSEQ-SRA)	80.55* (24)	.95	.04	.13

Note. CFI, comparative fit index; RMSEA, root-mean-square error of approximation; SRMR, standardised root mean-square residual.

* $p < .001$

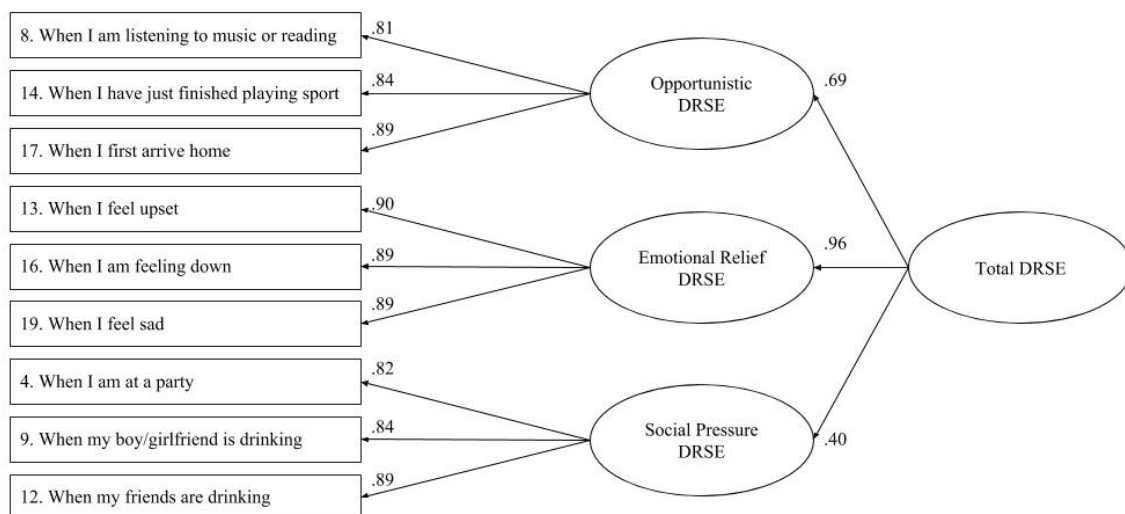


Figure 5.3 Confirmatory factor analysis model of the DRSEQ-SRA.

Note. Ellipses represent latent constructs, rectangles represent measured variables.

Association with Alcohol Use.

In order to assess convergent validity, regressions between alcohol consumption, alcohol use and each scale were conducted using Dataset 2 (see Tables 5.5 and 5.6). Both the DRSEQ-RA and the DRSEQ-SRA significantly explained 18% and 19% of the variance in total AUDIT-C scores and both significantly explained 18% of the variance in alcohol consumption. The DRSEQ-RA Emotional Relief subscale did not significantly predict AUDIT score but did significantly positively predict AUDIT-C score. Further, the DRSEQ-SRA Emotional Relief subscale was significantly positively related to AUDIT and AUDIT-C score use. These positive associations are the opposite direction of expected effects (higher self-efficacy predicting higher consumption/alcohol use). However, when the DRSEQ-RA and DRSEQ-SRA Emotional Relief subscales were each regressed on the AUDIT and AUDIT-C without including the other subscales in the model, the relationships were in the expected direction; that is, significant and negative. This provides evidence that the positive relationships may be due to suppression effects.

In order to assess gender effects, regressions were also run for males and females separately. The DRSEQ-SRA explained 18% of variance in the AUDIT-C for males, $_{adj}R^2 = .18$, $F(3, 621) = 46.62$, $p < .001$, and 18% for females, $_{adj}R^2 = .18$, $F(3, 616) = 46.35$, $p < .001$. The DRSEQ-SRA explained 19% of variance in the total AUDIT for males, $_{adj}R^2 = .19$, $F(3, 621) = 50.02$, $p < .001$, and 17% for females, $_{adj}R^2 = .17$, $F(3, 616) = 44.53$, $p < .001$. The emotional relief subscale did not significantly predict AUDIT consumption for females, $B = -.02$, $t(616) = .64$, $p = .522$, but was a significant predictor when analysed regressed on the AUDIT-C alone, $B = -.29$, $t(618) = -7.38$, $p < .001$. Some evidence of non-invariance between sexes was detected, but this could not be formally evaluated due to convergence issues in multi-group models.

Table 5.5

Associations between DRSEQ-RA and DRSEQ-SRA subscales and AUDIT scores (Dataset 2; N = 1,285).

Regression model	Predictors	B	β	sr^2	t	R	adjR²	F-test (df)
1. DRSEQ-RA	Social Pressure	-.21	-.35	0.07	-10.62**	.43	.18	94.08 (3, 1280)**
	Emotional Relief	.05	.10	0.00	1.73			
	Opportunistic	-.12	-.21	0.01	-3.94**			
2. DRSEQ-SRA	Social Pressure	-.34	-.35	0.08	-10.98**	.43	.19	98.77 (3, 1280)**
	Emotional Relief	.19	.11	0.00	2.34*			
	Opportunistic	-.30	-.24	0.02	-5.71**			
3. DRSEQ-SRA, Emotional Relief	Emotional Relief	-.33	-.29	.09	-10.98**	.29	.09	120.64 (1, 1282)**

** $p < .001$, * $p < .05$.

Table 5.6

Associations between DRSEQ-RA and DRSEQ-SRA subscales and AUDIT-C scores (Dataset 2; N = 1,285).

Regression model	Predictors	B	β	sr^2	t	R	adjR²	F-test (df)
1. DRSEQ- RA	Social Pressure	-.10	-.40	0.09	-12.16**	.43	.18	94.90 (3, 1280)**
	Emotional Relief	.03	.12	0.00	2.25*			
	Opportunistic	-.04	-.17	0.01	-3.22**			
2. DRSEQ- SRA	Social Pressure	-.16	-.39	0.09	-12.14**	.43	.18	94.53 (3, 1280)**
	Emotional Relief	.05	.11	0.00	2.33*			
	Opportunistic	-.09	-.18	0.01	-4.20**			
3. DRSEQ- SRA, Emotional Relief	Emotional Relief	-.13	-.29	.07	-9.95**	.27	.07	99.08 (1, 1282)**

** $p < .001$.

Reliability.

Reliability analyses were conducted using Cronbach's alpha. Reliability for both scales and each subscale was excellent (see Table 5.7). Cronbach's alpha ranged from .95 - .98 for the DRSEQ-RA and from .93 - .96 for the DRSEQ-SRA.

Table 5.7

Cronbach's Alpha reliability coefficients for the DRSEQ-RA and the DRSEQ-SRA scales and subscales (Dataset 2; N = 1,285).

Scale and Subscales	Cronbach's alpha
DRSEQ-RA Total	0.97
DRSEQ-RA Opportunistic	0.96
DRSEQ-RA Emotional Relief	0.98
DRSEQ-RA Social Pressure	0.95
DRSEQ-SRA Total	0.94
DRSEQ-SRA Opportunistic	0.94
DRSEQ-SRA Emotional Relief	0.96
DRSEQ-SRA Social Pressure	0.93

Discussion

The aims of this study were to create a shortened version of the adolescent drinking refusal self-efficacy scale, the DRSEQ-RA and to solidify the psychometric properties of the original scale. This scale is the only adolescent measure of drinking-refusal self-efficacy, as far as the authors are aware. Using a large sample (N = 2,609) of adolescents, we utilised NIRT and factor analyses to evaluate the 19-item DRSEQ-RA and developed a 9-item version of the scale, the DRSEQ-SRA. The psychometric properties of the scales were then assessed. On the whole, both scales demonstrated strong psychometric properties.

The DRSEQ-RA and the DRSEQ-SRA each accounted for 18% of variance in alcohol consumption and 18% and 19% of variance in total alcohol use respectively. Therefore, both scales demonstrate good convergent validity and may be useful in identifying adolescents at risk of early alcohol use. The DRSEQ-SRA explained a similar amount of variance as the DRSEQ-RA, indicating that the shortened scale has a similar level of predictive power as the full-length scale. Both scales also demonstrated excellent total scale and subscale reliability,

as measured by Cronbach's alpha, and mostly good fit to the data within CFAs. Taken together, the results indicate that the DRSEQ-SRA is a robust and clinically valid measure of adolescent drinking refusal self-efficacy, and is comparable to the 211% longer DRSEQ-RA.

The drinking refusal self-efficacy of participants was high, especially within the Opportunistic and Emotional Relief subscales. However, the scales were able to distinguish those participants with very low drinking-refusal self-efficacy, indicating that it could be useful for screening purposes. Additionally, there was a range of self-rated drinking refusal self-efficacy ability within the Social Pressure subscale, resulting in greater levels of distinction between higher levels of drinking refusal self-efficacy. This is consistent with existing literature suggesting that early alcohol consumption is greatly influenced by social contexts and expectations (Aas et al., 1995; Jones et al., 2001). However, the Emotional Relief and Opportunistic subscales warrant inclusion, as these scales may provide clinical utility and predictive power in at-risk populations. Possible gender invariance was also observed, however, overall the results indicate that the DRSEQ-SRA is appropriate for male and female adolescents. However, it is recommended that further invariance testing is pursued in a future study with an older adolescent sample which may prevent similar statistical artifacts. It may also be interesting to expand on this line of research by mapping the differing progressions of male and female adolescent drinking refusal self-efficacy. To understand how the DRSEQ-RA and the DRSEQ-SRA would perform with adolescents with lower drinking refusal self-efficacy, further testing should be performed with different populations, e.g., older adolescents and adolescents with existing alcohol use problems.

The high smoothing parameters required for the NIRT curves limited interpretation of the OCCs and the ICCs. However, the graphs are able to provide useful information about levels of discrimination across drinking refusal self-efficacy and were interpreted in combination with the Mokken scalability coefficients. While Mokken analyses assume monotonicity, this assumption was assessed for each item and only one non-significant violation was found. Therefore, it is unlikely that the Mokken results were affected.

Conclusions

In conclusion, a 9-item version of the DRSEQ-RA was developed using a large sample of adolescents. The shortened scale, the DRSEQ-SRA, retained the high psychometric properties of the full-length scale. Both scales explained a large proportion of the variance in

adolescent alcohol consumption and use (18%-19%). Given that drinking refusal self-efficacy is a strong predictor of concurrent and future adolescent alcohol use, an efficient measure of this construct could be ideal for screening, clinical use, prevention program settings as well as research settings, where brevity of assessment is desirable.

Supplementary Materials

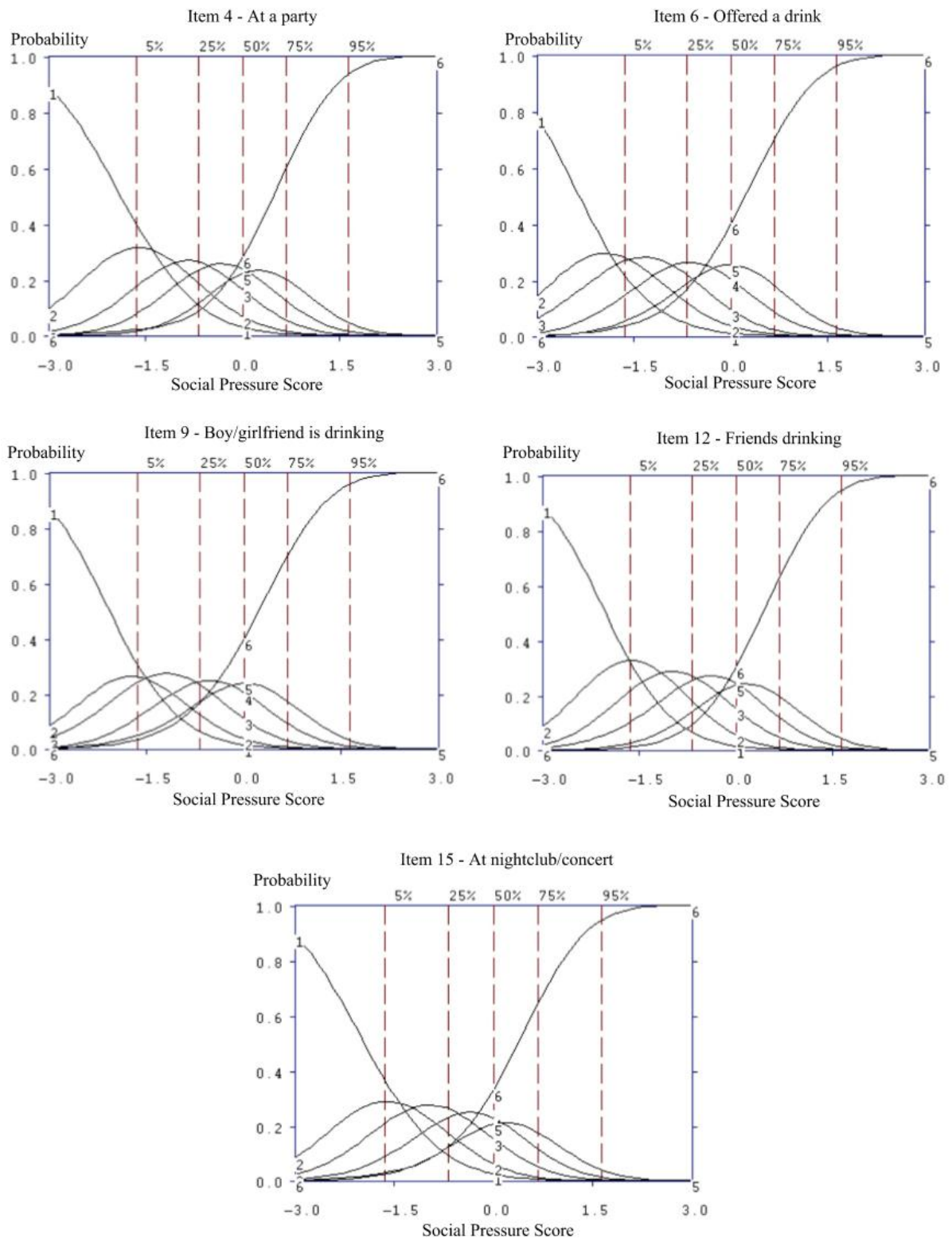


Figure 5.4. OCCs for the Social Pressure subscale.

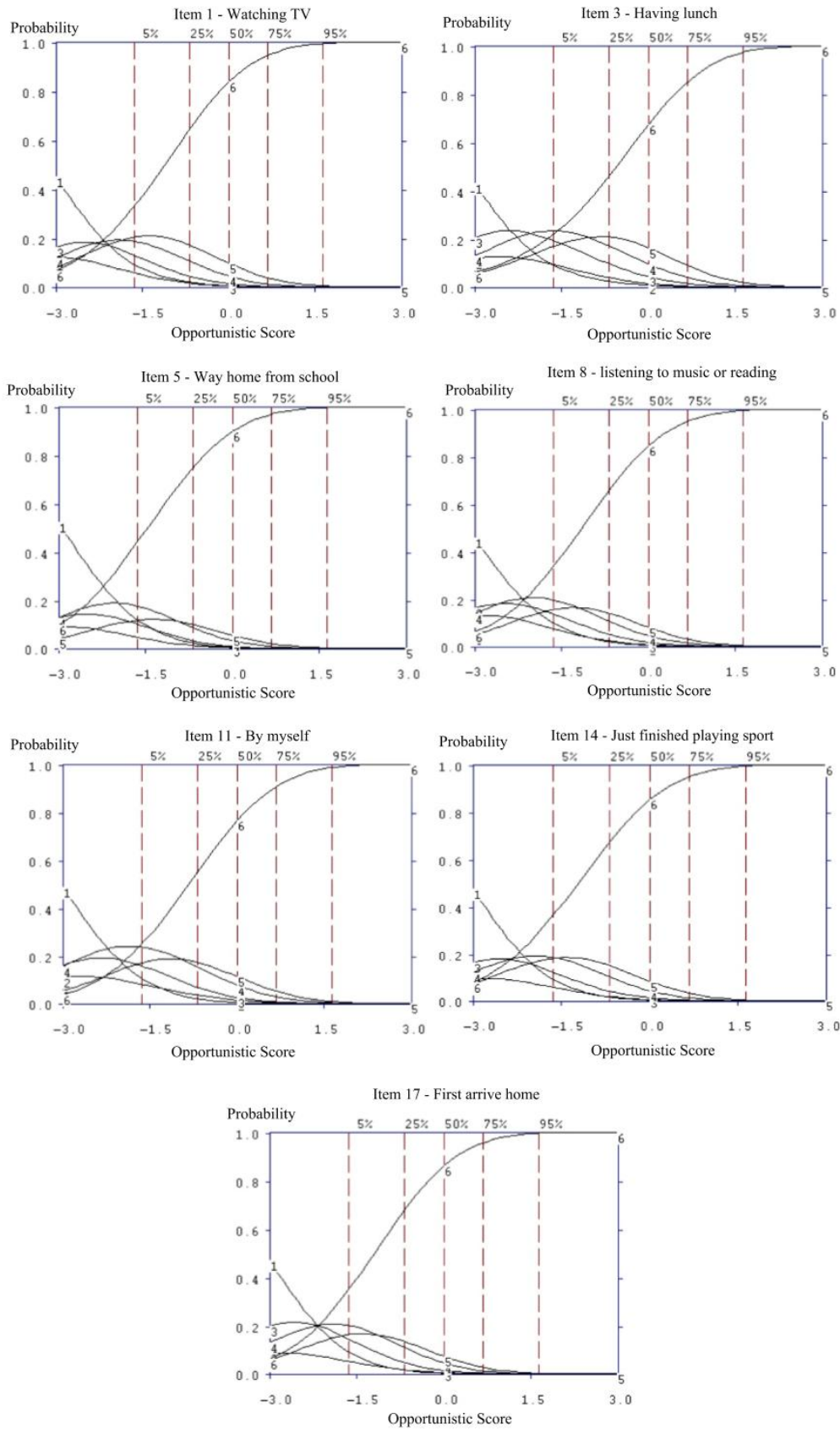


Figure 5.5. OCCs for the Opportunistic subscale.

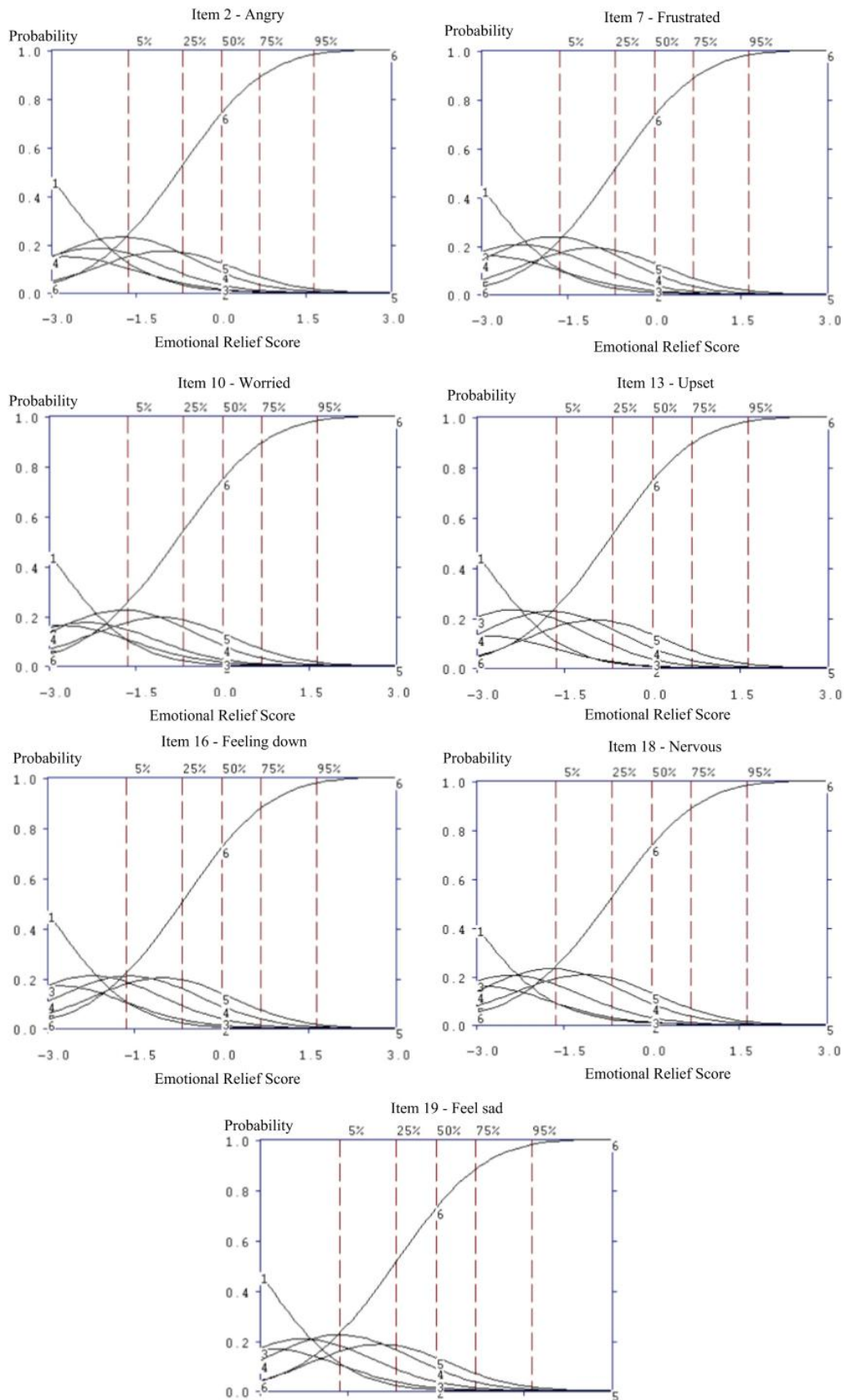


Figure 5.6. OCCs for the Emotional Relief subscale.

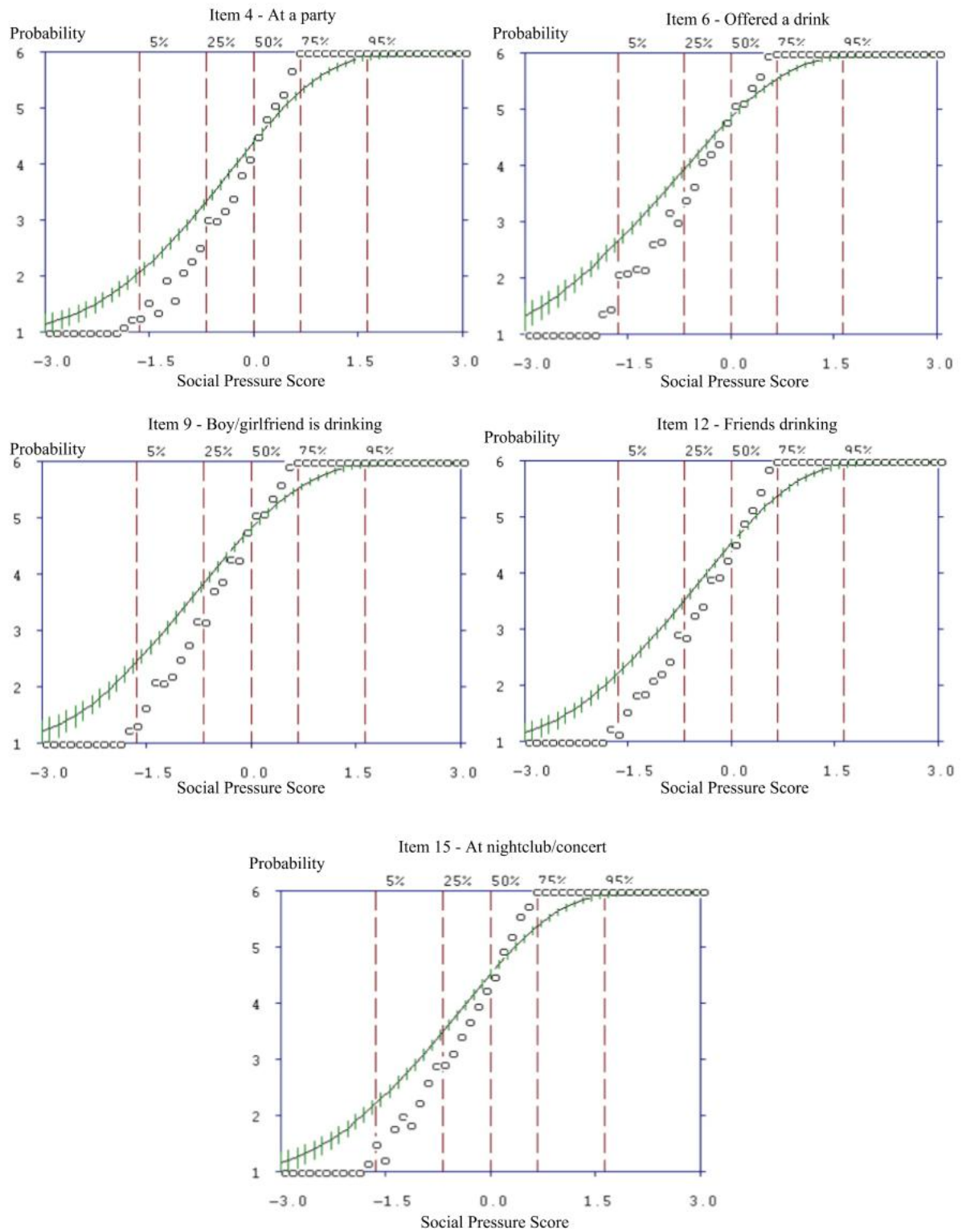


Figure 5.7. ICCs for the Social Pressure subscale.

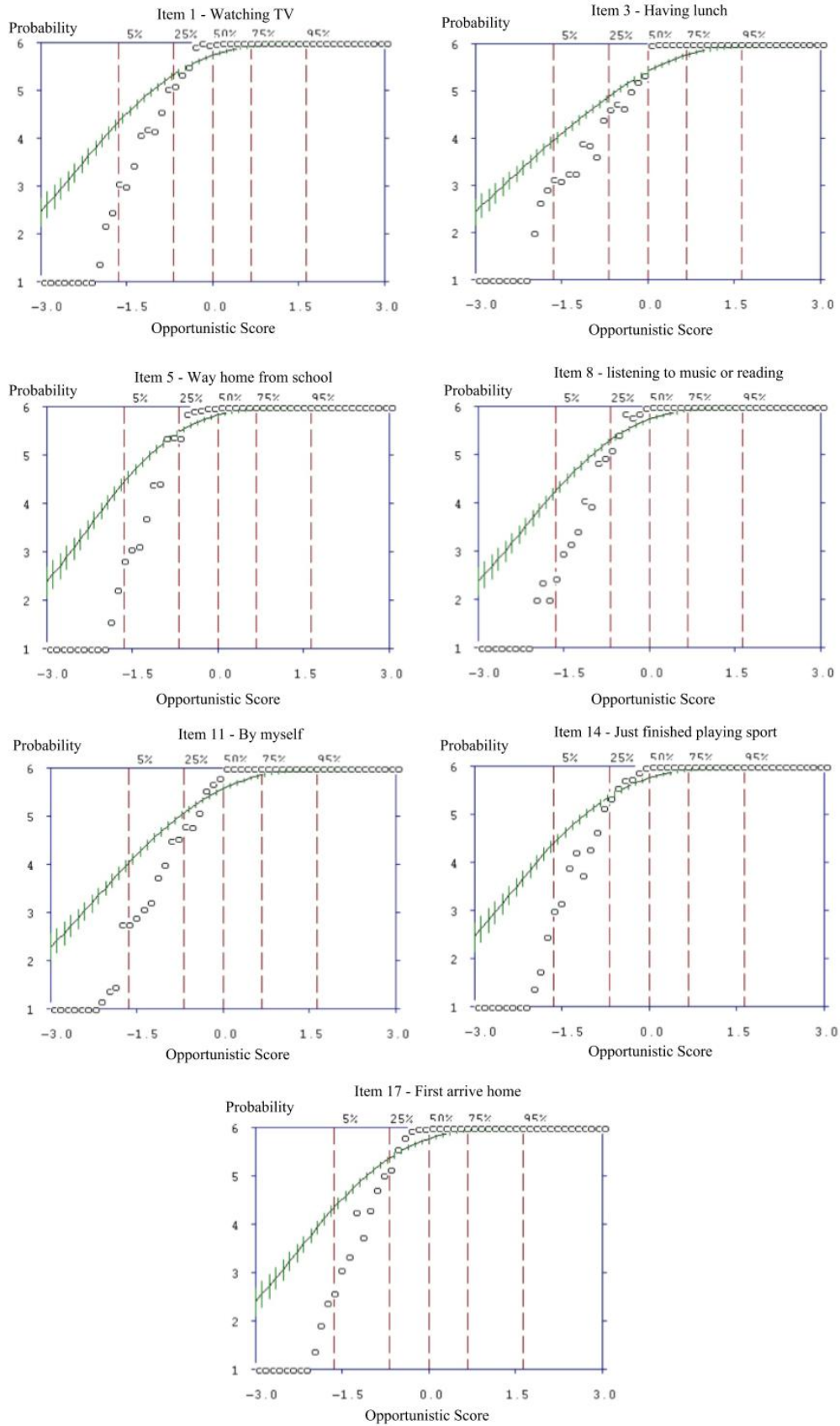


Figure 5.8. ICCs for the Opportunistic subscale.

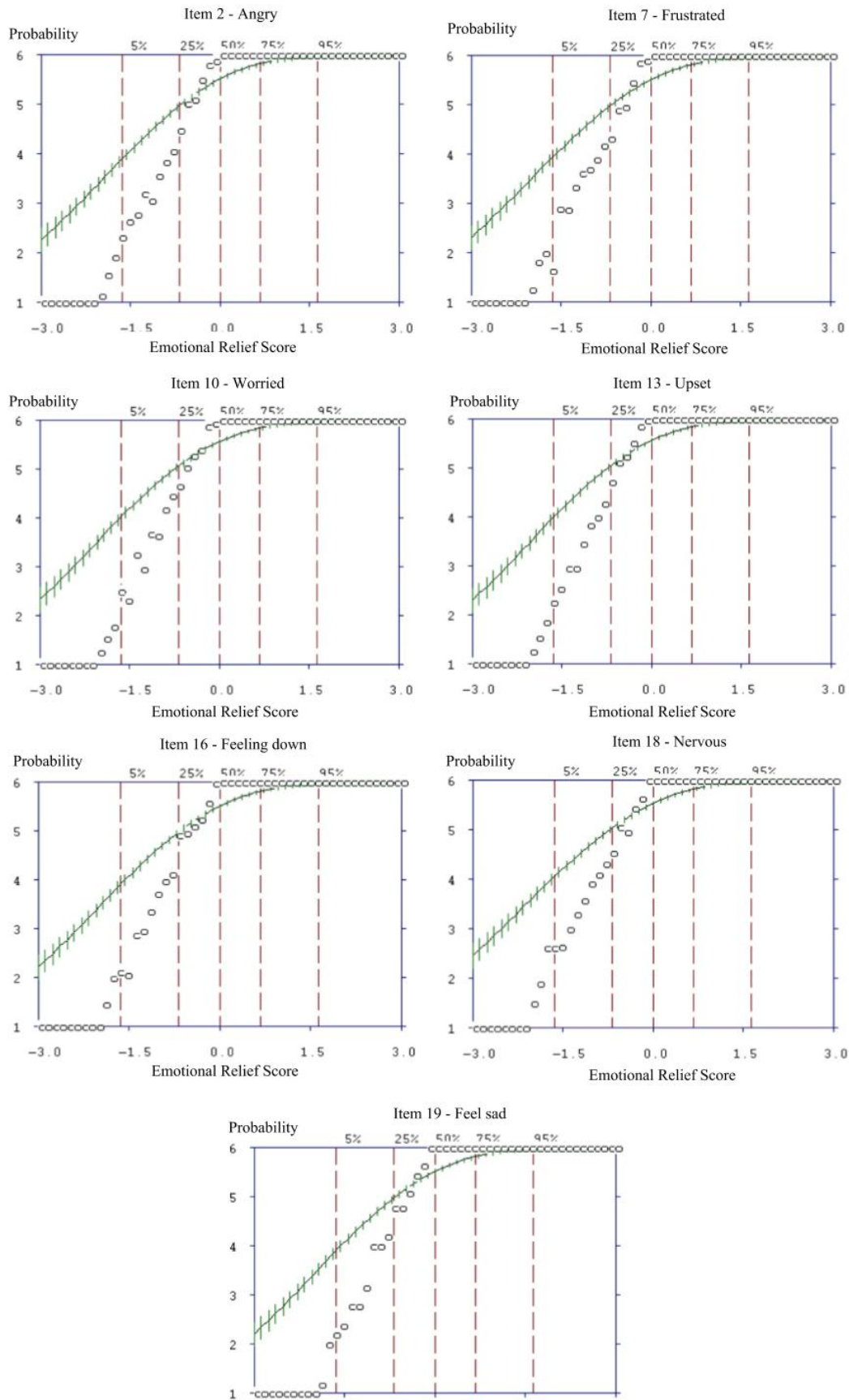


Figure 5.9. ICCs for the Emotional Relief subscale.

Drinking Refusal Self-Efficacy Questionnaire-Shortened Revised Adolescent
version (DRSEQ-SRA)

Directions:

The following items ask you to describe your ability to handle drinking situations. Your answers will be completely anonymous so please try to answer as honestly as you can.

The following pages contain a list of situations in which people may find themselves drinking alcohol. Please circle the number beside each statement which best describes how much you could resist drinking in each case.

I am very sure I could NOT resist drinking	I most likely would NOT resist drinking	I probably could NOT resist drinking	I probably could resist drinking	I most likely could resist drinking	I am very sure I could resist drinking
1	2	3	4	5	6

1.	When I am at a party	1	2	3	4	5	6
2.	When I am listening to music or reading	1	2	3	4	5	6
3.	When my boy/girlfriend is drinking	1	2	3	4	5	6
4.	When my friends are drinking	1	2	3	4	5	6
5.	When I feel upset	1	2	3	4	5	6
6.	When I have just finished playing sport	1	2	3	4	5	6
7.	When I am feeling down	1	2	3	4	5	6
8.	When I first arrive home	1	2	3	4	5	6
9.	When I feel sad	1	2	3	4	5	6

Drinking Refusal Self-Efficacy Questionnaire-Revised Adolescent Version
(DRSEQ-RA)

Directions:

The following items ask you to describe your ability to handle drinking situations. Your answers will be completely anonymous so please try to answer as honestly as you can. The following pages contain a list of situations in which people may find themselves drinking alcohol. Most people find it easier to resist drinking in some of these situations than others. Please circle the number beside each statement which best describes how much you could resist drinking in each case.

I am very sure I could NOT resist drinking	I most likely would NOT resist drinking	I probably could NOT resist drinking	I probably could resist drinking	I most likely could resist drinking	I am very sure I could resist drinking
1	2	3	4	5	6

1.	When I am watching TV	1	2	3	4	5	6
2.	When I am angry	1	2	3	4	5	6
3.	When I am having lunch	1	2	3	4	5	6
4.	When I am at a party	1	2	3	4	5	6
5.	When I am on my way home from school	1	2	3	4	5	6
6.	When someone offers me a drink	1	2	3	4	5	6
7.	When I feel frustrated	1	2	3	4	5	6
8.	When I am listening to music or reading	1	2	3	4	5	6
9.	When my boy/girlfriend is drinking	1	2	3	4	5	6
10.	When I am worried	1	2	3	4	5	6
11.	When I am by myself	1	2	3	4	5	6
12.	When my friends are drinking	1	2	3	4	5	6
13.	When I feel upset	1	2	3	4	5	6
14.	When I have just finished playing sport	1	2	3	4	5	6
15.	When I am at a nightclub/concert	1	2	3	4	5	6
16.	When I am feeling down	1	2	3	4	5	6
17.	When I first arrive home	1	2	3	4	5	6
18.	When I feel nervous	1	2	3	4	5	6
19.	When I feel sad	1	2	3	4	5	6

Chapter 6. Prospective Relationships Between Cognitive and Personality Risk Factors for Adolescent Alcohol Consumption

Overview

This chapter comprises a version of a manuscript published in *Addictive Behaviors* (citation below). The key concepts of the present thesis, drinking refusal self-efficacy, alcohol expectancies, rash impulsiveness, and reward drive, are tested prospectively using the bSCT model (Gullo et al., 2010). This is relevant to the current work as it improves understanding of how these risk factors form over time for different age groups. Through this understanding, unique pathways of intervention can be extrapolated, informing future interventions attempts.

Patton, K. A., Gullo, M. J., Connor, J. P., Chan, G. C. K., Kelly, A. B., Catalano, R. F., & Toumbourou, J. W. (2018). Social cognitive mediators of the relationship between impulsivity traits and adolescent alcohol use: Identifying unique targets for prevention. *Addictive Behaviors*, *84*, 79–85. doi:10.1016/j.addbeh.2018.03.031

Abstract

Background The mechanism linking impulsivity to adolescent alcohol use is unclear. We prospectively evaluated the cognitive mechanisms outlined in two-factor impulsivity models. **Methods** Two cohorts of adolescents followed for three years were included in the study (younger cohort: N = 908, aged 10–12 years at Time 1; older cohort: N = 943, aged 12–15 years at Time 1). Constructs measured included two impulsivity factors (reward drive, rash impulsiveness), cognitive mechanisms (positive social alcohol expectancies, drinking-refusal self-efficacy), family and community risk factors, and alcohol use. **Results** Data were analysed using structural equation modelling controlling for family and community risk factors, and mediation tested. Impulsivity traits predicted cognitive mechanisms and these in turn predicted alcohol use in both cohorts ($\chi^2 = 1,139.79$, $df = 249$, $p < .001$, CFI = .92, SRMR = .06, RMSEA = .04). Drinking-refusal self-efficacy and positive social alcohol expectancies mediated the effects of rash impulsiveness and reward drive, respectively, on alcohol use. In general, positive social alcohol expectancies had larger effects for the younger cohort and drinking-refusal self-efficacy had larger effects for older cohort. **Conclusions** The current study details the prospective interactive influences of impulsivity and cognitive risk factors on adolescent alcohol use. The findings have direct implications for prevention and treatment programs, providing information about possible high-impact targets for intervention.

Introduction

Adolescent alcohol use is associated with alcohol related problems, including mortality (Australian Institute of Health and Welfare, 2014; Geels et al., 2012; Keyes, Li, & Hasin, 2012). Due to the trajectory of alcohol use from adolescence to adulthood (Duncan, Duncan, & Strycker, 2006; Stoolmiller et al., 2012), prevention through identification and targeting of key risk factors is a commonly proposed solution to reducing alcohol-related harm. Consequently, there has been a push to identify modifiable psychological risk factors that predict alcohol use in adolescents.

Impulsivity is a strong predictor of adolescent alcohol use and problematic consumption (Moffitt et al., 2011; Stautz & Cooper, 2013; Stautz, Dinc, & Cooper, 2017). The adolescent developmental period is associated with increased impulsivity and sensitivity to reward due to rapid neurological changes which occur while the cognitive and emotional regulatory neurological systems are still developing (Gullo & Dawe, 2008; Steinberg, 2008). While there is debate over the structure of trait impulsivity, including the precise number of factors (Hamilton et al., 2015, 2016; Whiteside & Lynam, 2001), recent empirical studies (Sharma, Kohl, Morgan, & Clark, 2013; Stautz, Dinc, & Cooper, 2017) and a theoretical review (Gullo, Loxton, & Dawe, 2014) implicate two factors of impulsivity as uniquely involved in substance use.

According to the 2-Component Approach to Reinforcing Substances model (2-CARS; (Gullo & Dawe, 2008; Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010), impulsivity is composed of dual systems: one characterised by reward sensitivity and appetitive motivation, related to lower order mesolimbic dopamine systems (*reward sensitivity/drive*); and another characterised by difficulty inhibiting approach behaviour in light of negative future consequences, related to higher order prefrontal serotonergic systems (*“rash” impulsiveness*; Gullo et al., 2014a; Steinberg and Chein, 2015; Steinberg, 2008). Reward drive and rash impulsiveness create separate pathways of alcohol risk (Gullo, Ward, Dawe, Powell, & Jackson, 2011). Reward sensitive adolescents have a higher drive to seek the rewarding aspects of alcohol use, whereas rash impulsive adolescents may have a reduced capacity to inhibit the impulse to engage in drinking, end a drinking session, or to withdraw from drinking patterns once they are established (Dawe, Gullo, & Loxton, 2004).

Evidence from cross-sectional and prospective structural equation modelling analyses show that impulsivity traits have distinct associations with alcohol-related cognition; specifically, Reward Drive predicts increases in positive alcohol expectancies and Rash Impulsiveness predicts decreases in drinking-refusal self-efficacy (Connor, Gullo, Feeney, & Young, 2011; Gullo & Dawe, 2008; Gullo et al., 2010). Positive alcohol expectancies reflect an individual's positive beliefs about the outcomes of drinking. Adolescents, in particular, are driven by the expectations of social reward relating to alcohol use (Jester et al., 2015; Tomlinson & Brown, 2012). Drinking refusal self-efficacy pertains to an individual's belief in their ability to refrain from drinking alcohol in varying contexts (Oei & Baldwin, 1993). Both of these cognitive factors are robust predictors of hazardous alcohol use as well as earlier alcohol use onset (Connor, George, Gullo, Kelly, & Young, 2011; Ehret, Ghaidarov, & LaBrie, 2014; Morawska & Oei, 2005) and are of particular utility in alcohol naïve and adolescent populations as they can be derived from vicarious as well as direct experience (e.g., observing alcohol effects on parents; Bandura, 1977, 1986).

The 2-CARS model has been expanded to include the Social Cognitive Theory (SCT) risk factors, a model known as the bioSocial Cognitive Theory (bSCT) (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010). The bSCT theorises that rash impulsiveness and reward drive are, in part, mediated through positive alcohol expectancies and drinking refusal self-efficacy. Reward drive is hypothesised to produce a learning bias for the rewarding/positive outcomes of drinking, which then increases alcohol use and undermines drinking refusal self-efficacy. In contrast, rash impulsiveness' impact on alcohol misuse is thought to be mediated by reduced drinking refusal self-efficacy, which then increases alcohol misuse. That is, individuals who know they tend to act without considering the consequences of their actions may be less likely to believe they can refuse alcohol in a tempting situation. It is proposed that this creates a self-fulfilling prophecy that is reinforced when realised. Several studies now empirically support the bSCT model in a range of populations (Gullo, St. John, et al., 2014; Harnett, Lynch, Gullo, Dawe, & Loxton, 2013; Kabbani & Kambouropoulos, 2013; Leamy, Connor, Voisey, Young, & Gullo, 2016; Papinczak et al., in press).

While these studies have furthered the understanding of the mechanisms of impulsivity on alcohol use, no study has examined prospective mediation of impulsivity-related risk by cognition. Prospective analyses are pertinent for adolescent populations as

recent evidence has shown the importance of considering developmental perspectives on substance use interventions (Onrust, Otten, Lammers, & Smit, 2016). Onrust and colleagues (2016) found in their systematic review and meta-regression analysis that characteristics of intervention programs had differing effects at different developmental stages. For example, teaching drinking refusal skills – a common prevention technique that presumably increases self-efficacy – actually increased alcohol use in early and mid-adolescents, and only reduced drinking for late adolescents.

This study prospectively examined the relationships between impulsivity, alcohol-related cognition, and growth in alcohol consumption and hazardous alcohol use in adolescents, controlling for family and community risk factors. Data were analyzed from 1,911 Australian adolescents who took part in the International Youth Development Study (IYDS; McMorris, Hemphill, Toumbourou, Catalano, & Patton, 2007). The hypothesized model based on the bSCT model is depicted in Figure 6.1. It is hypothesised that (a) drinking refusal self-efficacy will mediate the relationship between rash impulsiveness and alcohol misuse, (b) positive alcohol expectancies will mediate the relationship between reward drive and alcohol misuse, and (c) drinking refusal self-efficacy will mediate the relationship between positive alcohol expectancies and alcohol misuse. It is also hypothesised that the older and younger cohorts will differ in the size but not direction of proposed effects. We expect that as adolescents progress from elementary to early-adolescence and early to mid-adolescence, the impact of drinking refusal self-efficacy will increase as their exposure to alcohol increases (i.e., stronger positive DRSE-alcohol association). We also expect that the older adolescents' positive expectations of alcohol use will increase as their perceptions of reward and social influence become more salient and appealing, consistent with stage-relevant developmental tasks and cognitive maturation (i.e., significantly larger positive slope on expectancy growth) (Onrust et al., 2016).

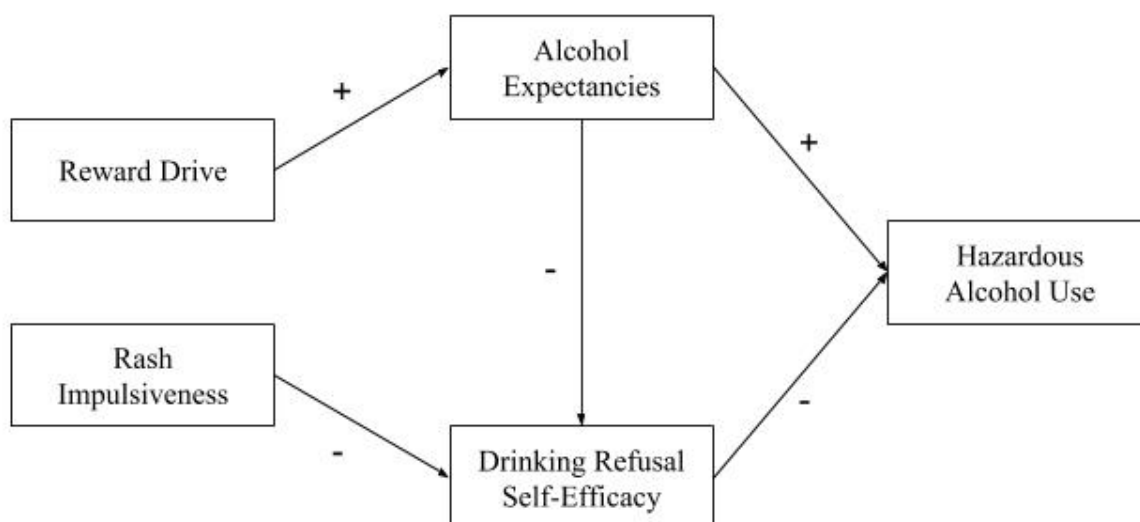


Figure 6.1 A simplified diagrammatic illustration of the hypothesised mediating relationships between variables in the current study

Method

Participants

The current study utilised the first three time points for two Australian cohorts (N = 1,911), Cohort 1 (younger cohort) and Cohort 2 (older cohort), from the International Youth Development Study (IYDS) (see Table 6.1 for participant information). The IYDS is an ongoing international longitudinal panel survey of three cohorts of Australian students (Grades 5, 7, and 9) in Victoria, Australia and Washington, the United States of America (only Australian data were used in the present study), obtained through two-stage cluster random sampling aimed at replication of state proportions of grade-level size and state versus public school distribution to ensure representativeness. This strategy was successful, with the IYDS data closely matching state diversity and school-type proportions (McMorris et al., 2007). In keeping with our focus on processes in early alcohol use, our analyses did not utilise the 9th grade (oldest) cohort. Further, there were only two waves of data collection in the 9th grade cohort and hence data from that cohort was unsuitable for our analysis. We requested and were granted access to the younger two cohorts only. The IYDS includes validated brief measures of impulsivity-related traits and comprehensive assessment of family and community risk factors for alcohol use (e.g., perceived availability of drugs, family conflict). The IYDS has utilised extensive strategies to achieve retention rates of up to 99% (Scholes-Balog, Hemphill, Reid, Patton, & Toumbourou, 2013). A detailed description of the

recruitment and data collection processes was given by McMorris, Hemphill, Toumbourou, Catalano, and Patton (2007).

Table 6.1

Demographic information.

		T1 (2002)	T2 (2003)	T3 (2004)	
Younger Cohort (N = 908)	Age range	10 – 12 years	11 – 13 years	12 – 14 years	
	Age <i>M</i> (<i>SD</i>) years	10.98(.40)	11.94(.40)	12.96(.40)	
	Grade	5	6	7	
	Gender - #female (%)	476 (52.4%)			
	Reward Drive: range, <i>M</i> (<i>SD</i>)	1-6, 2.47(1.78)	-	-	
	Average Rash Impulsiveness: range, <i>M</i> (<i>SD</i>)	1-4, 1.73(.60)	-	-	
	Drinking refusal self-efficacy: range, <i>M</i> (<i>SD</i>)	0-4, 3.25(.95)	0-4, 3.09(1.11)	0-4, 2.72(1.42)	
	Positive social alcohol expectancies: range, <i>M</i> (<i>SD</i>)	1-5, 1.70(1.15)	1-5, 1.70(1.14)	1-5, 2.07(1.25)	
	Average Family risk, <i>M</i> (<i>SD</i>)	1.50(.45)	-	-	
	Average Community risk, <i>M</i> (<i>SD</i>)	1.49(.38)	-	-	
	Older Cohort (N = 943)	Age range	12 – 15 years	13 – 15 years	14 – 16 years
		Age <i>M</i> (<i>SD</i>) years	12.92(.41)	13.96(.40)	14.98(.40)
Grade		7	8	9	
Gender - #female (%)			491 (52.1%)		
Reward Drive: range, <i>M</i> (<i>SD</i>)		1-6, 2.30(1.68)	-	-	
Average Rash Impulsiveness: range, <i>M</i> (<i>SD</i>)		1-4, 1.90(.56)	-	-	
Drinking refusal self-efficacy: range, <i>M</i> (<i>SD</i>)		0-4, 2.58(1.48)	0-4, 2.01(1.69)	0-4, 1.45(1.71)	
Positive social alcohol expectancies: range, <i>M</i> (<i>SD</i>)		1-5, 2.49(1.41)	1-5, 2.75(1.29)	1-5, 3.05(1.21)	
Average Family risk, <i>M</i> (<i>SD</i>)		1.63(.53)	-	-	
Average Community risk, <i>M</i> (<i>SD</i>)		1.72(.48)	-	-	

Measures

Impulsivity

Rash impulsiveness was assessed at Time 1 using the IYDS Impulsivity scale, which comprises three items on a 4-point Likert scale (1 ‘NO!’ to 4 ‘YES!’), e.g., “It’s important to think before you act”. **Reward drive** was measured at Time 1 using the item “How many times have you done what feels good no matter what?” (1 ‘Never’ to 6 ‘Once a week or more’). This item was derived from the 3-item IYDS Sensation Seeking scale. The other two items of the scale were not retained to reduce contamination by content related to disinhibition or insensitivity to danger (e.g., "Done something dangerous because someone dared you to do it?"), which the 2-CARS model considers to be more aligned with rash impulsiveness (Dawe & Loxton, 2004). The single item was strongly correlated with the three-item scale, younger cohort $r = .78$, older cohort $r = .80$, giving evidence for construct validity of the single item. Both the IYDS impulsivity and sensation seeking scales have previously shown significant associations with adolescent alcohol problems (Mason, Toumbourou, Herrenkohl, Hemphill, & Patton, 2011).

Alcohol use.

At Time 1, adolescents were assessed on drinking status (‘Never’ drank; drank ‘Once or more’). At Times 2 and 3, both cohorts were asked about their current drinking (30 days), drinking in the past year (1 ‘Never’ to 8 ‘40+ times’) and their binge drinking (occasions of five or more drinks in last two weeks; 1 ‘None’ to 6 ‘10 or more times’). For analysis, these items at Time 2 and 3 were combined into latent factors, allowing our alcohol outcome to include several aspects of the construct and adding comprehensiveness to otherwise single-item scales (Muthén, 2002).

Alcohol-related cognitions.

Proxy measures were chosen for alcohol related cognition, measured at all three time points. To measure **positive (social) alcohol expectancies**: “What are the chances you would be seen as cool if you: began drinking regularly, that is, at least once or twice a month?” (1 ‘No or Very Little Chance’ to 5 ‘Very Good Chance’). While this is a single-item measure, previous research has found that social alcohol expectations are a robust influence on adolescent alcohol consumption (Patton et al., 2017; Young-Wolff et al., 2015). **Social pressure drinking refusal self-efficacy (DRSE)** was measured using the item DRSE1 “You

are at a party at someone's house, and one of your friends offers you a drink containing alcohol. What would you say or do?" ('Drink it' (0) vs. 'Don't drink it' (+1)). That is, it assessed the predicted outcome of a hypothetical future situation involving an alcohol offer. The second item (DRSE2) was, "Do you think you would handle this situation well: Peer offers you a drink at a party?" (1 'NO!' to 4 'YES!'). That is, the item assessed perceived confidence in the participant's ability to enact this intended behavior. A product term of DRSE1xDRSE2 was computed to weight the response to DRSE2 and reflect strength of confidence in refusing alcohol. In this way, the product reflects established measures of social pressure refusal self-efficacy (e.g., the Social Pressure subscale of the Drinking Refusal Self-Efficacy Questionnaire-Revised Adolescent Version; Young, Hasking, Oei, & Loveday, 2007) by creating a score that indexes participants' confidence in their ability to refuse an offer of alcohol from a peer. The drinking refusal self-efficacy score showed significant negative correlations with positive alcohol expectancy items as would be expected based on previous literature and drinking refusal self-efficacy.

Other risk factors.

Family risk factors consisted of family history of antisocial behaviour, family conflict, and poor family management, which are known correlates of adolescent alcohol use (Habib et al., 2010; Kelly et al., 2011). Community risk factors were based on community disorganization, laws and norms favourable to drug use, and perceived availability of drugs (Smith et al., 2013). Composite variables for both overall risk factors were calculated to reduce model complexity (Jöreskog & Sörbom, 1989).

Analytical procedure.

The prospective relationships between variables were analyzed in Amos (version 22) using structural equation modelling (SEM). A multigroup SEM was used to examine the relationships between impulsivity variables (rash impulsiveness and reward drive), cognitive variables (positive alcohol expectancies and drinking refusal self-efficacy, and Time 2 and Time 3 alcohol use in the younger cohort (group 1) and the older cohort (group 2). Unconditional latent growth models and alcohol use outcomes showed good fit to the data (see table 6.4 in the supplementary materials). Mediation hypotheses were tested using bootstrapped indirect effects (2000 samples) (Hayes, 2009) and the distribution of the products of coefficients method in RMediation, which demonstrates more accurate Type I error rates and more power than other, more commonly used tests, including bias-corrected

confidence intervals (Tofighi & MacKinnon, 2011). Both methods produce Confidence Intervals but *p*-values are only given for bootstrapped indirect and direct effects as *p*-values are not calculated in the distribution of the products of coefficients method of mediation. Multigroup SEM (older vs younger cohort) was used to account for and test cohort differences. Significance was tested using χ^2 difference tests comparing a multi-group model (older and younger cohorts) in which paths of interest were constrained to equality across cohorts and a second model where paths were free to vary. A reduction in model fit when paths are constrained to equality indicates moderation by developmental stage.

The hypothesised structural model (see Figures 6.2 and 6.3) included reward drive and the latent factor of rash impulsiveness predicting 3-time-point growth curve models of positive alcohol expectancies (positive growth) and drinking refusal self-efficacy (negative growth) as well as the latent factors of Time 2 and Time 3 alcohol use. Family risk and community risk composite variables were calculated and introduced into the model as covariates, with direct paths to Time 2 and Time 3 alcohol use and covariances with rash impulsiveness, reward drive, Time 1 alcohol use, and each other. These covariances and covariates are not shown in Figures 6.2 and 6.3 for clarity of exposition.

The χ^2 test statistic, comparative fit index (CFI), the standardized root mean-square residual (SRMR), and the root mean-square error of approximation (RMSEA) were used to examine model fit. The model was considered as having good fit if the values approached $CFI \geq .95$, $RMSEA \leq .06$, and $SRMR \leq .08$ (Hu & Bentler, 1999; Marsh, Hau, & Wen, 2004).

Results

Sixty (3.14%) students were removed from the dataset based on negative responses to at least one of the honesty measures for each time point (e.g., reporting use of a fake illicit substance). After deletion, 1,851 students remained. Little's Missing Completely at Random (MCAR; Little, 1988) test was non-significant for the older cohort, $\chi^2(DF = 1,140) = 1,218.78, p = .052$. For the younger cohort, data were not missing completely at random according to Little's MCAR test, $\chi^2(DF = 722) = 1450.54, p < .001$. However, there was no clear pattern to missing data based on T1 variables. For example, some rash impulsiveness items were related to increased missingness on Wave 3 alcohol variables. However, rash impulsiveness items were not related to missingness on other variables. Additionally, there

was no variable missing more than 5%. Therefore it was considered appropriate to impute missing data using the Expectation Maximisation method in SPSS (version 22).

Alcohol use.

Approximately half of the adolescents in both cohorts reported having consumed a full alcoholic beverage at Time 1. In Waves 2 and 3, the older cohort showed higher rates of binge drinking, current drinking, and past-year drinking (see Table 6.2).

Table 6.2

Frequency of participants scoring greater than “none” or “never” on alcohol measures and average alcohol use for each alcohol measure across cohorts and time-points.

Alcohol use		Younger cohort (N = 908)		Older cohort (N = 943)	
		N > “none/never” (%)	M (SD)	N > “none/never” (%)	M (SD)
T1	Ever consumed full alcoholic drink	465 (51.2%)	N/A	553 (58.6%)	N/A
T2	Ever binge drank	46 (5.1%)	1.06 (.30)	170 (17.9%)	1.30 (.78)
	Currently drinking	231 (25.4%)	1.36 (.79)	415 (44.0%)	1.76 (1.19)
	Drank in past year	271 (29.8%)	1.48 (1.59)	531 (56.3%)	2.45 (1.86)
T3	Ever binge drank	69 (7.6%)	1.12 (.48)	295 (31.4%)	1.57 (1.05)
	Currently drinking	263 (29.0%)	1.49 (.99)	569 (60.4%)	2.17 (1.40)
	Drank in past year	334 (36.8%)	1.79 (1.39)	678 (72.0%)	3.25 (2.19)

Prospective structural model fit.

The mediating factors of drinking refusal self-efficacy and positive social alcohol expectancies were both fitted as multi-group latent (linear) growth curve models. Drinking

refusal self-efficacy showed poor fit to the data, $M_{\text{intercept}}(SE) = 2.96(0.3)$, $\text{Variance}_{\text{intercept}}(SE) = .77(.06)$, $ps < .001$, $M_{\text{slope}}(SE) = .39(0.2)$, $\text{Variance}_{\text{slope}}(SE) = .24(.03)$, $ps < .001$, $\chi^2 = 706.22$, $df = 11$, $p < .001$, $CFI = .17$, $RMSEA = .19$, as did positive social alcohol expectancies, $M_{\text{intercept}}(SE) = 2.08(0.3)$, $\text{Variance}_{\text{intercept}}(SE) = .96(.06)$, $ps < .001$, $M_{\text{slope}}(SE) = .24(0.2)$, $\text{Variance}_{\text{slope}}(SE) = .16(.02)$, $ps < .001$, $\chi^2 = 517.64$, $df = 11$, $p < .001$, $CFI = .26$, $RMSEA = .16$. However, when these factors were added to the larger multigroup SEM, the model showed acceptable-to-good fit to the data across both younger and older cohorts, $\chi^2 = 1,139.79$, $df = 249$, $p < .001$, $CFI = .92$, $SRMR = .06$, $RMSEA = .04$ (see Figure 6.2). When run without the multigroup inclusion, negative error variances were present, indicating the appropriateness of the present multigroup approach and moderation analyses.

Prospective relationships between predictors and alcohol use.

In the final multigroup model, higher rash impulsiveness was related to decreased drinking refusal self-efficacy (intercept) for both cohorts as well as decreased growth in drinking refusal self-efficacy for the younger cohort. This, in turn, predicted increased alcohol use, as higher drinking refusal self-efficacy (intercept) was related to decreased future alcohol use. Higher reward drive was related to increased positive alcohol expectancies (intercept) for the older cohort but not the younger cohort.

Higher positive alcohol expectancies were positively related to alcohol use in both cohorts. For the younger cohort, positive alcohol expectancies at Time 1 (intercept) predicted increased alcohol use at Time 2. For the older cohort, adolescents with high positive alcohol expectancies at Time 1 (intercept) had less growth of positive alcohol expectancies (slope) over time. This suggests a possible ceiling effect. For this cohort, positive alcohol expectancies growth (slope) was associated with increased alcohol use at Time 3.

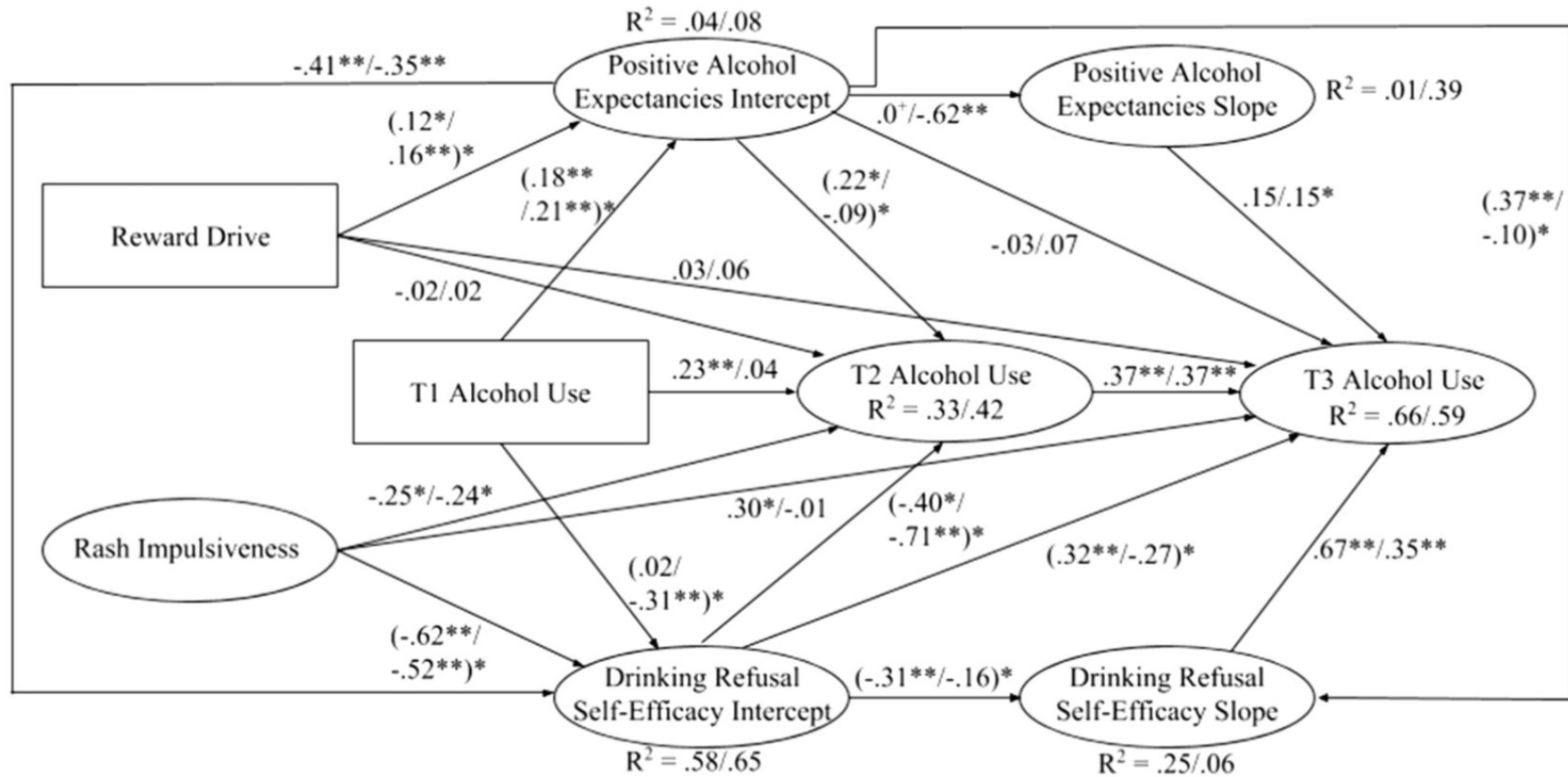


Figure 6.2 Structural equation modelling analysis for both cohorts (younger cohort/older cohort) controlling for Time 1 (T1) alcohol use, family risk, and community risk. Paths in brackets significantly moderated. * $p < .05$ ** $p < .001$. Note. Drinking refusal self-efficacy slope is negative. †constrained to be .0 due to floor effect.

Mediation analyses

Hypothesis (a). For the younger cohort, rate of drinking refusal self-efficacy decrease (slope) mediated the relationship between rash impulsiveness and Time 3 alcohol use (95% CI [-.146, -.009]) and Time 2 alcohol use (95% CI [.011, .111]). For the older cohort, drinking refusal self-efficacy at Time 1 (intercept) mediated the relationship between rash impulsiveness and Time 2 and Time 3 alcohol use, 95% CIs [.196, .518] and [.064, .366], respectively. It is theorised that these strong mediational effects resulted in the negative paths between rash impulsiveness and Time 2 alcohol use for both cohorts, as the variance is explained by drinking refusal self-efficacy.

Hypothesis (b). Reward drive impacted Time 2 and 3 alcohol use through positive alcohol expectancies and drinking refusal self-efficacy. Reward drive significantly indirectly affected T2 alcohol use, $\beta = .008$, 95% CIs [.003, .016], $p = .002$. While the direct and indirect effects of reward drive on Time 3 alcohol use were not significant, the total effect was, presumably due to reward drive's effect on Time 2 drinking, positive alcohol expectancies and consequently drinking refusal self-efficacy, $\beta = .036$, 95% CIs [.009, .066].

Hypothesis (c). Initial drinking refusal self-efficacy (intercept) also mediated the relationship between initial positive alcohol expectancies (intercept) and Time 2 alcohol use for the younger cohort (95% CI [.006, .07]) and both Time 2 and Time 3 alcohol use for the older cohort, 95% CIs [.07, .185] and [.023, .131], respectively.

Moderation analyses

Multigroup SEM analyses was used to examine moderated relationships between the older and younger cohort. Only significant paths were tested for non-equivalence. The model fit of the model with path coefficients not constraint to be the same between the young and old cohort was significantly better than the constraint model, unconstrained $\chi^2(df) = 1157.16(256)$, constrained $\chi^2(df) = 1694.30(285)$, χ^2 difference = 537.14 (26), $p < .001$. Chi-square difference tests revealed significant differences between the older and younger cohorts (see Table 6.3 and Figure 6.2). In general, the effects of positive alcohol expectancies were stronger for the younger

cohort and the effects of drinking refusal self-efficacy were stronger for the older cohort (see supplementary materials for description of specific effects).

Table 6.3

Path level moderation analyses of cohort.

Path	Younger cohort standardised coefficient	Older cohort standardised coefficient
RD --> PAE Intercept	.12*	.16*
PAE intercept --> T2 Alcohol use	.22*	-.09
T1 Alcohol use --> PAE intercept	.18**	.21**
T2 Alcohol use --> Family risk	.06	.12*
DRSE intercept --> T2 Alcohol use	-.40*	-.71**
DRSE intercept --> T3 Alcohol use	.32**	.27**
DRSE intercept --> DRSE slope	-.31**	-.16
RI --> DRSE intercept	-.62**	-.52**
T1 Alcohol use --> DRSE intercept	.02	-.31**
PAE intercept --> DRSE slope	.37**	-.10

Note. Only paths variant at $p < .05$ shown, $p < .05$, ** $p < .001$, * $p < .05$. DRSE = drinking refusal self-efficacy, PAE = positive alcohol expectancies, RI = rash impulsiveness, RD = reward drive.

Discussion

This large scale study ($N = 1,911$) tested prospective mediation relationships between impulsivity and social cognitive risk factors for adolescent alcohol use. The hypothesised model was based on the bioSocial Cognitive Theory (bSCT) model of substance use (Gullo, St. John, et al., 2014). Results demonstrated the selective influence for reward drive and rash impulsiveness on the development of alcohol-related beliefs and adolescent drinking over two subsequent years.

Our findings show that pathways to alcohol use change during early to mid-adolescence, providing unique targets for intervention. Positive alcohol expectancies convey greater risk for future alcohol use in younger adolescents, both directly and through their deleterious impact on drinking-refusal self-efficacy. Steeper decline in drinking-refusal self-efficacy from age 11–13 years was the largest predictor of later alcohol use. It is likely that positive expectancies undermine drinking-refusal self-efficacy as adolescents may find it more difficult to resist a substance they believe will lead to rewarding social consequences, i.e., social approval and facilitation (Gullo et al., 2010). This is consistent with the positive associations between the two cognitive domains reported here. It likely also reflects greater exposure to situations in which peer alcohol offers are encountered.

The results suggest that alcohol expectancies and drinking-refusal self-efficacy provide high-impact targets for adolescents aged 11–13 years. Past research has shown that simple, direct strengthening of drinking-refusal skills through training can have adverse effects in early and middle adolescence, increasing alcohol use (Onrust et al., 2016). Our findings suggest another pathway to improving self-efficacy may be decreasing positive expectancies and targeting rash impulsiveness, which also predicted drinking refusal self-efficacy. In this way, the proposed model may provide new avenues for prevention by identifying how to target key cognitive mechanisms to better effect.

For older adolescents aged 13–15 years in particular, impulsivity emerged as a risk factor for drinking through effects on cognitive mediators. Reward drive emerged as a risk factor for greater alcohol use in the older cohort, possibly due to the presence of a bidirectional relationship with reinforcement sensitivity with the older cohort's increased exposure to alcohol use (Lopez-Vergara et al., 2012). Reward drive at age 13 was associated with drinking at age 15 through its effect on the other factors within the model (i.e., positive alcohol expectancies and their effect on drinking-refusal self-efficacy), indicating its increasing influence on alcohol use as adolescents age. As age was associated with increased alcohol use, this suggests that impulsivity may garner risk for increased alcohol use through experiential rather than vicarious alcohol learning. Rash impulsiveness also had larger impacts on later drinking for the older cohort, supporting the increased impact of impulsivity during this period. Much of this risk was conveyed through

reduced drinking-refusal self-efficacy skills. Adolescents with higher rash impulsiveness may have a reduced capacity to end a drinking session, withdraw from drinking patterns once they are established, or to inhibit the impulse to engage in risk-taking behaviour (Dawe et al., 2004). The mediating role of drinking-refusal self-efficacy suggests this could encourage a self-fulfilling prophecy of poor drinking control, further exacerbating risk. Thus, approaches targeting impulsivity directly, e.g., mindfulness meditation (Robinson, Ladd, & Anderson, 2014), and drinking-refusal self-efficacy may be particularly effective at this age. As discussed earlier, the latter may best be achieved by targeting other components of the hypothesized causal chain. A worthwhile area of future research may be to investigate non-additive (interaction) effects of impulsivity on cognition, as adolescents may be high in both rash impulsiveness and reward drive. Additionally, further investigation could be conducted on the effects of these interventions on high impulsivity adolescents, as previous targeted interventions have found that effects may be stronger for these individuals (Conrod et al., 2008, 2006; Lammers et al., 2017). The subsample of high-impulsivity adolescents was not adequately powered for this analysis in the present study.

A limitation of the present study is the use of proxy measures for some constructs. While brief measures are not uncommon in large-scale survey studies, the use of validated measures of alcohol-related cognition is preferable but can be impractical. Importantly, (Connor, George, Gullo, Kelly, & Young, 2011) reported similar effect sizes between social cognitive mediators and alcohol use in a comparable population, suggesting that the effects found in the present study may not have been severely impacted by measurement. Additionally, Time 1 alcohol use was measured dichotomously, which reduced the sensitivity in controlling for baseline differences in alcohol use. Further investigation is needed into whether the current model is applicable to older adolescents and those with more drinking experience. Even so, the support of the model in adolescents as young as 11 and 13 years old is promising. Thus, the model may demonstrate applicability for early interventions.

A further limitation is the overlap of several measurement occasions. While the bSCT model showed the interrelationships of impulsivity and cognitive risk factors over 3 time-points, some of the associations between impulsivity and cognitive risk-factors were cross-sectional, due

to the need for a minimum of three time points to construct a growth curve. Interpretations of causality are somewhat limited by this. However, care was taken in the model of reducing cross-sectional relationships. Therefore, we do believe that there is initial evidence of prospective mediation in the current analysis. Future research could solidify the causal mediational links through inclusion of a larger number of assessment occasions. Previous dual-process analyses have shown that contexts of risk and the processes by which trait impulsivity impacts on “risky” behaviour can alter the effects of impulsivity (Wiers, Ames, Hofmann, Krank, & Stacy, 2010). This paper points to several cognitive processes behind the effects of impulsivity but future research could expand on this by looking at the effects of implicit and explicit expectancies.

In conclusion, the present study provides evidence for the importance of drinking-refusal self-efficacy and positive alcohol expectancies as mediators of the impact of impulsivity on the development of future adolescent alcohol use. We found that the model was predictive across two large, representative cohorts of young adolescents across three years. While this model should be replicated with purpose-made social cognition measures and in older cohorts, it may be useful in understanding the interplay of personality and cognitive influences on adolescent alcohol use and inform targets for prevention programs.

Supplementary Materials

Table 6.4.

Model fit for cognitive factor Latent Growth Curve models (LGM) and alcohol use outcomes for the older and younger cohorts.

		χ^2	df	CFI	SRMR	RMSEA	AIC
Younger cohort	Drinking refusal self-efficacy LGM	12.79*	3	0.97	0.02	0.06	24.79
	Positive social alcohol expectancies LGM	28.16**	3	0.91	0.02	0.10	40.16
	Alcohol use	38.24**	10	0.99	0.03	0.06	88.24
Older cohort	Drinking refusal self-efficacy LGM	18.77**	3	0.97	0.02	0.08	30.77
	Positive social alcohol expectancies LGM	20.67**	3	0.96	0.03	0.08	32.67
	Alcohol use	77.86**	10	0.98	0.04	0.09	127.86

** $p < .001$, * $p < .05$

Detailed explanation of paths moderated by cohort

For the younger cohort, positive alcohol expectancies at Time 1 increased drinking at Time 2 and contributed to steeper decline in drinking refusal self-efficacy over the three years, where neither of these relationships were significant for the older cohort. For the older cohort, higher Time 1 drinking refusal self-efficacy significantly decreased Time 2 alcohol use at a greater rate than for the younger cohort and decreased Time 3 alcohol use where there was no significant relationship for the younger cohort. Despite this, high drinking refusal self-efficacy at T1 was related to less decline in drinking refusal self-efficacy over time for the younger cohort and not for the older cohort. Cohort also had effects on the relative strength of impulsivity on the cognitive mediators. The relationship between reward drive and positive alcohol expectancies was stronger for the older cohort and the relationship between rash impulsiveness and drinking refusal self-efficacy was stronger for the younger cohort. Finally, Time 1 alcohol use was related

to decreased drinking refusal self-efficacy and slightly increased positive alcohol expectancies for the older cohort compared with the younger cohort.

Table 6.5.

Correlation (Spearman's Rho) matrix for Younger Cohort (N = 908).

	RD	RI	PAE			DRSE			Life AU	Past Year AU			Binge AU		Current AU		F Risk C	C Risk C
	T1 RD	T1 RI	T1	T2	T3	T1	T2	T3	T1	T2	T3	T2	T3	T2	T3	T1	T1	
T1 RD	1	.18**	.12**	.09**	.11**	-0.01	-0.03	-0.05	.20**	.09**	.11**	-0.02	0.04	.07*	.07*	.18**	.22**	
T1 RI	.18**	1	.16**	.12**	.13**	-.22**	-.14**	-.16**	.23**	.11**	.18**	0.06	.11**	.14**	.18**	.37**	.27**	
T1 PAE	.12**	.16**	1	.31**	.24**	-.14**	-.10**	-.13**	.11**	.15**	.15**	.08*	.10**	.14**	.13**	.24**	.28**	
T2 PAE	.09**	.12**	.31**	1	.41**	-0.03	-.26**	-.26**	.14**	.25**	.25**	.12**	.17**	.25**	.22**	.16**	.21**	
T3 PAE	.11**	.13**	.24**	.41**	1	-0.03	-.19**	-.35**	.09**	.17**	.27**	0.06	.25**	.17**	.28**	.17**	.20**	
T1 DRSE	-0.01	-.22**	-.14**	-0.03	-0.03	1	.26**	.22**	-.08*	-0.05	-.09**	0.02	-0.06	-0.04	-.08*	-.17**	-.15**	
T2 DRSE	-0.03	-.14**	-.10**	-.26**	-.19**	.26**	1	.41**	-.09**	-.21**	-.27**	-.19**	-.17**	-.22**	-.25**	-.18**	-.18**	
T3 DRSE	-0.05	-.16**	-.13**	-.26**	-.35**	.22**	.41**	1	-.07*	-.22**	-.39**	-.17**	-.35**	-.22**	-.38**	-.17**	-.19**	
T1 Life AU	.20**	.23**	.11**	.14**	.09**	-.08*	-.09**	-.07*	1	.35**	.28**	.14**	0.06	.30**	.24**	.39**	.26**	
T2 Past Year AU	.09**	.11**	.15**	.25**	.17**	-0.05	-.21**	-.22**	.35**	1	.43**	.30**	.22**	.73**	.40**	.25**	.21**	
T3 Past Year AU	.11**	.18**	.15**	.25**	.27**	-.09**	-.27**	-.39**	.28**	.43**	1	.14**	.38**	.38**	.78**	.28**	.20**	
T2 Binge AU	-0.02	0.06	.08*	.12**	0.06	0.02	-.19**	-.17**	.14**	.30**	.14**	1	.18**	.33**	.21**	.15**	.14**	
T3 Binge AU	0.04	.12**	.10**	.17**	.25**	-0.06	-.17**	-.35**	0.06	.22**	.38**	.18**	1	.22**	.45**	.19**	.13**	
T2 Current AU	.07*	.14**	.14**	.25**	.17**	-0.04	-.22**	-.22**	.30**	.73**	.38**	.33**	.22**	1	.39**	.22**	.19**	
T3 Current AU	.07*	.18**	.13**	.22**	.28**	-.08*	-.25**	-.38**	.24**	.40**	.78**	.21**	.45**	.39**	1	.29**	.18**	
T1 F Risk C	.18**	.37**	.24**	.16**	.17**	-.17**	-.18**	-.17**	.39**	.25**	.28**	.15**	.19**	.22**	.29**	1	.47**	
T1 C Risk C	.21**	.27**	.28**	.21**	.20**	-.15**	-.18**	-.19**	.26**	.21**	.20**	.14**	.13**	.20**	.18**	.47**	1	

Note. ** $p < .001$, * $p < .05$ RI = Rash Impulsiveness, RD = Reward Drive, PAE = Positive Alcohol Expectancies, DRSE = Drinking Refusal Self-Efficacy, Life AU = Lifetime Alcohol Use, F Risk C = Family Risk Composite, C Risk C = Community Risk Composite

Table 6.6.

Correlation (Spearman's Rho) matrix for Older Cohort (N = 943).

	RD		PAE			DRSE			Life AU			Binge AU		Current AU		F Risk C	C Risk C
	T1 RD	T1 RI	T1	T2	T3	T1	T2	T3	T1	T2	T3	T2	T3	T2	T3	T1	T1
T1 RD	1	.31**	.20**	.12**	.11**	-.22**	-.20**	-.18**	.19**	.19**	.19**	.17**	.20**	.18**	.19**	.25**	.19**
T1 RI	.31**	1	.19**	.09**	0.04	-.32**	-.22**	-.22**	.21**	.21**	.19**	.16**	.21**	.20**	.18**	.32**	.30**
T1 PAE	.20**	.19**	1	.42**	.29**	-.30**	-.22**	-.11**	.16**	.20**	.17**	.12**	.15**	.12**	.14**	.25**	.29**
T2 PAE	.12**	.09**	.42**	1	.46**	-.18**	-.28**	-.21**	.18**	.27**	.23**	.20**	.16**	.28**	.17**	.20**	.23**
T3 PAE	.11**	0.04	.29**	.46**	1	-.10**	-.19**	-.28**	.11**	.20**	.30**	.13**	.26**	.19**	.25**	.11**	.12**
T1 DRSE	-.22**	-.32**	-.30**	-.18**	-.10**	1	.42**	.32**	-.30**	-.30**	-.29**	-.23**	-.28**	-.24**	-.27**	-.35**	-.35**
T2 DRSE	-.20**	-.22**	-.22**	-.28**	-.19**	.42**	1	.49**	-.34**	-.48**	-.45**	-.38**	-.38**	-.43**	-.41**	-.33**	-.33**
T3 DRSE	-.18**	-.22**	-.11**	-.21**	-.28**	.32**	.49**	1	-.27**	-.40**	-.56**	-.29**	-.46**	-.38**	-.52**	-.27**	-.23**
T1 Life AU	.19**	.21**	.16**	.18**	.11**	-.30**	-.34**	-.27**	1	.43**	.38**	.22**	.26**	.35**	.35**	.34**	.31**
T2 Past Year AU	.19**	.21**	.20**	.27**	.20**	-.30**	-.47**	-.40**	.43**	1	.58**	.48**	.47**	.76**	.52**	.35**	.31**
T3 Past Year AU	.19**	.19**	.17**	.23**	.30**	-.29**	-.45**	-.56**	.38**	.58**	1	.38**	.61**	.52**	.82**	.35**	.32**
T2 Binge AU	.17**	.16**	.12**	.18**	.13**	-.23**	-.38**	-.29**	.22**	.48**	.38**	1	.43**	.55**	.41**	.27**	.28**
T3 Binge AU	.20**	.21**	.15**	.16**	.26**	-.28**	-.38**	-.46**	.26**	.47**	.61**	.43**	1	.48**	.64**	.32**	.26**
T2 Current AU	.18**	.20**	.12**	.28**	.19**	-.24**	-.43**	-.38**	.35**	.76**	.52**	.55**	.48**	1	.52**	.30**	.29**
T3 Current AU	.19**	.18**	.14**	.17**	.25**	-.27**	-.41**	-.52**	.35**	.53**	.82**	.41**	.64**	.53**	1	.32**	.26**
T1 F Risk C	.25**	.32**	.25**	.20**	.11**	-.35**	-.33**	-.27**	.34**	.35**	.35**	.27**	.32**	.29**	.32**	1	.53**
T1 C Risk C	.19**	.30**	.29**	.23**	.12**	-.35**	-.33**	-.23**	.31**	1	.32**	.28**	.26**	.29**	.26**	.53**	1

Note. ** $p < .001$, * $p < .05$ RI = Rash Impulsiveness, RD = Reward Drive, PAE = Positive Alcohol Expectancies, DRSE = Drinking Refusal Self-Efficacy, Life AU = Lifetime Alcohol Use, F Risk C = Family Risk Composite, C Risk C = Community Risk Composite

Chapter 7. Additive effectiveness of mindfulness meditation to a school-based brief cognitive-behavioural alcohol intervention for adolescents

Overview

This chapter details a Randomised Control Trial to test the translational capacity of central components of the preceding work. A Cognitive Behavioural Therapy (CBT) intervention is evaluated with the aim of targeting alcohol-related cognitions. The additive effects of Mindfulness Meditation to the CBT program as a strategy to target rash impulsiveness is also investigated in comparison to an active control condition, which included Progressive Muscle Relaxation as a parallel intervention. Both active conditions were compared to an assessment-only control. The chapter currently has been submitted to a peer-reviewed journal.

Abstract

Objective: This randomized controlled trial is the first study to evaluate the additive efficacy of mindfulness meditation to brief school-based universal Cognitive Behavior Therapy (CBT+MM) for adolescent alcohol consumption. Previous studies have lacked strong controls for non-specific effects and treatment mechanisms remain unclear. The present study compared a CBT+MM condition to an active control CBT intervention with Progressive Muscle Relaxation (CBT+PMR) for non-specific effects, and an assessment-only control (AoC). Further, impulsivity and associated cognitive constructs that are hypothesized to relate to intervention effects were also analyzed. **Method:** Cluster sampling was used to recruit Australian adolescents ($N = 404$, 62% female) aged 13-17 years (Mean age = 14.99 years, $SD = .66$ years) of mostly Australian/New Zealand or European descent. School classes were randomized to one of the three intervention conditions (CBT+PMR = 8 classes, CBT+MM and AoC = 7 classes, respectively) and adolescents completed pre-intervention, post-intervention, 3-month and 6-month follow-up assessments, including measures of alcohol consumption, mindfulness, impulsivity, and the alcohol-related cognitions of alcohol expectancies and drinking refusal self-efficacy. **Results:** Multi-level modelling analyses revealed that both intervention conditions reduced the growth of alcohol consumption compared to the AoC ($B = -0.18$, $p = .014$), although CBT+MM was no more effective than CBT+PMR, $B = -0.06$, $p = .484$. Negative alcohol expectancies increased for adolescents in the intervention conditions compared to the AoC ($B = 1.09$, $p = .012$), as did positive alcohol expectancies, $B = 1.30$, $p = .008$. There was no effect of interventions on measures of mindfulness, drinking refusal self-efficacy, or impulsivity. **Conclusions:** There was no evidence of mindfulness-specific effects beyond existing effects of CBT within a brief universal school-based CBT intervention. Hypothesized mechanisms of change were largely unsupported.

Introduction

According to the World Health Organization (2014), 46.1% of 15-19 year-olds identify as current or former drinkers. Further, the pattern of use for this age group includes higher rates of monthly heavy episodic drinking compared to older alcohol users (World Health Organization, 2014). Adolescent alcohol use has been associated with decreased cognitive abilities (Nguyen-Louie et al., 2015), increased social problems, such as criminal offenses and employment issues (Jennings, Piquero, Rocque, & Farrington, 2015), high school non-completion (Kelly et al., 2015), and social anxiety (Spear, 2014), and reduced brain matter volume (Luciana, Collins, Muetzel, & Lim, 2014) and subsequent neurocognitive effects, including reduced memory, attention, and executive functioning (Lisdahl, Gilbert, Wright, & Shollenbarger, 2013). Due to the high prevalence of adolescent alcohol use and the associated consequences, prevention approaches have been proposed to ameliorate harms (Tripodi, Bender, Litschge, & Vaughn, 2010).

The meta-analysis of adolescent alcohol treatments by Tripodi and colleagues (2010) concluded that individual and several family-based adolescent alcohol treatment programs have shown large effects in reducing alcohol use for adolescents aged 12-19 years. Effects decrease over time (Tripodi et al., 2010). Interestingly, brief interventions also showed large effect sizes for a number of studies (Tripodi et al., 2010). Similarly, brief school-based interventions have shown low-to-mixed short-term evidence (Carney, Myers, Louw, & Okwundu, 2016). As schools provide an opportunity for maximum breadth of intervention targets (McLellan & Meyers, 2004), improving the effects of school-based interventions may provide an avenue for high impact.

The focus on mechanisms of change within interventions has been widely recommended to pinpoint areas of maximum impact for intervention and to identify the causal pathways of intervention effects within existing programs (Gaume, McCambridge, Bertholet, & Daepfen, 2014; O'Leary-Barrett et al., 2016). Further, there is evidence that intervention targets may produce differential effects according to the age of the intervention group (Onrust et al., 2016). A model of risk that can elucidate the inter-relationships between risk factors may assist intervention effort through identifying unique mechanisms by which to target these factors.

Within adult alcohol use treatment interventions, drinking refusal self-efficacy and alcohol expectancies are considered to be key factors in explaining onset and maintenance of

alcohol use disorders as well as mechanisms of treatment outcomes (Coates et al., 2018; Connor et al., 2016; Magill, Kiluk, McCrady, Tonigan, & Longabaugh, 2015). Drinking refusal self-efficacy refers to one's belief in their ability to refuse alcohol and alcohol expectancies encompass positive and negative beliefs regarding likely outcomes of alcohol consumption (Connor et al., 2016; Magill et al., 2015). Despite their importance in adult treatment and their prospective association with adolescent alcohol use (Connor et al., 2011), there has been little research into whether these factors influence adolescent intervention outcomes (Black & Chung, 2014). Adults drink more frequently while adolescents have higher single occasion consumption, and adolescent use is associated with higher rates of mood, conduct disorders, and future alcohol-related problems (Deas, Riggs, Langenbucher, Goldman, & Brown, 2000). Due to the differing clinical profiles, it cannot be assumed that adults and adolescents will respond similarly to treatments and hypothesized treatment mechanisms (Deas et al., 2000). Developmental differences could be substantial. Indeed, targeting refusal skills can actually increase alcohol use in middle adolescence, rather than decrease it (Onrust et al., 2016).

If targeting drinking refusal self-efficacy is important, but addressing it directly can be detrimental during adolescence, interventions could improve efficacy through targeting related factors. Adolescence is a unique risk period for the development of alcohol use and dependence due, in part, to neurodevelopmental changes involving reduced executive functioning (especially impulse control) within the context of increased sensitivity to reward (Robert & Schumann, 2017). It is no surprise then, that while other personality factors such as neuroticism, agreeableness, and openness (Chassin, Flora, & King, 2004), as well as individual differences in depression, stress, and emotion regulation (Gigsby, Forster, Unger, & Sussman, 2016) contribute to adolescent alcohol use, impulsivity is consistently found to be a large predictor of alcohol consumption and problems, especially amongst adolescents (Gigsby et al., 2016; Stautz & Cooper, 2013). Additionally, adolescents are particularly influenced by social dynamics, which influence appraisals and perceived drinking norms (Colder et al., 2017).

Elevated reward drive (also referred to as trait Reward Drive, Approach Motivation, or Sensation Seeking) has been hypothesized to facilitate the formation of positive alcohol expectancies, which in turn increase alcohol use (Gullo et al., 2010a). On the other hand, high rash impulsivity (trait Rash Impulsiveness, Disinhibition, or Lack of Premeditation) undermines drinking refusal self-efficacy, predicting increased use (Gullo et al., 2010a).

Additionally, high positive expectancies and low negative expectancies are thought to decrease drinking refusal self-efficacy, which in turn predicts higher consumption (Gullo et al., 2010a). This bioSocial Cognitive Theory (bSCT) of substance use has been supported in community samples (Gullo et al., 2010a; Harnett et al., 2013; Kabbani & Kambouropoulos, 2013), and treatment-seeking cannabis and alcohol dependent adults (Gullo, St. John, et al., 2014; Papinczak et al., 2018), as well as adolescent populations (Patton, Gullo, et al., 2018).

It is clear from this research that alcohol-related cognitions impact alcohol use and that these cognitions are influenced by individual differences in appetitive and inhibitory processes. Cognitive Behavioral Therapy (CBT) is uniquely placed to target alcohol-related cognitions directly and perhaps interrupt the link between impulsivity and cognitions (Loree, Lundahl, & Ledgerwood, 2015), as well as indirectly impacting drinking refusal self-efficacy through altering alcohol expectancies (Connor et al., 2016; Patton, Gullo, et al., 2018). Current promising interventions have utilized CBT to target individual personality risk factors for adolescent alcohol use, including impulsivity traits (Conrod et al., 2013). For example, previous interventions have targeted boredom-susceptibility and reward-seeking cognitions in adolescents identified to have high sensation seeking (Conrod et al., 2008). It is possible that the effectiveness of these programs is driven by targeting these general cognitions regarding alcohol (such as expectancies and self-efficacy) as well as personality-risk specific cognitions. However, the effect of these interventions appears to be more robust for reward drive-related impulsivity and may not be equally effective in targeting rash impulsiveness-related traits (Conrod et al., 2008, 2006). This could explain the comparable effectiveness of universal cognitive-based alcohol programs (Teesson et al., 2017).

Therefore there may be room for increased effectiveness in CBT methods of targeting rash impulsiveness in school-based interventions. This may help to explain the mixed evidence for the effectiveness of school-based alcohol use intervention programs (Carney et al., 2016; Onrust et al., 2016). The findings that the effects of CBT for adolescent alcohol prevention interventions are strongest for high impulsivity adolescents and that impulsivity impacts a major cognitive mechanism of CBT (drinking refusal self-efficacy) lends support to the theory that targeting impulsivity directly may improve intervention effectiveness. We hypothesize that mindfulness meditation may be a more appropriate strategy to target rash impulsiveness. Mindfulness meditation involves deliberate attention on the present with non-judgmental acceptance of present moment experiences, which is theoretically consistent with managing rash, inattentive impulses (Papies, Barsalou, & Custers, 2012). Brief meditation

has been shown to improve attention and self-regulation (Tang et al., 2007) and increase brain white matter (Tang, Lu, Fan, Yang, & Posner, 2012). As adolescence is a period of both reward sensitivity and reduced impulse control (Stautz, Dinc, & Cooper, 2017) and each imparts unique risks for alcohol use (Gullo et al., 2010), finding effective strategies to target both factors of impulsivity could improve the efficacy of current intervention approaches.

Mindfulness is a complementary technique to CBT (Beck & Haigh, 2014).

Mindfulness interventions have gained empirical support for their efficacy as a treatment for adult and adolescent mental health problems (Khoury et al., 2013; Zoogman, Goldberg, Hoyt, & Miller, 2015). Further, there is preliminary support for the addition of mindfulness training to adolescent alcohol misuse interventions (Harris et al., 2017). It is thought that mindfulness may interrupt the link between motivations for use and behavior (Ostafin, Bauer, & Myxter, 2012). Previous studies investigating mindfulness often utilize a waitlist control group or do not include an active treatment comparison group in their design (Khoury et al., 2013; Zoogman et al., 2015). This lack of active comparison results in uncertainty as to the specific vs non-specific (e.g., relaxation) effects of mindfulness (Davidson, 2010; Goyal et al., 2014), especially when it is combined with a previously validated treatment approach, such as CBT. Therefore, a procedure such as Progressive Muscle Relaxation, which invokes relaxation but not increased objectivity regarding one's internal experience, known as decentering, which is considered a key component of mindfulness (Feldman, Greeson, & Senville, 2010), would be an appropriate active control.

The present study aimed to investigate the effect of a CBT-based adolescent alcohol use prevention intervention. Further, we aimed to identify whether Mindfulness Meditation (MM) would produce additional effectiveness to the CBT approach. To investigate this thoroughly, we utilized PMR as an active control for non-specific relaxation effects where adolescents received CBT (i.e., CBT+PMR). Both of these active conditions (CBT+MM and CBT+PMR) were compared to an assessment-only control group. We hypothesized that both interventions would reduce the growth in alcohol use over a six-month period post-intervention compared to the assessment-only control and that the CBT+MM condition would be superior to the CBT+PMR intervention. We also aimed to investigate possible mechanisms of effect of the intervention by conducting secondary analyses on other outcome variables including drinking refusal self-efficacy, positive and negative alcohol expectancies, and mindfulness ability. We predicted that both CBT interventions would decrease positive alcohol expectancies and increase negative alcohol expectancies and drinking refusal self-

efficacy compared to the assessment-only control, but that mindfulness would increase only for the CBT+MM condition.

Methods

Ethical clearance and trial registration and reporting

The trial was granted ethical clearance by the University of Queensland Behavioural and Social Sciences Ethical Review Committee (#2015000875), Brisbane Catholic Education (#196), and was registered with the Australian New Zealand Trials Registry (ACTRN12616000077460). The Journal Article Reporting Standards (JARS; American Psychological Association, 2008) have been used to guide the current report.

Power

Originally the analysis was planned as Structural Equation Modelling (SEM) (ACTRN12616000077460). The sample size was determined assuming intra-class variance of 0.4 (Heo & Leon, 2010). The meta-analysis by Sedlmeier et al. (2012) found moderate psychological effect sizes for meditation compared to relaxation ($r = .21$). However, a systematic review of mindfulness for adult substance use treatment found effect sizes ranged from small to moderate (Zgierska et al., 2009). Due to these findings and the robust active control in the present study a small effect size was assumed ($\beta = .14$). Based on these estimates, number of time points, degrees of freedom and analysis requirements and assuming a 20% attrition rate over time, a baseline sample of 441 students was sought (Kim, 2005; Muthén & Curran, 1997). Multi-Level Modelling (MLM) was considered more appropriate for the data after data collection (see analytical procedure section). Using the approach for MLM (Hox, 2002; Snijders, 2005), post hoc power analysis indicates that the study had power of .80 ($\alpha = .05$) to detect a $\beta = .12$ effect size of CBT+MM vs CBT+PMR within the current sample.

Participants and anonymised matching procedure

Four-hundred and ninety-nine students in Grade 9 or 10 (typically 13–15 years of age) from 6 schools were approached to participate in the study, of which 468 provided informed consent and were randomized. Grade 9 and 10 students were sought in order to deliver the prevention intervention earlier than the average age of onset of 15.7 years for Australian adolescents (Australian Institute of Health and Welfare, 2014). Twenty-five schools in urban South-East Queensland were initially contacted for possible inclusion in the study, out of which, six schools agreed to participate. Informed consent was gained from participants and

their parent or guardian. A cluster randomization procedure was utilized with an intention-to-treat approach, and 468 students were randomized by KP using an online random number generator to CBT+PMR, CBT+MM, or Control conditions within class clusters in each school (see Figure 1 for CONSORT flow diagram). That each school participated in all three conditions allowed for greater certainty that variation between conditions was not due to randomization artifacts. Participants were not incentivized to complete the intervention. However, all but one school opted for their students to receive skills reminder SMS. Students went into a pool to receive a gift voucher to a local electronics store and replies to these messages resulted in more chances to receive a voucher.

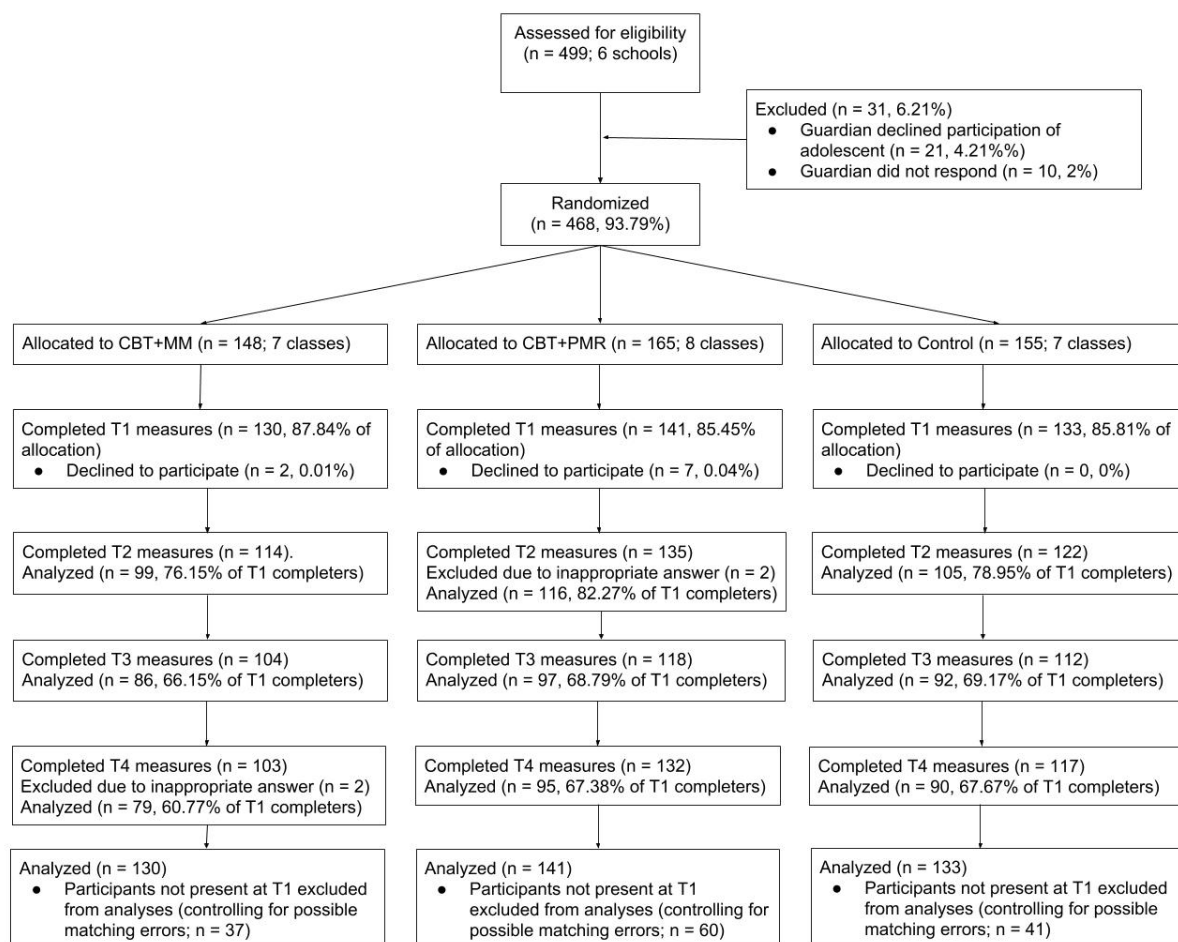


Figure 7.1. Participant flow across assessment occasions.

Note. “Inappropriate answers” refers to identifiably false or nonsensical responses.

Exclusions to control for possible matching errors are described in the method section.

Participants were anonymized using a nine-item code per the procedure of Schnell and colleagues (2010). The codes were manually matched across time points using

Levenshtein string distance function in Microsoft Excel and cross-checking with mobile phone numbers, if provided (Schnell, Bachteler, & Reiher, 2010). The majority of participants (75%) were matched to at least one other time point (see Table 7.3 in supplementary materials), which was considered a high matching rate given that losses of up to 50% can be reported for two time-point anonymized matching (Schnell et al., 2010). However, the total number of participants at the completion of data collection ($N = 542$) was greater than the number of allocated participants at Time 1 (total N allocated = 468). This was interpreted as a) possible failures in matching resulting in a single participant present at several time points appearing as several individuals or b) collection of data from participants who were not consented to participate (e.g., due to change in class or newly enrolled students during follow-up period). To correct for the latter possibility, data were restricted to participants present at Time 1 and all cases across Times 2, 3, and 4 who were not matched to a case at Time 1 were removed to conform to study ethics approval (final sample $N = 404$, 74.54% of all data initially collected).

Participants were aged 13-17 years (Mean age = 14.99, $SD = .66$ years), and 62% were female ($N = 251$). In the final analyses, there were 130 adolescents in the CBT+PMR condition (8 classes), 141 in the CBT+PMR condition (7 classes), and 133 in the AoC condition (7 classes). There were no significant pre-intervention differences between participants in each condition for demographic, predictor, and outcome measures. Most participants lived within medium affluence families and had Australian or European backgrounds (see Table 7.5 for baseline characteristics). Sixty-five percent of participants provided data at 6-month follow-up (Time 4). However, 75% provided data at 3 or more of the 4 assessment occasions.

Interventions

The intervention involved a universal Cognitive Behavioral Therapy (CBT) program. The interventions were delivered by one or two facilitators in class groups of 8-23 students. The facilitators were not blinded to condition. Adolescents in the two intervention conditions were introduced to the cognitive model of the interplay between thoughts, emotions, and behaviors (Beck, 1976) and were taught techniques to identify, challenge, and change “unhelpful” cognitions. The techniques were first applied to general stress and negative emotions before then being applied to alcohol use. Specifically, class-generated cognitions regarding possible alcohol use at a hypothetical party. The adolescents were also taught either Progressive Muscle Relaxation (CBT+PMR condition) or mindful breathing exercises

(CBT+MM). The CBT+PMR condition participants were introduced to PMR as a technique to reduce stress through recognizing and relaxing tension. The CBT+MM condition was taught MM as a technique to reduce inattention and to increase present-moment awareness. Both intervention conditions were given access to condition-specific websites with resources on the CBT and PMR/MM techniques, including recordings of PMR/MM exercises used in sessions. See Table 7.4 in supplementary materials for session outlines.

Procedure

The intervention was designed to be 110 minutes in total (plus 80 minutes for completing assessments), delivered over 3 sessions. Due to practical considerations within each school, total intervention time differed between schools. The 6 schools ranged in intervention time from 110-220 minutes with an average intervention time of 173 minutes. The intervention was delivered by students completing masters or doctorate-level psychology programs who were trained in the intervention by a doctoral-level instructor. Assessment measures were completed prior to the intervention (Time 1), immediately post-intervention (Time 2), 3-months post-intervention (Time 3), and 6-months post-intervention (Time 4). The control group completed the measures only.

Measures

Alcohol use. Alcohol use was measured using the 10-item Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). The first three items of the AUDIT assess frequency of alcohol use (0 = 'Never'; 4 = '4 or more times a week'), typical quantity of drinks in a single occasion (0 = '1 or 2'; 4 = '10 or more'), and frequency of binge use (6+ standard drinks; 0 = 'Never'; 4 = 'Daily or almost daily'). These three items are widely used as a stand-alone scale of alcohol consumption, known as the AUDIT-C (Bush et al., 1998). The average Cronbach's alpha over the four time-points for the AUDIT-C was .38. Cronbach's alpha can be impacted by non-normal distributions (Sheng & Sheng, 2012), so the positive skew in the current sample may have impacted this score. Non-parametric correlations to assess test-retest reliability showed significant moderate to strong associations between all assessment occasions at $p < .001$. Effect sizes ranged between $r(259) = .54$ (Time 1 with Time 3) to $r(304) = .65$ (Time 1 with Time 2).

Alcohol-related cognitions. Positive and Negative Alcohol Expectancies were measured using the 21-item Drinking Expectancy Questionnaire – Adolescent version (DEQ-A; Connor et al., 2011; Patton et al., 2017). The scale comprises two positive expectancy

subscales (*increased confidence*, 6-items; and *tension reduction*, 5-items) and two negative expectancy subscales (*cognitive and motor impairment*, 5-items; and *negative mood*, 4-items). Items are measured on a 5-point Likert scale (1 = ‘Strongly Disagree’; 5 = ‘Strongly Agree’). The average Cronbach’s alphas over the four time-points for the two positive subscales combined and the two negative subscales combined were both .97.

Drinking Refusal Self-Efficacy (DRSEQ) was measured using the 19-item Drinking Refusal Self-Efficacy Questionnaire–Revised Adolescent version (DRSEQ-RA), which is measured using a 6-point Likert scale (1 = ‘I am very sure I could NOT resist drinking’; 6 = ‘I am very sure I could resist drinking’; Patton et al., 2018; Young, Hasking, Oei, & Loveday, 2007). The subscales of the DRSEQ-RA relate to *opportunistic* (7-items), *social pressure* (5-items), and *emotional relief* (7-items) drinking refusal self-efficacy contexts. The average Cronbach’s alpha over the four time-points was .98.

Impulsivity. Reward Drive (RD) was measured using the 10-item shortened Sensitivity to Reward Scale (SR-S), which is measured using binary response options (1 = ‘YES’, 2 = ‘NO’) (Cooper & Gomez, 2008). The average Cronbach’s alpha over the four time-points was .78. Rash Impulsiveness was measured using the 8-item Barratt Impulsiveness Scale–Brief (BIS-B), which allows 4 response options (1 = ‘Rarely/Never’; 4 = ‘Almost always/Always’; Steinberg, Sharp, Stanford, & Tharp, 2013). The average Cronbach’s alpha over the four time-points was .77.

Family Affluence. Socio-economic background was measured using the Family Affluence Scale–II (FAS-II), which is a 4-item scale developed for the WHO Health Behavior in School-Aged Children survey (Boyce, Torsheim, Currie, & Zambon, 2006). An example item is “How many computers does your family own” (0 = ‘None’; 3 = ‘Two or more’). The FAS was validated by the WHO across 35 countries, achieving good criterion validity when compared to country Gross Domestic Product (Boyce et al., 2006). Reliability has also been established through comparison with parent responses to items (Currie et al., 2008). The scale is recommended for use in research evaluating adolescent health and Socio-Economic Status (Boyce et al., 2006).

Mindfulness. The 14 item Mindful Attention Awareness Scale-Adolescent (MAAS-A; Brown, West, Loverich, & Biegel, 2011) was used to assess change in mindfulness over time. Items, e.g. “I rush through activities without being really attentive to them” are measured on a 6-point Likert scale (1 = ‘Almost always’; 6 = ‘Almost never’). The average Cronbach’s alpha

over the four time-points was .95. Participants were also asked about their previous mindfulness experience (1=“No, never”; 2=“Only a few times”; 3=“Many times but not anymore”; 4=“I currently practice mindfulness meditation”).

Analytical procedure

Multilevel modelling (MLM) was conducted in MLwiN (version 2.30). Those analyzing the data were not masked to intervention conditions. Originally Structural Equation Modelling (SEM) was planned (ACTRN12616000077460). However, MLM was considered more appropriate due to the variability observed between recruited schools in intervention length, follow-up times and SMS reminder support. This is supported by the variance partition coefficient (VPC) for the AUDIT-C MLM analysis, which showed that 8% of the variance in alcohol consumption was explained by school-level variation, $VPC = .08$. SEM analyses revealed similar outcomes to those presented in the current paper. The SEM outcomes can be made available upon request. Due to MLM’s ability to chart individual growth trajectories, it is also robust to attrition, using full information maximisation likelihood (FIML) estimation – an optimal means of handling missing data (Graham, 2009; Hallgren & Witkiewitz, 2013).

Three-level models were built with assessment time-points (level 1) nested within participants (level 2), nested within schools (level 3). Gender, age, and family affluence were included as level 2 covariates with the latter two being grand mean-centred. Full iterative generalized least squares (IGLS) was used to estimate the models. Two-condition contrasts per outcome measure were calculated using contrast codes (J. Cohen, Cohen, West, & Aiken, 2003). These comparisons were based on the hypothesised outcomes for the outcome measures. For alcohol, impulsivity, and cognition outcomes, Contrast 1 compared both intervention conditions to the control and Contrast 2 compared the intervention conditions, as predicted. For the mindfulness outcome variable, Contrast 1 compared the CBT+MM condition to the other two conditions and Contrast 2 compared CBT+PMR to assessment-only control (see Tables 7.6, 7.7, and 7.8 in supplementary materials for contrast codes used). The contrasts were entered into the MLM models as level 2 predictors, along with Time (coded 0, 1, 2, 3) as a level 1 predictor, and cross-level interaction terms between Time and the Contrasts were calculated and added to the model. Random intercepts were specified. Plots of residuals at each level were examined to check assumptions and outliers. The tested models were specified as follows: $Outcome_{ijk} = \beta_{0jk} + \beta_1 Male_{jk} + \beta_2 Age_{jk} + \beta_3$

$$\text{FamilyAffluence}_{jk} + \beta_4 \text{TimePoint}_{ijk} + \beta_5 \text{Contrast1}_{jk} + \beta_6 \text{Contrast2}_{jk} + \beta_7$$

$$\text{TimePoint.Contrast1}_{ijk} + \beta_8 \text{TimePoint.Contrast2}_{ijk} + e_{ijk}$$

Results

Missing data. The majority of the sample (73.7%) provided responses at three or all four time-points (see Table 7.3). A fewer number of participants (15.4%) were present at two time-points and 10.6% attended Time 1 only. The descriptives for the outcome variables are given in Table 7.1. Little's Missing Completely at Random (MCAR; Little, 1988) test was significant for, $\chi^2(\text{DF} = 3,892) = 4,377.32, p < .001$. However, separate variance t-tests showed that there were no significant differences between present and missing participants on variables at times 2-3 based on time 1 variable values.

Covariates. There was a significant positive effect of Age on the AUDIT-C, indicating that older adolescents had higher consumption rates at Time 1. Older adolescents also had significantly higher positive and negative alcohol expectancies at Time 1. Male adolescents had significantly lower positive and negative alcohol expectancies as well as higher drinking refusal self-efficacy at Time 1 compared to female adolescents. However, male adolescents also showed significantly higher Reward Drive compared to female adolescents at Time 1.

Alcohol use. The multilevel models analyzed the growth of the outcome measures over time and whether this growth was impacted by intervention condition, age, gender or family affluence. The results of each MLM analysis (unstandardized regression coefficients) are outlined in Table 7.2 and represented visually in Figure 7.2.

As expected due to the age of the population, AUDIT-C scores were low, but there was a significant interaction between Time and Contrast 1, such that participating in either intervention significantly decreased the growth in AUDIT-C scores compared to assessment-only control. The treatment effect size was standardized by comparing the hypothesized model's deviance ($-2 * \text{Log-Likelihood}$) to that of a model in which the treatment Contrast parameter was constrained to zero. This difference in model fit is equivalent to a chi-square value, which was then converted a Cohen's d of -.14. Contrary to hypothesis, CBT+MM did not produce a significantly larger effect on alcohol growth compared to CBT+PMR, as indicated by the non-significant Time x Contrast 2 interaction (see Table 7.2).

Table 7.1.

Descriptive statistics for the outcome variables at each time-point.

Outcome variable	Time-point	Minimum	Maximum	Mean	SD
Alcohol consumption (AUDIT-C)	Time 1	0	8	0.94	1.66
	Time 2	0	7	0.81	1.58
	Time 3	0	11	0.76	1.70
	Time 4	0	11	0.93	1.85
Drinking Refusal Self-Efficacy Social Pressure Subscale (DRSEQ-RA SP)	Time 1	5	30	25.79	6.47
	Time 2	5	30	25.50	6.58
	Time 3	5	30	27.26	5.37
	Time 4	5	30	26.25	6.76
Positive Alcohol Expectancies (DEQ-A Pos)	Time 1	11	55	23.99	11.14
	Time 2	11	46	23.54	10.89
	Time 3	11	55	22.15	11.44
	Time 4	11	55	23.33	11.36
Negative Alcohol Expectancies (DEQ-A Neg)	Time 1	10	50	21.33	10.00
	Time 2	10	41	20.80	9.59
	Time 3	10	50	20.02	10.37
	Time 4	10	50	20.80	10.01
Reward Drive (SR-S)	Time 1	0	10	4.46	2.47
	Time 2	0	10	4.49	2.68
	Time 3	0	10	4.45	2.97
	Time 4	0	10	4.37	3.11
Rash Impulsiveness (BIS-B)	Time 1	8	30	16.96	4.29
	Time 2	8	30	16.92	4.29
	Time 3	8	32	16.75	3.89
	Time 4	8	30	17.22	3.89
Mindfulness (MAAS-A)	Time 1	14	84	58.70	14.27
	Time 2	14	84	58.05	15.90
	Time 3	14	84	61.31	17.57
	Time 4	14	84	56.38	17.84

Table 7.2.

Multilevel Modelling results for alcohol use, alcohol-related cognitions, impulsivity factors, and mindfulness with condition contrasts[^] (N = 404).

Outcome	Estimate	Intercept, β_{0jk}	Male _{jk}	Age _{jk}	Family Affluence _{jk}	Contrast1 _{jk}	Contrast2 _{jk}	Time _{ijk}	Time. Contrast1 _{ijk}	Time. Contrast2 _{ijk}	σ^2_e	σ^2_{v0}	σ^2_{u0}	-2*log likelihood
Alcohol Consumption (AUDIT-C)	Unstandardised coefficient	0.94	-0.17	0.26 ⁺	0.05	-0.11	-0.14	0.03	-0.18 ⁺	-0.06				
	SE	0.07	0.14	0.11	0.07	0.17	0.19	0.03	0.07	0.08	1.54	0.24	1.16	4535.31
	z	-	-1.19	2.48	0.76	-0.62	-0.72	0.97	-2.46	-0.70				
	p	-	0.234	0.013	0.447	0.535	0.472	0.332	0.014	0.484				
Negative Alcohol Expectancies (DEQ-A Negative)	Unstandardised coefficient	20.91	-1.91 ⁺	1.50 ⁺	0.56	-0.17	-1.48	-0.09	1.09 ⁺	-0.94				
	SE	0.40	0.82	0.62	0.41	0.20	1.01	1.14	0.44	0.50	59.13	0	40.43	8740.68
	z	-	-2.33	2.43	1.38	-0.84	-1.47	-0.08	2.50	-1.88				
	p	-	0.020	0.015	0.168	0.401	0.142	0.936	0.012	0.060				
Positive Alcohol Expectancies (DEQ-A Positive)	Unstandardised coefficient	23.49	-1.84 ⁺	1.67 ⁺	0.76	-0.22	-1.97	-0.28	1.30 ⁺	-0.85				
	SE	0.44	0.92	0.69	0.46	0.23	1.14	1.28	0.49	0.56	73.65	0	51.47	8988.38
	z	-	-2.00	2.41	1.66	-0.94	-1.73	-0.22	2.67	-1.50				
	p	-	0.046	0.016	0.097	0.347	0.084	0.826	0.008	0.134				
Social Pressure	Unstandardised coefficient	25.88	1.22 ⁺	- 1.01 ⁺	0.05	0.12	1.57 ⁺	-0.71	-0.05	-0.19	22.91	1.35	17.56	7815.47

Drinking	<i>SE</i>	0.26	0.53	0.40	0.27	0.13	0.65	0.73	0.27	0.31				
Refusal Self-	<i>z</i>	-	2.29	-2.52	0.20	0.93	2.43	-0.97	-0.18	-0.62				
Efficacy	<i>p</i>													
(DRSEQ-RA		-	0.022	0.012	0.841	0.352	0.015	0.332	0.857	0.535				
SP)														
	Unstandardised													
	coefficient	4.47	0.55 ⁺	0.28	-0.08	0.01	0.58 ⁺	-0.20	0.01	0.06				
Reward Drive	<i>SE</i>	0.12	0.23	0.18	0.12	0.06	0.28	0.31	0.12	0.14	3.72	0	3.83	5149.16
(SR-S)	<i>z</i>	-	2.37	1.59	-0.70	0.09	2.07	-0.65	0.07	0.42				
	<i>p</i>	-	0.018	0.112	0.484	0.928	0.038	0.516	0.944	0.674				
	Unstandardised													
	coefficient	17.12	-0.05	-0.03	0.20	0.15 ⁺	-0.03	-0.38	-0.03	0.11				
Rash	<i>SE</i>	0.19	0.39	0.30	0.20	0.06	0.44	0.49	0.13	0.15	5.04	1.23	11.16	6280.75
Impulsiveness	<i>z</i>	-	-0.11	-0.08	1.04	2.38	-0.06	-0.78	-0.20	0.71				
(BIS-B)	<i>p</i>	-	0.912	0.936	0.298	0.017	0.952	0.435	0.841	0.478				
	Unstandardised													
	coefficient	58.30	-1.17	-1.62	-0.20	-0.49	0.45	-0.99	-0.21	0.54				
Mindfulness	<i>SE</i>	0.64	1.33	1.01	0.67	0.36	1.66	1.88	0.77	0.86	165.79	0	97.03	9547.46
(MAAS-A)	<i>z</i>	-	-0.88	-1.60	-0.30	-1.37	0.27	-0.52	-0.28	0.63				
	<i>p</i>	-	0.379	0.110	0.764	0.171	0.787	0.603	0.779	0.529				

Note. Boldface indicates $p < .05$, ⁺ indicates significant unstandardized coefficient at $p < .05$. ^See supplementary materials for contrasts used.

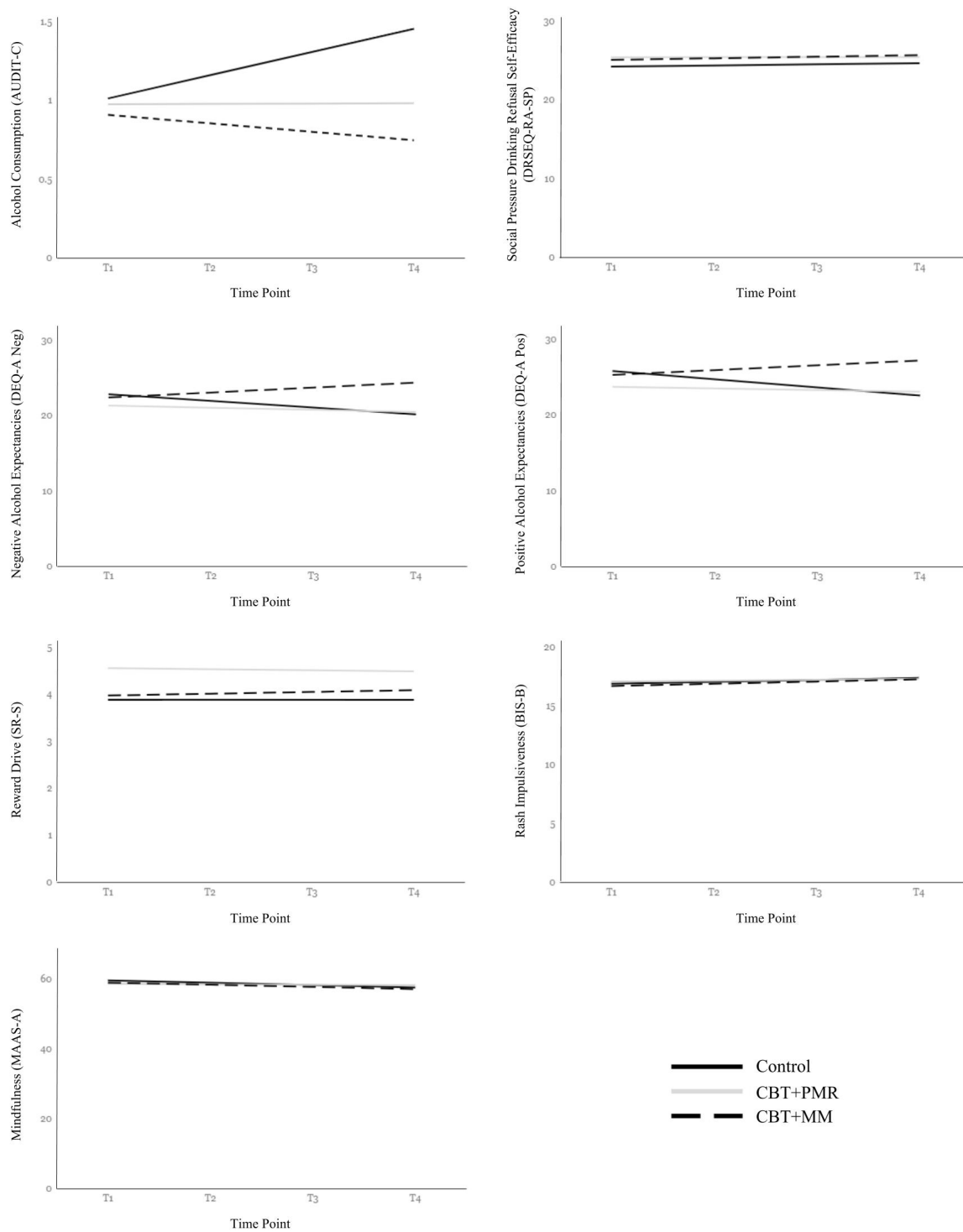


Figure 7.2. Estimated Multilevel Model plots of outcome slopes weighted by contrasts across the four time-points. Note. Model included age, gender, and family affluence as level 2 covariates.

Alcohol-related cognitions. There was a significant interaction between Time and Contrast 1 for both positive and negative alcohol expectancies, indicating that adolescents in the intervention conditions had significantly higher growth in these expectancies compared to those in the assessment-only control condition. Examination of the residuals plots for drinking refusal self-efficacy total revealed severe deviation from normality. The social pressure subscale of DRSEQ was analysed instead because the distribution of residuals met normality assumptions and was the more relevant subscale for this population (H. Aas et al., 1995; Jester, Wong, et al., 2015; Jones et al., 2001; Tomlinson & Brown, 2012; Young-Wolff et al., 2015). Social pressure refusal self-efficacy subscale scores correlated highly with the DRSE total score at each time-point (correlations ranges from $r = .84$ for Time 1 to $r = .88$ for Time 4, $ps < .001$). The results revealed that adolescents in the intervention conditions had significantly higher social pressure drinking refusal self-efficacy than the control group at Time 1, but that the growth over time was not impacted by condition. This may be partly due to the finding that social pressure drinking refusal self-efficacy did not significantly change, on average, across the 4 time-points. Correlations were run and significant moderately sized associations were found between social pressure drinking refusal self-efficacy and alcohol consumption at each time-point, Time 1 $r(381) = -.42, p < .001$, Time 2 $r(303) = -.49, p < .001$, Time 3 $r(264) = -.48, p < .001$, Time 4 $r(247) = -.44, p < .001$. There was also evidence of a prospective association after controlling for Time 1 alcohol consumption with Time 1 social pressure drinking refusal self-efficacy explained 5% of unique variance in Time 2 alcohol consumptions ($sr^2 = .05; p < .001$), 8% of unique variance in Time 3 alcohol consumption ($sr^2 = .08; p < .001$), and 5% of unique variance in Time 4 alcohol consumption ($sr^2 = .05; p < .001$).

Impulsivity. The results indicated that the intervention groups had significantly higher Reward Drive at Time 1 compared to the control group, but it did not significantly change over time overall and this was not moderated by condition. While Rash Impulsivity significantly increased over the four time-points, growth was not moderated by intervention condition.

Mindfulness. There were no significant effects of condition, time, or the covariates on the MAAS-A. Including previous mindfulness experience in the model did not alter effects. However, greater previous mindfulness experience was significantly related to increased mindfulness over time on the MAAS-A. Despite this, associations between mindfulness and alcohol consumption were small. Nonparametric correlations showed very weak concurrent

correlations between these factors at Time 1, $r(365) = -.13, p = .011$, and Time 2, $r(296) = -.13, p = .021$, but non-significant associations at Times 3 and 4. Prospective associations were also non-significant or very weak. Regressions showed that only Time 2 mindfulness predicted Time 4 alcohol consumption, $\beta = -.14, t(212) = -2.03, p = .043$, and that it accounted for 1% of variance, $\text{adj}R^2 = .01, F(1, 212) = 4.13, p = .043$. The small variance suggests that even if mindfulness had increased due to intervention efforts, it may not have impacted alcohol use.

Discussion

This study is the first test of the additive effectiveness of Mindfulness Meditation to a brief universal Cognitive Behavior Therapy (CBT+MM) intervention for adolescent alcohol use using a robust active control. The biosocial Cognitive theory (bSCT) model was utilized to identify evidence-based risk factors for intervention. It was theorized that CBT may directly target alcohol expectancies and, in doing so, indirectly affect refusal self-efficacy and also address the risk conveyed by the impulsivity factor of Reward Drive (theoretically, expectancies mediate the effect of Reward Drive, and expectancy effects on alcohol use are mediated by refusal self-efficacy). The addition of MM was proposed to directly target Rash Impulsiveness, which is theorized to have a direct effect on alcohol use and an indirect effect through lowering refusal self-efficacy. The effect of CBT+MM condition on adolescent alcohol use outcomes was compared with an active control of CBT combined with Progressive Muscle Relaxation (CBT+PMR) and an assessment-only control group. The effects of these interventions on possible mechanisms of change were also investigated, including drinking refusal self-efficacy, positive and negative alcohol expectancies, reward drive, rash impulsivity, and mindfulness. The results showed that CBT reduced the growth in alcohol use and increased both positive and negative alcohol expectancies but that there was no evidence that Mindfulness had an additive effect beyond the effects of relaxation.

Previous research has found encouraging evidence for mindfulness as an effective treatment for adolescent mental health problems (Zoogman et al., 2015). However, the meta-analysis by Khoury and colleagues (2013) found that only 35 (approximately 17%) of their 209 included studies included an active psychological control condition, with most studies utilizing a pre-post design or comparing a mindfulness-based therapy to a waitlist control. A second meta-analysis by Zoogman et al. (2015) considered that 60% of the 20 included studies had an active treatment. However, their definition of active control included the health and other school classes taken by the students, which could be interpreted as

treatment-as-usual. These consistent methodological issues leave ambiguity as to benefit of mindfulness compared to existing treatments (Sedlmeier et al., 2012) and prompted the use of a robust active comparison condition to control for the non-specific effects of mindfulness in the present study.

The finding that there was no difference between the CBT+MM and the CBT+PMR condition is consistent with previous research concluding that mindfulness-based treatments do not provide benefits above CBT with relaxation approaches for broader mental health diagnoses including depression and anxiety (Farias et al., 2016; Khoury et al., 2013). However, this is the first evidence of no additional benefit in youth alcohol use prevention. While the lack of change in mindfulness over time could mean that the adolescents were not trained in or applying mindfulness effectively, previous interventions have shown effects with only a few mindfulness sessions (Sedlmeier et al., 2012). Further, a recent RCT found that a school-based mindfulness intervention did not improve depression, anxiety, or eating disorder symptoms and that adolescent home practice of mindfulness did not moderate these effects (Johnson, Burke, Brinkman, & Wade, 2017). It is also possible that the effects are smaller due to the non-clinical nature of the sample (Zoogman et al., 2015). Accordingly, the present results suggest that the addition of mindfulness may not improve adolescent substance use outcomes beyond existing CBT and relaxation treatments.

Mindfulness meditation also did not have a significant impact on adolescent impulsivity. There was an increase in both reward drive and rash impulsiveness across the 6-months included in the present study and there was no effect of CBT+MM or CBT+PMR on this growth. The finding that impulsivity increases across adolescence replicates previous research (Littlefield, Stevens, Ellingson, King, & Jackson, 2016). That neither intervention condition decreased the growth in impulsivity may seem counterintuitive given previous success targeting these personality factors (Conrod et al., 2013). However, it is unclear whether previous interventions are altering the impulsivity personality traits themselves or changing the way in which individuals act on their impulses (e.g., to express them in more adaptive ways). Indeed, there are divergent theoretical perspectives on whether the traits themselves can be altered (Harkness & Lilienfeld, 1997; Magidson, Roberts, Collado-Rodriguez, & Lejuez, 2014). Therefore, future research could investigate whether CBT and mindfulness interventions moderate the pathways by which impulsivity imparts risk for alcohol use, e.g., through drinking refusal self-efficacy and alcohol expectancies (Gullo, Loxton, et al., 2017; Gullo et al., 2010a). Additionally, the use of self-report instruments may

have affected the ability to detect treatment effects. Future studies should seek to employ teacher- and parent-rated scales, and behavioral measures of impulsivity, if practical (Ferne et al., 2013). What these findings confirm is that adolescence is a period of increasing elevated impulsivity and therefore elevated risk for alcohol use (Stautz et al., 2017).

Both intervention conditions produced a reduction in the growth of alcohol consumption over the 6-month period compared to the assessment-only control. These findings are noteworthy, considering that reduction in adolescent alcohol use due to early intervention is a greater predictor of reduced future problematic drinking than personality and mental health risk factors (O’Leary-Barrett et al., 2016). Promisingly, our effect size is greater than recent meta-analytic estimates of the effect sizes for CBT-based universal alcohol use programs for similarly-aged adolescents, which were non-significant (Onrust et al., 2016). Despite this, the role of social cognitive factors as potential mechanisms of change received mixed support. Alcohol expectancies did change over time, dependent on treatment condition. Both CBT interventions showed an increase in positive and negative expectancies compared to the assessment-only control group. There was also a trending effect ($p = .06$) of a larger increase in negative alcohol expectancies over time for the CBT+MM condition. Increased negative expectancies are associated with reduced adolescent drinking (Colder et al., 2017) and therefore may have contributed to the reduced consumption outcomes in the intervention conditions. However, the increase in positive expectancies was unexpected. Despite their increased positive expectancies, the intervention conditions had reduced the growth of alcohol consumption compared to the control. One possible explanation is that the increase in expectations of positive outcomes is that reduced consumption and, therefore, less hazardous consumption, may have produced more positive alcohol experiences. Further research into the dynamic effect of initial positive treatment response on psychological risk factors like expectancies is required to support this.

Drinking refusal self-efficacy, which is a robust predictor of CBT alcohol outcomes in adult populations (Connor et al., 2016; Magill et al., 2015), did not increase or decrease across the 6-months, even for adolescents in the intervention conditions. The average social pressure drinking refusal self-efficacy scores at each time-point in the sample ranged from 25.50 to 27.26 of a possible total of 30, showing possible evidence of a ceiling effect. While it was expected that drinking refusal self-efficacy would decrease over time and with exposure to alcohol use, it is possible that 6-months was not sufficient to capture this effect, especially with the low levels of alcohol consumption within the present sample. Prospective

relationships between drinking refusal self-efficacy and alcohol consumption at each time-point show drinking refusal self-efficacy was associated with higher consumption. Due to the importance of this factor indicated by previous research (Black & Chung, 2014; Connor et al., 2011, 2016; Magill et al., 2015), it is plausible that with increased exposure to alcohol contexts, the high self-efficacy of these adolescents will reduce their risk of future misuse.

Another possibility regarding the current drinking refusal self-efficacy findings is that the present study potentially intervened too early to see an impact. Drinking refusal self-efficacy was associated with future drinking in this study; however, previous research shows that targeting this factor in late adolescence produces greater effects (Onrust et al., 2016). This may be due to the phenomenon seen in the present study that drinking refusal self-efficacy is high prior to experience with alcohol. In this age-group it may be more effective to target related constructs such as rash impulsiveness and alcohol expectancies, as in the present study, as improvements in these factors may have future “knock on” effects on drinking refusal self-efficacy. Future research could use age as a moderator to further explore the age-effects. The present results show the benefit of a theoretically driven model of biosocial cognitive risk (such as the bSCT) which can provide a deeper understanding of the dynamic interplay between adolescent alcohol use and risk factors to inform treatment targets and the age of optimal effect.

The present study had limitations. Firstly, although a post-hoc MLM power analysis indicated that the current study had adequate power to detect effects, the study had a moderately sized sample. Due to the robust control and small-to-medium effects, a larger sample may be beneficial in future studies to further evaluate effects and group comparisons. As effects of alcohol-interventions often reduce over time (Tripodi et al., 2010), future studies could also evaluate the effects of the addition of mindfulness to CBT over a longer follow-up period. There was also variation in the delivery of the interventions due to practical considerations and one school opted not to include SMS follow-up skills reminders. While the current study attempted to incorporate this variation into the analysis through the use of multi-level modelling, a more standardized approach would be recommended in future trials. While facilitators were trained to deliver interventions in a standardised manner, and received regular supervision by a clinical psychologist (MJG), video recording of sessions for independent fidelity rating was beyond the scope of the study.

Future research may also wish to consider the content of chosen active controls. The inclusion of PMR as an active control for Mindfulness is considered a strength of the current study. This is due to the hypotheses that impulsivity would be impacted by the mindfulness-specific effects of decentering (Bernstein et al., 2015; Feldman et al., 2010). However, mindfulness mechanisms are also thought to include attention regulation, body awareness, emotional regulation and perspective alteration (Hölzel et al., 2011). While PMR and Mindfulness have differential impacts on stress (Gao, Curtiss, Liu, & Hofmann, 2017), anxiety, and depression (Lancaster, Klein, & Knightly, 2016), both involve directed attention and can therefore increase constructs considered to be components of mindfulness (Gao et al., 2017). Therefore, active controls in mindfulness interventions should be chosen based on the aspect of mindfulness considered central to the intervention effects.

This study is the first to compare a mindfulness-enhanced CBT intervention for adolescent alcohol use to CBT with an active relaxation control. The findings support the use of CBT as an effective universal intervention to reduce the growth in adolescent alcohol consumption. The addition of mindfulness meditation to the brief CBT intervention was not found to have a benefit beyond that of the active CBT control (progressive muscle relaxation). An investigation of associated outcomes found support for the theory that alcohol expectancies may be an important precursor to alcohol consumption but that drinking refusal self-efficacy may gain increasing importance in predicting misuse as contact with alcohol increases. Both rash impulsivity and reward drive increased over time, supporting theories of increasing risk for substance use in mid-adolescence. Our findings highlight the need for robust, well-controlled studies of alcohol interventions that are guided by strong theory to elucidate the complex mechanisms of action (and inaction; Magill & Longabaugh, 2013).

Supplementary materials

Table 7.3.

Participant matching outcomes (N = 404).

Present	Time-point	<i>N</i>	Percent	Overall <i>N</i>	Overall Percent
One time-point	T1	43	10.6	43	10.6
	T1 and T2	46	11.4		
Two time-points	T1 and T3	7	1.7	63	15.6
	T1 and T4	10	2.5		
	T1, T2, and T3	44	10.9		
Three time-points	T1, T2, T4	30	7.4	98	24.2
	T1, T3, T4	24	5.9		
Four time-points	T1, T2, T3, T4	200	49.5	200	49.5

Table 7.4.

Intervention content for 3 and 6 session programs

3 sessions	6 sessions	Content
Session 1	Session 1	<ul style="list-style-type: none"> • Introduction of facilitators and parental consent. • Questionnaires, information sheet, and participant consent.
	Session 2	<ul style="list-style-type: none"> • Psychoeducation. • Introduction to mindfulness (MM)/ Introduction to PMR (PMR) • Mindful eating. (MM)/ Stress and the body exercise. (PMR) • Mindfulness of the breath and body (MM)/ Progressive Muscle Relaxation meditation (PMR) • Summary and home practice 1.
Session 2	Session 3	<ul style="list-style-type: none"> • Welcome back and home practice review 1. • Introducing the cognitive model. • Cognitive model example 2.
	Session 4	<ul style="list-style-type: none"> • Cognitive challenging. • Cognitive distortions. • Sitting Mindfulness of Thoughts practice (MM) / Sitting PMR Practice (PMR) • Summary and home practice 2.
Session 3	Session 5	<ul style="list-style-type: none"> • Welcome back and home practice review 2 • Cognitive model reminder • Thoughts about alcohol
	Session 6	<ul style="list-style-type: none"> • Summary and explanation of follow-ups and SMS. • Post intervention questionnaires.

Table 7.5.

Participant demographic variables (N = 404).

Demographic variable		<i>N</i>	%
Parental Background	Australian/New Zealander	77	19.06
	Aboriginal/Torres Strait Islander	8	1.98
	European	130	32.18
	Asian	33	8.17
	Polynesian/Melanesian	7	1.73
	North American	3	0.74
	African	6	1.49
	Mixed nationality parentage	20	4.95
	Unsure/did not respond	120	29.70
Where participant was born	Australia/New Zealand	357	88.37
	Europe	16	3.96
	Asia	23	5.69
	Polynesia/Melanesia	2	0.50
	North America	2	0.50
	Africa	4	0.99
Language mostly spoken at home	English	369	91.34
	Other	33	8.17
	Missing	2	0.50
Family Affluence	Low affluence	25	6.19
	Medium affluence	334	82.67
	High affluence	43	10.64
	Missing	2	0.50
Who participants live with	Mother	30	7.43
	Father	12	2.97
	Both mother and father	338	83.66
	Other	22	5.45
	Missing	2	0.50

Table 7.6.

Contrast codes for alcohol consumption and impulsivity models

Condition	Contrast 1a	Contrast 2a
CBT+MM	0.33	0.5
Ax only	-0.66	0
CBT+PMR	0.33	-0.5

Table 7.7.

Contrast codes for social cognition models

Condition	Contrast 1b	Contrast 2b
CBT+MM	0.33	-0.5
Ax only	-0.66	0
CBT+PMR	0.33	0.5

Table 7.8.

Contrast codes for mindfulness models

Condition	Contrast 1c	Contrast 2c
CBT+MM	0.66	0
Ax only	-0.33	-0.5
CBT+PMR	-0.33	0.5

Chapter 8: General Discussion

This thesis aimed to investigate and intervene upon psychological risk factors for adolescent alcohol use. A literature review was conducted and it was found that, while prevention of problem alcohol use can be highly impactful on future alcohol problems, few current interventions target known risk factors and the mechanisms of effect of successful interventions are unclear. The bioSocial Cognitive Theory (bSCT) of substance use risk is a promising framework to explain the interplay of several cognitive and personality risk factors for adolescent alcohol use. The model predicts that the impulsivity factors of Reward Drive and Rash Impulsiveness impart separate pathways of risk through mediational relationships with cognitions relating to drinking refusal self-efficacy and alcohol expectancies (Gullo et al., 2010a). Chapters 4-8 comprise research conducted into the measurement of these constructs, utility of the model for predicting the prospective risk of adolescent alcohol use, and the process and outcomes of a Randomised Controlled Trial (RCT) aimed at targeting adolescent impulsivity and alcohol-related cognitions.

Summary of findings

Chapters 4 and 5 aimed to validate and reduce the length of existing measures of adolescent drinking refusal self-efficacy and alcohol expectancies. Shorter measures would increase scale utility for time-limited contexts, including research and clinical settings. This research reduced a 24-item expectancy scale and a 19-item drinking refusal self-efficacy scale into two 9-item scales, the Drinking Refusal Self-Efficacy Questionnaire – Shortened Revised Adolescent version (DRSEQ-SRA) and the Drinking Expectancy Questionnaire – Shortened Adolescent version (DEQ-A). Both scales demonstrated good fit to the data and had similar psychometric properties to the full scales. The shortened scales showed good reliability and each explained 18% of the variance in adolescent alcohol consumption in the study sample. These findings support previous studies showing that shortened scales can demonstrate comparable psychometric properties and predictive power to longer scales (Fromme & D’Amico, 2000). Both scales are proposed for use in research into mechanisms of risk and intervention effects, where the reduction in test administration time and participant fatigue may be of value.

Chapter 6 was the first prospective investigation of the bSCT model of adolescent alcohol use risk. As expected, high rash impulsiveness predicted lower drinking refusal self-efficacy and this, in turn, predicted increased future alcohol consumption. Positive (social)

alcohol expectancies predicted reduced drinking refusal self-efficacy, which also predicted increased future alcohol consumption. There was evidence that higher reward drive increased positive social alcohol expectancies, that is, having increased responsiveness and drive for rewarding outcomes of actions increased expectations that alcohol use would result in social facilitation. These expectancies were associated with reduced drinking refusal self-efficacy and thus, reward drive impacted alcohol use indirectly through alcohol expectancies and subsequent effects on drinking refusal self-efficacy. Overall, drinking refusal self-efficacy declined over time and was a more robust predictor of alcohol consumption than alcohol expectancies. However, both cognitive factors contributed to risk. A strength of the study was the use of two parallel cohorts, which allowed for comparison of the effects of the model for early and mid-adolescents. This comparison indicated that positive (social) alcohol expectancies had larger effects for the younger cohort (aged 11-13 years), and drinking-refusal self-efficacy had larger effects for older cohort (aged 13-15 years). This study showed that the bSCT model was appropriate for predicting alcohol consumption up to two years in the future across two cohorts of adolescents. It confirmed that impulsivity factors impart risk through their effects on alcohol-related cognitions and therefore provide unique, age-relevant targets for prevention interventions.

Chapter 7 provides details of designing and running a school-based brief alcohol use intervention. The final study included 404 year-9 and 10 adolescents who were cluster randomised into assessment-only control or one of two active conditions. The active conditions consisted of Cognitive Behavioural Therapy (CBT) techniques with either Mindfulness Meditation (CBT+MM) or a Progressive Muscle Relaxation control (CBT+PMR). The CBT component of the intervention aimed to target the risk factors identified by previous research, including the study reported in Chapter 6, namely drinking refusal self-efficacy and alcohol expectancies. Mindfulness was included for evaluation as a theoretically consistent intervention addition with the aim of reducing impulsivity, another risk factor implicated in the present thesis. Indeed, the study reported in Chapter 6 found that Rash Impulsiveness still directly predicted some variance in future alcohol use. It was not completely mediated by cognition. The intervention conditions both decreased the growth of alcohol consumption from baseline to the 6-month follow-up. The decreased growth may be the result of a significant increase in negative alcohol expectancies for the active conditions compared to the control. However, positive alcohol expectancies also increased for the active condition versus the control condition. This finding was thought to be explained by a possible

interaction with drinking motives, i.e., not only what they thought would happen but what they wished would happen. Motives have been proposed as a mediator of the relationship between alcohol expectancies and drinking outcomes (Engels, Wiers, Lemmers, & Overbeek, 2005; Kuntsche et al., 2007). However, this hypothesis would have to be confirmed in future research. Drinking refusal self-efficacy was stable over time and did not alter according to condition membership. Both rash impulsiveness and reward drive increased equally over time for all three conditions. There was no evidence that mindfulness changed over time, even for the CBT+MM condition. The main conclusions for this research are that brief universal CBT-based interventions can delay growth in adolescent alcohol use although the mechanisms of effect are as yet unclear. Further, mindfulness did not appear to improve outcomes beyond existing CBT effects. To our knowledge, this was the first study to include an adequate active control (PMR) when assessing the utility of mindfulness for adolescent substance use prevention.

Theoretical implications and future directions

Taken together, this body of research is the first to prospectively model the bSCT model of alcohol risk due to trait impulsivity and alcohol-related cognitions and then to investigate the effect of a brief intervention targeting these mechanisms. This research differs from previous investigations into adolescent impulsivity and cognitive risk through the inclusion of both reward drive and rash impulsiveness and through specifying the relationship between rash impulsiveness and drinking refusal self-efficacy. It was felt that this better incorporated current proposals that impulsivity is a two-factored construct with separate paths of risk for alcohol use (Gullo et al., 2010a; Stautz et al., 2017). Overall, the present research showed the utility of having a unified model of risk on which to base intervention efforts and that cognitive and personality risk factors interact to produce risk.

The current thesis further supports the utility of the 2-CARS model of substance use risk and conducted some of the first testing of the expanded bSCT model of alcohol risk in adolescent populations. The 2-CARS model proposes that impulsivity is comprised of rash impulsiveness and reward drive and that these factors affect alcohol use in differential ways (2-CARS) (Dawe et al., 2004). The bSCT model expands on this by theorising that the way in which the 2-CARS model imparts risk is through cognitive mediators (Gullo et al., 2010a). That is, rash impulsiveness affects alcohol use through decreasing drinking refusal self-efficacy, and reward drive produces risk through increasing positive alcohol expectancies (Gullo et al., 2010a). In the current research, both rash impulsiveness and reward drive were

found to be related to future alcohol consumption through their impact on cognitive mediators (Chapter 6). Interestingly, rash impulsiveness proved to be the more robust of the two impulsivity factors, despite reward drive/sensation seeking being commonly cited as a major driver of adolescent alcohol use (Sargent, Tanski, Stoolmiller, & Hanewinkel, 2010; Stautz & Cooper, 2013; Stautz et al., 2017). However, we did find that reward drive gained increased importance for the older cohort included in the analysis (13-15 years old) compared to the younger cohort. It may be the case that with increased exposure to alcohol with age, the rewarding aspects of alcohol become more salient and therefore desirable, creating an avenue for reward drive to take effect. Recent research has also found that self-reported ability to control behaviour at age 11 predicted alcohol use at age 16. This effect was magnified for participants with high reward sensitivity, indicating that ability to control rash impulsive-like behaviour produces risk independently and in combination with reward drive over the adolescent period (Peeters, Oldehinkel, & Vollebergh, 2017). Further, the RCT in Chapter 7 found that both rash impulsiveness and reward drive increased over time, replicating previous findings (Collado, Felton, MacPherson, & Lujuez, 2014; Littlefield et al., 2016; Niv, Tuvblad, Raine, Wang, & Baker, 2012; Romer & Hennessy, 2007). Both the findings from the prospective model (Chapter 6) and the RCT (Chapter 7) in the present thesis confirm that impulsivity may be an increasingly important risk factor as adolescents age. A novel finding is that the risk imparted by these factors may develop differentially, with rash impulsiveness decreasing drinking refusal self-efficacy from early adolescence and reward drive gaining importance in mid to late-adolescence.

The results presented in Chapter 6 build upon previous findings that both drinking refusal self-efficacy and alcohol expectancies predict adolescent alcohol consumption but that drinking refusal self-efficacy is a stronger predictor (Connor et al., 2008, 2011; Ehret et al., 2014). Much of the predictive power of drinking refusal self-efficacy arises from its relationships with other variables. Drinking refusal self-efficacy consistently mediates (fully or partially) the relationships between alcohol expectancies and rash impulsiveness on substance use (Baldwin et al., 1993; Connor et al., 2008, 2011, 2014; Gullo et al., 2010a; Harnett et al., 2013). These relationships were supported by findings in Chapter 6, which was the first demonstration that the mediational relationships between rash impulsiveness, reward drive and the cognitive risk factors of drinking refusal self-efficacy and positive (social) alcohol expectancies impacted adolescent alcohol use up to two years in the future. The theoretically consistent relationships over time show that the bSCT model is appropriate for

modelling prospective risk, the implication being that interventions could interrupt these processes.

The findings throughout the current research showed that socially-related drinking refusal self-efficacy and alcohol expectancies were consistently the greatest predictors within each cognitive domain. This supports previous research that adolescent alcohol use and alcohol-related cognitions are greatly impacted by social contexts (Aas et al., 1995; Jester, Wong, et al., 2015; Jones et al., 2001; Smith et al., 1995; Tomlinson & Brown, 2012; Young-Wolff et al., 2015). While adolescents are more driven by positive alcohol expectancies (and social cognitions in particular), by adulthood, the importance of negative alcohol expectancies increases (Leigh & Stacy, 2004), with adult alcohol use driven by a range of expectancies (Gullo et al., 2010a). Despite this trajectory of growth in negative expectancies, our findings from Chapter 4 showed that adolescents do hold negative expectancies and that these contribute unique variance in alcohol consumption. Further, abstinent adolescents had higher negative alcohol expectancies than those who had initiated alcohol use. Future research could further investigate the development of these negative expectancies over time, which the current thesis aimed to facilitate through the development of a shortened adolescent expectancy scale that includes both positive and negative expectancies.

The RCT outcomes presented in Chapter 7 showed that alcohol expectancies may be more implicated in adolescent prevention than drinking refusal self-efficacy, despite the latter being a more robust predictor of future drinking. These findings are consistent with initial conceptualisations of the interaction between alcohol expectancies and drinking refusal self-efficacy as central to the acquisition of alcohol use and abuse (Oei & Baldwin, 1993). However, it is inconsistent with previous findings that drinking refusal self-efficacy is central mechanism of treatment effects in alcohol treatment and relapse outcomes (Black & Chung, 2014; Cho, 2005; Connor et al., 2016). It is possible that drinking refusal self-efficacy becomes a higher impact mechanism once alcohol is being consumed at higher rates. At low use, as in the sample in Chapter 7, adolescents have reduced exposure to alcohol use, thus limiting the conditioning effect between alcohol use and behavioural outcomes (Oei & Baldwin, 1993). They also encounter fewer alcohol-related problems, which may assist in explaining the differences in impacts of cognitions between adolescents and adults. The lower consumption and thus fewer desirable or undesirable outcomes restricts the “testing” of perhaps exaggerated positive beliefs regarding their self-efficacy over drinking control and may, therefore, reduce change in self-efficacy. This possible explanation is supported by the

very high adolescent drinking refusal self-efficacy found in the present body of research utilising relatively alcohol naïve participants. It is also supported by previous findings that there is a reduced effect of socially-related alcohol expectancies on alcohol consumption for adolescents with less exposure to alcohol contexts due to being socially anxious, presumably due to the same lack of testing and subsequent reinforcement or challenge of beliefs (Tomlinson & Brown, 2012). However, as adolescents age and have increased exposure to situations involving alcohol the importance of these beliefs may increase. These time-dependent beliefs and traits highlight the need for extended follow-up periods in adolescent RCTs as mechanisms of effect may become apparent at different developmental ages.

Based on our findings and those of previous research we aimed to identify targets we theorised may improve intervention efficacy, including rash impulsivity and indirectly rather than directly targeting drinking refusal self-efficacy. Mindfulness has previously been shown to improve executive and inhibitory control (Chambers et al., 2009; Mrazek et al., 2013; Oberle et al., 2012; Semple & Burke, 2012; Teper et al., 2013) and thus was proposed as possible “antidote” to maladaptive impulsive actions. Our findings showed that this was not the case, as mindfulness did not increase over time and the CBT+Mindfulness condition did not show improvement compared to an active control. The lack of change in mindfulness could have been due to insufficient mindfulness training. However, in adult populations, even a few sessions of meditation have previously been shown to produce effects and interventions longer than one month may not produce additional effects (Sedlmeier et al., 2012). Another hypothesis is that adolescents did not incorporate the mindfulness meditation into their routines adequately, but this is countered by a recent RCT showing that home practice of mindfulness did not moderate the effects of a school-based mindfulness intervention on depression, anxiety, or eating disorder symptoms (Johnson et al., 2017). To our knowledge, this is the first RCT comparing mindfulness-enhanced CBT to an adequate active control for adolescent substance use outcomes. Its findings add to a growing body of research showing that, while mindfulness may be therapeutic when compared to no intervention, it is not more effective than existing psychological interventions in targeting mental health outcomes (Farias et al., 2016; Johnson et al., 2017; Khoury et al., 2013).

When targeting the bSCT risk factors in the RCT (Chapter 7 and 8) we found that CBT did reduce growth in alcohol consumption over time. However, the mechanisms of effect were less clear than predicted. Alcohol expectancies did alter in the active treatment conditions, but both positive and negative expectancies increased. Further, drinking refusal

self-efficacy, a robust predictor of adult alcohol intervention outcomes (Connor et al., 2016), did not change over time or according to condition. Despite this, it is promising that the intervention was effective given that universal intervention programs are generally considered to have mixed evidence (Carney et al., 2016), and interventions targeting drinking refusal self-efficacy during mid-adolescence may produce detrimental effects (Onrust et al., 2016). One explanation for our results is that aiming to improve psychosocial functioning rather than only focusing on substance use has been shown to improve alcohol outcomes in school-based interventions (Stockings et al., 2016). It is possible that our approach to teaching CBT and PMR/MM as a life-skill to combat negative emotions and thoughts before transitioning to discussing how this relates to alcohol-related thoughts specifically may have benefitted outcomes. Further, our positive results may be due to our use of the bSCT model to choose intervention targets. Our aim to base intervention targets on known bSCT pathways of risk may have reduced the possible detrimental effects of directly targeting drinking refusal self-efficacy. Our CBT interventions targeted rash impulsiveness through mindfulness and alcohol expectancies more directly through cognitive challenging with the aim to indirectly target drinking-refusal self-efficacy. This indicates the unique strength of the bSCT model, which allowed us to indirectly target specific risk factors based on theoretically supported links with other variables.

It is not uncommon that mechanisms of action within adolescent treatment approaches are not as predicted. O’Leary-Barrett and colleagues (2016) investigated the mechanisms of effect in a brief personality-targeted school-based CBT intervention for alcohol use. They found that intervention effects over two years were not predicted by changes in internalising or externalising problems or changes in the targeted personality traits of impulsivity, sensation seeking, anxiety, or hopelessness (O’Leary-Barrett et al., 2016). Instead, improvements in future alcohol outcomes were predicted by initial intervention effects on alcohol use. That is, the delay of alcohol use onset and problem use decreased future problem use (O’Leary-Barrett et al., 2016). Despite intervening using cognitive strategies, measures of alcohol-related cognitions were not included as potential mediators of intervention effects. Therefore, direct comparison of mechanisms of effects with the present research cannot be made. As in our study, these findings do not fully explain the mechanism of the initial alcohol effects that successfully prevented future harm.

The findings from the present body of research show that two questions are important to consider when designing intervention programs for adolescent alcohol use – when to

intervene and how to choose intervention targets. The bSCT model has been shown to provide useful information to answer the latter question. Through an understanding of prospective associations between risk factors, interventions can be designed to increase efficacy. An example can be found in our decision to focus on rash impulsiveness and alcohol expectancies in order to indirectly target drinking refusal self-efficacy. This may have prevented the potential adverse effects that can be found when directly targeting this factor (Onrust et al., 2016).

The first question of when to intervene is more complex. The importance of considering age in risk and prevention was apparent throughout the thesis. As discussed above, positive social alcohol expectancies had a greater impact for the younger cohort in Chapter 6 whereas drinking refusal self-efficacy was a more robust predictor for older cohort. Additionally, reward drive was a greater predictor in the older cohort. In the RCT, older adolescents had significantly greater alcohol consumption, higher positive and negative alcohol expectancies, and reduced drinking refusal self-efficacy and impulsivity increased over time. These findings of differential effects of risk factors according to age support recent findings of altered efficacy of intervention targets depending on the adolescent developmental period. Onrust and colleagues (2016) found that, while early adolescents were responsive to the majority of school-based intervention strategies, the largest effect sizes were found for older adolescents. The RCT in the present study targeted mid-adolescence, which is a developmental period where there is little intervention success and even possible detrimental effects (Onrust et al., 2016). Taken together, prevention interventions may be of best effect if the developmental stage is considered when deciding intervention targets and at which age to intervene.

It is possible that one of the reasons that the mechanisms of effect were not as predicted in our RCT is the age chosen to intervene and the length of follow-up. We chose to intervene before the average age of onset in order to best reduce future alcohol use through delayed initiation and growth. However, this meant that adolescents had little exposure to personal alcohol use, resulting in very high baseline drinking refusal self-efficacy. Thus, the intervention outcomes could show very little impact on a key bSCT variable. It is possible that intervening in late adolescence would allow for greater visible impact on mechanisms shown to develop later in adolescence, such as reward drive and drinking refusal self-efficacy. Indeed, in their meta-analysis, Onrust and colleagues (2016) found that the largest effect sizes by far for universal adolescent substance use interventions were produced in late-

adolescence. So should interventions occur in late adolescence? Perhaps, but in the study by O’Leary-Barrett and colleagues (2016) mentioned above, where they investigated mechanisms of intervention effectiveness over a two-year period, the greatest mechanism of change was delayed initiation of alcohol bingeing and problems. Thus, the argument swings back to intervening earlier rather than later. It is also possible that the reason no effects were found in the RCT in the present research is due to restricted follow-up period. It is conceivable that intervention effects on the factors known to provide greater risk in late adolescence will become apparent with time. While this cannot be assumed, it provides an interesting area for future research. An intervention in early to mid-adolescence where participants are followed until late adolescence or early adulthood and mechanisms of interest as well as outcomes are examined longitudinally may help to answer this question of when to intervene.

Limitations

In addition to the limitations discussed throughout the previous chapters, several further considerations should be added to the interpretation of the present body of research. Firstly, all research was conducted with participants of similar backgrounds; that is, Australian school students from single denomination schools with parents of predominantly European descent. Previous reviews have cautioned against generalising school-based alcohol use intervention approaches to diverse populations (Carney et al., 2016). However, the increase in impulsivity during adolescence appears to be a cross-cultural phenomenon (Steinberg et al., 2017). Further, previous research has shown that race, ethnicity, income and family structure explain only a small proportion of adolescent risk behaviour (Blum et al., 2000). Therefore, the findings in the present research should be interpreted as specific to a particular demographic and future research should investigate the appropriateness of the risk factors to diverse cohorts and backgrounds.

Secondly, there is evidence that genetics play an important role in the development of adolescent alcohol consumption (Heinrich et al., 2016) and even alcohol cognitions after use onset (Young-Wolff et al., 2015). Research investigating genetic influences on the bSCT risk factors has been conducted in adult populations (Connor et al., 2008; Gullo, St. John, et al., 2014; Leamy et al., 2016; Swagell et al., 2012). It was not within the scope of the current thesis to include genetic factors. However, future research should endeavour to better understanding the role of genetics in the formation of early drinking patterns and alcohol-related cognition.

Finally, the alcohol-related cognitions investigated in the present thesis were explicit in nature. That is, they are consciously accessible. A parallel body of research has found that desires, biases and associations that are not consciously accessible (implicit cognitions) may help to explain why people engage in risky behaviours such as substance use despite explicit knowledge of harms (Stacy & Wiers, 2010). These processes are impacted by executive control and working memory (Grenard, Ames, & Stacy, 2008; Stacy & Wiers, 2010; Thush et al., 2008). While this program is not itself without theoretical and measurement limitations, implicit cognitions could be a useful addition to future models (Stacy & Wiers, 2010).

Concluding remarks

In conclusion, this thesis showed that a two-factor impulsivity model differentially affects alcohol-related cognitions over time and that age impacts these relationships. This increased understanding allows for unique intervention targets according to age and the possibility of indirectly targeting mechanisms of interest through known relationships with other risk factors. What is clear in both previous research and the present studies is that the cognitive and personality factors are consistently implicated in alcohol risk and targeting these risk factors appears to be successful in reducing alcohol consumption. However, whether the observed beneficial effects are produced by actually affecting these mechanisms is less clear. The lack of clarity is most likely due to the complex interplay of these risk factors, which points to the need for a comprehensive theoretical framework, such as the bSCT model.

It is clear that the future of adolescent alcohol-use programs is to intercede on specific risk factors, whether it be through universal or targeted programs. This research suggests that the relative impact of mechanisms of risk may be affected by age and that understanding of the inter-connecting pathways of risk can assist in indirectly targeting key mechanisms. The present research demonstrated that this approach successfully reduced the growth in alcohol use in a school-based RCT. This was an exciting finding due to previously mixed effects for prevention interventions and evidence that delaying problem alcohol use improves future outcomes (O’Leary-Barrett et al., 2016). The findings indicated that altering alcohol expectancies may be associated with intervention effects. However, mindfulness did not seem to impact rash impulsiveness, or improve alcohol outcomes. Conversely, drinking refusal self-efficacy, which is a common mechanism of change in adult alcohol use treatments (Connor et al., 2016), did not appear to effect prevention outcomes over a 6-month period but was strongly associated with prospective risk. It was hypothesised that relative alcohol

naivety contributed to this finding and that longer follow-ups or interventions in older adolescents may produce larger effects for this factor. Future research should attempt to disentangle the effects of risk versus mechanisms of intervention effects through larger RCTs with extended follow-up periods and by continuing to investigate possible associated mechanisms of action, such as the development of expectancies and their impact on drinking motives, changes in drinking refusal self-efficacy, and increases in impulsivity. Overall, the present research contributes to the ongoing efforts to improve adolescent alcohol use interventions through increasing the prospective understanding of high impact risk factors and through showing that targeting these risk factors can be effective in reducing adolescent alcohol consumption.

References

- Aas, H., Klepp, K. I., Laberg, J. C., & Aarø, L. E. (1995). Predicting adolescents' intentions to drink alcohol: Outcome expectancies and self-efficacy. *Journal of Studies on Alcohol*, *56*, 293–9. <https://doi.org/10.15288/jsa.1995.56.293>
- Aas, H. N., Leigh, B. C., Anderssen, N., & Jakobsen, R. (1998). Two-year longitudinal study of alcohol expectancies and drinking among Norwegian adolescents. *Addiction*, *93*, 373–384.
- Adamson, S. J., Sellman, J. D., & Frampton, C. M. A. (2009). Patient predictors of alcohol treatment outcome: A systematic review. *Journal of Substance Abuse Treatment*, *36*(1), 75–86. <https://doi.org/10.1016/j.jsat.2008.05.007>
- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*(3), 317–332. <https://doi.org/10.1007/BF02294359>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- American Psychological Association. (2008). Reporting standards for research in psychology: Why do we need them? What might they be? *American Psychologist*, *63*(9), 839–851. <https://doi.org/10.1037/0003-066X.63.9.839>
- Anderson, K. G., Smith, G. T., & Fischer, S. F. (2003). Women and acquired preparedness: Personality and learning implications for alcohol use. *Journal of Studies on Alcohol*, *64*(May), 384–392.
- Asato, M. R., Terwilliger, R., Woo, J., & Luna, B. (2010). White matter development in adolescence: A DTI study. *Cerebral Cortex*, *20*(9), 2122–2131. <https://doi.org/10.1093/cercor/bhp282>
- Australian Institute of Health and Welfare. (2011). *Young Australians: Their health and wellbeing 2011*. Canberra: AIHW.
- Australian Institute of Health and Welfare. (2014). *National Drug Strategy Household Survey detailed report: 2013*. Canberra: AIHW.
- Australian Institute of Health and Welfare. (2017). *National Drug Strategy Household Survey (NDSHS) 2016 key findings*. Canberra: AIHW.
- Baldwin, A. R., Oei, T. P. S., & Young, R. (1993). To drink or not to drink: The differential role of alcohol expectancies and drinking self-efficacy in quantity and frequency of alcohol consumption. *Cognitive Therapy and Research*, *17*(6), 511–530.
- Bandura, A. (1977). *Social learning theory. Social Learning Theory*. Oxford, England: Prentice-Hall.
- Bandura, A. (1986). *Social foundations of thought and action*. Englewood Cliffs NJ: Prentice-Hall.
- Bandura, A., Ross, D., & Ross, S. A. (1961). Transmission of aggression through imitation of aggressive models. *Journal of Abnormal and Social Psychology*, *63*(3), 575–582. <https://doi.org/10.1037/h0045925>
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International University Press.
- Beck, A. T., & Haigh, E. A. P. (2014). Advances in cognitive theory and therapy: the generic cognitive model. *Annual Review of Clinical Psychology*, *10*(December 2013), 1–24.

<https://doi.org/10.1146/annurev-clinpsy-032813-153734>

- Bekman, N. M., Goldman, M. S., Worley, M. J., & Anderson, K. G. (2011). Pre-adolescent alcohol expectancies: Critical shifts and associated maturational processes. *Experimental and Clinical Psychopharmacology*, *19*(6), 420–432. <https://doi.org/10.1037/a0025373>
- Bernstein, A., Hadash, Y., Lichtash, Y., Tanay, G., Sherherd, K., & Fresco, D. M. (2015). Decentering and related constructs: A critical review and metacognitive processes model. *Perspectives on Psychological Science*, *10*(5), 599–617. <https://doi.org/10.1177/1745691615594577>
- Biegel, G. M., Brown, K. W., Shapiro, S. L., & Schubert, C. M. (2009). Mindfulness-based stress reduction for the treatment of adolescent psychiatric outpatients: A randomized clinical trial. *Journal of Consulting and Clinical Psychology*, *77*(5), 855–866. <https://doi.org/10.1037/a0016241>
- Black, J. J., & Chung, T. (2014). Mechanisms of change in adolescent substance use treatment: How does treatment work? *Substance Abuse*, *35*(November), 344–351. <https://doi.org/10.1080/08897077.2014.925029>
- Blum, R. W., Beuhring, T., Shew, M. L., Bearinger, L. H., Sieving, R. E., & Resnick, M. D. (2000). The effects of race/ethnicity, income, and family structure on adolescent risk behaviors. *American Journal of Public Health*, *90*(12), 1879–1884. <https://doi.org/10.2105/AJPH.90.12.1879>
- Boden, J. M., Fergusson, D. M., & Horwood, L. J. (2013). Alcohol misuse and criminal offending: Findings from a 30-year longitudinal study. *Drug and Alcohol Dependence*, *128*(1–2), 30–6. <https://doi.org/10.1016/j.drugalcdep.2012.07.014>
- Bögels, S., Hoogstad, B., van Dun, L., de Schutter, S., & Restifo, K. (2008). Mindfulness training for adolescents with externalizing disorders and their parents. *Behavioural and Cognitive Psychotherapy*, *36*(02), 193–209. <https://doi.org/10.1017/S1352465808004190>
- Bonomo, Y. A., Bowes, G., Coffey, C., Carlin, J. B., & Patton, G. C. (2004). Teenage drinking and the onset of alcohol dependence: A cohort study over seven years. *Addiction*, *99*(12), 1520–1528. <https://doi.org/10.1111/j.1360-0443.2004.00846.x>
- Bootzin, R. R., & Stevens, S. J. (2005). Adolescents, substance abuse, and the treatment of insomnia and daytime sleepiness. *Clinical Psychology Review*, *25*(5), 629–644. <https://doi.org/10.1016/j.cpr.2005.04.007>
- Bowen, S., Witkiewitz, K., Clifasefi, S. L., Grow, J., Chawla, N., Hsu, S. H., ... E, D. (2014). Relative Efficacy of Mindfulness-Based Relapse Prevention, Standard Relapse Prevention, and Treatment as Usual for Substance Use Disorders. *JAMA Psychiatry*, *71*(5), 547. <https://doi.org/10.1001/jamapsychiatry.2013.4546>
- Boyce, W., Torsheim, T., Currie, C., & Zambon, A. (2006). The Family Affluence Scale as a Measure of National Wealth: Validation of an adolescent self-report measure. *Social Indicators Research*, *78*(3), 473–487.
- Britton, W. B., Bootzin, R. R., Cousins, J. C., Hasler, B. P., Peck, T., & Shapiro, S. L. (2010). The Contribution of Mindfulness Practice to a Multicomponent Behavioral Sleep Intervention Following Substance Abuse Treatment in Adolescents: A Treatment-Development Study. *Substance Abuse*, *31*(2), 86–97. <https://doi.org/10.1080/08897071003641297>
- Brown, K. W., West, A. M., Loverich, T. M., & Biegel, G. M. (2011). Assessing adolescent

- mindfulness: Validation of an Adapted Mindful Attention Awareness Scale in adolescent normative and psychiatric populations. *Psychological Assessment*, 23, 1023–1033. <https://doi.org/10.1037/a0021338>
- Brown, S. A., Christiansen, B. A., & Goldman, M. S. (1987). The alcohol expectancy questionnaire: An instrument for the assessment of adolescent and adult alcohol expectancies. *Journal of Studies on Alcohol*, 48(5), 483–91. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/3669677>
- Brown, S. A., Goldman, M. S., & Christiansen, B. A. (1985). Do alcohol expectancies mediate drinking patterns of adults? *Journal of Consulting and Clinical Psychology*, 53(4), 512–519. <https://doi.org/10.1037/0022-006X.53.4.512>
- Brown, S. A., Goldman, M. S., Inn, A., & Anderson, L. R. (1980). Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *Journal of Consulting and Clinical Psychology*, 48(4), 419–426. <https://doi.org/10.1037/0022-006X.48.4.419>
- Bush, K., Kivlahan, D. R., McDonell, M. B., Fihn, S. D., & Bradley, K. A. (1998). The AUDIT Alcohol Consumption Questions (AUDIT-C). *Archives of Internal Medicine*, 158, 1789–1795. <https://doi.org/10.1097/00000374-199811000-00034>
- Carney, T., Myers, B. J., Louw, J., & Okwundu, C. I. (2016). Brief school-based interventions and behavioural outcomes for substance-using adolescents. *Cochrane Database of Systematic Reviews*, 2016(1). <https://doi.org/10.1002/14651858.CD008969.pub3>
- Castellanos, N., & Conrod, P. (2006). Brief interventions targeting personality risk factors for adolescent substance misuse reduce depression, panic and risk-taking behaviours. *Journal of Mental Health*, 15(6), 645–658. <https://doi.org/10.1080/09638230600998912>
- Chambers, R. A., Taylor, J. R., & Potenza, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *The American Journal of Psychiatry*, 160(6), 1041–1052. <https://doi.org/10.1176/appi.ajp.160.6.1041>
- Chambers, R., Gullone, E., & Allen, N. B. (2009). Mindful emotion regulation: An integrative review. *Clinical Psychology Review*, 29(6), 560–572. <https://doi.org/10.1016/j.cpr.2009.06.005>
- Chassin, L., Flora, D. B., & King, K. M. (2004). Trajectories of alcohol and drug use and dependence from adolescence to adulthood: The effects of familial alcoholism and personality. *Journal of Abnormal Psychology*, 113(4), 483–498. <https://doi.org/10.1037/0021-843X.113.4.483>
- Cho, H. (2005). Readiness to change, norms, and self-efficacy among heavy-drinking college students. *Journal of Studies on Alcohol*, 67(1), 131–138. <https://doi.org/10.15288/jsa.2006.67.131>
- Christiansen, B. A., & Goldman, M. S. (1983). Alcohol-related expectancies versus demographic/background variables in the prediction of adolescent drinking. *Journal of Consulting and Clinical Psychology*, 51(2), 249–257. <https://doi.org/10.1037/0022-006X.51.2.249>
- Christiansen, B. A., Goldman, M. S., & Inn, A. (1982). Development of alcohol-related expectancies in adolescents: Separating pharmacological from social-learning influences. *Journal of Consulting and Clinical Psychology*, 50(3), 336–344. <https://doi.org/10.1037/0022-006X.50.3.336>

- Clayton, R. R., Cattarello, A., & Walden, K. P. (1991). Sensation seeking as a potential mediating variable for school-based prevention intervention: A two-year follow-up of DARE. *Health Communication, 3*(4), 229–239. https://doi.org/10.1207/s15327027hc0304_5
- Coates, J. M., Gullo, M. J., Feeney, G. F. X., Young, R. M., Dingle, G. A., & Connor, J. P. (2018). Alcohol expectancies pre-and post-Alcohol Use Disorder treatment: Clinical implications. *Addictive Behaviors*.
- Cohen, E., Feinn, R., Arias, A., & Kranzler, H. R. (2007). Alcohol treatment utilization: Findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Drug and Alcohol Dependence, 86*(2–3), 214–221. <https://doi.org/10.1016/j.drugalcdep.2006.06.008>
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Mahwah, N.J.: Laurence Erlbaum Associates.
- Colder, C. R., Read, J. P., Wiczorek, W. F., Eiden, R. D., Lengua, L. J., Hawk, L. W., ... Lopez-Vergara, H. I. (2017). Cognitive appraisals of alcohol use in early adolescence: Psychosocial predictors and reciprocal associations with alcohol use. *Journal of Early Adolescence, 37*(4), 525–558. <https://doi.org/10.1177/0272431615611256>
- Colder, C. R., Read, J. P., Wiczorek, W. F., Eiden, R. D., Lengua, L. J., Hawk, L. W., ... Lopez-Vergara, H. I. (2017). Cognitive appraisals of alcohol use in early adolescence. *The Journal of Early Adolescence, 37*(4), 525–558. <https://doi.org/10.1177/0272431615611256>
- Collado, A., Felton, J. W., MacPherson, L., & Lujuez, C. W. (2014). Longitudinal trajectories of sensation seeking, risk taking propensity, and impulsivity across early to middle adolescence. *Addictive Behaviors, 39*(11), 1580–1588. <https://doi.org/10.1016/j.addbeh.2014.01.024>
- Collins, D. J., & Lapsley, H. M. (2008). *The costs of tobacco, alcohol and illicit drug abuse to Australian society in 2004/05. National Drug Strategy Monograph*. Canberra. <https://doi.org/10.1037/e674312010-001>
- Connor, J. P., George, S. M., Gullo, M. J., Kelly, A. B., & Young, R. M. (2011). A prospective study of alcohol expectancies and self-efficacy as predictors of young adolescent alcohol misuse. *Alcohol and Alcoholism, 46*(2), 161–9. <https://doi.org/10.1093/alcalc/agr004>
- Connor, J. P., Gudgeon, E. T., Young, R. M., & Saunders, J. B. (2007). The relationship between alcohol expectancies and drinking restraint in treatment seeking alcohol dependent patients. *Addictive Behaviors, 32*(7), 1461–1469. <https://doi.org/10.1016/j.addbeh.2006.11.001>
- Connor, J. P., Gullo, M. J., Feeney, G. F. X., Kavanagh, D. J., & Young, R. M. (2014). The relationship between cannabis outcome expectancies and cannabis refusal self-efficacy in a treatment population. *Addiction, 109*, 111–119. <https://doi.org/10.1111/add.12366>
- Connor, J. P., Haber, P. S., & Hall, W. D. (2016). Alcohol use disorders. *The Lancet, 387*(10022), 988–998. [https://doi.org/10.1016/S0140-6736\(15\)00122-1](https://doi.org/10.1016/S0140-6736(15)00122-1)
- Connor, J. P., Young, R. M., Saunders, J. B., Lawford, B. R., Ho, R., Ritchie, T. L., & Noble, E. P. (2008). The A1 allele of the D2 dopamine receptor gene region, alcohol expectancies and drinking refusal self-efficacy are associated with alcohol dependence severity. *Psychiatry Research, 160*(1), 94–105.

<https://doi.org/10.1016/j.psychres.2007.06.030>

- Conrod, P. J. (2016). Personality-Targeted interventions for substance use and misuse. *Current Addiction Reports*, 3, 426–436. <https://doi.org/10.1007/s40429-016-0127-6>
- Conrod, P. J., Castellanos-Ryan, N., & Mackie, C. (2011). Long-term effects of a personality-targeted intervention to reduce alcohol use in adolescents. *Journal of Consulting and Clinical Psychology*, 79(3), 296–306. <https://doi.org/10.1037/a0022997>
- Conrod, P. J., Castellanos, N., & Mackie, C. (2008). Personality-targeted interventions delay the growth of adolescent drinking and binge drinking. *Journal of Child Psychology and Psychiatry*, 49(2), 181–190. <https://doi.org/10.1111/j.1469-7610.2007.01826.x>
- Conrod, P. J., O’Leary-Barrett, M., Newton, N. C., Topper, L., Castellanos-Ryan, N., Mackie, C., & Girard, A. (2013). Effectiveness of a selective, personality-targeted prevention program for adolescent alcohol use and misuse: A cluster randomized controlled trial. *JAMA Psychiatry*, 70(3), 334–42. <https://doi.org/10.1001/jamapsychiatry.2013.651>
- Conrod, P. J., Stewart, S. H., Comeau, N., & Maclean, A. M. (2006). Efficacy of cognitive-behavioral interventions targeting personality risk factors for youth alcohol misuse. *Journal of Clinical Child and Adolescent Psychology*, 35(4), 550–63. https://doi.org/10.1207/s15374424jccp3504_6
- Conrod, P. J., Stewart, S. H., Pihl, R. O., Cote, S., Fontaine, V., & Dongier, M. (2000). Efficacy of Brief Coping Skills Interventions That Match Different Personality Profiles of Female Substance Abusers. *Psychology of Addictive Behaviors*, 14(3), 231–242. <https://doi.org/10.1037//0893-164X.14.3.231>
- Cooper, A., & Gomez, R. (2008). The development of a short form of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire. *Journal of Individual Differences*, 29(2), 90–104. <https://doi.org/10.1027/1614-0001.29.2.90>
- Copeland, A. L., Proctor, S. L., Terlecki, M. A., Kulesza, M., & Williamson, D. A. (2014). Do positive alcohol expectancies have a critical developmental period in pre-adolescents? *Journal of Studies on Alcohol and Drugs*, 75(6), 945–952. <https://doi.org/doi:10.15288/jsad.2014.75.945>
- Corbin, W. R., Iwamoto, D. K., & Fromme, K. (2011). A comprehensive longitudinal test of the acquired preparedness model for alcohol use and related problems. *Journal of Studies on Alcohol and Drugs*, 72, 602–10. <https://doi.org/http://dx.doi.org/10.15288/jsad.2011.72.602>
- Cuijpers, P. (2002). Effective ingredients of school-based drug prevention programs: A systematic review. *Addictive Behaviours*, 27(6), 1009–1023. <https://doi.org/10.1111/j.1467-9280.2008.02196.x>
- Currie, C., Molcho, M., Boyce, W., Holstein, B., Torsheim, T., & Richter, M. (2008). Researching health inequalities in adolescents: The development of the Health Behaviour in School-Aged Children (HBSC) Family Affluence Scale. *Social Science and Medicine*, 66, 1429–1436. <https://doi.org/10.1016/j.socscimed.2007.11.024>
- Cyders, M. A., Smith, G. T., Spillane, N. S., Fischer, S., Annus, A. M., & Peterson, C. (2007). Integration of impulsivity and positive mood to predict risky behavior: Development and validation of a measure of positive urgency. *Psychological Assessment*, 19(1), 107–118. <https://doi.org/10.1037/1040-3590.19.1.107>
- Davidson, R. J. (2010). Empirical Explorations of Mindfulness: Conceptual and

- Methodological Conundrums. *Emotion*, *10*(1), 8–11. <https://doi.org/10.1037/a0018480>
- Dawe, S., Gullo, M. J., & Loxton, N. J. (2004). Reward drive and rash impulsiveness as dimensions of impulsivity: Implications for substance misuse. *Addictive Behaviors*, *29*(August 2015), 1389–1405. <https://doi.org/10.1016/j.addbeh.2004.06.004>
- Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience and Biobehavioral Reviews*, *28*(3), 343–51. <https://doi.org/10.1016/j.neubiorev.2004.03.007>
- Deas, D., Riggs, P., Langenbucher, J., Goldman, M., & Brown, S. (2000). Adolescents are not adults: Developmental in considerations alcohol users. *Alcoholism, Clinical and Experimental Research*, *24*(2), 232–7. <https://doi.org/http://dx.doi.org/10.1111/j.1530-0277.2000.tb04596.x>
- Deplus, S., Billieux, J., Scharff, C., & Philippot, P. (2016). A Mindfulness-Based Group Intervention for Enhancing Self-Regulation of Emotion in Late Childhood and Adolescence: A Pilot Study. *International Journal of Mental Health and Addiction*, *14*(5), 775–790. <https://doi.org/10.1007/s11469-015-9627-1>
- Diep, P. B., Schelleman-Offermans, K., Kuntsche, E., De Vries, N., & Knibbe, R. A. (2016). Direct and indirect effects of alcohol expectancies through drinking motives on alcohol outcomes among students in Vietnam. *Addictive Behaviors*, *52*, 115–122. <https://doi.org/10.1016/j.addbeh.2015.09.009>
- Dietrich, T., Rundle-Thiele, S., Schuster, L., Drennan, J., Russell-Bennett, R., Leo, C., ... Connor, J. P. (2015). Differential segmentation responses to an alcohol social marketing program. *Addictive Behaviors*, *49*, 68–77. <https://doi.org/10.1016/j.addbeh.2015.05.010>
- Dissabandara, L. O., Loxton, N. J., Dias, S. R., Dodd, P. R., Daghli, M., & Stadlin, A. (2014). Dependent heroin use and associated risky behaviour: The role of rash impulsiveness and reward sensitivity. *Addictive Behaviors*, *39*(1), 71–76. <https://doi.org/10.1016/j.addbeh.2013.06.009>
- Egan, S. T., Kambouropoulos, N., & Staiger, P. K. (2010). Rash-impulsivity, reward-drive and motivations to use ecstasy. *Personality and Individual Differences*, *48*(5), 670–675. <https://doi.org/10.1016/j.paid.2010.01.010>
- Ehret, P. J., Ghaidarov, T. M., & LaBrie, J. W. (2014). Can you say no? Examining the relationship between drinking refusal self-efficacy and protective behavioral strategy use on alcohol outcomes. *Addictive Behaviors*, *38*(4), 1898–1904. <https://doi.org/10.1016/j.addbeh.2012.12.022>
- Elek, E., Wagstaff, D. A., & Hecht, M. L. (2010). Effects of the 5th and 7th grade enhanced versions of the keepin' it REAL substance use prevention curriculum. *Journal of Drug Education*, *40*(1), 61–79. <https://doi.org/10.2190/DE.40.1.e>
- Ellickson, P. L., Tucker, J. S., & Klein, D. J. (2003). Ten-year prospective study of public health problems associated with early drinking. *Pediatrics*, *111*(5), 949–955. <https://doi.org/10.1542/peds.111.5.949>
- Embretson, S. E., & Diehl, K. A. (2000). Item Response Theory. In *Encyclopedia of Psychology* (Vol. 4, pp. 378–380). American Psychological Association. <https://doi.org/10.1037/10519-153>
- Engels, R. C. M. E., Wiers, R., Lemmers, L., & Overbeek, G. (2005). Drinking motives, alcohol expectancies, self-efficacy, and drinking patterns. *Journal of Drug Education*, *35*(2), 147–166. <https://doi.org/10.2190/6Q6B-3LMA-VMVA-L312>

- Ennett, S. T., Tobler, N. S., Ringwalt, C. L., & Flewelling, R. L. (1994). How effective is drug abuse resistance education? A meta-analysis of project DARE outcome evaluations. *American Journal of Public Health, 84*(9), 1394–1401. <https://doi.org/10.2105/AJPH.84.9.1394>
- Ewing, B. A., Tucker, J. S., Miles, J. N. V., Shih, R. A., Kulesza, M., Pedersen, E. R., & D'Amico, E. J. (2015). Early Substance Use and Subsequent DUI in Adolescents. *Pediatrics, 136*(5), 1–8. <https://doi.org/10.1542/peds.2015-1143>
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods, 4*(3), 272–299. <https://doi.org/10.1037/1082-989X.4.3.272>
- Farias, M., Wikholm, C., Delmonte, R., Sedlmeier, P., Eberth, J., Schwarz, M., ... McMahan, D. (2016). What is mindfulness-based therapy good for? *The Lancet Psychiatry, 3*(11), 1012–1013. [https://doi.org/10.1016/S2215-0366\(16\)30211-5](https://doi.org/10.1016/S2215-0366(16)30211-5)
- Feduccia, A. A., Chatterjee, S., & Bartlett, S. E. (2012). Neuronal nicotinic acetylcholine receptors: neuroplastic changes underlying alcohol and nicotine addictions. *Frontiers in Molecular Neuroscience, 5*(August), 1–18. <https://doi.org/10.3389/fnmol.2012.00083>
- Feldman, G., Greeson, J., & Senville, J. (2010). Differential effects of mindful breathing, progressive muscle relaxation, and loving kindness meditation on decentering and negative reactions to repetitive thoughts. *Behaviour Research and Therapy, 48*(10), 1002–1011. <https://doi.org/10.1016/j.brat.2010.06.006>.Differential
- Feldstein Ewing, S. W., Sakhardande, A., & Blakemore, S.-J. (2014). The effect of alcohol consumption on the adolescent brain: A systematic review of MRI and fMRI studies of alcohol-using youth. *NeuroImage: Clinical, 5*, 1–17. <https://doi.org/10.1016/j.nicl.2014.06.011>
- Feola, T. W., de Wit, H., & Richards, J. B. (2000). Effects of d-amphetamine and alcohol on a measure of behavioral inhibition in rats. *Behavioral Neuroscience, 114*(4), 838–848. <https://doi.org/10.1037//0735-7044.114.4.838>
- Fernie, G., Peeters, M., Gullo, M. J., Christiansen, P., Cole, J. C., Sumnall, H., & Field, M. (2013). Multiple behavioural impulsivity tasks predict prospective alcohol involvement in adolescents. *Addiction, 108*, 1916–1923. <https://doi.org/10.1111/add.12283>
- Foxcroft, D. R., & Tsertsvadze, A. (2011). Universal family-based prevention programs for alcohol misuse in young people. *Cochrane Database of Systematic Reviews, 9*(9), CD009308. <https://doi.org/10.1002/14651858.CD009308>
- Fromme, K., & D'Amico, E. J. (2000). Measuring adolescent alcohol outcome expectancies. *Psychology of Addictive Behaviors, 14*(2), 206–212. <https://doi.org/10.1037/0893-164X.14.2.206>
- Fromme, K., Stroot, E., & Kaplan, D. (1993). Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire. *Psychological Assessment, 5*(1), 19–26. <https://doi.org/10.1037/1040-3590.5.1.19>
- Gao, C., Ogeil, R., & Lloyd, B. (2014). *Alcohol's burden of disease in Australia*. Canberra.
- Gao, L., Curtiss, J., Liu, X., & Hofmann, S. G. (2017). Differential Treatment Mechanisms in Mindfulness Meditation and Progressive Muscle Relaxation. *Mindfulness*. <https://doi.org/10.1007/s12671-017-0869-9>
- Gaume, J., McCambridge, J., Bertholet, N., & Daepfen, J. B. (2014). Mechanisms of action of brief alcohol interventions remain largely unknown - A narrative review. *Frontiers in*

- Psychiatry*, 5(AUG), 1–9. <https://doi.org/10.3389/fpsyt.2014.00108>
- Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., ... Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, 2(10), 861–863. <https://doi.org/10.1038/13158>
- Gigsby, T. J., Forster, M., Unger, J. B., & Sussman, S. (2016). Predictors of alcohol-related negative consequences in adolescents: A systematic review of the literature and implications for future research. *Journal of Adolescence*, 48, 18–35. <https://doi.org/10.1016/j.adolescence.2016.01.006>
- Goossens, F. X., Lammers, J., Onrust, S. A., Conrod, P. J., de Castro, B. O., & Monshouwer, K. (2016). Effectiveness of a brief school-based intervention on depression, anxiety, hyperactivity, and delinquency: a cluster randomized controlled trial. *European Child & Adolescent Psychiatry*, 25(6), 639–48. <https://doi.org/10.1007/s00787-015-0781-6>
- Gorman, D. M. (2015). ‘Everything works’: The need to address confirmation bias in evaluations of drug misuse prevention interventions for adolescents. *Addiction*, 110, 1539–1540. <https://doi.org/10.1111/add.12954>
- Goyal, M., Singh, S., Sibinga, E. M. S., Gould, N. F., Rowland-Seymour, A., Sharma, R., ... Haythornthwaite, J. a. (2014). Meditation programs for psychological stress and well-being: a systematic review and meta-analysis. *JAMA Internal Medicine*, 174(3), 357–68. <https://doi.org/10.1001/jamainternmed.2013.13018>
- Graham, J. W. (2009). Missing data analysis: making it work in the real world. *Annual Review of Psychology*, 60, 549–576. <https://doi.org/10.1146/annurev.psych.58.110405.085530>
- Grant, B. F., & Dawson, D. A. (1997). Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse*, 9, 103–109. [https://doi.org/10.1016/S0899-3289\(97\)90009-2](https://doi.org/10.1016/S0899-3289(97)90009-2)
- Grant, B. F., Stinson, F. S., & Harford, T. C. (2001). Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: A 12-year follow-up. *Journal of Substance Abuse*, 13(4), 493–504. [https://doi.org/10.1016/S0899-3289\(01\)00096-7](https://doi.org/10.1016/S0899-3289(01)00096-7)
- Grenard, J. L., Ames, S. L., & Stacy, A. W. (2008). Working memory capacity moderated the predictive effects of drug-related associations on substance use. *Psychology of Addictive Behaviors*, 22(3), 426–432. <https://doi.org/10.1037/0893-164X.22.3.426>
- Griffin, K. W., & Botvin, G. J. (2010). Evidence-Based Interventions for Preventing Substance Use Disorders in Adolescents. *Child and Adolescent Psychiatric Clinics of North America*, 19, 505–526. <https://doi.org/10.1016/j.chc.2010.03.005>
- Gullo, M. J., & Dawe, S. (2008). Impulsivity and adolescent substance use: Rashly dismissed as “all-bad”? *Neuroscience and Biobehavioral Reviews*, 32(8), 1507–18. <https://doi.org/10.1016/j.neubiorev.2008.06.003>
- Gullo, M. J., Dawe, S., Kambouropoulos, N., Staiger, P. K., & Jackson, C. J. (2010a). Alcohol expectancies and drinking refusal self-efficacy mediate the association of impulsivity with alcohol misuse. *Alcoholism, Clinical and Experimental Research*, 34(8), 1386–99. <https://doi.org/10.1111/j.1530-0277.2010.01222.x>
- Gullo, M. J., Dawe, S., Kambouropoulos, N., Staiger, P. K., & Jackson, C. J. (2010b). Supplementary material. *Alcoholism, Clinical and Experimental Research*.

- Gullo, M. J., Loxton, N. J., & Dawe, S. (2014). Impulsivity: Four ways five factors are not basic to addiction. *Addictive Behaviors*, *39*(11), 1547–56. <https://doi.org/10.1016/j.addbeh.2014.01.002>
- Gullo, M. J., Loxton, N. J., Price, T., Voisey, J., Young, R. M., & Connor, J. P. (2017). A laboratory model of adolescent impulsivity and alcohol use. *Behaviour Research and Therapy*, *97*, 52–63. <https://doi.org/10.1016/j.brat.2017.07.003>
- Gullo, M. J., Matveeva, M., Feeney, G. F. X., Young, R. M., & Connor, J. P. (2017). Social cognitive predictors of treatment outcome in cannabis dependence. *Drug and Alcohol Dependence*, *170*, 74–81. <https://doi.org/10.1016/j.drugalcdep.2016.10.030>
- Gullo, M. J., St. John, N., Young, R. M., Saunders, J. B., Noble, E. P., & Connor, J. P. (2014). Impulsivity-related cognition in alcohol dependence: Is it moderated by DRD2/ANKK1 gene status and executive dysfunction? *Addictive Behaviors*, *39*(11), 1663–9. <https://doi.org/10.1016/j.addbeh.2014.02.004>
- Gullo, M. J., Ward, E., Dawe, S., Powell, J., & Jackson, C. J. (2011). Support for a two-factor model of impulsivity and hazardous substance use in British and Australian young adults. *Journal of Research in Personality*, *45*(1), 10–18. <https://doi.org/10.1016/j.jrp.2010.11.002>
- Hallgren, K. A., & Witkiewitz, K. (2013). Missing data in alcohol clinical trials: A comparison of methods. *Alcoholism, Clinical and Experimental Research*, *37*(12), 2152–2160. <https://doi.org/10.1111/acer.12205>
- Hansen, W. B., & McNeal, R. B. (1997). How DARE works: an examination of program effects on mediating variables. *Health Education and Behavior*, *24*, 165–176.
- Harkness, A. R., & Lilienfeld, S. O. (1997). Individual differences science for treatment planning: Personality traits. *Psychological Assessment*, *9*(4), 349–360. <https://doi.org/10.1037/1040-3590.9.4.349>
- Harnett, P. H., Lynch, S. J., Gullo, M. J., Dawe, S., & Loxton, N. (2013). Personality, cognition and hazardous drinking: Support for the 2-Component Approach to Reinforcing Substances Model. *Addictive Behaviors*, *38*(12), 2945–2948. <https://doi.org/10.1016/j.addbeh.2013.08.017>
- Harris, J. S., Stewart, D. G., & Stanton, B. C. (2017). Urge Surfing as Aftercare in Adolescent Alcohol Use: a Randomized Control Trial. *Mindfulness*, *8*(1), 144–149. <https://doi.org/10.1007/s12671-016-0588-7>
- Hartley, C. A., & Somerville, L. H. (2015). The neuroscience of adolescent decision-making. *Current Opinion in Behavioral Sciences*, *5*, 108–115. <https://doi.org/10.1016/j.cobeha.2015.09.004>
- Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., ... Grant, B. F. (2013). DSM-5 criteria for Substance Use Disorders: Recommendations and rationale. *American Journal of Psychiatry*, *170*(8), 834–851. <https://doi.org/10.1176/appi.ajp.2013.12060782>
- Hayes, S. C., Strosahl, K., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.
- Hecht, M. L., Marsiglia, F. F., Elek, E., Wagstaff, D. A., Kulis, S., Dustman, P., & Miller-Day, M. (2003). Culturally grounded substance use prevention: An evaluation of the keepin' it R.E.A.L. curriculum. *Prevention Science*, *4*(4), 233–248. <https://doi.org/10.1023/A:1026016131401>

- Heinrich, A., Müller, K. U., Banaschewski, T., Barker, G. J., Bokde, A. L. W., Bromberg, U., ... Nees, F. (2016). Prediction of alcohol drinking in adolescents: personality-traits, behavior, brain responses, and genetic variations in the context of reward sensitivity. *Biological Psychology, 118*, 79–87. <https://doi.org/10.1016/j.biopsycho.2016.05.002>
- Heo, M., & Leon, A. C. (2010). Sample sizes required to detect two-way and three-way interactions involving slope differences in mixed-effects linear models. *Journal of Biopharmaceutical Statistics, 20*(March 2015), 787–802. <https://doi.org/10.1080/10543401003618819>
- Himelstein, S., Saul, S., Garcia-Romeu, A., & Pinedo, D. (2014). Mindfulness Training as an Intervention for Substance User Incarcerated Adolescents: A Pilot Grounded Theory Study. *Substance Use & Misuse, 49*(5), 560–570. <https://doi.org/10.3109/10826084.2013.852580>
- Hoffmann, N. G., & Kopak, A. M. (2015). How well do the DSM-5 alcohol use disorder designations map to the ICD-10 disorders? *Alcoholism: Clinical and Experimental Research, 39*(4), 697–701. <https://doi.org/10.1111/acer.12685>
- Hölzel, B. K., Carmody, J., Vangel, M., Congleton, C., Yerramsetti, S. M., Gard, T., & Lazar, S. W. (2011). Mindfulness practice leads to increases in regional brain gray matter density. *Psychiatry Research, 191*(1), 36–43. <https://doi.org/10.1016/j.psychres.2010.08.006>. Mindfulness
- Hopwood, C. J., & Donnellan, M. B. (2010). How should the internal structure of personality inventories be evaluated? *Personality and Social Psychology Review, 14*(3), 332–46. <https://doi.org/10.1177/1088868310361240>
- Jaworska, N., & MacQueen, G. (2015). Adolescence as a unique developmental period. *Journal of Psychiatry & Neuroscience, 40*(5), 291–293. <https://doi.org/10.1503/jpn.150268>
- Jayne, M., & Valentine, G. (2015). “It makes you go crazy”: Children’s knowledge and experience of alcohol consumption. *Journal of Consumer Culture*, (forthcoming). <https://doi.org/10.1177/1469540515574450>
- Jennings, W. G., Piquero, A. R., Rocque, M., & Farrington, D. P. (2015). The effects of binge and problem drinking on problem behavior and adjustment over the life course: Findings from the Cambridge Study in Delinquent Development. *Journal of Criminal Justice, 43*(6), 453–463. <https://doi.org/10.1016/j.jcrimjus.2015.09.002>
- Jester, J. M., Steinberg, D. B., Heitzeg, M. M., & Zucker, R. A. (2015). Coping expectancies, not enhancement expectancies, mediate trauma experience effects on problem alcohol use: A prospective study from early childhood to adolescence. *Journal of Studies on Alcohol and Drugs, 76*(1988), 781–789. <https://doi.org/http://dx.doi.org/10.15288/jsad.2015.76.781>
- Jester, J. M., Wong, M. M., Cranford, J. A., Buu, A., Fitzgerald, H. E., & Zucker, R. a. (2015). Alcohol expectancies in childhood: Change with the onset of drinking and ability to predict adolescent drunkenness and binge drinking. *Addiction, 110*, 71–79. <https://doi.org/10.1111/add.12704>
- Johnson, C., Burke, C., Brinkman, S., & Wade, T. (2017). A randomized controlled evaluation of a secondary school mindfulness program for early adolescents: Do we have the recipe right yet? *Behaviour Research and Therapy, 99*, 37–46. <https://doi.org/10.1016/j.brat.2017.09.001>
- Jones, B. T., & McMahon, J. (1993). The reliability of the Negative Alcohol Expectancy

- Questionnaire and its use. *Journal of the Association of Nurses in Substance Abuse*, 12, 15–16.
- Jones, B. T., & McMahon, J. (1994). Negative alcohol expectancy predicts post-treatment abstinence survivorship: The whether, when and why of relapse to a first drink. *Addiction*, 89, 1653–1665. <https://doi.org/10.1111/j.1360-0443.1994.tb03766.x>
- Jones, B. T., Will, C., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction*, 96(1), 57–72. <https://doi.org/10.1046/j.1360-0443.2001.961575.x>
- Kabat-Zinn, J. (1990). *Full catastrophe living: using the wisdom of your body and mind to face stress, pain and illness*. New York: Delacorte.
- Kabbani, R., & Kambouropoulos, N. (2013). Positive expectancies and perceived impaired control mediate the influence of reward drive and rash impulsiveness on alcohol use. *Personality and Individual Differences*, 54(2), 294–297. <https://doi.org/10.1016/j.paid.2012.08.008>
- Kadden, R. M., & Litt, M. D. (2011). The role of self-efficacy in the treatment of substance use disorders. *Addictive Behaviors*, 36(12), 1120–1126. <https://doi.org/10.1016/j.addbeh.2011.07.032>
- Kelly, A. B., Evans-Whipp, T. J., Smith, R., Chan, G. C. K., Toumbourou, J. W., Patton, G. C., ... Catalano, R. F. (2015). A longitudinal study of the association of adolescent polydrug use, alcohol use and high school non-completion. *Addiction*, 110, 627–635. <https://doi.org/10.1111/add.12829>
- Kelly Yvonne, Goisis Alice, Sacker Amanda, Cable Noriko, Watt Richard G., & Britton Annie. (2015). What influences 11-year-olds to drink? Findings from the Millennium Cohort Study. *BMC Public Health*, 16, 169. <https://doi.org/10.1186/s12889-016-2847-x>
- Khan, A., Lewis, C., & Lindenmayer, J.-P. (2011). Use of NON-PARAMETRIC Item Response Theory to develop a shortened version of the Positive and Negative Syndrome Scale (PANSS). *BMC Psychiatry*, 11(1), 178. <https://doi.org/10.1186/1471-244X-11-178>
- Khoury, B., Lecomte, T., Fortin, G., Masse, M., Therien, P., Bouchard, V., ... Hofmann, S. G. (2013). Mindfulness-based therapy: A comprehensive meta-analysis. *Clinical Psychology Review*, 33(6), 763–771. <https://doi.org/10.1016/j.cpr.2013.05.005>
- Kim, K. H. (2005). The relation among fit indexes, power, and sample size in structural equation modeling. *Structural Equation Modeling*, 12(3), 368–390. <https://doi.org/10.1207/s15328007sem1203>
- Knight, R. G., & Godfrey, H. P. D. (1993). The role of alcohol-related expectancies in the prediction of drinking behaviour in a simulated social interaction. *Addiction*, 88(8), 1111–1118. <https://doi.org/10.1111/j.1360-0443.1993.tb02130.x>
- Koob, G. F., & Bloom, F. E. (1988). Cellular and molecular mechanisms of drug dependence. *Science*, 242(4879), 715–723. Retrieved from <http://www.jstor.org/stable/1702139>
- Koob, G. F., Roberts, A. J., Schulteis, G., Parsons, L. H., Heyser, C. J., Hyytiä, P., ... Weiss, F. (1998). Neurocircuitry targets in ethanol reward and dependence. *Alcoholism, Clinical and Experimental Research*, 22(1), 3–9. <https://doi.org/00000374-199802000-00001> [pii]
- Kuntsche, E. (2017). “Do grown-ups become happy when they drink?” Alcohol expectancies among preschoolers. *Experimental and Clinical Psychopharmacology*, 25(1), 24–30.

- Kuntsche, E., Knibbe, R., Engels, R., & Gmel, G. (2007). Drinking motives as mediators of the link between alcohol expectancies and alcohol use among adolescents. *Journal of Studies on Alcohol and Drugs*, *68*(1), 76–85. <https://doi.org/10.15288/jsad.2007.68.76>
- Kuntsche, E., Knibbe, R., Gmel, G., & Engels, R. (2005). Why do young people drink? A review of drinking motives. *Clinical Psychology Review*, *25*, 841–861. <https://doi.org/10.1016/j.cpr.2005.06.002>
- Lammers, J., Goossens, F., Conrod, P., Engels, R., Wiers, R. W., & Kleinjan, M. (2017). Effectiveness of a selective alcohol prevention program targeting personality risk factors: Results of interaction analyses. *Addictive Behaviors*, *71*, 82–88. <https://doi.org/10.1016/j.addbeh.2017.02.030>
- Lancaster, S. L., Klein, K. P., & Knightly, W. (2016). Mindfulness and relaxation: A comparison of brief, laboratory-based interventions. *Mindfulness*, *7*(3), 614–621. <https://doi.org/10.1007/s12671-016-0496-x>
- Laslett, A.-M., Wilkinson, C., Room, R., Livingston, M., Ferris, J., & Mugavin, J. (2014). Alcohol's harm to others: An overview of Australian work and results so far. *Australian Epidemiologist*, *21*(2), 10–11.
- Lattimore, P., Fisher, N., & Malinowski, P. (2011). A cross-sectional investigation of trait disinhibition and its association with mindfulness and impulsivity. *Appetite*, *56*(2), 241–248. <https://doi.org/10.1016/j.appet.2010.12.007>
- Leamy, T. E., Connor, J. P., Voisey, J., Young, R. M., & Gullo, M. J. (2016). Alcohol misuse in emerging adulthood: Association of dopamine and serotonin receptor genes with impulsivity-related cognition. *Addictive Behaviors*, *63*, 29–36. <https://doi.org/10.1016/j.addbeh.2016.05.008>
- Lee, N. K., Greely, J., & Oei, T. P. S. (1999). The relationship of positive and negative alcohol expectancies to patterns of consumption of alcohol in social drinkers. *Addictive Behaviors*, *24*(3), 359–369. [https://doi.org/10.1016/S0306-4603\(98\)00091-4](https://doi.org/10.1016/S0306-4603(98)00091-4)
- Lee, N. K., & Oei, T. P. S. (1993). The importance of alcohol expectancies and drinking refusal self-efficacy in the quantity and frequency of alcohol consumption. *Journal of Substance Abuse*, *5*(4), 379–390. [https://doi.org/10.1016/0899-3289\(93\)90006-W](https://doi.org/10.1016/0899-3289(93)90006-W)
- Leigh, B. C. (1987). Beliefs about the effects of alcohol on self and others. *Journal of Studies on Alcohol*, *48*(5), 467–475. <https://doi.org/10.15288/jsa.1987.48.467>
- Leigh, B. C. (1989). In search of the Seven Dwarves: issues of measurement and meaning in alcohol expectancy research. *Psychological Bulletin*, *105*(3), 361–373. <https://doi.org/10.1037/0033-2909.105.3.361>
- Leigh, B. C., & Stacy, A. W. (1993). Alcohol outcome expectancies: Scale construction and predictive utility in higher order confirmatory models. *Psychological Assessment*, *5*(2), 216–229. <https://doi.org/10.1037/1040-3590.5.2.216>
- Leigh, B. C., & Stacy, A. W. (2004). Alcohol expectancies and drinking in different age groups. *Addiction*, *99*(2), 215–227. <https://doi.org/10.1046/j.1360-0443.2004.00641.x>
- Lemstra, M., Bennett, N., Nannapaneni, U., Neudorf, C., Warren, L., Kershaw, T., & Scott, C. (2010). A systematic review of school-based marijuana and alcohol prevention programs targeting adolescents aged 10–15. *Addiction Research & Theory*, *18*(February), 84–96. <https://doi.org/10.3109/16066350802673224>
- Li, W., Howard, M. O., Garland, E. L., McGovern, P., & Lazar, M. (2017). Mindfulness treatment for substance misuse: A systematic review and meta-analysis. *Journal of*

- Substance Abuse Treatment*, 75, 62–96. <https://doi.org/10.1016/j.jsat.2017.01.008>
- Linehan, M. M. (1987). Dialectical Behavior Therapy for Borderline Personality Disorder: Theory and method. *Bulletin of the Menninger Clinic*, 51(3), 261–276.
- Linehan, M. M. (1993). *Cognitive Behavioural Therapy of Borderline Personality Disorder*. New York: Guilford Press.
- Lisdahl, K. M., Gilbert, E. R., Wright, N. E., & Shollenbarger, S. (2013). Dare to delay? The impacts of adolescent alcohol and marijuana use onset on cognition, brain structure, and function. *Frontiers in Psychiatry*, 4, 1–19. <https://doi.org/10.3389/fpsy.2013.00053>
- Little, R. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, 83(404), 1198–1202.
- Littlefield, A. K., Stevens, A. K., Ellingson, J. M., King, K. M., & Jackson, K. M. (2016). Changes in negative urgency, positive urgency, and sensation seeking across adolescence. *Personality and Individual Differences*, 90, 332–337. <https://doi.org/10.1016/j.paid.2015.11.024>
- Lopez-Vergara, H. I., Colder, C. R., Hawk Jr, L. W., Wiczorek, W. F., Eiden, R. D., Lengua, L. J., & Read, J. P. (2012). Reinforcement sensitivity theory and alcohol outcome expectancies in early adolescence. *American Journal of Drug and Alcohol Abuse*, 38(2), 130–134. <https://doi.org/10.3109/00952990.2011.643973>. Reinforcement
- Loree, A. M., Lundahl, L. H., & Ledgerwood, D. M. (2015). Impulsivity as a predictor of treatment outcome in substance use disorders: Review and synthesis. *Drug and Alcohol Review*, 34(2), 119–134. <https://doi.org/10.1111/dar.12132>
- Lovinger, D., White, G., & Weight, F. (1989). Ethanol inhibits NMDA-activated ion current in hippocampal neurons. *Science*, 243(4899), 1721–1724. <https://doi.org/10.1126/science.2467382>
- Luciana, M., Collins, P. F., Muetzel, R. L., & Lim, K. O. (2014). Effects of alcohol use initiation on brain structure in typically developing adolescents. *Am J Drug Alcohol Abuse*, 39(6), 612–626. <https://doi.org/10.3109/00952990.2013.837057>. Effects
- MacLaren, V. V., Fugelsang, J. A., Harrigan, K. A., & Dixon, M. J. (2012). Effects of impulsivity, reinforcement sensitivity, and cognitive style on pathological gambling symptoms among frequent slot machine players. *Personality and Individual Differences*, 52(3), 390–394. <https://doi.org/10.1016/j.paid.2011.10.044>
- Magidson, J. F., Roberts, B. W., Collado-Rodriguez, A., & Lejuez, C. W. (2014). Theory-driven intervention for changing personality: Expectancy value theory, behavioral activation, and conscientiousness. *Developmental Psychology*, 50(5), 1442–1450. <https://doi.org/10.1037/a0030583>
- Magill, M., Kiluk, B. D., McCrady, B., Tonigan, J. S., & Longabaugh, R. (2015). Active ingredients of treatment and client mechanisms of change in behavioural treatments for Alcohol Use Disorders: Progress 10 years later. *Alcoholism: Clinical & Experimental Research*, 39(10), 1852–1862. <https://doi.org/10.1111/acer.12848>
- Magill, M., & Longabaugh, R. (2013). Efficacy combined with specified ingredients: A new direction for empirically supported addiction treatment. *Addiction*, 108(5), 874–881. <https://doi.org/10.1111/add.12013>
- Manning, M., Smith, C., & Mazerolle, P. (2013). The societal costs of alcohol misuse in Australia. *Trends and Issues in Crime and Criminal Justice*, (454), 1–6.
- Marsh, H. W., Hau, K.-T., & Wen, Z. (2004). In search of golden rules: Comment on

- hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) findings. *Structural Equation Modeling*, *11*(3), 320–341. https://doi.org/10.1207/s15328007sem1103_2
- Marsiglia, F. F., Kulis, S., Yabiku, S. T., Nieri, T. A., & Coleman, E. (2011). When to intervene: Elementary school, middle school or both? Effects of keepin' It REAL on substance use trajectories of Mexican heritage youth. *Prevention Science*, *12*(2), 48–62. <https://doi.org/10.1007/s11121-010-0189-y>
- McCambridge, J., McAlaney, J., & Rowe, R. (2011). Adult consequences of late adolescent alcohol consumption: A systematic review of cohort studies. *PLoS Medicine*, *8*(2). <https://doi.org/10.1371/journal.pmed.1000413>
- McCarthy, D. M., Kroll, L. S., & Smith, G. T. (2001). Integrating disinhibition and learning risk for alcohol use. *Experimental and Clinical Psychopharmacology*, *9*(4), 389–398. <https://doi.org/10.1037//1064-1297.9.4.389>
- McLellan, A. T., & Meyers, K. (2004). Contemporary addiction treatment: A review of systems problems for adults and adolescents. *Biological Psychiatry*, *56*(10), 764–770. <https://doi.org/10.1016/j.biopsych.2004.06.018>
- McMahon, J., & Jones, B. T. (1993). The negative alcohol expectancy questionnaire. *Journal of the Association of Nurses in Substance Abuse*, *12*, 17.
- McMurray, M. S., Amodeo, L. R., & Roitman, J. D. (2015). Consequences of adolescent ethanol consumption on risk preference and orbitofrontal cortex encoding of reward. *Neuropsychopharmacology*, (September), 1–10. <https://doi.org/10.1038/npp.2015.288>
- Meijer, R. R., & Baneke, J. J. (2004). Analyzing psychopathology items: A case for Nonparametric Item Response Theory modeling. *Psychological Methods*, *9*(3), 354–368. <https://doi.org/10.1037/1082-989X.9.3.354>
- Mewton, L., Slade, T., McBride, O., Grove, R., & Teesson, M. (2011). An evaluation of the proposed DSM-5 alcohol use disorder criteria using Australian national data. *Addiction*, *106*, 941–950. <https://doi.org/10.1111/j.1360-0443.2010.03340.x>
- Mihic, S. J., Ye, Q., Wick, M. J., Koltchine, V. V., Krasowski, M. D., Finn, S. E., ... Harrison, N. L. (1997). Sites of alcohol and volatile anaesthetic action on GABA(A) and glycine receptors. *Nature*, *389*(6649), 385–9. <https://doi.org/10.1038/38738>
- Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., ... Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences of the United States of America*, *108*(7), 2693–8. <https://doi.org/10.1073/pnas.1010076108>
- Mokken, R. J. (1971). *A theory and procedure of scale analysis: With applications in political research*. The Hague, The Netherlands: Mouton.
- Morawska, A., & Oei, T. P. S. (2005). Binge drinking in university students: A test of the cognitive model. *Addictive Behaviors*, *30*, 203–218. <https://doi.org/10.1016/j.addbeh.2004.05.011>
- Mrazek, M. D., Franklin, M. S., Phillips, D. T., Baird, B., & Schooler, J. W. (2013). Mindfulness training improves working memory capacity and GRE performance while reducing mind wandering. *Psychological Science*, *24*(5), 776–81. <https://doi.org/10.1177/0956797612459659>
- Murphy, C., & MacKillop, J. (2012). Living in the here and now: Interrelationships between impulsivity, mindfulness, and alcohol misuse. *Psychopharmacology*, *219*(2), 1–15.

<https://doi.org/10.1007/s00213-011-2573-0>.Living

- Muthén, B. O., & Curran, P. J. (1997). General longitudinal modeling of individual differences in experimental designs: A latent variable framework for analysis and power estimation. *Psychological Methods*, 2(4), 371–402. <https://doi.org/10.1037/1082-989X.2.4.371>
- National Health and Medical Research Council. (2009). *Australian Guidelines to Reduce Health Risks from Drinking Alcohol. Australian guidelines to reduce health risks from drinking alcohol*. <https://doi.org/10.1037/e509232012-001>
- Nestler, E. J. (2005). Is there a common molecular pathway for addiction? *Nature Neuroscience*, 8(11), 1445–9. <https://doi.org/10.1038/nn1578>
- Newton-Howes, G., & Boden, J. M. (2015). Relation between age of first drinking and mental health and alcohol and drug disorders in adulthood: Evidence from a 35 year cohort study. *Addiction*. <https://doi.org/10.1111/add.13230>
- Nguyen-Louie, T., Castro, N., Matt, G. E., Squeglia, L. M., Brumback, T., & Tapert, S. (2015). Effects of emerging alcohol and marijuana use behaviors on adolescents' neuropsychological functioning over four years. *Journal of Studies on Alcohol and Drugs*, 76(5), 738–748.
- Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., ... Zucker, R. A. (2006). Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45(4), 468–75. <https://doi.org/10.1097/01.chi.0000199028.76452.a9>
- Niv, S., Tuvblad, C., Raine, A., Wang, P., & Baker, L. A. (2012). Heritability and longitudinal stability of impulsivity in adolescence. *Behavior Genetics*, 42(3), 378–392. <https://doi.org/10.1007/s10519-011-9518-6>
- O'Donnell, A., Anderson, P., Newbury-Birch, D., Schulte, B., Schmidt, C., Reimer, J., & Kaner, E. (2014). The impact of brief alcohol interventions in primary healthcare: A systematic review of reviews. *Alcohol and Alcoholism*, 49(1), 66–78. <https://doi.org/10.1093/alcalc/agt170>
- O'Leary-Barrett, M., Castellanos-Ryan, N., Pihl, R. O., & Conrod, P. J. (2016). Mechanisms of personality-targeted intervention effects on adolescent alcohol misuse, internalizing and externalizing symptoms. *Journal of Consulting and Clinical Psychology*, 84(5), 438–452. <https://doi.org/10.1037/ccp0000082>
- O'Leary-Barrett, M., Topper, L., Al-Khudhairy, N., Pihl, R. O., Castellanos-Ryan, N., Mackie, C. J., & Conrod, P. J. (2013). Two-year impact of personality-targeted, teacher-delivered interventions on youth internalizing and externalizing problems: A cluster-randomized trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(9), 911–920. <https://doi.org/10.1016/j.jaac.2013.05.020>
- Oberle, E., Schonert-Reichl, K. A., Lawlor, M. S., & Thomson, K. C. (2012). Mindfulness and Inhibitory Control in Early Adolescence. *The Journal of Early Adolescence*, 32(4), 565–588. <https://doi.org/10.1177/0272431611403741>
- Oggers, C. L., Caspi, A., Nagin, D. S., Piquero, A. R., Slutske, W. S., Milne, B. J., ... Moffitt, T. E. (2008). Is it important to prevent early exposure to drugs and alcohol among adolescents? *Psychological Science*, 19(10), 1037–1044. <https://doi.org/10.1111/j.1467-9280.2008.02196.x>

- Oei, T. P. S., & Baldwin, A. R. (1993). Expectancy theory: A two-process model of alcohol use and abuse. *Journal of Studies on Alcohol and Drugs*, 55(5), 525–534. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7990462>
- Oei, T. P. S., & Burrow, T. (2000). Alcohol expectancy and drinking refusal self-efficacy: A test of specificity theory. *Addictive Behaviors*, 25(4), 499–507. [https://doi.org/10.1016/S0306-4603\(99\)00044-1](https://doi.org/10.1016/S0306-4603(99)00044-1)
- Oei, T. P. S., Hasking, P. A., & Young, R. M. (2005). Drinking refusal self-efficacy questionnaire-revised (DRSEQ-R): A new factor structure with confirmatory factor analysis. *Drug and Alcohol Dependence*, 78(3), 297–307. <https://doi.org/10.1016/j.drugalcdep.2004.11.010>
- Oei, T. P. S., & Morawska, A. (2004). A cognitive model of binge drinking: The influence of alcohol expectancies and drinking refusal self-efficacy. *Addictive Behaviors*, 29(1), 159–179. [https://doi.org/10.1016/S0306-4603\(03\)00076-5](https://doi.org/10.1016/S0306-4603(03)00076-5)
- Onrust, S. A., Otten, R., Lammers, J., & Smit, F. (2016). School-based programmes to reduce and prevent substance use in different age groups: What works for whom? Systematic review and meta-regression analysis. *Clinical Psychology Review*, 44, 45–59. <https://doi.org/10.1016/j.cpr.2015.11.002>
- Ostafin, B. D., Bauer, C., & Myxter, P. (2012). Mindfulness Decouples the Relation Between Automatic Alcohol Motivation and Heavy Drinking. *Journal of Social and Clinical Psychology*, 31(7), 729–745. <https://doi.org/10.1521/jscp.2012.31.7.729>
- Papies, E. K., Barsalou, L. W., & Custers, R. (2012). Mindful attention prevents mindless impulses. *Social Psychological and Personality Science*, 3(3), 291–299. <https://doi.org/10.1177/1948550611419031>
- Papinczak, Z. E., Connor, J. P., Feeney, G. F. X., Young, R. M., & Gullo, M. J. (2017). Treatment seeking in cannabis dependence: The role of social cognition. *Drug and Alcohol Dependence*, 170, 142–146. <https://doi.org/10.1016/j.drugalcdep.2016.11.005>
- Papinczak, Z. E., Connor, J. P., Harnett, P., & Gullo, M. J. (2018). A biosocial cognitive model of cannabis use in emerging adulthood. *Addictive Behaviors*, 76, 229–235. <https://doi.org/10.1016/j.addbeh.2017.08.011>
- Patton, K. A., Connor, J. P., Rundle-Thiele, S., Dietrich, T., Young, R. M., & Gullo, M. J. (2018a). Measuring adolescent drinking-refusal self-efficacy: Development and validation of the Drinking Refusal Self-Efficacy Questionnaire-Shortened Adolescent version (DRSEQ-SRA). *Addictive Behaviors*, 81, 70–77. <https://doi.org/10.1016/j.addbeh.2018.02.007>
- Patton, K. A., Connor, J. P., Rundle-Thiele, S., Dietrich, T., Young, R. M., & Gullo, M. J. (2018b). Validation of the Adolescent Drinking Expectancy Questionnaire and development of a short form. *Drug and Alcohol Review*, 37(3), 396–405. <https://doi.org/10.1111/dar.12567>
- Patton, K. A., Gullo, M. J., Connor, J. P., Chan, G. C. K., Kelly, A. B., Catalano, R. F., & Toumbourou, J. W. (2018). Social cognitive mediators of the relationship between impulsivity traits and adolescent alcohol use: Identifying unique targets for prevention. *Addictive Behaviors*, 84, 79–85. <https://doi.org/10.1016/j.addbeh.2018.03.031>
- Peeters, M., Oldehinkel, T., & Vollebergh, W. (2017). Behavioral Control and Reward Sensitivity in Adolescents' Risk Taking Behavior: A Longitudinal TRAILS Study. *Frontiers in Psychology*, 8(February), 1–11. <https://doi.org/10.3389/fpsyg.2017.00231>

- Peters, J. R., Erisman, S. M., Upton, B. T., Baer, R. a., & Roemer, L. (2011). A Preliminary Investigation of the Relationships Between Dispositional Mindfulness and Impulsivity. *Mindfulness*, 2, 228–235. <https://doi.org/10.1007/s12671-011-0065-2>
- Peters, L., Sunderland, M., Andrews, G., Rapee, R. M. R. M., & Mattick, R. P. R. P. (2012). Development of a short form Social Interaction Anxiety (SIAS) and Social Phobia Scale (SPS) using nonparametric item response theory: The SIAS-6 and the SPS-6. *Psychological Assessment*, 24(1), 66–76. <https://doi.org/10.1037/a0024544>
- Pfefferbaum, A., Rohl, T., Pohl, K. M., Lane, B., Chu, W., Kwon, D., ... Sullivan, E. V. (2015). Adolescent development of cortical and white matter structure in the NCANDA sample: Role of sex, ethnicity, puberty, and alcohol drinking. *Cerebral Cortex*, 1–21. <https://doi.org/10.1093/cercor/bhv205>
- Quinn, P. D., & Harden, K. P. (2013). Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. *Development and Psychopathology*, 25(1), 223–39. <https://doi.org/10.1017/S0954579412000284>
- R: A language and environment for statistical computing. (2014). Vienna, Austria. Retrieved from <http://www.r-project.org/>.
- Raes, F., Griffith, J. W., Gucht, K. Van Der, & Williams, J. M. G. (2014). School-based prevention and reduction of depression in adolescents: A cluster-randomized controlled trial of a mindfulness group program. *Mindfulness*, 5, 477–486. <https://doi.org/10.1007/s12671-013-0202-1>.
- Ramsay, J. O. (2000). TestGraf. A program for the graphical analysis of multiple-choice tests and questionnaire data [Computer software and manual]. Retrieved from <http://www.psych.mcgill.ca/misc/fda/downloads/testgraf/>
- Regier, D. A., Kuhl, E. A., & Kupfer, D. J. (2013). The DSM-5: Classification and criteria changes. *World Psychiatry*, 12(2), 92–98. <https://doi.org/10.1002/wps.20050>
- Robert, G. H., & Schumann, G. (2017). Reinforcement related behaviors and adolescent alcohol abuse: from localized brain structures to coordinated networks. *Current Opinion in Behavioral Sciences*, 13, 106–116. <https://doi.org/10.1016/j.cobeha.2016.11.008>
- Robinson, J. M., Ladd, B. O., & Anderson, K. G. (2014). When you see it, let it be: Urgency, mindfulness and adolescent substance use. *Addictive Behaviors*, 39(6), 1038–1041. <https://doi.org/10.1016/j.addbeh.2014.02.011>
- Romer, D., & Hennessy, M. (2007). A biosocial-affect model of adolescent sensation seeking: The role of affect evaluation and peer-group influence in adolescent drug use. *Prevention Science*, 8(2), 89–101. <https://doi.org/10.1007/s11121-007-0064-7>
- Rose, R. J., Winter, T., Viken, R. J., & Kaprio, J. (2014). Adolescent alcohol abuse and adverse adult outcomes: Evaluating confounds with drinking-discordant twins. *Alcoholism: Clinical and Experimental Research*, 38(8), 2314–2321. <https://doi.org/10.1111/acer.12491>
- Rosseel, Y. (2012). lavaan: An R Package for Structural Equation Modeling. *Journal of Statistical Software*, 48(2), 1–36.
- Rossow, I., & Kuntsche, E. (2013). Early onset of drinking and risk of heavy drinking in young adulthood-A 13-year prospective study. *Alcoholism, Clinical and Experimental Research*, 37 Suppl 1(January), E297-304. <https://doi.org/10.1111/j.1530-0277.2012.01924.x>

- Rundle-Thiele, S., Russell-Bennett, R., Leo, C., & Dietrich, T. (2013). Moderating teen drinking: Combining social marketing and education. *Health Education, 113*(5), 392–406. <https://doi.org/10.1108/HE-07-2012-0041>
- Rundle-Thiele, S., Schuster, L., Dietrich, T., Russell-Bennett, R., Drennan, J., Leo, C., & Connor, J. P. (2015). Maintaining or changing a drinking behavior? GOKA's short-term outcomes. *Journal of Business Research, 68*(10), 2155–2163. <https://doi.org/10.1016/j.jbusres.2015.03.015>
- Sargent, J. D., Tanski, S. E., Stoolmiller, M., & Hanewinkel, R. (2010). Using sensation seeking to target adolescents for substance use interventions. *Addiction, 105*(3), 506–514. <https://doi.org/10.1111/j.1360-0443.2009.02782.x>
- Satorra, A., & Bentler, P. M. (1994). Corrections to test statistics and standard errors in covariance structure analysis. In A. von Eye & C. C. Clogg (Eds.), *Latent variables analysis: Applications for developmental research* (pp. 399–419). Thousand Oaks, CA: SAGE Publications Ltd.
- Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika, 66*(4), 507–514. <https://doi.org/10.1007/BF02296192>
- Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993a). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. *Addiction, 88*(6), 791–804. <https://doi.org/10.1111/j.1360-0443.1993.tb02093.x>
- Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993b). Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption-II. *Addiction, 88*(6), 791–804. <https://doi.org/10.1111/j.1360-0443.1993.tb02093.x>
- Schafer, J. L. (1999). Multiple Imputation: A Primer. *Statistical Methods in Medical Research, 8*(1), 3–15.
- Schafer, J., & Leigh, B. C. (1996). A comparison of factor structures of adolescent and adult alcohol effect expectancies. *Addictive Behaviors, 21*(3), 403–408. [https://doi.org/10.1016/0306-4603\(95\)00045-3](https://doi.org/10.1016/0306-4603(95)00045-3)
- Schnell, R., Bachteler, T., & Reiher, J. (2010). Improving the use of self-generated identification codes. *Evaluation Review, 34*(5), 391–418. <https://doi.org/10.1177/0193841X10387576>
- Scott-Sheldon, L. A. J., Terry, D. L., Carey, K. B., Garey, L., & Carey, M. P. (2012). Efficacy of expectancy challenge interventions to reduce college student drinking: A meta-analytic review. *Psychology of Addictive Behaviors, 26*(3), 393–405. <https://doi.org/10.1037/a0027565>
- Sedlmeier, P., Eberth, J., Schwarz, M., Zimmermann, D., Haarig, F., Jaeger, S., & Kunze, S. (2012). The psychological effects of meditation: A meta-analysis. *Psychological Bulletin, 138*(6), 1139–1171. <https://doi.org/10.1037/a0028168>
- Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness based cognitive therapy for depression—A new approach to preventing relapse*. New York: Guilford Press.
- Semple, R. J., & Burke, C. A. (2012). Mindfulness-Based Treatment for Children and Adolescents. In P. C. Kendall (Ed.), *Child and adolescent therapy: Cognitive-behavioral procedures* (4th ed., pp. 411–426). New York: Guilford Press.

- Settles, R. F., Cyders, M. A., & Smith, G. T. (2010). Longitudinal validation of the Acquired Preparedness Model of drinking risk. *Psychology of Addictive Behaviors, 24*(2), 198–208. <https://doi.org/10.1037/a0017631>
- Sheng, Y., & Sheng, Z. (2012). Is coefficient alpha robust to non-normal data? *Frontiers in Psychology, 3*(FEB), 1–13. <https://doi.org/10.3389/fpsyg.2012.00034>
- Shnitko, T. A., Spear, L. P., & Robinson, D. L. (2015). Adolescent binge-like alcohol alters sensitivity to acute alcohol effects on dopamine release in the nucleus accumbens of adult rats. *Psychopharmacology, 233*(3), 361–371. <https://doi.org/10.1007/s00213-015-4106-8>
- Sijtsma, K., & Molenaar, I. W. (2002). *Introduction to Nonparametric Item Response Theory*. Thousand Oaks, CA: Sage.
- Smith, G. T., & Anderson, K. G. (2001). Adolescent risk for alcohol problems as acquired preparedness: A model and suggestions for intervention. In P. M. Monti, S. M. Colby, & T. A. O’Leary (Eds.), *Adolescents, alcohol, and substance abuse: Reaching teens through brief interventions* (pp. 109–144). New York: Guilford.
- Smith, G. T., Goldman, M. S., Greenbaum, P. E., & Christiansen, B. A. (1995). Expectancy for social facilitation from drinking: the divergent paths of high-expectancy and low-expectancy adolescents. *Journal of Abnormal Psychology, 104*(1), 32–40. <https://doi.org/10.1037/0021-843X.104.1.32>
- Spear, L. P. (2014). Adolescents and alcohol: Acute sensitivities, enhanced intake, and later consequences. *Neurotoxicology and Teratology, 41*, 51–59. <https://doi.org/10.1016/j.ntt.2013.11.006>
- Squeglia, L. M., Boissoneault, J., Van Skike, C. E., Nixon, S. J., & Matthews, D. B. (2014). Age-related effects of alcohol from adolescent, adult, and aged populations using human and animal models. *Alcoholism: Clinical and Experimental Research, 38*(10), 2509–2516. <https://doi.org/10.1111/acer.12531>
- Squeglia, L. M., Jacobus, J., Nguyen-Louie, T. T., & Tapert, S. F. (2014). Inhibition during early adolescence predicts alcohol and marijuana use by late adolescence. *Neuropsychology, 28*(5), 782–790. <https://doi.org/10.1037/neu0000083>
- Squeglia, L. M., Rinker, D., Bartsch, H., Castro, N., Chung, Y., Dale, A. M., ... Tapert, S. F. (2014). Brain volume reductions in adolescent heavy drinkers. *Developmental Cognitive Neuroscience, 9*, 117–125. <https://doi.org/10.1016/j.dcn.2014.02.005>
- Squeglia, L. M., Tapert, S. F., Sullivan, E. V., Jacobus, J., Meloy, M. J., Rohlfing, T., & Pfefferbaum, A. (2015). Brain development in heavy-drinking adolescents. *American Journal of Psychiatry, 172*(6), 531–542. <https://doi.org/10.1176/appi.ajp.2015.14101249>
- Stacy, A. W., & Wiers, R. W. (2010). Implicit cognition and addiction: A tool for explaining paradoxical behaviour. *Annual Review of Clinical Psychology, 6*, 551–575. <https://doi.org/10.1146/annurev.clinpsy.121208.131444>
- Stautz, K., & Cooper, A. (2013). Impulsivity-related personality traits and adolescent alcohol use: A meta-analytic review. *Clinical Psychology Review, 33*(4), 574–92. <https://doi.org/10.1016/j.cpr.2013.03.003>
- Stautz, K., Dinc, L., & Cooper, A. J. (2017). Combining trait models of impulsivity to improve explanation of substance use behaviour. *European Journal of Personality, 31*(January), 118–132. <https://doi.org/10.1002/per.2091>
- Stein, L. A. R., Katz, B., Colby, S. M., Barnett, N. P., Golembeske, C., Lebeau-Craven, R., &

- Monti, P. M. (2007). Validity and reliability of the Alcohol Expectancy Questionnaire-Adolescent, brief. *Journal of Child & Adolescent Substance Abuse, 16*(2), 115–127. https://doi.org/10.1300/J029v16n02_06
- Steinberg, L., Icenogle, G., Shulman, E. P., Breiner, K., Chein, J., Bacchini, D., ... Takash, H. M. S. (2017). Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation. *Developmental Science, (October 2016)*, e12532. <https://doi.org/10.1111/desc.12532>
- Steinberg, L., Sharp, C., Stanford, M. S., & Tharp, A. T. (2013). New tricks for an old measure: the development of the Barratt Impulsiveness Scale-Brief (BIS-Brief). *Psychological Assessment, 25*(1), 216–26. <https://doi.org/10.1037/a0030550>
- Stockings, E., Hall, W. D., Lynskey, M., Morley, K. I., Reavley, N., Strang, J., ... Degenhardt, L. (2016). Prevention, early intervention, harm reduction, and treatment of substance use in young people. *The Lancet Psychiatry, 3*, 280–296. [https://doi.org/10.1016/S2215-0366\(16\)00002-X](https://doi.org/10.1016/S2215-0366(16)00002-X)
- Swagell, C. D., Lawford, B. R., Hughes, I. P., Voisey, J., Feeney, G. F. X., Daal, A. Van, ... Young, R. M. (2012). DRD2 C957T and Taq IA Genotyping Reveals Gender Effects and Unique Low-Risk and High-Risk Genotypes in Alcohol Dependence. *Alcohol and Alcoholism, 47*(4), 397–403. <https://doi.org/10.1093/alcalc/ags047>
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using Multivariate Statistics* (5th ed.). New York: Allyn and Bacon.
- Tang, Y., Lu, Q., Fan, M., Yang, Y., & Posner, M. I. (2012). Mechanisms of white matter changes induced by meditation. *Proceedings of the National Academy of Sciences, 109*(26), 10570–10574. <https://doi.org/10.1073/pnas.1207817109>
- Tang, Y., Ma, Y., Wang, J., Fan, Y., Feng, S., Lu, Q., ... Posner, M. I. (2007). Short-term meditation training improves attention and self-regulation. *Proceedings of the National Academy of Sciences, 104*(43), 17152. <https://doi.org/10.1073/pnas.0707678104>
- Tarter, R. E., Kirisci, L., Mezzich, A., Cornelius, J. R., Pajer, K., Vanyukov, M., ... Clark, D. (2003). Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. *American Journal of Psychiatry, 160*(6), 1078–1085. <https://doi.org/10.1176/appi.ajp.160.6.1078>
- Teesson, M., Newton, N. C., Slade, T., Carragher, N., Barrett, E. L., Champion, K. E., ... Conrod, P. J. (2017). Combined universal and selective prevention for adolescent alcohol use: A cluster randomized controlled trial. *Psychological Medicine, 47*(10), 1761–1770. <https://doi.org/10.1017/S0033291717000198>
- Teper, R., Segal, Z. V., & Inzlicht, M. I. (2013). Inside the mindful mind: How mindfulness enhances emotion regulation through improvements in executive control. *Current Directions in Psychological Science, 22*(6), 449–454. <https://doi.org/10.1177/0963721413495869>
- Thush, C., Wiers, R. W., Ames, S. L., Grenard, J. L., Sussman, S., & Stacy, A. W. (2008). Interactions between implicit and explicit cognition and working memory capacity in the prediction of alcohol use in at-risk adolescents. *Drug Alcohol Dependence, 94*(1–3), 116–124. <https://doi.org/10.1016/j.drugalcdep.2007.10.019>. Interactions
- Tomlinson, K. L., & Brown, S. A. (2012). Self-medication or social learning? A comparison of models to predict early adolescent drinking. *Addictive Behaviors, 37*(2), 179–186. <https://doi.org/10.1016/j.addbeh.2011.09.016>

- Trantham-Davidson, H., Centanni, S. W., Garr, S. C., New, N. N., Mulholland, P. J., Gass, J. T., ... Chandler, L. J. (2016). Binge-like alcohol exposure during adolescence disrupts dopaminergic neurotransmission in the adult prelimbic cortex. *Neuropsychopharmacology, preview*, 1–33. <https://doi.org/10.1038/npp.2016.190>
- Tripodi, S. J., Bender, K., Litschge, C., & Vaughn, M. G. (2010). Interventions for Reducing Adolescent Alcohol Abuse. *Archives of Pediatrics & Adolescent Medicine*, 164(1), 85–91. <https://doi.org/10.1001/archpediatrics.2009.235>
- van der Ark, L. A. (2007). Mokken Scale Analysis in R. *Journal of Statistical Software*, 20(11), 183–208. <https://doi.org/10.1007/s11336-007-9034-z>
- van der Ark, L. A. (2012). New Developments in Mokken Scale Analysis in R. *Journal of Statistical Software*, 48(5), 1–27. <https://doi.org/10.18637/jss.v048.i05>
- Warner, L. A., & White, H. R. (2003). Longitudinal effects of age at onset and first drinking situations on problem drinking. *Substance Use & Misuse*, 38(14), 1983–2016. <https://doi.org/10.1081/JA-120025123>
- Whiteford, H. A., Degenhardt, L., Rehm, J., Baxter, A. J., Ferrari, A. J., Erskine, H. E., ... Vos, T. (2013). Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *The Lancet*, 382(13), 1575–1586. [https://doi.org/10.1016/S0140-6736\(13\)61611-6](https://doi.org/10.1016/S0140-6736(13)61611-6)
- Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, 30(4), 669–689. [https://doi.org/10.1016/S0191-8869\(00\)00064-7](https://doi.org/10.1016/S0191-8869(00)00064-7)
- Wiers, R. W., Ames, S. L., Hofmann, W., Krank, M., & Stacy, A. W. (2010). Impulsivity, impulsive and reflective processes and the development of alcohol use and misuse in adolescents and young adults. *Frontiers in Psychology*, 1, 1–12. <https://doi.org/10.3389/fpsyg.2010.00144>
- Witkiewitz, K., Lustyk, M. K. B., & Bowen, S. (2013). Retraining the addicted brain: A review of hypothesized neurobiological mechanisms of mindfulness-based relapse prevention. *Psychology of Addictive Behaviors*, 27(2), 351–365. <https://doi.org/10.1037/a0029258>
- Wood, A. P., Dawe, S., & Gullo, M. J. (2013). The role of personality, family influences, and prosocial risk-taking behavior on substance use in early adolescence. *Journal of Adolescence*, 36(5), 871–881. <https://doi.org/10.1016/j.adolescence.2013.07.003>
- World Health Organization. (1992). *International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10)* (10th ed.). Geneva: WHO.
- World Health Organization. (2014). *Global status report on alcohol and health*. Geneva, Switzerland. Retrieved from http://www.who.int/substance_abuse/publications/global_alcohol_report/en/
- Yoshimoto, K., McBride, W. J., Lumeng, L., & Li, T. K. (1992). Alcohol stimulates the release of dopamine and serotonin in the nucleus accumbens. *Alcohol*, 9(1), 17–22. [https://doi.org/10.1016/0741-8329\(92\)90004-T](https://doi.org/10.1016/0741-8329(92)90004-T)
- Young-Wolff, K. C., Wang, P., Tuvblad, C., Baker, L. A., Raine, A., & Prescott, C. A. (2015). Drinking experience uncovers genetic influences on alcohol expectancies across adolescence. *Addiction*, 110, 610–618. <https://doi.org/10.1111/add.12845>
- Young, R. M., Connor, J. P., & Feeney, G. F. X. (2011). Alcohol expectancy changes over a 12-week cognitive-behavioral therapy program are predictive of treatment success.

- Journal of Substance Abuse Treatment*, 40(1), 18–25.
<https://doi.org/10.1016/j.jsat.2010.08.003>
- Young, R. M., Connor, J. P., Ricciardelli, L. A., & Saunders, J. B. (2006). The role of alcohol expectancy and drinking refusal self-efficacy beliefs in university student drinking. *Alcohol and Alcoholism*, 41(1), 70–75. <https://doi.org/10.1093/alcalc/agh237>
- Young, R. M., Gullo, M. J., Feeney, G. F. X., & Connor, J. P. (2012). Development and validation of the cannabis refusal self-efficacy questionnaire (CRSEQ) in adult cannabis users in treatment. *Drug and Alcohol Dependence*, 125(3), 244–51.
<https://doi.org/10.1016/j.drugalcdep.2012.02.018>
- Young, R. M., Hasking, P. A., Oei, T. P. S., & Loveday, W. (2007). Validation of the drinking refusal self-efficacy questionnaire-revised in an adolescent sample (DRSEQ-RA). *Addictive Behaviors*, 32(4), 862–8. <https://doi.org/10.1016/j.addbeh.2006.07.001>
- Young, R. M., & Knight, R. G. (1989). The Drinking Expectancy Questionnaire: A revised measure of alcohol-related beliefs. *Journal of Psychopathology and Behavioral Assessment*, 11(1), 99–112. <https://doi.org/10.1007/BF00962702>
- Young, R. M., & Oei, T. P. S. (1993). Grape expectations: The role of alcohol expectancies in the understanding and treatment of problem drinking. *International Journal of Psychology*, 28(3), 337–364. <https://doi.org/10.1080/00207599308246926>
- Young, R. M., & Oei, T. P. S. (1996). *Drinking expectancy profile (DEP) test manual*. Brisbane, Australia: Behaviour Research and Therapy Centre, University of Queensland.
- Young, R. M., Oei, T. P. S., & Crook, G. M. (1991). Development of a drinking self-efficacy questionnaire. *Journal of Psychopathology and Behavioral Assessment*, 13(L), 1–15.
<https://doi.org/10.1007/BF00960735>
- Zgierska, A., Rabago, D., Chawla, N., Kushner, K., Koehler, R., & Marlatt, A. (2009). Mindfulness meditation for substance use disorders: A systematic review. *Substance Abuse*, 30(4), 266–294. <https://doi.org/10.1080/08897070903250019>
- Zoogman, S., Goldberg, S. B., Hoyt, W. T., & Miller, L. (2015). Mindfulness Interventions with Youth: A Meta-Analysis. *Mindfulness*, 6(2), 290–302.
<https://doi.org/10.1007/s12671-013-0260-4>



THE UNIVERSITY OF QUEENSLAND
Institutional Human Research Ethics Approval

Project Title: Advancing Prevention Science: Alcohol Online Role-Play Simulation Curriculum

Chief Investigator: A/Prof Jason Connor, Prof Sharyn Rundle-Thiele

Supervisor: None

Co-Investigator(s): Dr Matt Gullo, Ms Kiri Patton, Prof Judy Drennan, Prof Rebekah Russell-Bennett, Dr Wei Wei Cheryl Leo, Dr Lisa Schuster, Mr Timo Dietrich, Ms Victoria Aldred, Mr John Percy

School(s): UQ Centre for Youth Substance Abuse Research; Social Marketing @ Griffith, Griffith University; QUT; Murdoch University; QLD Catholic Education Commission

Approval Number: 2014001623

Granting Agency/Degree: ARC Linkage Project Grant

Duration: 31st December 2017

Comments/Conditions:

Expedited review on the basis of approval from Griffith University HREC dated 25/02/2014

Note: if this approval is for amendments to an already approved protocol for which a UQ Clinical Trials Protection/Insurance Form was originally submitted, then the researchers must directly notify the UQ Insurance Office of any changes to that Form and Participant Information Sheets & Consent Forms as a result of the amendments, before action.

Name of responsible Committee:

Behavioural & Social Sciences Ethical Review Committee

This project complies with the provisions contained in the *National Statement on Ethical Conduct in Human Research* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:

Associate Professor John McLean

Chairperson

Behavioural & Social Sciences Ethical Review Committee

Signature

Date

1/12/2014



THE UNIVERSITY OF QUEENSLAND
Institutional Human Research Ethics Approval

Project Title: Cognitive-Behavioural Mediators and Moderators of the Relationship Between Impulsivity Traits and Adolescent Alcohol Use: Identifying Unique Targets for Prevention

Chief Investigator: Ms Kiri Patton

Supervisor: Dr Matthew Gullo

Co-Investigator(s): Matthew Gullo, Jason Connor, Gary Chan, Adrian Kelly, John Toumbourou, Richard F. Catalano

School(s): CYSAR

Approval Number: 2015000294

Granting Agency/Degree: Masters of Clinical Psychology/PhD

Duration: 31st December 2015

Comments/Conditions:

Expedited Review - Low Risk

Note: If this approval is for amendments to an already approved protocol for which a UQ Clinical Trials Protection/Insurance Form was originally submitted, then the researchers must directly notify the UQ Insurance Office of any changes to that Form and Participant Information Sheets & Consent Forms as a result of the amendments, before action.

Name of responsible Committee:

Behavioural & Social Sciences Ethical Review Committee

This project complies with the provisions contained in the *National Statement on Ethical Conduct in Human Research* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:

Associate Professor John McLean

Chairperson

Behavioural & Social Sciences Ethical Review Committee

Signature

Date

3/3/2015



THE UNIVERSITY OF QUEENSLAND
Institutional Human Research Ethics Approval

Project Title: A Cluster Randomised Controlled Trial of Brief Mindfulness-Enhanced Wellbeing Intervention for Adolescents

Chief Investigator: Ms Kiri Patton

Supervisor: Dr Matthew Gullo, A/Prof Jason Connor, Dr Jeanie Sheffield

Co-Investigator(s): None

School(s): Centre for Youth Substance Abuse Research; School of Psychology

Approval Number: 2015000875

Granting Agency/Degree: Masters of Clinical Psychology/PhD

Duration: 30th June 2017

Comments/Conditions:

Note: This approval is for amendments to an already approved protocol for which a UQ Clinical Trial Protection/Insurance Form was originally submitted. When the researcher must directly notify the UQ Insurance Office of any changes to that form and Participant Information Sheets & Consent Forms as a result of the amendments, before action.

Name of responsible Committee:

Behavioural & Social Sciences Ethical Review Committee

This project complies with the provisions contained in the *National Statement on Ethical Conduct in Human Research* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:

Associate Professor John McLean

Chairperson

Behavioural & Social Sciences Ethical Review Committee

Signature

Date

20/10/2015



Catholic Education Archdiocese of Brisbane

teaching • challenging • transforming

A11.096 id:cf ref:196

7 December 2015

Ms Kiri Patton
School of Psychology
McElwain Building
University of Queensland
St Lucia Qld 4072

Email kiri.patton@uqconnect.edu.au

Dear Ms Patton

The Brisbane Catholic Education Research Committee has met and considered your application to conduct the research, titled “A *cluster randomised controlled trial of brief mindfulness-enhanced wellbeing intervention for adolescents.*” Approval was granted by the committee for this research to be conducted.

Please note that participation in your project is at the discretion of the principal.

A condition of receiving this approval is that the following steps must be completed prior to working in the school.

1. Applicant to meet personally with each principal or their nominee and seek their approval to proceed.
2. Present this letter to the principal or their nominee.
3. Every researcher involved in the study, working in schools, must undertake a volunteer induction program for each school.
4. Every researcher involved in the study, working in schools, must be briefed by the principal or their nominee regarding:
 - a. Reporting procedures and requirement to be undertaken if a researcher, witnesses a situation where they believe a child experiences or is at risk of harm.
 - b. Reporting procedures and requirements to be undertaken if a child makes a disclosure to them.
 - c. Be able to identify the schools’ Student Protection Officers.

ALL CORRESPONDENCE:

Brisbane Catholic Education
GPO Box 1201
Brisbane QLD 4001
Phone: (07) 3033 7000
Fax: (07) 3844 5101
www.bne.catholic.edu.au
ABN 49 991 006 857



OFFICE LOCATIONS:

Dutton Park

243 Gladstone Road
Phone: (07) 3033 7000

West End

199 Montague Road
Phone: (07) 3033 7000

North Lakes

12 Endeavour Blvd
Phone: (07) 3490 1700

Springwood

5-7 Laurinda Crescent
Phone: (07) 3440 7900

**Fr Bernard O’Shea
Inservice Centre, Wliston**

19 Lovedale St
Phone: (07) 3033 7111

**Ngutana-Lui Aboriginal and
Torres Strait Islander Cultural
Studies Centre, Inala**

100 Lilac St
Phone: (07) 3033 7200